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DONALD ROSS VARDIMAN

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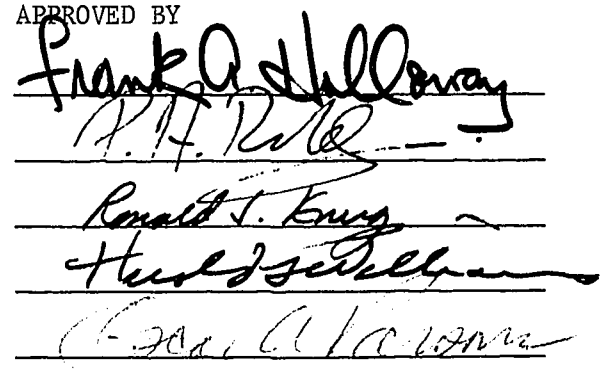
A RE-EXAMINATION OF LOCALIZATION OF FUNCTION

IN THE RAT NEOCORTEX

APPROVED BY



DISSERTATION COMMITTEE

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A RE-EXAMINATION OF LOCALIZATION OF FUNCTION IN THE RAT NEOCORTEX

CHAPTER I

INTRODUCTION AND STATEMENT OF THE PROBLEM

The history of Man's attempts to understand the precise nature by which his central nervous system (CNS) contributes to his behavior has been strongly influenced by the issue of localization versus non-localization of function (Head, 1926; Boring, 1957; Krech, 1963, Luria, 1966). Basically the question of localization of function asks the degree to which specific behaviors are controlled by specific neural structures. "The problem of localization is in its essence the problem of relation of structural and functional units in brain activities." (Vygotsky, 1965). Specificity of function is guardedly granted for many sensory and motor functions but the assignment of specific neuroanatomical control to complex behaviors is still an area of neuropsychological exploration (Luria, 1970). Thus the question of localization of function is a viable issue today and present attempts to understand complex behavioral processes, such as memory, either by biochemical or probabilistic neural models (John, 1967) point to the importance of determining the behavioral significance of a given CNS unit.

The history of neurobehavioral investigations can be roughly divided into three and perhaps four major eras, each of which has some relevance to the question of localization of function and is distinguishable from the others on the basis of its particular accomplishments at either the neuroanatomical level, the behavioral level or both. Three of these eras were: (1) a period of neurobehavioral naivete, (2) a period of advancement in both neuroanatomical and behavioral conceptualizations, and (3) a period which emphasized the necessity of studying behavior, in all of its complexity, in an analysis of neurobehavioral relationships. There are indications that future advances in neurobehavioral theory may depend, in part, upon an increasing emphasis on the possibility that complex behavioral states are the only unique properties of highly evolved neural systems--as opposed to such commonly ascribed properties as "mind", "consciousness", etc. (Sperry, 1964). This would constitute a fourth period of neurobehavioral investigations and theorizing. While having an element of chronological order in their occurrence, these periods are primarily defined in terms of men, their theories and their methods of investigating neurobehavioral relationships.

The following sections will deal briefly with the first three eras of neurobehavioral inquiry, including present doctrines concerning the localization of complex behaviors in the CNS. A more detailed consideration of a possible fourth era of neurobehavioral conceptualization will be reserved for the section of Chapter III which deals with the more general considerations of the neurobehavioral analysis employed in this presentation.

The Age of Neurobehavioral Naivete'

This era begins, of course, in Man's pre-recorded history and is embarrassingly evident in present day understandings of the biological determinants of behavior. However, progress in neurobehavioral understanding has indicated that the brain is the principal 'controller' of behavior. This has not always been accepted as the case. While attempts to understand brain functions are quite old, even the localization of the 'mind' at or within the brain was a matter of some doubt until the middle of the nineteenth century (Boring, 1957). Speculations concerning neurobehavioral relationships reached a ridiculous extreme in the teachings of phrenology which ascribed the control of highly complex behaviors, such as "cautiousness" and "individuality", to specific cortical areas whose relative importance in the overall behavior of a given individual was indicated by cranial bumps which reflected the relative size of the particular underlying cortical area. These bumps varied from individual to individual and therefore differences in behavior were explained through implied differences in cortical structure.

Historians generally credit the school of phrenology with popularizing the possibility that particular parts of the brain controlled particular 'organs' of separate mental faculties. This movement originated from the works of Franz Joseph Gall (1758-1828) and was formalized and given the name phrenology by his pupil J. G. Spurzheim (1776-1832). While phrenology made many assumptions which are not granted today, such as assuming a correlation between the shape of the skull and the conformations of the brain as well as the assumption that the behavior and personality of man were innately determined, its most

enduring principle was that of brain localization of function (Krech, 1963).

Despite Gall's expertise as an anatomist, the extreme views of phrenology were generally opposed by the scientific world in the early nineteenth century. Pierre Flourens (1794-1867), the French experimental neurologist, is the one man credited with discrediting phrenology (Boring, 1957; Krech, 1963). While introducing the ablation technique and the use of laboratory observations to the study of brain-behavior relationships, Flourens also divided the nervous system into six separate units which could be isolated and experimentally studied. These units were the cerebral hemispheres, the cerebellum, the corpora quadrigemina, the medulla oblongata, the spinal cord and the nerves. Although granting specific properties, functions, and effects to these units, Flourens considered the nervous system to have a unity which resulted from an action commune in addition to the action propre of the parts. "Unity was considered to be the great reigning principle; it was everywhere, it dominated everything. The nervous system thus formed but a unitary system." (Boring, 1957).

By bringing neurobehavioral studies into the laboratory and applying the ablation technique, "Flourens radically changed the original Gall-Spurzheim question and thereby altered the nature of the answer." (Krech, 1963, p. 40). In place of the uncontrolled clinical observations and anecdotal material of the phrenologists, Flourens surgically destroyed specific brain areas and observed changes in his subjects. However, Flourens' work was not without its limitations. While attacking phrenological accounts of cortical functioning Flourens

employed subjects with small amounts of cortex--hens and pigeons. The lack of adequate methods of recording, measuring and distinguishing behavior patterns and capacities obviously prejudiced Flourens' results as well as those of the phrenologists. The inadequacies of Flourens' methods can be seen in the following exerpts reported by Krech (1963) in which Flourens is addressing the question of whether

> ...all these perceptions and all these faculties occupy the same seat in the organs, or is there, for each of them, a seat different from that of the others?
>
> . . . . . .
>
> I carefully removed the whole anterior portion of the right cerebral lobe of a pigeon in successive layers, and the whole superior and middle of the left.
>
> Vision weakened more and more, very gradually, as the extirpation progressed, and was finally lost on both sides only when the layers, near the central node of both lobes, were removed.
>
> But as soon as vision was lost, so was audition, and, together with vision and audition, all intellectual and perceptual faculties.
>
> . . . . . .
>
> As the ablation progressed, vision diminished gradually and noticeably; audition weakened as did vision; all the other faculties in the same manner; as soon as one of these was completely lost, so were they all.
>
> . . . . . .
>
> ...as soon as one perception is lost, all are lost; as soon as one faculty disappears, all disappear. There are not, therefore, different sites for the different faculties, nor for the different perceptions.
>
> . . . . . .
>
> Unity is the grand principle which reigns.

Thus while Flourens deserves much credit for establishing neurobehavioral research as a laboratory science it is obvious that his techniques of behavioral evaluation fell short of currently accepted methods. In effect, his approach to the study of behavior was little, if any, advancement over the impressionistic techniques of phrenology. "This, of course, cannot be taken as a criticism of Flourens--we could

hardly expect him to invent and develop a new science of animal behavior in addition to a neurological technique." (Krech, 1963, p. 41).

Advances in Neurobehavioral Conceptualization: The Clinic and the Laboratory

Flourens' ideas of the action commune of the nervous system carried the day for nearly half a century but there was some opposition based upon clinical observations of limited paralysis and limited cerebral lesions. Head (1926) cites many writings of Bouillaud (1796-1881) to this effect. Gradually developments in the clinic and the laboratory began to suggest that the reigning principle of the nervous system might be that of the action propre of its perhaps previously undefined parts. The next period in the history of brain-behavior research belongs to the Clinicans--Bouillaud, Broca (1824-1880) and Hughlings Jackson (1835-1911) and another group of experimenters--Fritsch (1838-1927), Hitzig (1838-1907) and David Ferrier (1843-1928). These clinicans and experimenters constituted the French, English, and German champions of the localization hypothesis (Krech, 1963).

Despite Bouillaud's writings in 1825 and again in 1839 in support of the Gallian motion of localization, this position was not afforded much credence by the scientific community until Broca's startling pronouncement in 1861. Bouillaud had argued that the faculty of speech was situated in the anterior lobes. However, according to Head (1926) "...the bogey of phrenology as well as the findings that speech could be gravely affected although the lesion was not situated in the frontal lobes..." distracted from Bouillaud's assertions of localization. Discussion of localization continued over the years and once in

1848, Bouillaud offered five hundred francs to anyone who would bring him an example of a severe lesion of the anterior lobes of the brain unaccompanied by disturbance of speech (Head, 1926, p. 17).

In April, 1861, Paul Broca began a series of investigations which lead to the conclusion that the center for articulated speech is located at the base of the third frontal convolution of the left cerebral hemisphere. Boring states that Broca's famous observation was in itself very simple and that its merit lay in his "...careful examination of a clean-cut case which chance threw into his hands and in the immediacy with which he seized upon the broader implications." (Boring, 1957, p. 71). This "simple" observation, determined by "chance" does indeed appear to be an accident in the history of science. Or was it?

Head concludes that "...in 1861, the air was again full of the localization of cerebral functions, and this question was liable to crop up with any excuse, in scientific discussions." (Head, 1926, p. 17). And Boring relates that "...for many years French surgeons...had been doubting Flourens' doctrine of the unity of the nervous system and believing that they must find more specific localization of function within it." (Boring, 1957, p. 71). Thus by chance Broca, during this pregnant time, acquired a patient with "aphemia" (possessing the general faculty of language but unable to speak even though the musculature of the larynx and articulatory organs was capable of normal movement, there was no other paralysis to interfere with speech, and the patient was intelligent enough to speak) whose timely death allowed for an autopsy to be performed in time for Broca to present the brain before the Société d'Anthropologie the next day, April 18, 1861. Other cases soon

followed to support this one and soon the principle of localization was firmly entrenched in scientific circles. In 1874, Wernicke concluded "...the sensory images of speech..." are localized in a zone of the cortex of the left hemisphere.

> The descriptions of two isolated areas of the brain in which corresponding lesions led to disturbances of such different functions caused unprecedented activity on the part of investigators of cerebral localization. These cases provoked the suggestion that other mental processes, even the most complex, may be localized in comparatively small areas of the cerebral cortex and that, in fact the cortex is an aggregate of separate 'centers' whose cell groups are 'depots' of different mental faculties. (Luria, 1966, p. 11).

It was also about this time (1860's-1870's) that the famous English neurologist, Hughlings Jackson, first presented his ideas concerning the importance of the localization properties of epileptic seizures. "For him, the study of focal seizures became the starting point for the study of all seizures; the study of seizures became the starting point for the study of localization of function within the nervous system." (Penfield & Jasper, 1954). While Krech (1963) also includes Hughlings Jackson among the defenders of the localization of function position this might be the case only as it applies to Jackson's speculations about separate motor centers in the brain. Both Head (1926) and Luria (1966) consider Jackson to be against ideas of strict localization of function for speech but, as Head points out, Jackson himself is difficult to interpret on this point. Head quotes Jackson as saying

> Whilst I believe that the hinder part of the left frontal convolution is the part most often damaged, I do not localize speech in any such small part of the

> brain. To locate the damage which destroys speech and to localize speech are two different things. The damage is in my experience always in the region of the corpus striatum. (Head, 1926, p. 50).

Luria interprets Jackson as saying that the localization of a sympton following a circumscribed lesion in the CNS is not equivalent to the localization of the particular function (Luria, 1966, p. 18). However, Head (1926) points out that few people recognized that Jackson did not postulate a separate center for speech.

Apart from the findings of Broca and Wernicke two other developments about this time contributed to the view that complex mental processes can be narrowly localized in circumscribed areas of the cerebral cortex. Both Luria (1966) and Boring (1957) emphasize the influence of (1) British Associationism, which proposed that human mental activity is based upon the association of sensation and ideas, and (2) progress in anatomy and physiology as important factors in the favorable reception of the ideas of the localizationists in the second half of the nineteenth century. The suspicion that the cortex was replete with various depots for the control of their respective behaviors received much encouragement from discoveries in histological techniques which demonstrated that the brain was composed of an almost infinite number of separate cells as opposed to the concept of homogeneous neural mass.

> For the associationists, mind is composed of an infinitude of separate ideas, just as the brain is constituted of an infinitude of cells. But these ideas are bound together into more complex ideas or into higher mental processes by a huge number of associations, just as the nerve cells are connected by fibers.... The important point is that the new picture of the brain... bore a close resemblance to the new picture of the mind

> that associationism yielded...the new knowledge of the division of the brain into many tiny connected units implied that sometime further separation of localized mental functions, like ideas, was to be sought. (Boring, 1957, p. 70).

The trend toward an increasing acceptance of the position of narrow localization of function was further strengthened by laboratory findings, during the early 1870's, which suggested a possible 'structural' bases for behavior. That is, the discoveries of Fritsch and Hitzig (1870) which showed that stimulation of certain cortical areas was followed by contraction of certain muscles was soon followed by Betz' (1874) finding that the cortical zone discovered by Fritsch and Hitzig was structurally distinguishable from adjacent cortical areas. The motor areas were found to structurally differ from other cortical zones by the presence of giant pryamidal cells.

The localization of motor activity to discrete brain sites, by electrical stimulation procedures, influenced Ferrier who employed this technique in an examination of the cortices of several species. "The net result of Ferrier's work was the final (to date) establishment of the principle of localization of sensory and motor function in the brain." (Krech, 1963, p. 49). While Ferrier located vision in the occipital lobes it was Munk who in accordance with (1) Johannes Muller's doctrine of specific nerve energies which encouraged a belief in the existence of five centers, one for each sense, and (2) the facts of neuroanatomy which included the semi-decussation of the optic nerves at the chiasma, demonstrated that the removal of an occipital lobe is not followed by complete blindness in either eye but instead there is blindness for half of the visual field for each eye (hemianopia) (Boring,

1957, p. 75). Function could be understood in terms of structure.

From the localization of phrenology to the unity of Flourens and now back again to a localized "scientific phrenology", the pendulum of opinion swayed--to and fro. First one, then the other, and now the first again! Fickle science! Or is it? With respect to phrenology perhaps the question is inappropriate but both the action commune of Flourens and the localized aphemia of Broca carried the weight of scientific support for their respective day. Both Flourens' and Broca's findings were accepted by the scientific community because they employed the "scientific method". Why then the dissimilarity of results?

Boring credits Fritsch and Hitzig, as referring to "...the discrepancy between dogma (the action commune of Flourens?) and experiment to technique; 'the method', they remarked, 'creates the results.'" (Boring, 1957, p. 73). However, Krech states that Flourens "...radically changed the original Gall-Spurzheim question and thereby altered the nature of the answer," (Krech, 1963, p. 40) and credits Flourens with stating, in 1842, "It is the method which gives the results" and to this Krech adds "...the method having first shaped the question." Method, therefore, appears to be all important, shaping both the question and the answer. What then determines method? Would it be too bold to answer--"theory"?

The Behavioral Emphasis

A new method of neurobehavioral investigation, born of a new theoretical orientation, was to be launched in the early nineteen hundreds. By this time psychology had become divorced from philosophy and was an independent science which advocated that behavior be studied

objectively and measured quantitatively. The importance of this new emphasis to future neurobehavioral research was monumental.

> The progress in neural-behavioral research had thus far spiraled around significant advances in neurological techniques--from the careful anatomical studies of Gall and Spurzheim, through the skillful ablation procedures of Flourens, to the electrical stimulation techniques of Fritsch and Hitzig and of Ferrier. The development of the other variable--behavior--lagged behind....in the laboratory no advances in the measurement of behavior had been made since the time of Flourens. Impressionistic observations of behavior was still the custom in the laboratory.
>
> With the growth of the new science of animal psychology--at the very end of the nineteenth century--this one sided development was changed. (Krech, 1963, p. 54).

The new discipline, animal psychology, allowed for a new method--objective and quantitative analysis of behavior. One of the first to employ this new method as an approach to neurobehavioral problems was Shepherd Ivory Franz (1874-1933), who began extirpating brain tissue to see what loss in behavior would result (Herrnstein & Boring, 1966). Franz' work is historically important for at least three reasons: (1) behavior was examined in terms of its objective complexity, such as the learning (problem solving behavior) and retention of habits, instead of either such broad terms as "will" and "intellect" or the traditional simple sensory reactions or motor reflexes, and the effects of destroying various neural regions were evaluated in terms of these objectively identifiable complex behaviors; (2) the pendulum of localization began to swing back toward Flourens, and perhaps toward Jackson, and away from the "scientific phrenology" of Ferrier and others; and (3) Franz had a student named Karl Spencer Lashley (1890-1959).

From studies dealing with the recovery of habits which were

lost following one and even two-stage brain removal surgeries (1902; cited in Krech, 1963) and observations on the apparent lack of localization or "fixity" or "definiteness of connection" of specific motor behaviors (1915; in Herrnstein & Boring, 1966) Franz concluded that the localization of function position was weak on both behavioral and anatomical grounds. "Franz's contribution seemed largely to be negative: cerebral localization is not precise." (Herrnstein & Boring, 1966). His student, K. S. Lashley, whose experiments, in his own words, "...constitute the first attempt to apply quantitative methods to both neurological and behavioral data..." and who is considered to be "...one of the most influential and productive psychologists who has worked in the field of neural-behavioral studies," (Krech, 1963, p. 55) left a much deeper mark upon the field of neurobehavioral investigations.

Lashley also concluded that localization was "not precise" but to summarize his work as merely supporting the earlier positions of Flourens and Franz would be both an understatement and an error. The importance of Lashley's work is best understood not just in terms of what he did, and found, but in terms of the manner in which he undertook to examine behavioral theory in the laboratory and the impact of his work upon nearly all behavioral theorizing from that date on.

In 1929, Lashley published Brain Mechanisms and Intelligence: A Quantitative Study of Injuries to the Brain, the work for which he is most remembered and in which he formulated the now famous laws of "equipotentiality" and "mass action". Lashley coined the term "equipotentiality" in describing the functional significance of the rat cortex and defined the term as "...the apparent capacity of any intact part of

a functional area to carry out, with or without reduction in efficiency, the functions, which are lost by destruction of the whole." (Lashley, 1929, p. 25). Lashley also popularized the concept of "mass action" to further describe neural functioning. The law of mass action states

> ...that the equipotentiality is not absolute but is subject to a law of mass action whereby the efficiency of performance of an entire complex function may be reduced in proportion to the extent of brain injury within an area whose parts are not more specialized for one component of the function than for another. (Lashley, 1929, p. 25).

D. O. Hebb (1963) introduced a publication of Lashley's 1929 book by saying "It is not far-fetched to date the beginning of the modern period of psychology from the publication of this book in 1929." The validity of this statement is best understood in terms of the points that Hebb emphasized, in the introduction, concerning psychological behavioral theorizing prior to and around the time that Lashley began his life's work.

Hebb argues that behavior theory <u>per se</u> was strongly influenced by the 1929 book. "The behavior theory that existed before its publication was mostly cast in neurological terms, and concerned ideas that could never be the same again.... None of this carefree neurologizing was possible after 1930, at least not for a psychologist." Hebb points out that from Wundt onward psychology had been predominantly physiological psychology, "...however fanciful..." In the decades preceeding Lashley's work "consciousness" had meant "cortical" and automatic "unconscious" habits were simply shortcircuited and handled "subcortically". Physiological behavioral theory had become dependent upon arguments utilizing concepts such as synaptic resistance, detailed

localizations of cortical functions, and new paths from point to point in the cortex for new habits. "Now, suddenly, it appeared from Lashley's work that such ideas were fantasy, not science." The destruction of the neurological basis for the then current behavior theory was not the guiding purpose of Lashley's initial explorations in this area. Far from it!

Lashley was a thoroughgoing behaviorist having encountered John B. Watson at Johns Hopkins, in 1912, when Watson was preparing what Hebb calls "the Behaviorist manifesto". Lashley was only twenty-two years old at the time and the next six years during which he collaborated closely with Watson in a number of studies undoubtedly influenced him strongly. During this time Lashley met another man who was to strongly influence his future--Shepherd Ivory Franz. Watson's monistic explanation of behavior included ideas of synaptic modification and the formation of stimulus-response connections through the cortex. While with Franz, Lashley assisted in two studies of the effect of cortical extirpations on habit in the rat. Lashley later began the work, which was the basis for the 1929 book, of demonstrating the soundness of Watson's ideas of the neural counterpart of observable behavior. "But every experiment he did came out wrong." He finally abandoned this aspect of the theory but Hebb states that Lashley "...never abandoned Watson's real aim of achieving a completely monistic and objective explanation of behavior."

Just exactly how did Lashley's experiments come out "wrong"? Current behavior theory held that learning or habit formation was the end result of chains of associations between stimuli and reflexes which

produced an overt series of reflexes (behavior) and that these associations or growth relationships between reflex components occurred in specific neural locations. Lashley concluded that

> The experimental findings have never fitted into such a scheme. Rather, they have emphasized the unitary character of every habit, the impossibility of stating any learning as a concatenation of reflexes, and the participation of large masses of nervous tissue in the functions rather than the development of restricted conduction-paths. (Lashley, 1929, p. 14).

Although there were instances of apparent habit localization such as a loss of retention of a double-latch box problem following frontal lesions but not others and loss of a brightness discrimination following posterior but not anterior lesions, the fact that identical lesions prior to training did not affect rate of learning contributed heavily to Lashley's conclusions that all behaviors were probably not highly localized in cerebral depots. While demonstrating that for habits involving unique visual properties (such as the elevated maze) localization may be an important factor, for other tasks which are not so dependent upon a particular sensory mode, such as Maze III, this was not the case. Equipotentiality was said to hold for "...the association areas and for functions more complex than simple sensitivity or motor co-ordination." (Lashley, 1929, p. 25).

Lashley's views of equipotentiality and mass action came from investigations of the effects of size and location of cortical lesions upon performance of rats in his number three (III) maze (Figure 1). Using this maze, some of Lashley's findings were:

> (1) The maze habit, formed before cerebral insult, is disturbed by lesions in any part of the cortex. The amount of reduction in efficiency in performance is proportional to the extent of injury and is independent

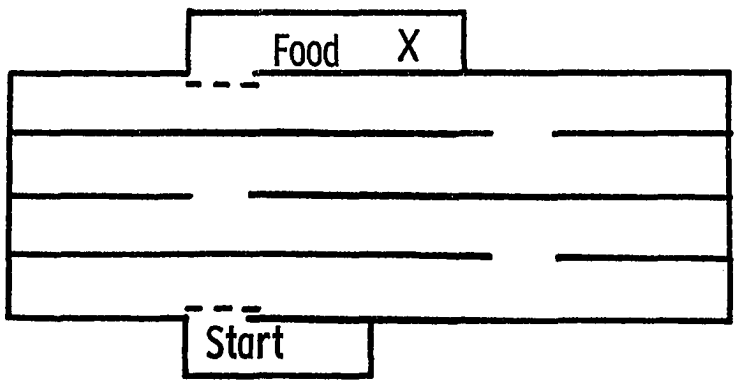

Figure 1. The eight cul-de-sac maze used by Lashley. The start-box and goal-box were equipped with swinging doors. (From Lashley, 1929)

> of locus. (2) The capacity to form maze habits is reduced by destruction of cerebral tissue. (3) The reduction is roughly proportional to the amount of destruction. (4) The same retardation in learning is produced by equal amounts of destruction in any of the cyto-architectural fields. Hence the capacity to learn the maze is dependent upon the amount of functional cortical tissue and not upon its anatomical specialization.

From these findings, Lashley concluded that

> ...the learning process and the retention of habits are not dependent upon any finely localized structural changes within the cerebral cortex. The results are incompatible with theories of learning by changes in synaptic structure, or with any theories which assume that particular neural integrations are dependent upon definite anatomical paths specialized for them. Integration cannot be expressed in terms of connections between specific neurons. (Lashley, 1929, p. 176).

Lashley's conclusions that no part of the rat neocortex is more essential than any other part of the neocortex for the mastery of a complex maze habit is widely accepted today (Chow, 1968; Deese, 1968; John, 1967). However, at least two lines of evidence suggest that a re-examination of some of Lashley's findings is in order. First, there is evidence that the rat neocortex is not equipotential with respect to maze mastery. Second, advances in techniques of behavioral analysis which have demonstrated the importance of many discrete brain loci in a wide variety of behaviors (such as the role of the septum in active versus passive avoidance) suggests the possibility that a more microscopic examination of maze behaviors *per se* could point to previously undetected localization properties in discrete cortical regions.

The following sections will deal with (1) evidence concerning localization of function in the rat cortex for the maze habit, (2) considerations of the complexity of behavior, (3) an analysis of the III

maze habit, (4) a question of neuroanatomical logic in approaching the study of localization and (5) the current problem.

Evidence for Localization

Pickett (1952), though not employing a Lashley type III maze, reported that anterior cortical lesions produced greater deficits upon maze retention than did posterior lesions. However, Thompson (1959a) using a water maze version of the Lashley III maze, found bilateral posterior cortical lesions produced greater deficits in maze behaviors than did bilateral anterior lesions. Posterior lesions resulted in significant deficits in both blind and nonblind subjects while anterior lesions produced deficits only in subjects which were enucleated prior to maze training. "It would seem, therefore, that large bilateral anterior lesions are effective in impairing learning only when visual cues are absent during the learning period." (Thompson, 1959a, p. 503). The finding that anterior lesions were primarily effective in blind subjects is also inconsistent with the concept of "mass action".

In a similar study, Thompson (1959b) found that, in subjects which learned the maze with vision intact and were later blinded, anterior lesions resulted in significantly more errors in retention than did posterior lesions. No significant retention deficit was observed in subjects with posterior lesions. In nonblind subjects posterior lesions did produce a significant retention deficit while anterior lesions had no significant effect on maze retention.

While the results of these studies are difficult to interpret, they do point to the possibility of localization of function in the rat neocortex with respect to maze mastery.

Behavioral Considerations

Lashley recognized the importance of including in his investigations of brain-behavior relationships a wide variety of tasks, especially those involving "...a more complex habit". Both behavioral definition and response complexity were important factors in determining task selection. In an early paper, with S. I. Franz, both of these factors were presented as justification for using the inclined-plane box in addition to the simple, one cul-de-sac, maze in assessing loss of habit following brain damage.

> The simple maze offered some disadvantages for a study of retention owing to the fact that it did not require a reaction that was sufficiently well defined to be certainly recognizable in the retention tests. It seemed best therefore to use some more complex habit in the later experiments for the sake of getting a more clearly defined series of activities and also with the possibility that the more complex habit, involving different types of reaction, might reveal a selective effect of the cerebral lesion upon certain types of activity. (Lashley & Franz, 1917, p. 86).

Thus while granting that complex habits could involve "...different types of reaction..." in terms of one task, the inclined-plane box, this was apparently not the case for the III maze habit which was considered to be unitary.

> None of the studies of learning or retention of the mazes after cerebral lesions has given the slightest indication that the maze habit is made up of independent associational elements.... The diversities of behavior, such as disorientation, tendencies to perseveration, and the like, correspond somewhat with the magnitude of lesion but not at all with the locus, and seemed to represent diverse degrees of deterioration rather than specific defects. (Lashley, 1929, p. 141).

These conclusions are not surprising in view of the unitary behavioral assessment employed by Lashley--culs-de-sac (the dead end

areas in each row) entered.

The III Maze Habit

There is a possibility that the primary dependent variable utilized by Lashley in assessing maze performance was not as sensitive to the various effects of brain damage as originally thought. In evaluating the effects of brain lesions upon the learning and retention of habits Lashley employed a wide variety of tasks but placed primary emphasis upon performance in the III maze. The reasoning for this is fairly straightforward. The III maze was believed to be the most complex of the various mazes utilized in Lashley's studies (with the possible exception of maze IV) having eight culs-de-sac and requiring alternate right and left turns in the true path (Figure 1). As entry into a cul-de-sac constituted the commonly used error measure and this maze had more culs-de-sac than the other mazes, then this was the most "complex" maze. The complexity was considered an important factor in assessing the effects of brain damage for several reasons. The more complex the problem, the greater the possibility that its solution was not dependent upon any particular sensory input. Also

> The simpler problem offers difficulties which are not much greater for animals with brain lesions than for normal ones; and correspondingly, the difficulty does not greatly increase with increasing magnitude of brain injury. The more complex problem, on the other hand, is more difficult for animals with lesions than for normals; and as the magnitude of the lesion increases, the difficulty of the problem becomes progressively greater. (Lashley, 1929, p. 74).

While the above conclusions were not applicable to all the various tasks--in learning the brightness discrimination or the double-platform box the operated animals showed no retardation--they are

consistent with the maze results where complexity is measured in terms of culs-de-sac. Thus the III maze was considered to be the most sensitive of the various tasks to the effects of brain damage, in both theoretical and empirical aspects, when performance was measured in terms of culs-de-sac entries and trial latencies.

Performance on the III maze can be evaluated in terms of behaviors other than simple cul-de-sac entry. Cul-de-sac entry *per se* is by no means a complete assessment of the habit complexity of the III maze. An inspection of Figure 1 reveals that the III maze is made up of several rows with each row being connected to the others via either alternate left or right end openings (doors). Thus the most efficient strategy for mastery of this maze habit would be upon entering row one, turn right, go through the door leading to row two, turn left, go through the door into row three, turn right, etc. Two classes of behavior essential for efficient maze maneuvering are immediately obvious: the rat must go through openings (doors) in order to get to the next row and upon entering a row the rat must alternate in either a right or left direction to the door leading to that row. These behaviors will be termed "door" and "alternation" behaviors respectively. Thus a rat which upon entering row one turned *left* and then also ran *past* the door leading into row two, turned around and ran past the same door again, and then returned and went through the door into row two, would have committed one *alternation error* and two *door errors* while in row one of the maze.

It would not be too difficult to imagine either the alternation error (behavior) as being related to the subject's ability to

remember the correct sequence of turns in the true path and the subject's own position with respect to this sequence, or the door error as being related to the degree and control of locomotor activity. A subject with a deficit in either spatial orientation or temporal sequencing could commit more alternation than door errors while a subject with visual-perceptual failings (cannot see the door) or with deficits in locomotor control (inability to stop) might commit more door than alternation errors. Thus, while the two errors could certainly have many common elements, it is quite possible that they could reflect separate and distinct behaviors required for efficient maze performance. The hypothetical rat above, scored by the culs-de-sac entered as the error measure, would simply have been observed to make two errors yet the same rat also made one alternation and two door errors. Thus utilizing both door and alternation error concepts in an analysis of III maze behaviors could represent a quantitative as well as a qualitative advancement over an analysis of maze performance solely in terms of culs-de-sac entered.

Furthermore, these two error measures have been found to be valid descriptors of behavior in terms of maze mastery. Defining a door error as being committed whenever a subject goes past a door, in either direction, without entering, which leads to the next row between the subject and the goal-box, a large number of door errors are possible for each row per trial on the maze. However, this error quickly drops (by five or six trials) to a very low level in the behavior of normal rats (White & Vardiman, 1964; White & McGee, 1964). Jackson & Strong (1969) and Bender, Hostetter & Thomas (1968) have shown this error to

be significantly elevated in subjects with limbic system damage (primarily the hippocampus).

The alternation error, which consists of the subject turning in the direction opposite the next door immediately upon entering a new row of the maze, can only be committed once per row. This is a more difficult behavior for normal subjects to master (White & Vardiman, 1964) and should be sensitive to subtle changes in behavior following brain damage. This would seem to be especially true if the brain damage resulted in a deficit in temporal sequencing ability.

In summary, it would appear that while there are at least two types of logically derivable behaviors (door and alternation) which can be evaluated during performance of the maze habit, performance measured purely in terms of culs-de-sac entered would fail to distinguish between these behaviors. A rat which always entered a blind cul immediately upon entering a row (alternation error) would not be distinguishable from one which always ran past a door into a cul (door error) prior to going through the door into the next row. To say that these behaviors are identical, and should be so judged, is to ignore the moment to moment changes in response requirement during a successful maze performance.

Neuroanatomical Considerations

Although the studies of Pickett (1952) and Thompson (1959) question the concept of rat neocortical equipotentiality, the results of these studies are so divergent that an interpretation is somewhat difficult. Pickett found that anterior cortical lesions severely impaired maze performance, as compared to posterior lesions, while

Thompson's findings were just the opposite of this.

The contradictory nature of these studies could be due to one or more of several factors: (1) different type mazes were used in the two studies; (2) different behaviors could have constituted "errors" in the two studies; (3) the use of only two cortical zones might have allowed for the inadvertent inclusion in one experimenter's "anterior" region, structures which another researcher would consider "posterior" and vice versa. In light of this last possibility, it would appear that studies of this nature could profit by employing three cortical zones: anterior, middle and posterior. Thus, even though boundaries between two areas may overlap, and therefore produce ambiguous or even contradictory results, with three areas one area should be significantly different from the other two, in terms of resulting behaviors, if true cortico-behavioral differences exist.

An examination of the various lesion sites employed by Lashley indicates that a rather non-systematic approach determined their selection. Thus while many subjects had lesions in common areas, there was usually enough individuality of lesion shape and extent to make direct comparisons between various sites somewhat difficult. It would also appear that most attempts to control for the size of the lesion were employed after histological examination and were not a part of the surgical procedure. While this type of comparison is logically allowable, the failure to control for mass and site as part of a systematic experimental analysis often resulted in few direct comparisons between subjects which were completely satisfactory in terms of site and amount of brain damage.

Another area which needs some clarification is the relative effects of cortical versus subcortical lesions upon maze behaviors. The reason for this is although Lashley interpreted most of his lesions in terms of "cortical" effects, many cases were reported with damage to subcortical structures. As there was no significant difference in maze performance (culs-de-sac entered) between cortically lesioned subjects with and without accompanying thalamic lesions, thalamic damage was considered to be relatively unimportant as was damage to the striatum, septum, or hippocampal structures "...and so we cannot ascribe the correlation to injury to subcortical structures." (Lashley, 1929, p. 64). The studies of Jackson & Strong (1969) and Bender, Hostetter and Thomas (1968) would indicate that this is not the case as the hippocampus is involved in one type of possible cul entry (the door error). Again, it should be noted that the failure to analyze the maze habit in terms of its components automatically precludes the possibility that different habit components may be differentially associated with the activity of different brain sites such as cortical versus subcortical structures.

Current theories of neurological functioning would certainly stress the importance of, at least, limbic mechanisms in a set of behaviors as complex as maze mastery. The previously mentioned studies of Jackson & Strong (1969) and Bender, Hostetter and Thomas (1968) demonstrate the importance of hippocampal functioning with regard to the execution of door errors in the Lashley III maze. Thomas, Moore, John & Hunt (1959), using the Lashley III maze with an error measure which in effect included door errors, found that septal lesions greatly

impaired maze performance. The findings of these studies suggest that much caution should be exercised when interpreting maze deficits in purely "cortical" terms.

The Problem

The previous discussion has suggested that it is not unusual for opinion and fact to vary from time to time concerning the issue of localization versus non-localization of function and that there exists a need to re-evaluate at least one presently held view of brain functioning; the equipotentiality of the rat neocortex with respect to maze mastery. With respect to the first suggestion, the implication was presented that the pendulum of opinion concerning this question has been moved by method as much as by fact. Here the suggestion was that theory dictates method which in turn generates fact. Somewhere in this collection of forces and events one finds the influence of certain individuals. The theory, the method and the individual all interact to produce the fact. A consideration of these factors led to the formation of the second suggestion. Explicitly these considerations were as follows: (1) current neurobehavioral theory could profit from new information concerning the question of localization of function; (2) much of the support for the non-localization of function position is based upon a behavioral measurement (culs-de-sac entry) which may not be sufficiently sensitive to distinguish between the functionings of various neural locations in the acquisition or retention of the III maze habit; (3) other evidence does suggest that the rat neocortex exhibits localization of function for the maze habit; (4) the use of both the door and alternation errors as dependent variables which might

selectively reflect changes in activity level, ability to orient in the maze, perseveration, etc., could contribute greatly to an analysis of maze behaviors; and (5) a systematic examination of three cortical zones, with each subject receiving lesions in only one zone, would help to eliminate confusion resulting from the individuality of lesions in some studies and should clarify some of the ambiguity of other studies of localization employing only two cortical zones.

CHAPTER II

BASIC INVESTIGATIONS

Experiment I

On the basis of the previous discussion an experiment was undertaken to investigate the role of three bilateral cortical zones in the acquisition of the III maze habit as measured by door and alternation errors.

Method

Subjects. Eighteen albino rats, approximately 90-110 days old, were randomly assigned to one of three cortical lesion groups such that there were six subjects (Ss) in each group. The lesion sites (groups) were designated anterior cortex (AC), middle cortex (MC) and posterior cortex (PC) (Figure 2).

Surgery. All surgery was performed in one session while the animal was anesthetized with sodium pentobarbital and was positioned in a Kopf stereotaxic apparatus. Bilateral cortical ablation was accomplished by suction removal of exposed cortex. Cortical destruction was primarily restricted to the dorsal surface of the brain, as far lateral as the temporal ridge. The three cortical ablation zones were defined in terms of cranial landmarks as follows: anterior zones--from the frontal pole to a point approximately 2 mm posterior to bregma; middle

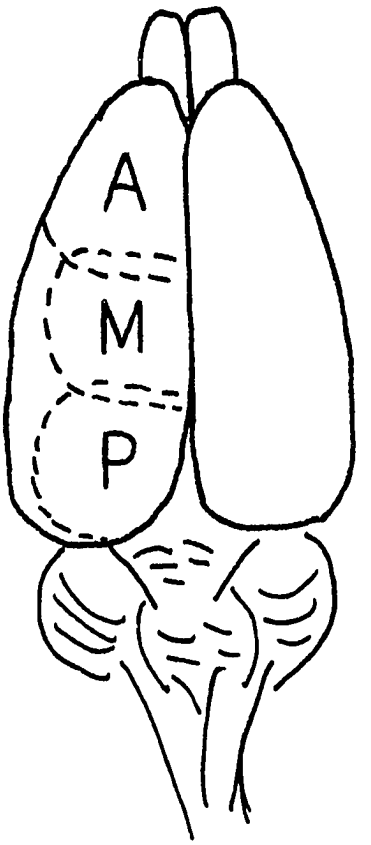

Figure 2. A unilateral representation of the three cortical zones used in this study.

zone--from bregma to a point approximately 5 mm posterior to bregma; and posterior zone--from a point approximately 5 mm posterior to bregma to lambda.

In order to make complete lesions over the prescribed surface areas it was necessary to remove all of the bone over that area. Much care was taken to insure that a minimal amount of CNS damage occurred during skull removal. This procedure consisted of these steps: (1) expose skull and scrape clean in prescribed area; (2) mark skull zone to be removed; (3) drill small hole through skull somewhere in prescribed zone; and (4) enlarge opening to desired size by use of small bone rongeurs. After some practice one can acquire the skills necessary to remove a large section of the skull without disrupting the dura.

Both hemisphere zones were exposed prior to the initiation of suction procedures as bleeding commenced concurrent with the invasion of the meninges. Continual flooding of the suctioning area with sterile physiological saline was necessary to remove blood and other fluids which produced visual interference.

After each hemisphere zone was suctioned, bleeding was checked by packing that area with sterile absorbable gelatin. An additional covering of gelatin was then placed over both zones. A thin layer of dental acrylic was deposited over the last gelatin layer and the scalp was sutured closed. Penicillin was topically applied to the wound and approximately 80,000 units of penicillin was injected intramuscularly.

Apparatus and procedures. The maze used in this study was a modification of Lashley's number III maze, having six rows instead of

four (Figure 3). The internal dimensions of this maze are: wall height - one foot; alley width - four inches; alley length - four feet; door width - four inches; start-box and goal-box width - four inches; start-box length - one foot; and goal-box length - eighteen inches. The floor of the maze was made from 1/4 inch aluminum wire mesh.

Each S was placed in the start-box for ten seconds and then allowed entry into the maze. As each row was entered the door was closed, by the experimenter (E), behind the S to prevent re-tracing. Each S was allowed access to wet lab chow mash for 30 seconds in the goal-box and then returned to the home cage. All Ss received one trial per day for twenty consecutive days.

The dependent variables for the Lashley III were door errors and alternation errors. The door error consists simply of a S going past a door (in either direction by at least half a body length) which leads to the next row or, in the case of row six, the goal-box. Several door errors may be made per row (Arrow #1, Figure 3). The alternation error consists of the S upon entering a new row turning into the blind cul-de-sac, by at least half a body length, which is opposite in direction from the path leading to the door to the next row (Arrow #2, Figure 3). Only one alternation error may be made per row. Both types of errors were determined by observation.

As each row was entered, and following commission of an error, the E pressed an appropriate key on a control box which was always held in the E's left hand. The right hand was used to operate recording switches and to replace doors to prevent the S's re-tracing between rows. The control box was equipped with eight switches which activated

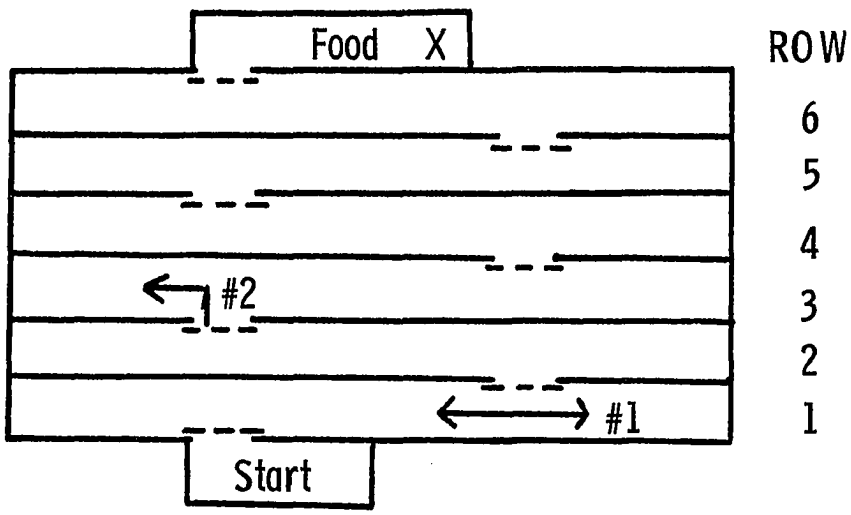

Figure 3. A modified version of the III maze having twelve culs-de-sac instead of eight or six rows instead of four. Arrow #1 represents a door error and arrow #2 represents an alternation error.

eight individual channels of a multi-channel Esterline-Angus event recorder. Six of the switches corresponded to each of the rows while the other two switches corresponded to door and alternation errors. The switch-pen combinations were so arranged that an easily readable permanent record of each S's maze performance, per trial, was obtained. The E first pressed the appropriate row switch and then pressed the appropriate error switch, if an error was committed. This allowed for an easy determination of what errors, if any, were committed in each row, per trial, by inspection of the chart record.

Approximately twenty-four hours after completion of maze training each S received a ten-minute period of activity assessment in an activity chamber which was designed to measure several types of activity (locomotor, head movement, etc.). The activity device used in this study was a LeHigh Valley photocell and quadrant counter apparatus which was housed in a sound-dampening room. A count was electronically made whenever a S interrupted any of several photocell beams.

All Ss were tested in all phases of this study while between 75% and 80% of their normal body weight. This was determined for each S as follows: (1) on the 11th post-operative day, while all Ss were on ad libidum food and water, each S was weighed at the same time of day that the S would be tested; (2) ad libidum weights were taken each day until a stable body weight was established or until five weights were obtained; (3) the average weight for the stable period of five weights obtained in step 2 was taken as the 100% body weight and each S was slowly deprived of food for a period of approximately ten days until the 75-80% body weight range was reached and then testing was initiated.

Handling, weighing and testing were done at the same time each day for each S and only one E handled any given S. Each S was tested by the same E throughout the study and the assignment of Ss to each E was such that no one E tested more than one-half of the Ss in each group. The group identity of each S was also unknown to the E.

Histology. All Ss were sacrificed and their brains profused with 10% formalin. Amount of cortical destruction was estimated by inspection of the intact brain similar to that described by Lashley (1929). That is, the extent of the superficial lesions were determined by inspecting the intact brain and "...laid off on diagrams of the brain with proportional dividers". The areas of these lesions were finally measured from the diagrams with a planimeter and expressed as a percentage of the total surface area. All brains were then microtomed and stained to allow for an examination of thalamic degeneration. Sectioning was at 40 micron intervals and every third section was stained such that every other stained section was either a cell body (cresylecht violet) or a tract stain (Weil).

Results and Discussion

The two maze behaviors under investigation, door and alternation errors, were not equally affected by the various cortical lesions. The alternation error exhibited striking equipotentiality. That is, all three cortical groups performed almost identically with respect to this behavior (Figure 4). Figure 4 also shows that the strong equipotentiality of the alternation error was not observed in the door error behaviors. The door error behavior was most pronounced in the middle cortical group. The high within-group variability of the

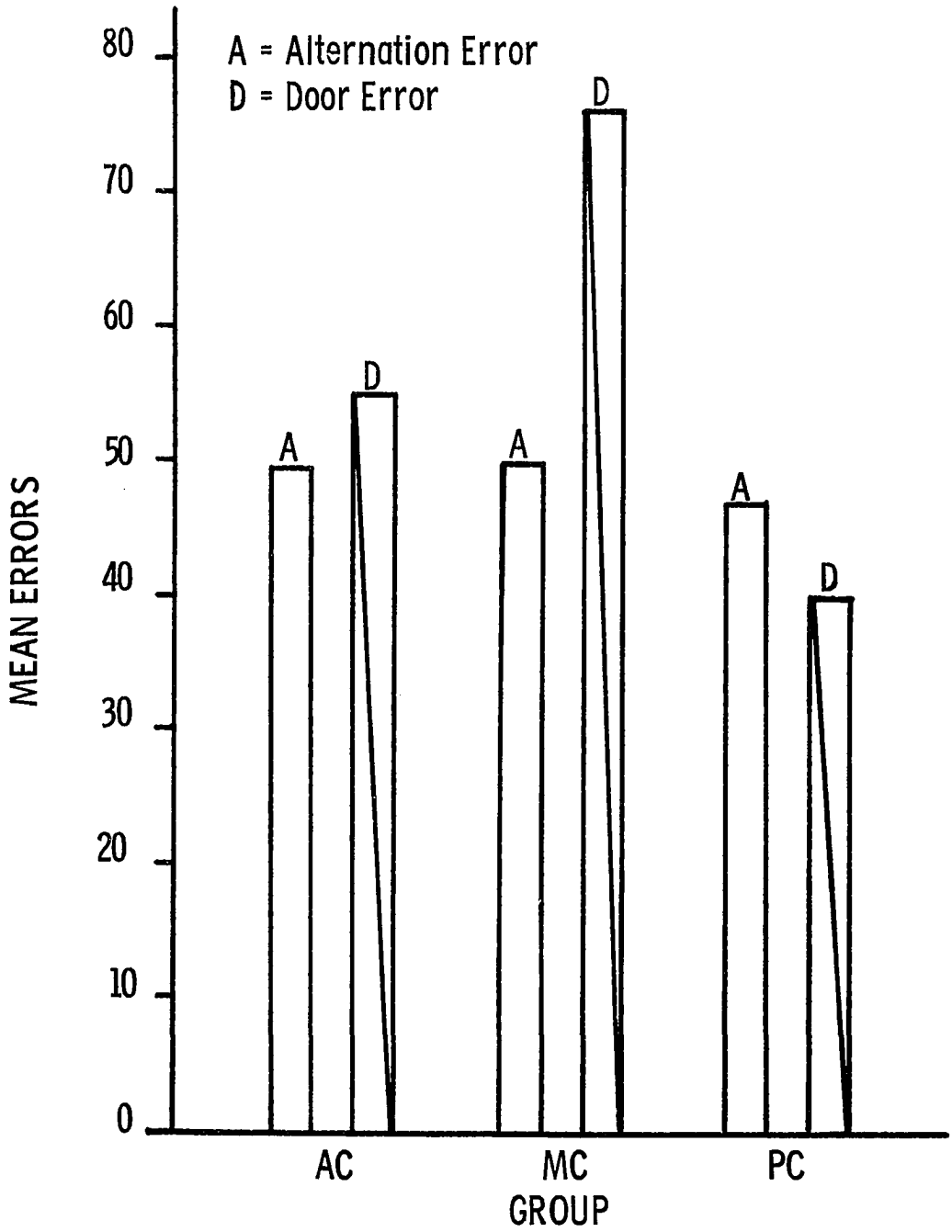

Figure 4. The mean performance of each of the cortical groups in terms of door and alternation errors.

door error behavior (Table 1) and the small number of cases in each

TABLE 1

TOTAL DOOR (De) AND ALTERNATION (Ae) ERRORS AND PERCENTAGE OF CORTICAL DESTRUCTION (%), BY SUBJECT, FOR THE THREE CORTICAL GROUPS

Anterior			Middle			Posterior		
%	De	Ae	%	De	Ae	%	De	Ae
11.3	47	37	17.5	135	63	21.4	52	46
13.0	24	42	22.9	57	48	10.7	53	53
24.0	63	67	33.6	132	48	26.6	42	44
9.6	90	41	25.7	60	56	20.3	21	43
10.0	58	53	16.0	31	37	22.3	33	49
12.0	42	56	23.1	32	44			
[1] 13.3	54.0	49.3	23.1	74.5	49.3	20.3	40.2	47.0
[2] 5.4	22.3	11.4	6.3	47.3	9.1	5.8	13.5	4.1

[1] Means

[2] Standard deviations

group preclude any possibility of significant statistical differences between groups as measured by this behavior. One S died in the PC group reducing the total number of Ss to seventeen. A Kruskal-Wallis one-way analysis of variance (Siegel, 1956) of the total door errors, for twenty trials for the three groups produced an H of 4.62 which approached significance at the .10 level of confidence with two degrees of freedom. Despite the low level of significance between groups in terms of door errors, an examination of the data raised some interesting questions concerning equipotentiality, mass action and behavior.

Table 1 shows that the amount of cortex removed was not equal for the three groups, with the AC receiving much less damage than either of the other two groups. Yet in terms of alternation errors the AC group was similar to the other groups. In terms of door errors the AC group was intermediate to the MC and PC groups. If mass is the crucial factor in error production then increases in the amount of cortex destroyed in the AC group to a level comparable to the other groups (for example--from 13% to 20%) should produce large increases in both errors. In that case the PC group would be much lower in terms of door errors than the other two groups and both the PC and MC groups would be lower than the AC group in terms of alternation errors. Therefore if mass is the critical factor in error production, given an equal amount of area destroyed and based on the data in Table 1, the PC group would make less door errors than the AC or MC groups while the AC group would make more alternation errors than either of the other groups.

If the rat neocortex is equipotential with respect to maze mastery and is therefore subject to the law of mass action in terms of neurobehavioral relationships then differences in amount of cortex destroyed should explain differences in observed behaviors. This was not the case for either error behavior. The correlations between errors and amount of cortical destruction were low and nonsignificant for both behaviors ($R = +0.076$ for door errors and $R = +.236$ for alternation errors).

The two behaviors were significantly correlated with each other ($R = +.53$, $p < .05$). This correlation was highest in the MC group ($R = +.90$, $p < .01$), lower in the PC group ($R = +.70$) and absent

in the AC group (R = + .08). Both errors were also significantly correlated with the ten minute activity score (alternation error: R = + .539, $p < .05$: door error; R = + .695, $p < .01$). However, there were no significant activity differences between groups as measured by Mann-Whitney U comparisons (Siegel, 1956).

These findings, while indicating that equipotentiality may be a valid concept in terms of alternation errors, do raise questions concerning the role of mass in terms of both maze behaviors and general activity. Thus the concept of equipotentiality is indirectly questioned by failings of the law of mass action as well as the finding that the door error does not appear to be as equipotential as the alternation error.

The two maze behaviors appear to be distinguishable in terms of both a logical analysis of the maze habit and in the manner in which they are affected by neural damage. One behavior, the alternation error, appears to be a model for the concept of equipotentiality but this is not the case for the door error. Both errors, however, appear to be independent of the concept of mass action. The findings raise new doubts concerning the generality of the laws of equipotentiality and mass action and suggest that while some behaviors may be subject to these laws other behaviors may not be so governed.

Experiment II

The findings of Experiment I, while questioning both the generality of the concepts of rat neocortical equipotentiality (for some behaviors) and mass action, also question the view that the maze habit is "unitary". Lashley (1929) concluded that there was no reason to

suspect that the maze habit is made up of independent associational elements since the "diversities" of behavior resulting from brain damage appeared to be related not to site of damage but only to amount of damage (see earlier quote on p. 20). However, when the maze habit is viewed in terms of door and alternation behaviors, one behavior (door errors) is related to locus of lesion (middle cortical zone) while the other behavior (alternation errors) is not related to a particular cortical site. Thus in terms of cortical neurobehavioral mechanisms the maze habit is not unitary but can be viewed as consisting of two, possibly more, behaviors--door and alternation behaviors.

The small number of Ss employed in Experiment I discouraged an extensive analysis of the data, yet the data did raise enough questions to warrant further investigations in this area. Specifically, some questions which need to be answered are: (1) can the results of Experiment I be replicated in terms of the effects of cortical damage upon the two maze behaviors; (2) how will subcortical damage affect these behaviors; and (3) what are the dynamics of these behaviors?

The first two questions can be answered by simply testing the acquisition of these behaviors in cortically and subcortically damaged rats. The third question, concerning the nature of the behaviors per se, is more difficult to approach. As mentioned in Chapter I, the two behaviors can be distinguished in terms of their operationally defined requirements. That is, the door error is clearly distinguishable from the alternation error. However, it is important to know more about these behaviors than that they are simply distinct.

As mentioned in Chapter I, the door error on the III maze

could be related to deficits in locomotor control or visual-perceptual failings. One measure of locomotor control could be the rate of acquisition and extinction of a straight runway response. Basic failings in the execution of locomotor behaviors would interfere with the acquisition of a running response while failings of locomotor inhibition would interfere with the extinction of that response.

Visual-perceptual failings might also relate to door error activities. That is the rat may fail to see or recognize the opening (door) in the row wall. Thus door error behaviors in the III maze may be related to the ability to detect and enter breaks (openings) in a wall surface. This type of task can be seen in the "door error maze" (Figure 5), which requires the S to turn into an opening in one of the two row walls. A comparison of performance on this task with the III maze door errors is relevant to questions about the generality of the door error behavior. That is, if door errors in the III maze are determined by properties of the maze *per se*, such as might be the case with the alternation errors, then there should be little correspondence between door error performance in the III maze and performance in the DEM.

As mentioned in Chapter I also, the alternation error is probably related to the S's ability to remember the correct sequence of turns in the true path and the S's own position with respect to this sequence. Therefore the alternation behavior could reflect more than just the ability to learn to turn left or right but also the ability to remember a particular sequence of alternations. Thus the alternation error might reflect more than a simple loss in the ability to alternate

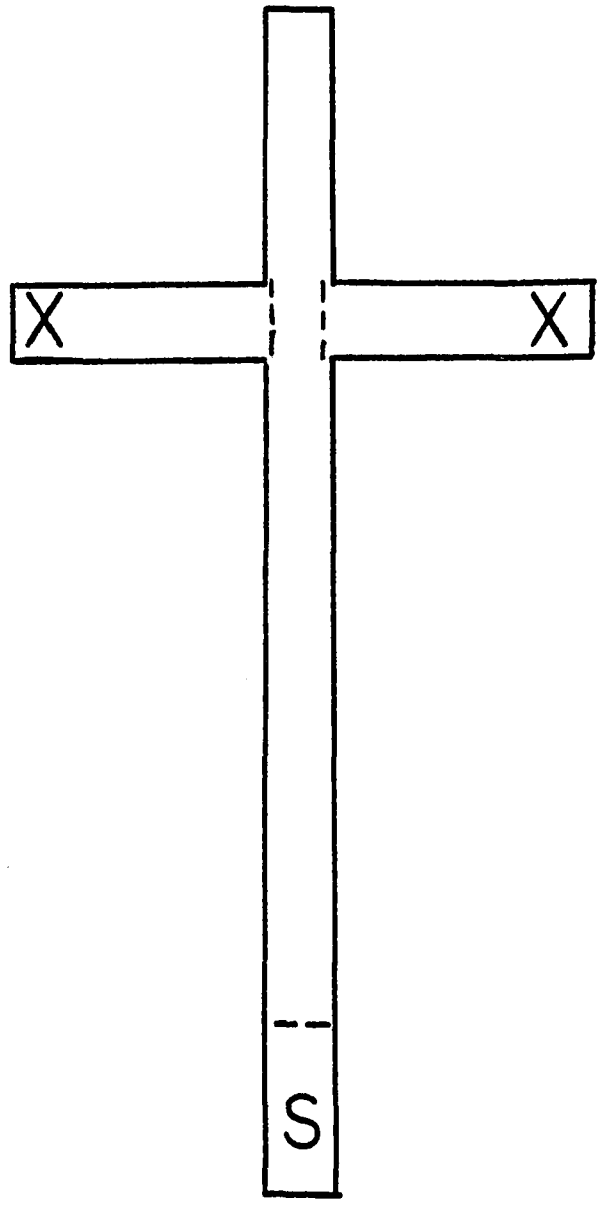

Figure 5. The door error maze (DEM). Only one arm of the apparatus is open at any one time (S = Start ; X = Food).

and lesions which interfere with the execution of the III maze alternation behaviors may not affect the ability to perform a simple alternation problem. If the ability to execute successful III maze alternation behaviors is dependent upon more than just the ability to learn to alternate but also involves the ability to learn a sequence of alternations then alternation behaviors in the III maze may not be related to alternation behaviors as measured in a conventional T-maze (Figure 6).

General activity level as well as habituation to a novel situation could also be related to maze mastery ability. Repeated exposures to an initially novel environment in which activity could be assessed would contribute information concerning both activity and habituation. A comparison of initial activity level, activity habituation and maze performance could aid in an understanding of the maze behaviors.

The present study was undertaken to replicate and extend the findings of the first study by including (1) in addition to the three cortical groups, two additional groups with damage to either the septum or dorsolateral hippocampus and (2) a series of behavioral tasks other than the acquisition of the III maze habit. The acquisition of the III maze habit was measured by door and alternation errors and trial latency.

To further aid in the interpretation of the analysis of the acquisition of the III maze habit, several additional behavioral measures were taken. An additional measure of door error tendency was measured by the use of a "door error maze" which presumably does not

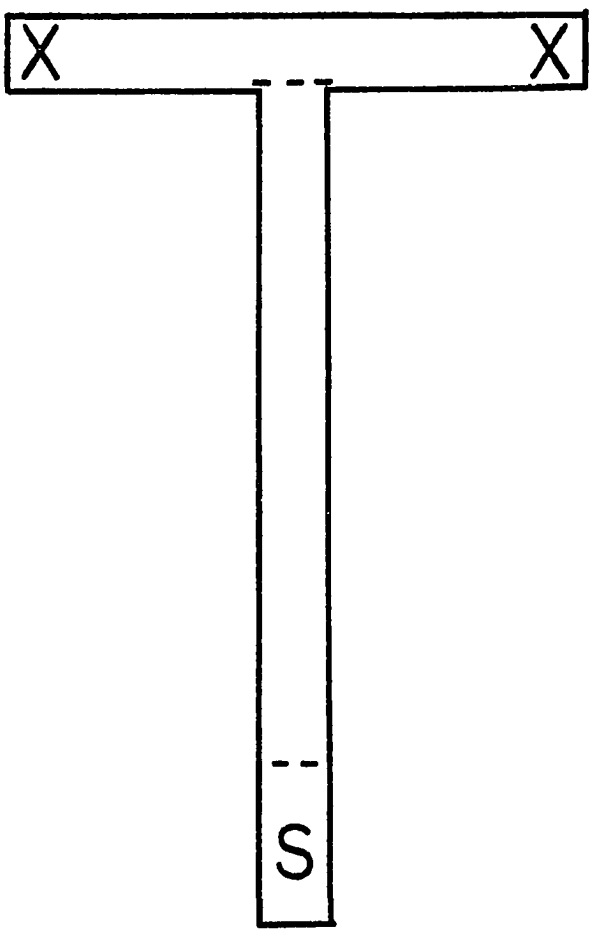

Figure 6. The alternation error maze (AEM).

have an alternation error component (Figure 5). A T-maze, presumably without a door error component, was also used to further aid in the interpretation of the alternation error behavior (Figure 6). Latency in a straight runway, with food reward, was also obtained to aid in interpreting possible motivational, motor, or other effects. Extinction training was given in the straight runway in an attempt to assess such factors as response perseveration and/or failures of inhibition. Spontaneous activity was measured for three ten-minute sessions (one session per day for three consecutive days) following the completion of all maze testing.

Method

Subjects. The Ss in this study were fifty-six male Sprague-Dawley rats approximately 200 days old at the initiation of testing. There were seven groups of eight Ss each. In addition to the three cortical groups employed in the first experiment the present study also included four additional groups. These were a group with septal destruction (SEP), damage to the dorsolateral hippocampus (DLH), a surgical control group (SCN) and a group of unoperated controls (CN).

Surgery. All surgery was performed in one session while the animal was anesthetized with sodium pentobarbital and positioned in a Kopf stereotaxic apparatus. Cortical destruction was achieved by the same procedure as in Experiment I and the same coordinates were employed. Bilateral septal and hippocampal lesions were accomplished with a Grass Model 2 Radio Frequency Lesioner. The subcortical lesion electrodes were unipolar and constructed from insulated Nichrome wire (diameter = 0.0159 inches), exposed at the tip for 0.5 to 0.75 mm.

The stereotaxic coordinates (Pellegrino and Cushman, 1967) for the SEP lesions were: antero--posterior plane = + 8.0, lateral plane = $\pm$ 0.5, and vertical plane = + 1.3; coordinates for the DLH lesions were: antero--posterior plane = + 1.8, lateral plane = $\pm$ 4.0, and vertical plane = + 0.5. The SCN were subjected to the same surgical procedures as the MC group except that the dura was not punctured and no cortex was removed. All Ss were given ten days for surgical recovery prior to the initiation of test procedures.

Deprivation and handling. All deprivation and handling procedures were identical to those employed in Experiment I.

Order of testing. On the 11th day of deprivation, once 75-80% body weights had been reached, the following order of testing was begun for each S: (1) one ten-minute period of activity recording each day for four consecutive days; (2) twenty-one days of acquisition on the Lashley III with one trial per day; (3) six days of acquisition on the door error maze with two trials per day; (4) six days of acquisition on the T-maze with two trials per day; (5) six days of acquisition in the straight runway with two trials per day; (6) six days of extinction in the straight runway; and (7) a final four day period of activity assessment with one ten-minute period per day for four days. Due to each S being run within the same 15 minute interval each day, in an attempt to control for possible circadian effects, no test session can require more than two trials per day.

Apparatus and procedures. The III maze used in this study was the same as that employed in Experiment I. In addition to door and alternation errors, row and trial latencies were also included as

dependent variables. A switch, depressed by the E's left hand, started a timer when the E raised the start-box door thus allowing the S access to row one; another switch, also operated by the E's left hand, was depressed thus stopping the timer, activated earlier, whenever the S entered the goal-box. The elapsed time was defined as trial latency. The switch which the E depressed to signify each row entry also activated a print-out counter which printed the total cumulative elapsed time prior to that print signal. Subtracting the first print-out from the second print-out gave the time spent (latency) in row one. All row latencies could thus be calculated by a simple subtraction of the different cumulative latencies. Admittedly this technique of latency assessment includes an element of the E's own reaction times but the latencies in question are usually quite large, several seconds at least, and therefore any appreciable treatment effects should be detected by this technique.

The activity measuring device used in this study was also the same as that employed in Experiment I, but the activity phase of this study was extended to four consecutive days. Both the maze and activity handling and testing procedures were identical to those employed in Experiment I.

The door error maze (Figure 5) consisted of a five-foot straight runway, four inches wide, with a goal-box one foot from the end on either of the two alley walls. The dimensions of the goal-box were: door width - four inches; length - eighteen inches; and width - four inches. One-half of the Ss in each group had the goal box on one side and the other Ss in each group had the goal-box on the opposite side.

The dependent variables were latency per trial and door errors. Latency per trial was the time elapsed between the S leaving the start-box and entering the goal-box. A door error consisted of the S going past the door (opening) to the goal-box.

The alternation error maze was a conventional T-maze (Figure 6). The length of the alley from start-box to choice point was four feet. The start-box section was one foot in length. The T-arms extended eighteen inches from either side of the start-box alley at right angles to the alley. The internal width of the alley and T-arms was four inches.

The dependent variables were latency per trial and alternation errors. Latency per trial was the time elapsed between the S's leaving the start-box and entering the arm of the T which contained the food. The alternation error consisted of the S turning in the direction opposite the goal-box at the choice point. The arm of the T that contained the food was opposite from the side of the door error maze that contained the goal-box for each S. Thus if a S found food on the left (reference to the start-box) side of the door error maze, then for that S the right side of the T-maze was the goal-box. Each S was allowed to find the goal-box even if an alternation error was made.

The straight runway was an alley six and one-half feet long and four inches wide, with one foot of one end being a start-box and eighteen inches of the other end being the goal-box. When the start-box was opened the S was simply required to go to the food. Latency from start-box to goal-box was the dependent variable.

Results

The data from this study which consisted of the III maze, door and alternation error behaviors, and day one of the activity testing phase, of the three cortical groups was considered to be a replication of the first experiment as surgery, testing procedures and other general factors were basically the same for both studies. Therefore the data from both experiments were pooled for some analyses dealing exclusively with cortical lesions. All other analyses dealt with the data from Experiment II.

The number of Ss in the analyses which pooled the cortically lesioned Ss in Experiments I and II were thirty-nine (N = 13 per group). In Experiment I the number of Ss in each group which survived until the completion of maze training was six, six and five for the AC, MC and PC groups respectively. The number of Ss in each of the respective groups which completed maze training in Experiment II were seven, eight and eight. One S was randomly selected from the MC group and discarded. This allowed for greater ease of statistical analyses as the available computerized analyses of variance programs required an equal number of observations per cell and discarding one S from the MC group resulted in thirteen Ss being in each of the cortical groups. In the analysis of the III maze data for Experiment II the number of Ss in each of the six groups was seven. One S in two of the groups died prior to the completion of training on the III maze and for ease of computations one S was randomly discarded from each of the other groups.

For all analyses of variance both the door and alternation errors and latencies were transformed according to Kirk (1968). For

door errors, where the variances were highly correlated with the means, the transformation was $x^1 = \log_{10} (x + 1.0)$ and for alternation errors, where there were many scores of zero and one, the transformation was $x^1 = x + 0.5$. Latencies were transformed to the logarithm of each value to reduce some of the positive skew associated with such data.

III Maze

Cortical effects. Only thirty-four of the thirty-nine cortically lesioned brains were available for an estimate of amount of cortical damage (topographical area) as three subjects died prior to the completion of all testing, and these brains were not saved for histological examination, and two other brains were inadvertently microtomed and partially destroyed before an estimate of cortical damage could be made.

Of the thirty-four brains, ten were in the AC group and twelve were in the MC and twelve in the PC groups. The mean amount of cortical destruction for the AC, MC and PC groups was 22.4%, 22.5% and 22.0% respectively (Appendix A). Neither a t-test between means nor a Mann-Whitney U test of ranks indicated a significant difference between any of the groups in terms of amount of cortical damage.

An examination of these brains confirmed the finding of the first study that amount of cortical damage (mass) was not significantly correlated with either error. Across groups, the correlations between amount of cortical damage (%) and errors were R = + 0.174 and R = + 0.141 for door and alternation errors respectively. Within groups, the same correlations were equally as low ranging from an R of + 0.164 between amount of destruction and door errors in the AC group to an

R of - 0.07 between amount of destruction and alternation errors in the MC group. Although the range of amount of cortical destruction is not very great, varying for 10.3% to 33.9%, higher correlations than those observed should have been obtained if the relationship between brain damage and maze behavior is governed by "mass action".

Other investigators (Saavedra, Pinto-Hamuy & Oberti, 1965) have also reported findings which were inconsistent with the general concept of mass action. While lesions to the anterior (frontomotor) and middle (somatosensory) cortical areas did produce behavioral deficits which correlated with lesion size, lesions to the visual (posterior) and auditory (lateral middle and posterior sites) failed to result in behavioral loss which was related to size of lesion.

Figures 7, 8 and 9 demonstrate that mass is not the critical factor in determining maze behavior in terms of door errors. While all three Ss had comparable lesions, in fact the AC and PC lesions were larger than the MC lesion, and performed comparably in terms of alternation errors, the MC lesioned S made many more door errors than the other two Ss.

Door error analysis--cortical groups. A Kruskal-Wallis one-way analysis of variance performed on total door errors for the pooled cortical groups indicated a significant difference between groups ($H = 9.819$, $df = 2$, $p < .01$). It can be seen in Figure 10 that the MC group displayed more door errors than the other two groups. Both the AC and PC groups differed significantly from the MC group (AC--MC, $t = 1.96$, $p < .05$; PC--MC, $t = 1.97$, $p < .05$) but failed to differ significantly from each other ($t = 0.226$). A four factor analysis of variance (Winer,

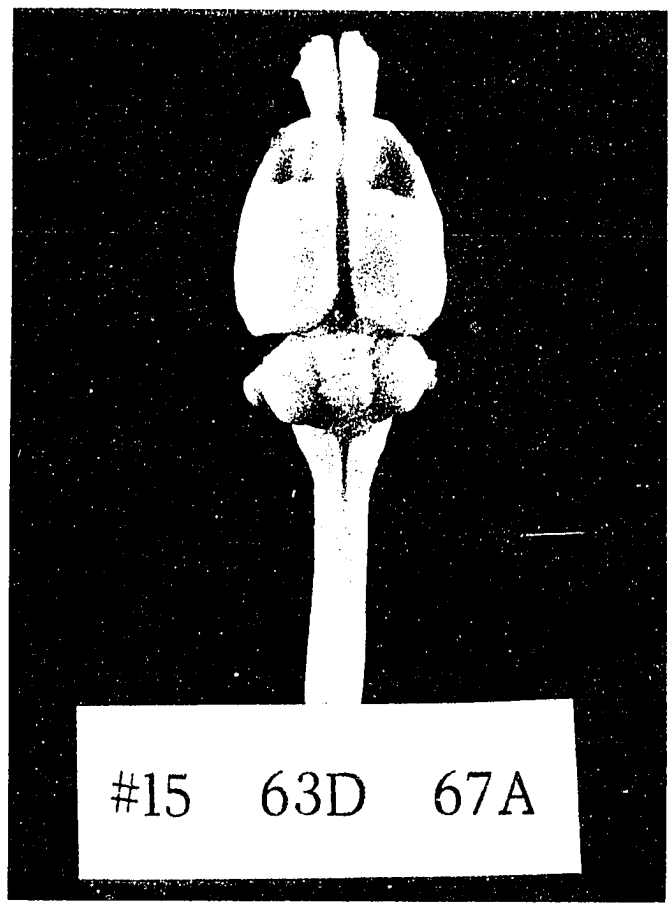

Figure 7. An example of an AC subject. The three numbers, from left to right, refer to (1) S #, (2) total door errors and (3) total alternation errors for that S.

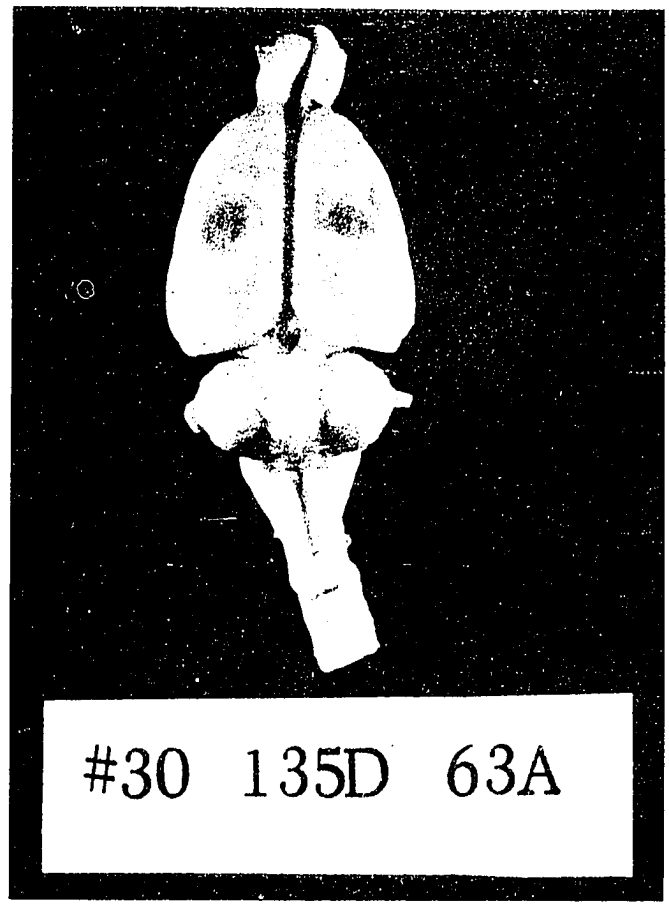

Figure 8. An example of a MC subject.

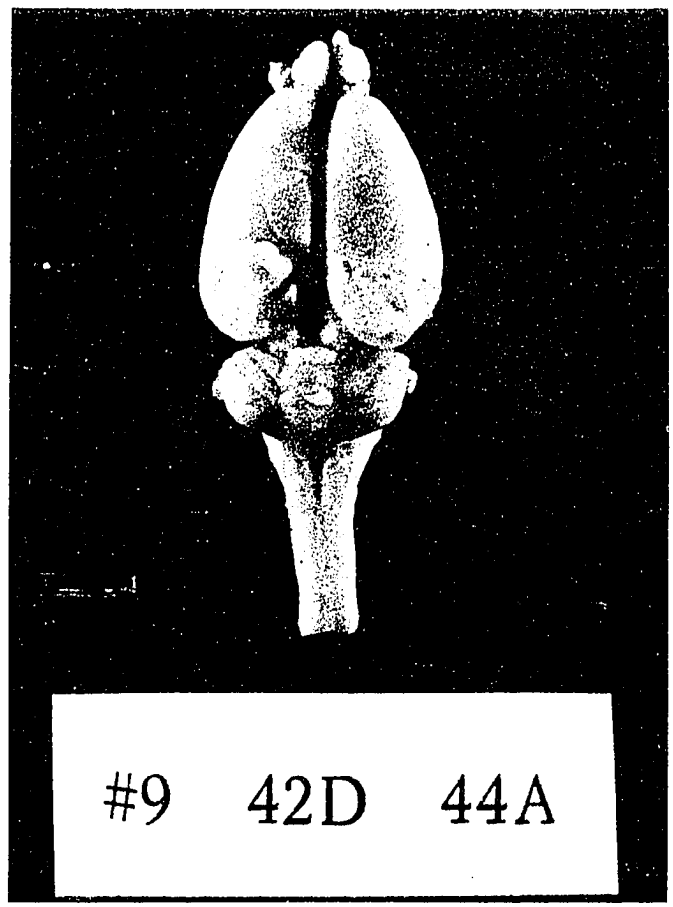

Figure 9. An example of a PC subject.

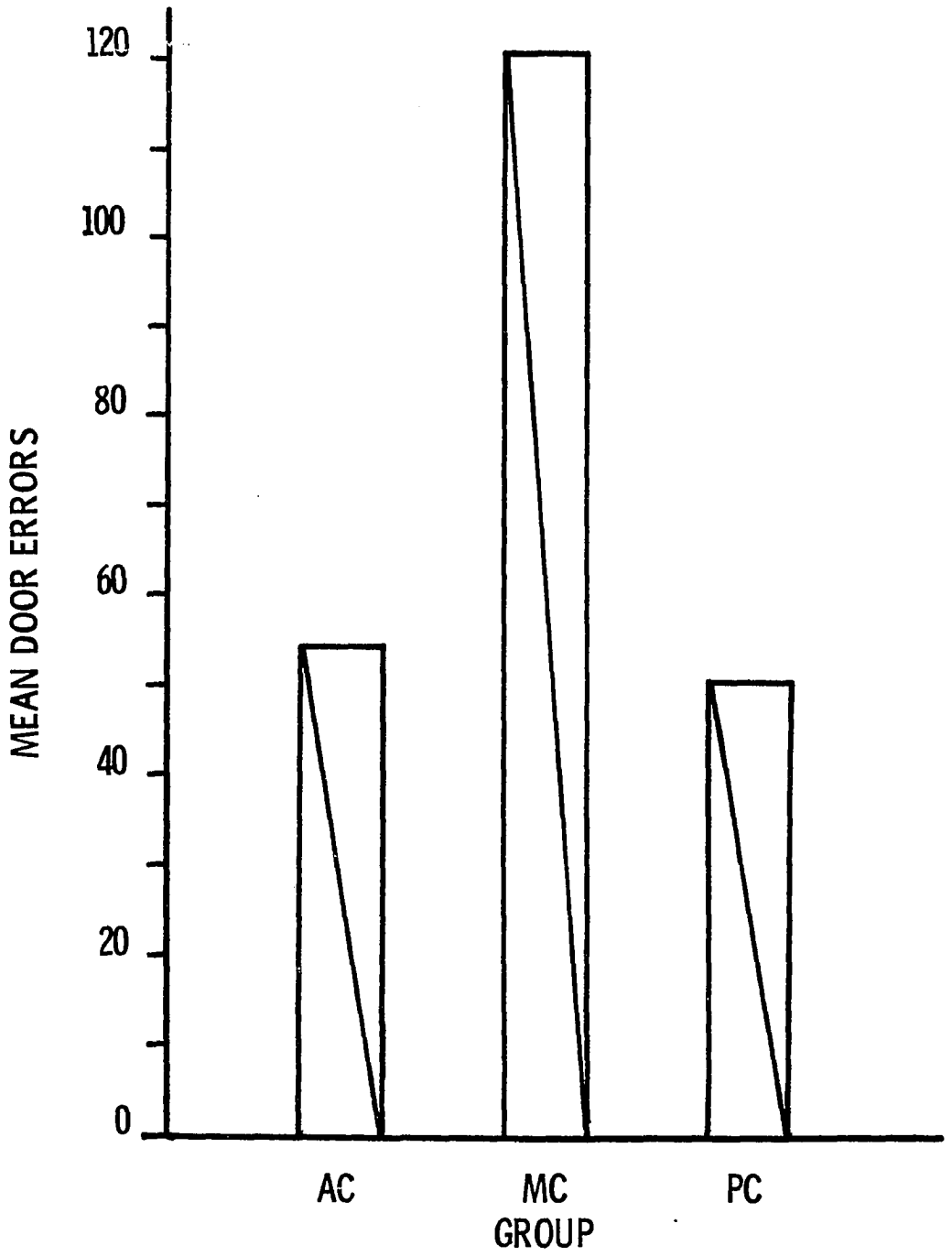

Figure 10. Mean door errors as a function of site of cortical damage.

1962, p. 328) was performed on these data also revealing a significant between groups effect (Table 2). The factors in this analysis were

TABLE 2

SUMMARY OF ANALYSIS OF VARIANCE FOR DOOR ERRORS FOR COMBINED CORTICAL GROUPS OF EXPERIMENT I AND II

Source	df[a]	MS	F
Between subjects	38		
G (groups)	2	5.373	3.913 [b]
Subj w. groups	36	1.344	
Within subjects	2301		
T (trials)	9 (1)	5.243	39.174 [c]
GT	18 (2)	0.465	3.481 [b]
T x subj w. groups	324 (36)	0.133	
R (row)	5 (1)	2.315	15.952 [c]
GR	10 (2)	0.167	1.154
R x subj w. groups	180 (36)	0.141	
TR	45 (2)	0.073	
GTR	90 (2)	0.074	
TR x subj w. groups	1620 (36)	0.074	

[a] conservative test df

[b] $p < .05$

[c] $p < .01$

groups (three--AC, MC and PC), trials (ten--the twenty acquisition trials were collapsed into ten blocks of two trials each), rows (the six rows of the maze) and replications (the thirteen Ss in each group). This analysis also revealed a significant trials effect, row effect and groups by trials interaction. The Greenhouse and Geisser procedure of

using conservative degrees of freedom in calculating F ratios for multi-factor experiments having repeated measures (Winer, 1962, p. 305-306) was used in calculating all F values reported in this study.

The row effect for door errors can be seen in Figure 11 and shows a typical goal-gradient effect. That is, the row furtherest from the goal-box (row one) shows a higher frequency of errors than does that row nearest to the goal (row six).

The trials effect (Figure 12) shows that although there was much initial difficulty in executing the door behaviors on the first few trials, this difficulty had greatly decreased by the last few trials. Thus the door error showed a steady decrease across trials.

The groups by trials interaction for door errors can be seen in Figure 13 which shows that while the AC and PC Ss had overcome by trialblock four the sudden difficulty experienced in trialblock two, the MC group did not overcome this difficulty until trialblock six.

Alternation error analysis--cortical groups. A Kruskal-Wallis one-way analysis of variance performed on the total alternation errors for the pooled cortical groups failed to indicate a significant difference between groups. A four factor analysis of variance of the transformed data (Table 3) revealed significant trial and row effects. The trials effect (Figure 14) is interesting in that while there was improvement across trials for all groups, this improvement was slight. Thus while being significant this improvement may not have been appreciable.

The row effect (Figure 15) was due to the extreme difficulty afforded by rows one, furtherest from the goal, and row six, the nearest

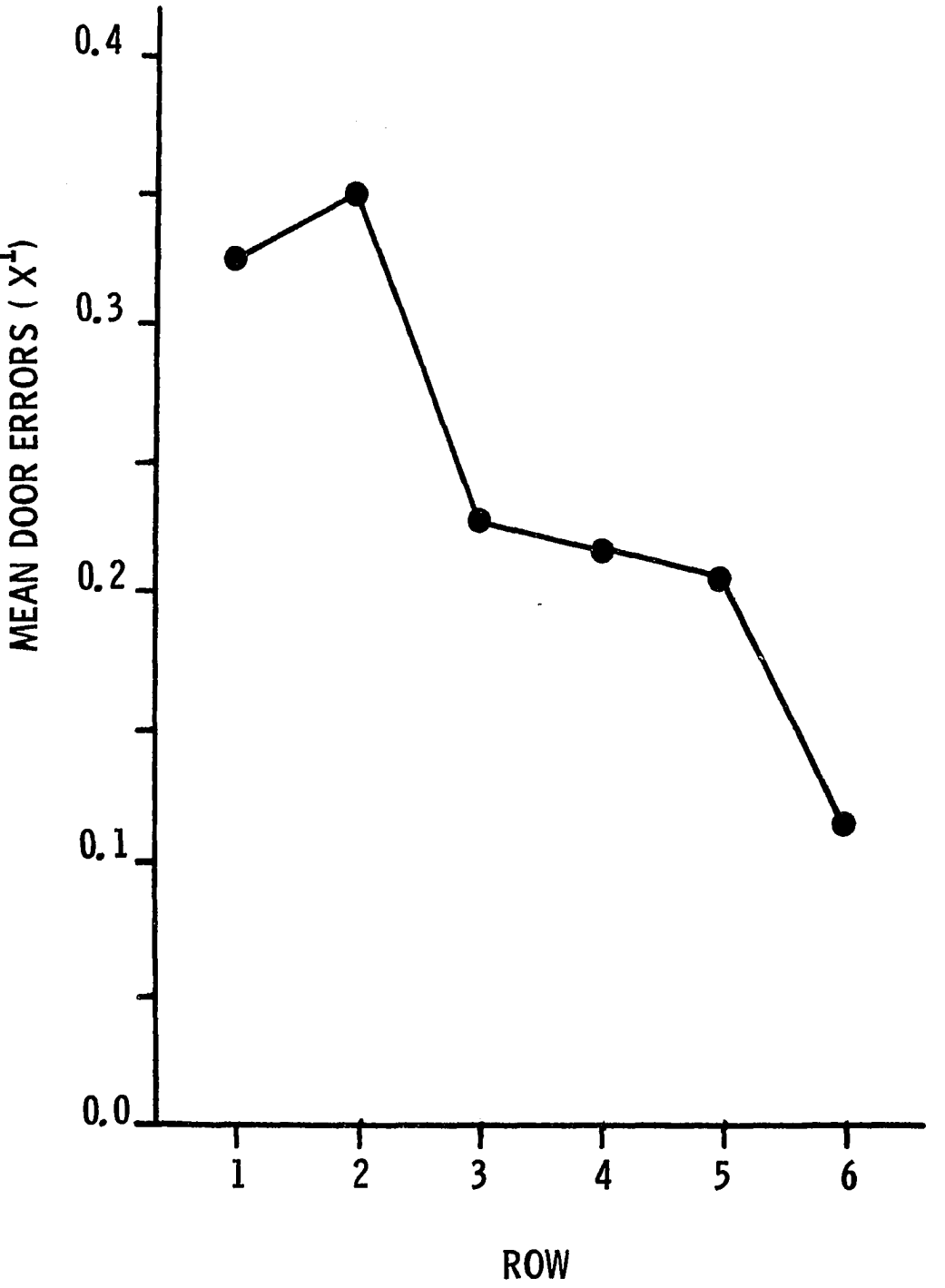

Figure 11. Mean door errors per row.

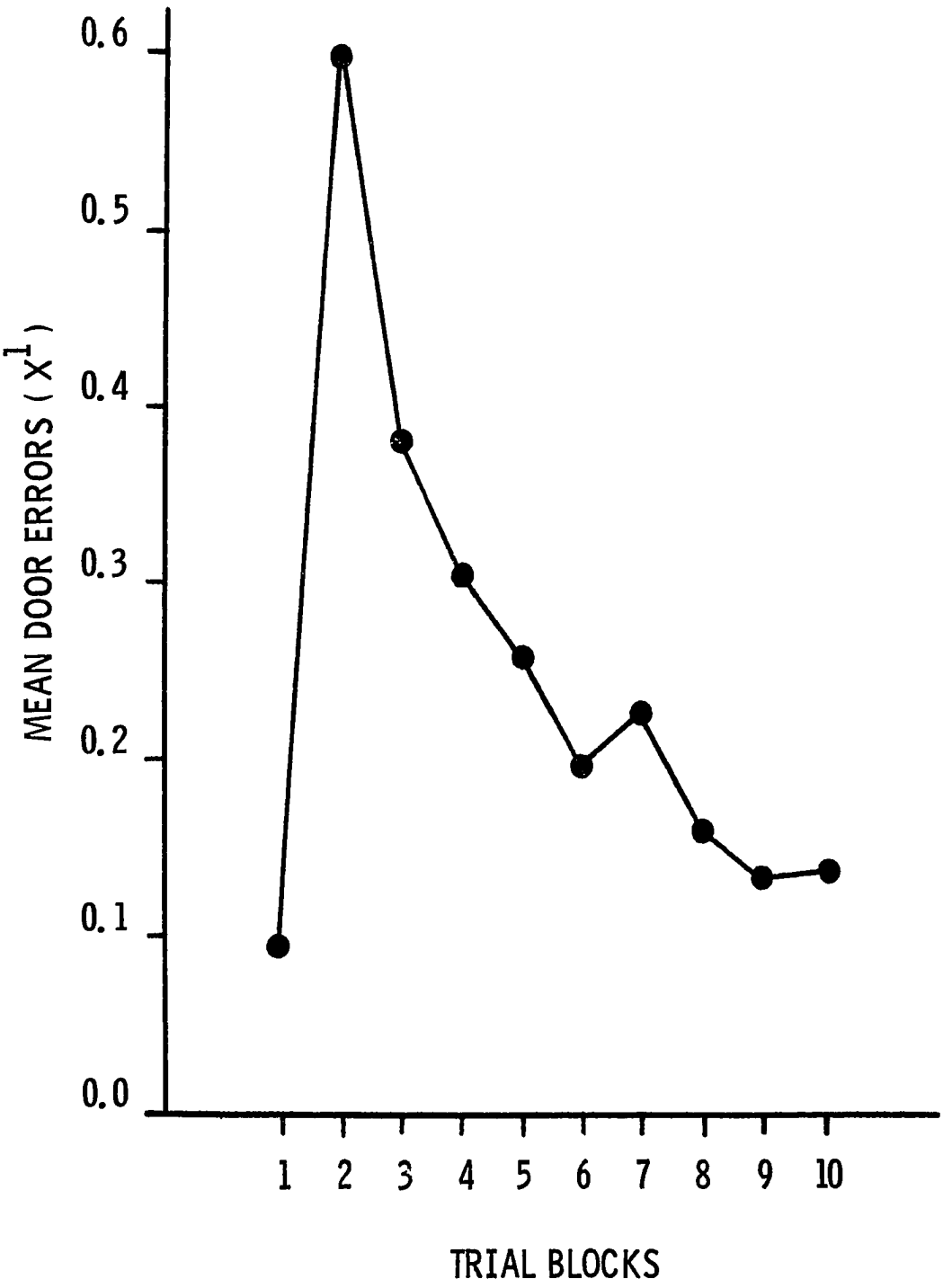

Figure 12. Mean door errors per trial block.

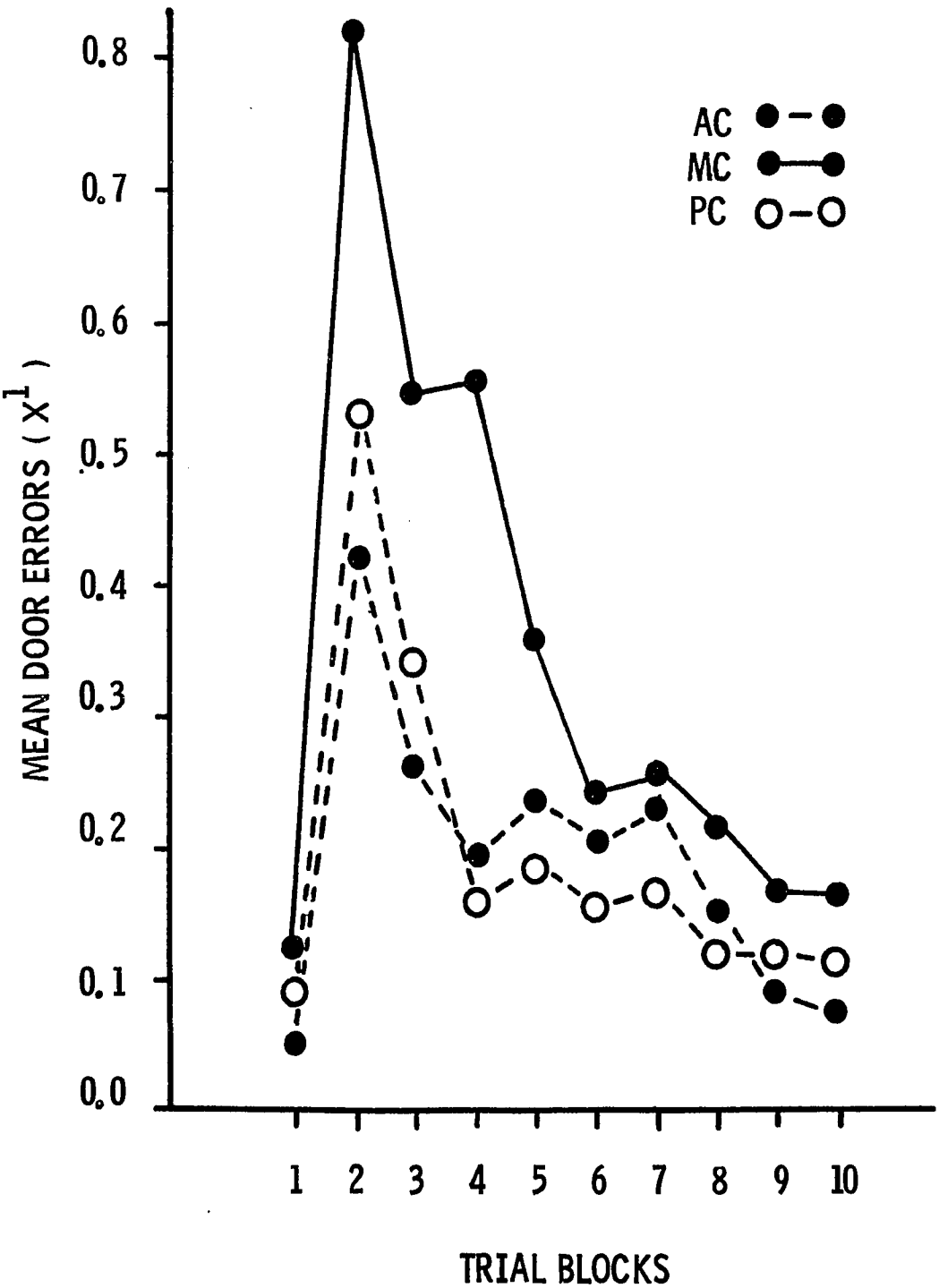

Figure 13. The groups by trials interaction

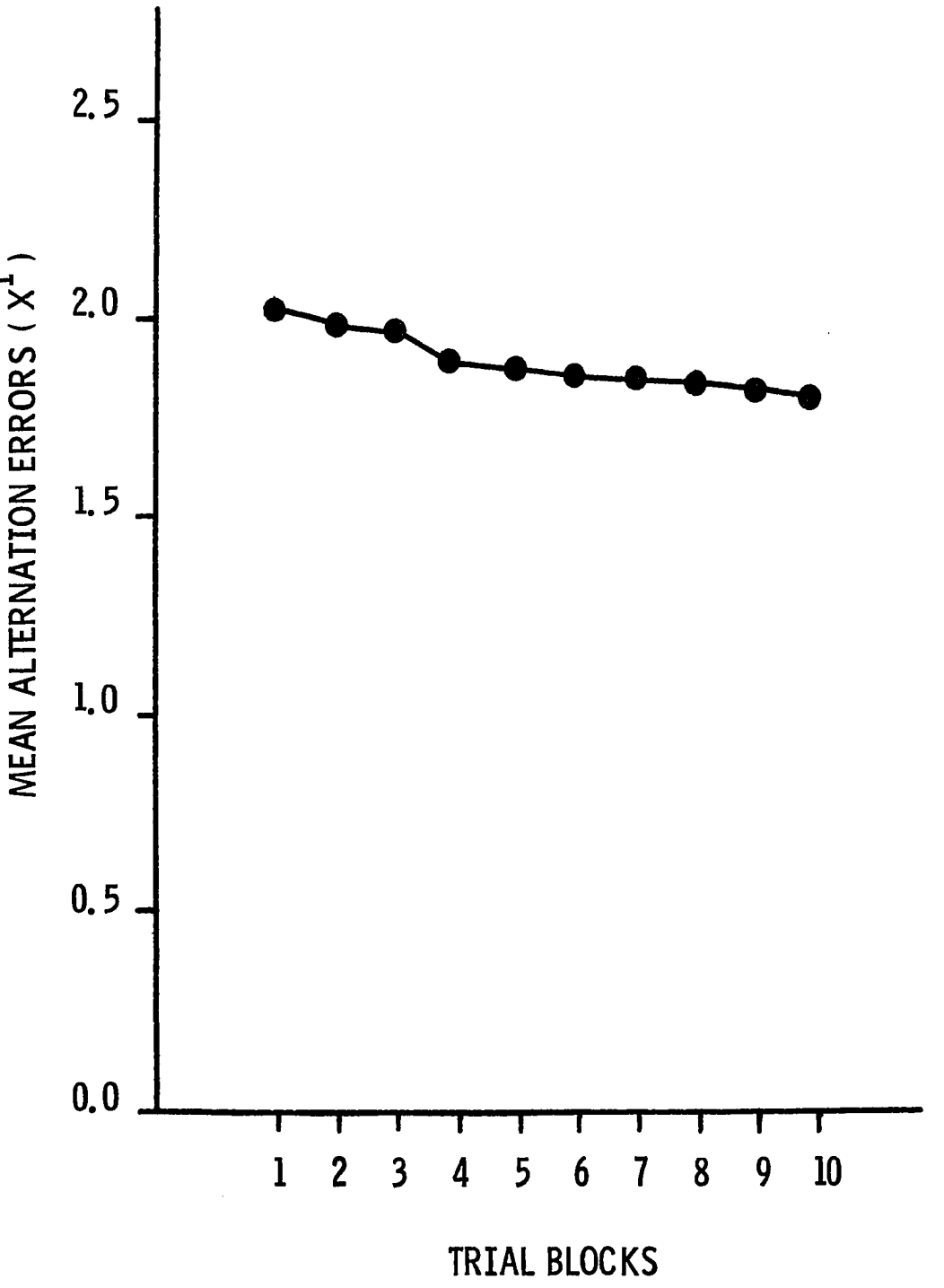

Figure 14. Mean alternation errors per trial.

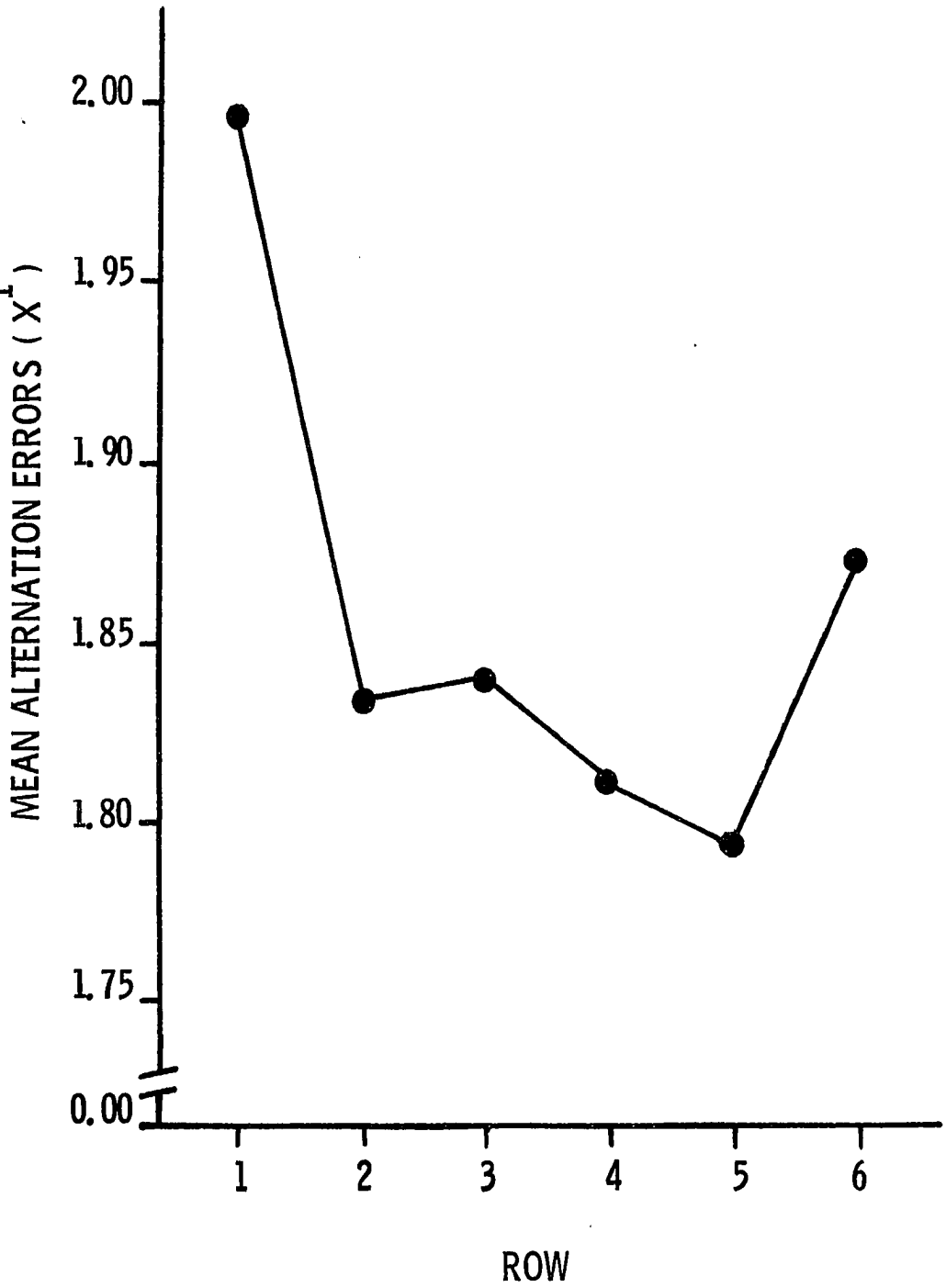

Figure 15. Mean alternation errors per row.

to the goal. Thus while the door error shows a goal-gradient effect, this is not the case for the alternation error.

TABLE 3

SUMMARY OF ANALYSIS OF VARIANCE FOR ALTERNATION ERRORS FOR COMBINED CORTICAL GROUPS OF EXPERIMENT I AND II

Source	df	MS	F
Between subjects	38		
G	2	1.583	2.496
Subj w. groups	36	0.634	
Within subjects	2301		
T	9 (1)	4.542	39.496 [a]
GT	18 (2)	0.218	1.896
T x subj w. groups	324 (36)	0.115	
R	5 (1)	2.014	10.070 [a]
GR	10 (2)	0.571	2.855
R x subj w. groups	180 (36)	0.200	
TR	45 (2)	0.104	1.0
GTR	90 (2)	0.113	1.0
TR x subj w. groups	1620 (36)	0.139	

[a] $p < .01$

Cortical, subcortical and normal behaviors. Figures 16, 17 and 18 show an example of SEP, DLH and SCN brains. The SEP lesions (Figure 16) were extensive, destroying most of the medial and lateral septum. The DLH lesions (Figure 17) produced little damage to non-hippocampal structures and were restricted to the DLH. An example of a SCN brain (Figure 18) is shown to demonstrate the herniation of these brains due to the brain tissue filling the cavity left from the skull

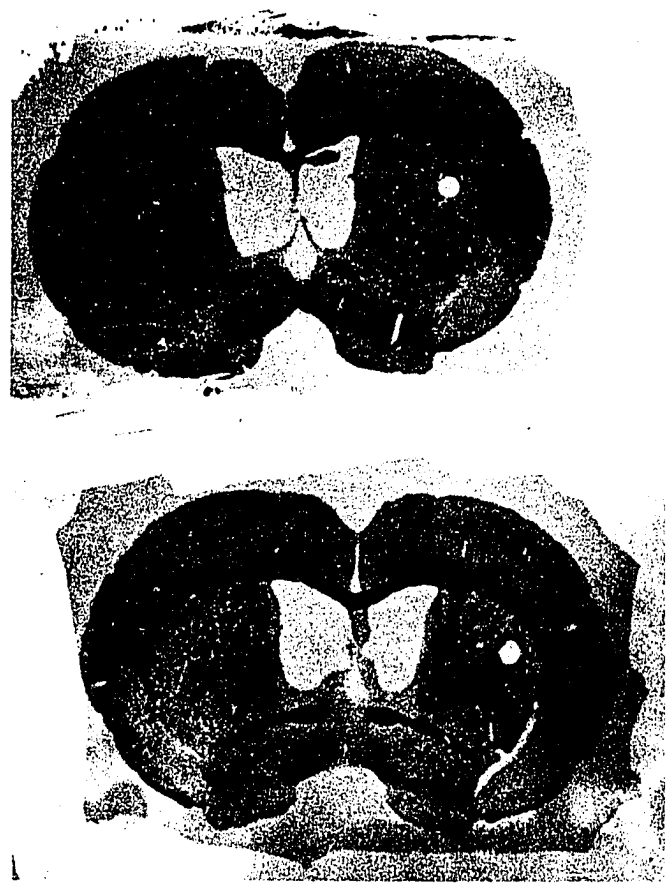

Figure 16. An example of a SEP lesion.

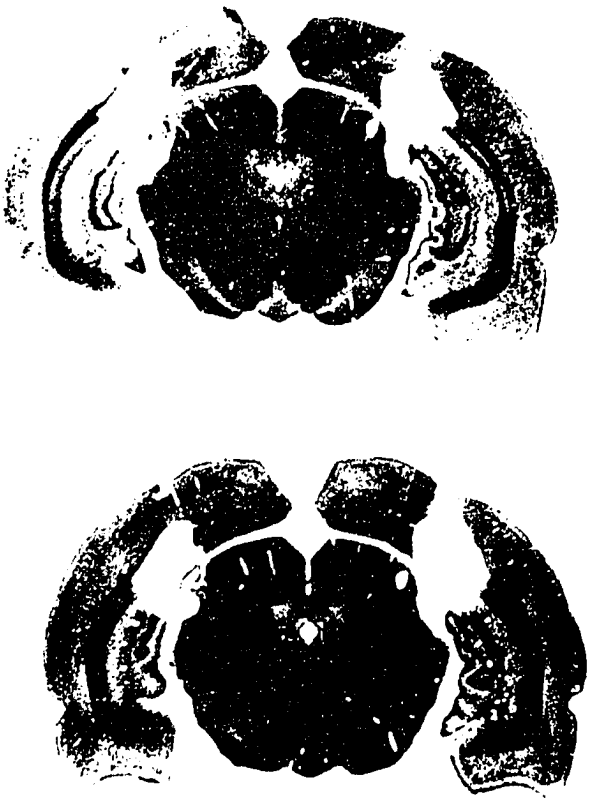

Figure 17. An example of a DLH lesion.

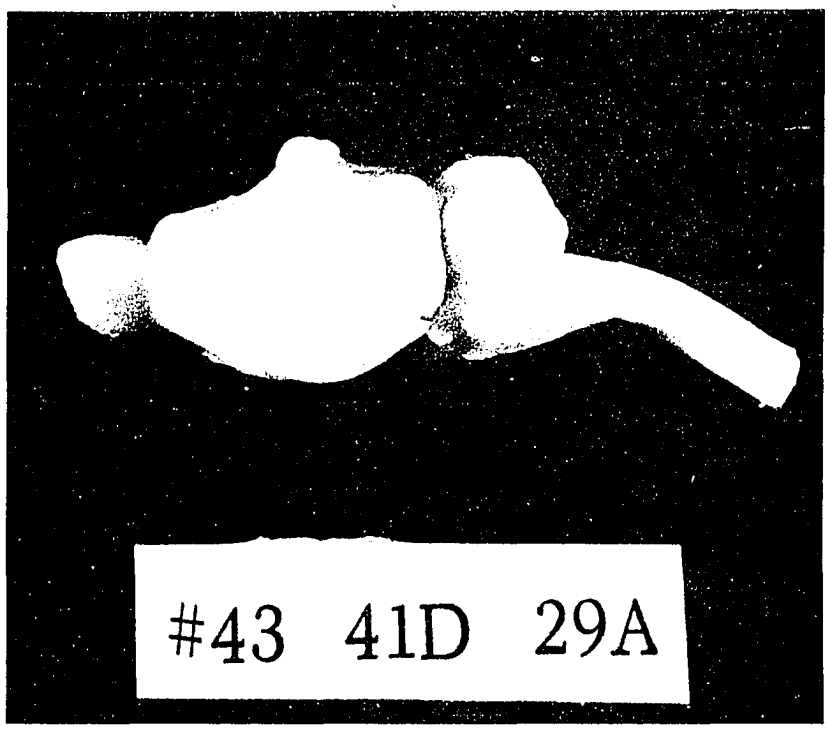

Figure 18. An example of a SCN brain.

removal.

Door error analysis--all groups. A Kruskal-Wallis one-way analysis of variance of the total door errors for each of the seven groups (AC, MC, PC, SEP, DLH, SCN and CN) indicated a significant difference between groups ($H = 18.88$, $df = 6$, $p < .01$). A four factor analysis of variance (groups x trials x rows x replications) revealed significant group, trial and row effects (Table 4).

TABLE 4

SUMMARY OF ANALYSIS OF VARIANCE FOR DOOR ERRORS IN III MAZE FOR ALL GROUPS

Source	df	MS	F
Between subjects	48		
G	6	4.177	3.674 [a]
Subj w. groups	42	1.137	
Within subjects	2891		
T	9 (1)	6.060	56.635 [a]
GT	54 (6)	0.219	2.046
T x subj w. groups	378 (42)	0.107	
R	5 (1)	2.795	18.149 [a]
GR	30 (6)	0.126	0.818
R x subj w. groups	210 (42)	0.154	
TR	45 (6)	0.109	1.579
GTR	270 (6)	0.073	1.058
TR x subj w. groups	1890 (42)	0.069	

[a] $p < .01$

The groups effect for door errors can be seen in Figure 19. Every group, except the PC Ss, differed significantly from the CN group on the basis of Mann-Whitney U (Siegel, 1956) comparisons (Table 5).

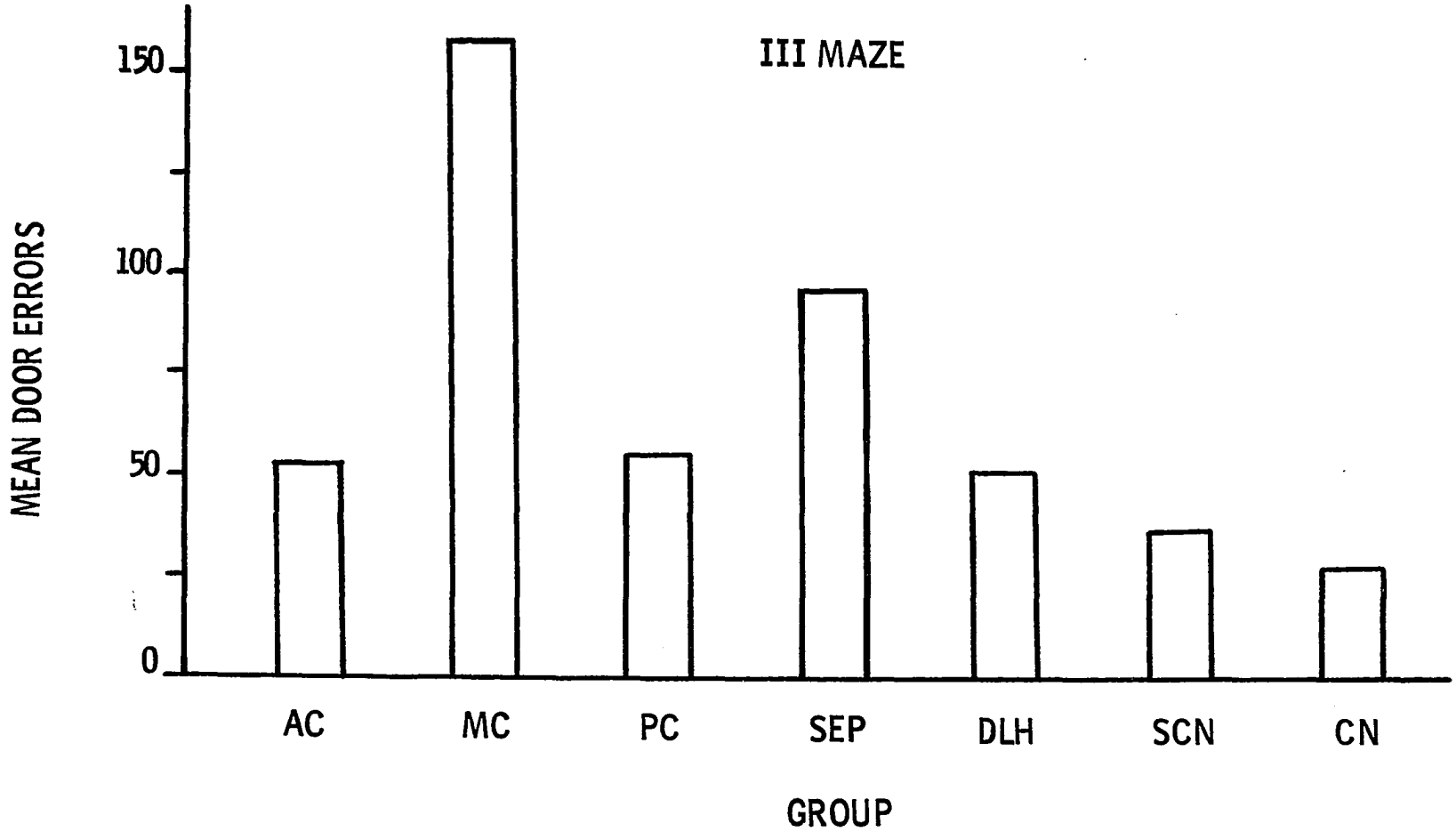

Figure 19. Mean door errors per group.

The other significant differences were between the MC Ss and the DLH Ss and the MC group and the SCN.

TABLE 5

MANN-WHITNEY U COMPARISONS OF TOTAL DOOR ERRORS FOR ALL GROUPS

	AC	MC	PC	SEP	DLH	SCN
CN	.05[a]	.001	*	.01	.002	.05
AC		*	*	*	*	*
MC			*	*	.04	.01
PC				*	*	*
SEP					*	*
DLH						*

* Non-significant

[a] All values represent two-tailed probabilities

The row and trials effects are not shown as they are virtual replicas of the same effects seen in Figures 11 and 12.

Alternation error analysis--all groups. A Kruskal-Wallis one-way analysis of variance of the total alternation errors for the seven groups revealed a significant difference between groups ($H = 20.97$, $df = 6$, $p < .01$). A four factor analysis of variance (groups x trials x rows x replications) indicated significant group, trial and row effects (Table 6). The group effect can be seen in Figure 20. Table 7 gives the Mann-Whitney U comparisons between the various groups and shows that every group except the SCN differs significantly from the CN. The MC group also differs significantly from the AC, SEP and SCN groups. There was also a significant difference between DLH and SEP lesioned

TABLE 6

SUMMARY OF ANALYSIS OF VARIANCE FOR ALTERNATION ERRORS IN III MAZE FOR ALL GROUPS

Source	df	MS	F
Between subjects	48		
G	6	5.599	6.813 [a]
Subj w. groups	42	0.838	
Within subjects	2891		
T	9 (1)	4.580	28.095 [a]
GT	54 (6)	0.268	1.641
T x subj w. groups	378 (42)	0.163	
R	5 (1)	3.047	10.470[a]
GR	30 (6)	0.400	1.374
R x subj w. groups	210 (42)	0.291	
TR	45 (6)	0.139	1.311
GTR	270 (6)	0.092	0.867
TR x subj w. groups	1890 (42)	0.106	

[a] $p < .01$

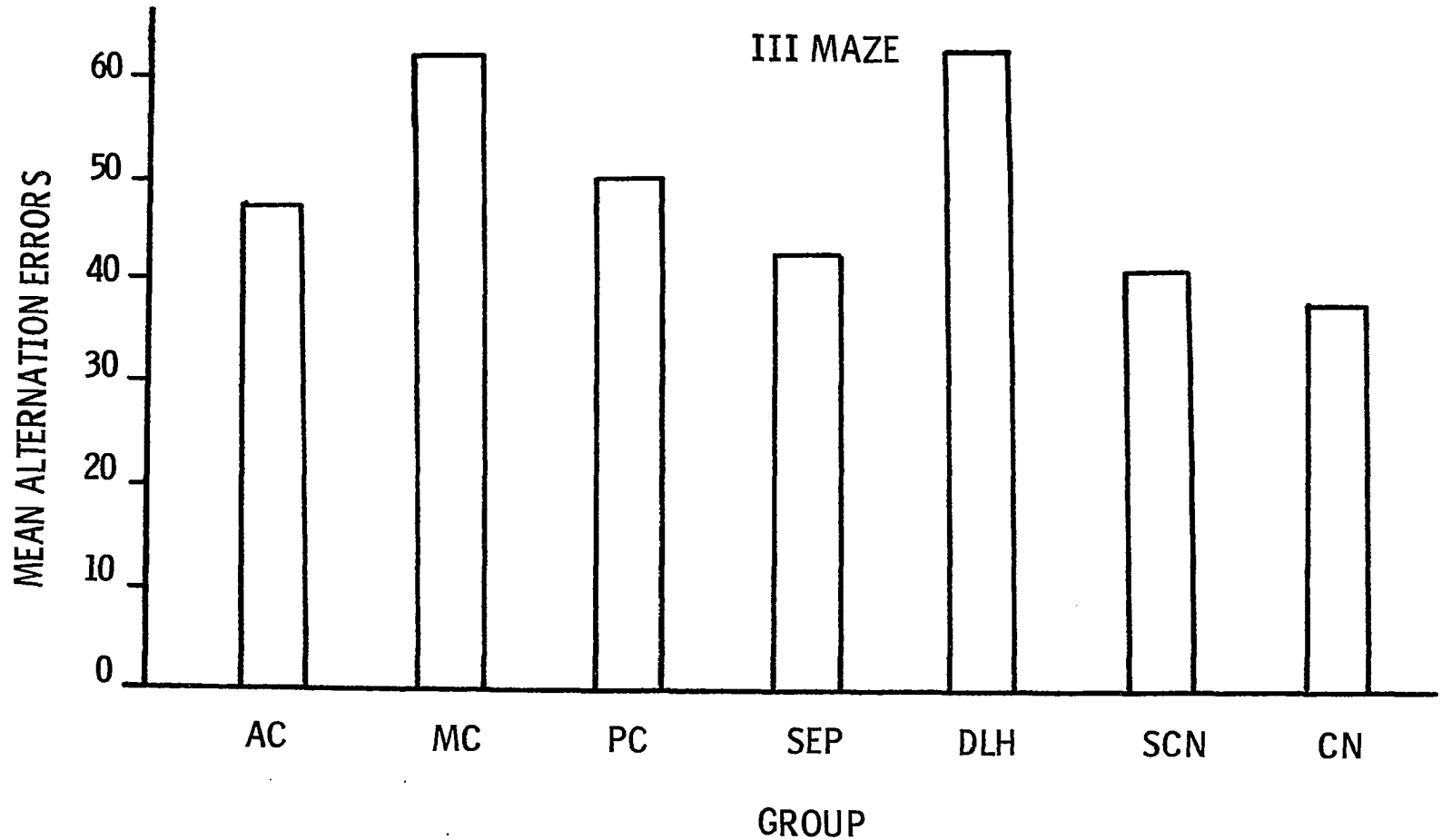

Figure 20. Mean alternation errors per group.

Ss as well as significant differences between the two control groups.

TABLE 7

MANN-WHITNEY U COMPARISONS OF TOTAL ALTERNATION ERRORS FOR ALL GROUPS

	AC	MC	PC	SEP	DLH	SCN
CN	.006	.002	.014	.01	.002	*
AC		.054	*	*	*	*
MC			*	.018	*	.014
PC				*	*	*
SEP					.028	*
DLH						.038

The row effect (not shown) was very similar to that seen for the cortical groups. That is, rows one and six were the most difficult rows for the alternation error, across groups. The trials effect, also not shown indicated a moderate improvement across trials.

Latency analysis--all groups. A four factor analysis of variance of the trial latencies failed to indicate a significant difference between groups but did show significant trial and row effects (Table 8). The trials effect, not shown, was due to a marked, steady improvement across trials. The row effect can be seen in Figure 21 and is interesting in that while row six is a difficult row with respect to the alternation error, latencies in this row were the shortest of any.

Door Error Maze (DEM)

Table 9 gives the results of the between group comparisons of the total errors on the DEM. These results closely parallel those seen

TABLE 8

SUMMARY OF ANALYSIS OF VARIANCE OF III MAZE LATENCIES FOR ALL GROUPS

Source	df	MS	F
Between subjects	48		
G	6	9.903	1.488
Subj w. groups	42	6.656	
Within subjects	2891		
T	9 (1)	32.536	92.063 [b]
GT	54 (6)	0.305	0.863
T x subj w. groups	378 (42)	0.353	
R	5 (1)	1.076	5.020 [a]
GR	30 (6)	0.230	1.072
R x subj w. groups	210 (42)	0.214	
TR	45 (6)	0.115	1.477
GTR	270 (6)	0.082	1.057
TR x subj w. groups	1890 (42)	0.078	

[a] $p < .05$

[b] $p < .01$

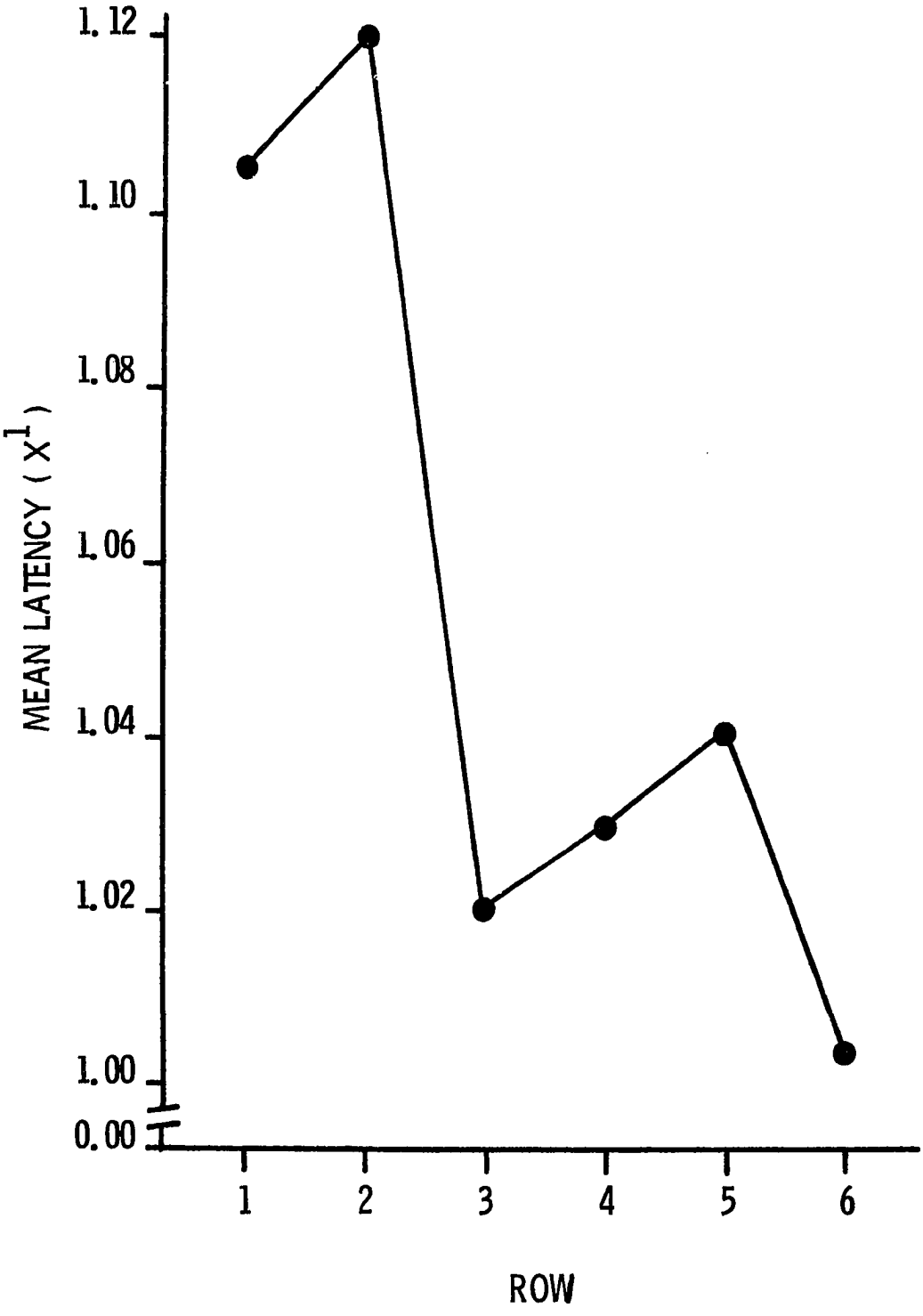

Figure 21. Mean latency per row.

in Table 5 for door errors on the III maze. The performance of the various groups on the DEM can be seen in Figure 22. With the exception of the DLH lesioned Ss all groups differ significantly from the CN. The MC group differs significantly from the AC group, the DLH group and both control groups.

TABLE 9

MANN-WHITNEY U COMPARISONS OF TOTAL ERRORS ON THE DOOR ERROR MAZE (DEM) FOR ALL GROUPS

	AC	MC	PC	SEP	DLH	SCN
CN	.06	.001	.02	.02	*	.01
AC		.04	*	*	*	*
MC			*	*	.06	.04
PC				*	*	*
SEP					*	*
DLH						*

A comparison of the various groups in terms of trial latencies (Figure 23) revealed that the SEP lesioned Ss had significantly lower latencies than the AC, MC and PC groups and the SCN Ss (Table 10).

Alternation Error Maze (AEM)

There were no significant differences between groups as measured either by errors (Figure 24) or trial latencies on this apparatus.

Straight Alley Maze (SAM)

There were no significant differences between groups in terms of trial latencies during the acquisition phase.

Figure 25 shows the mean performance of the various groups

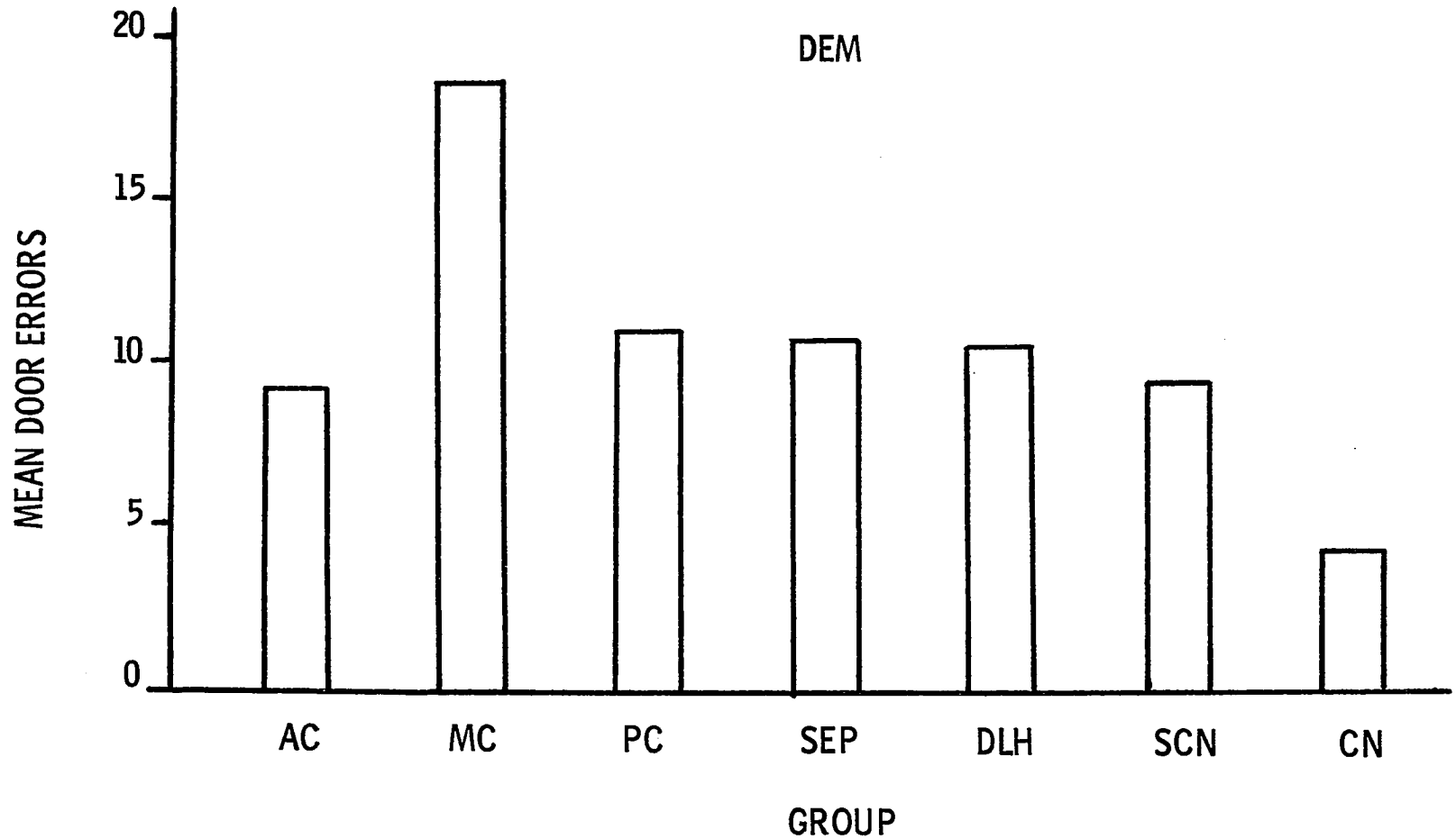

Figure 22. Mean door errors per group.

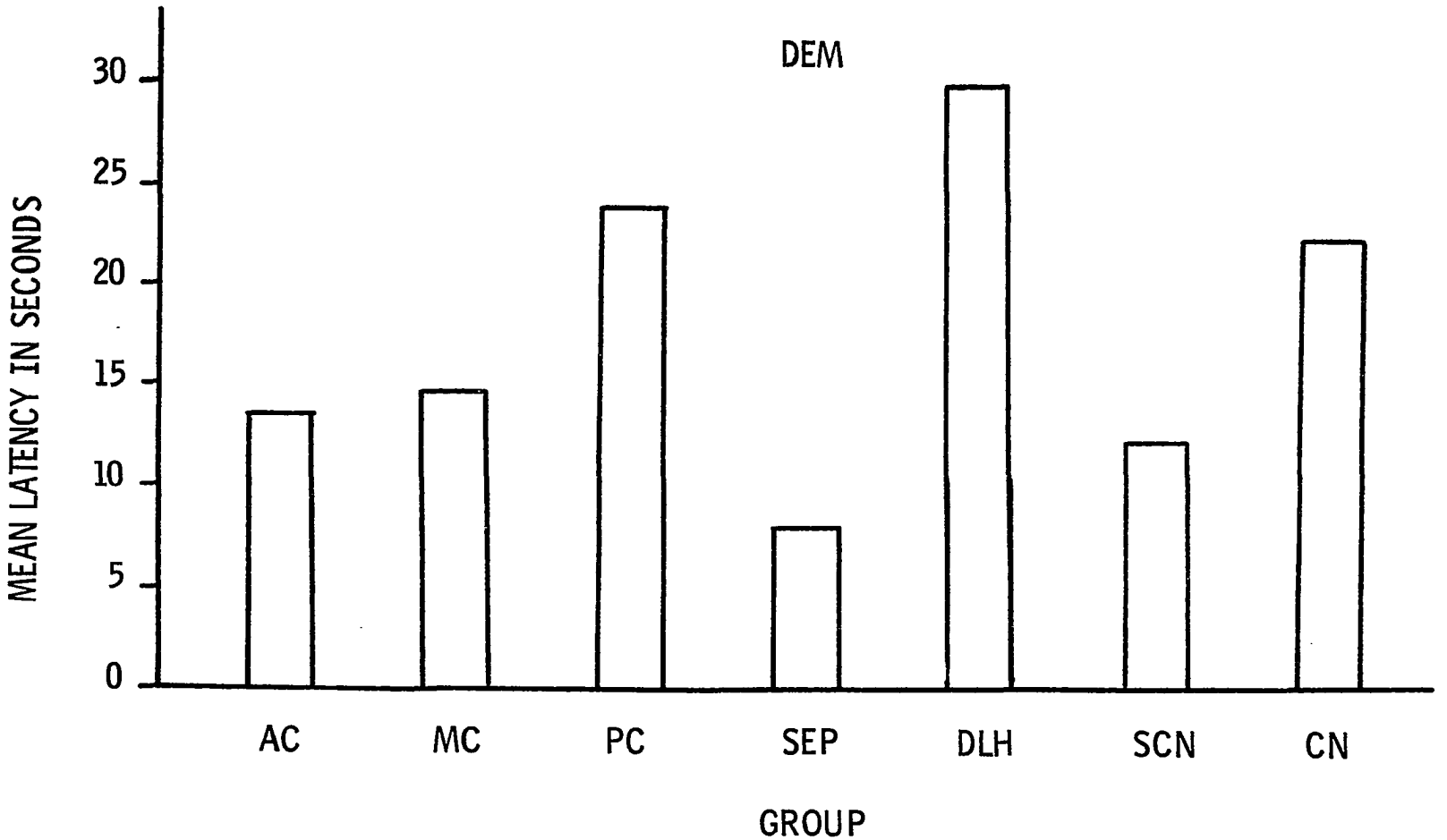

Figure 23. Mean latency per group.

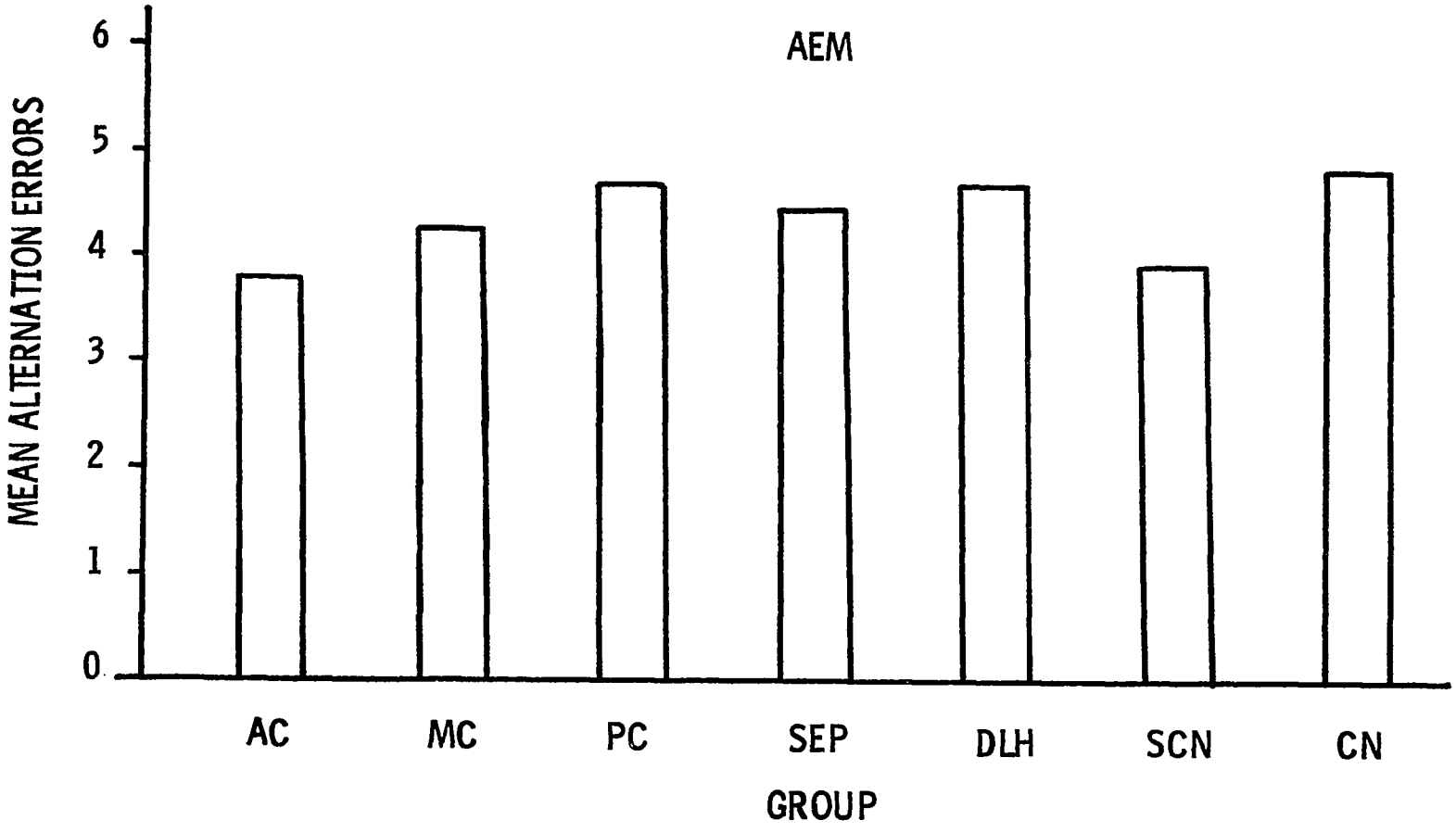

Figure 24. Mean alternation errors per group

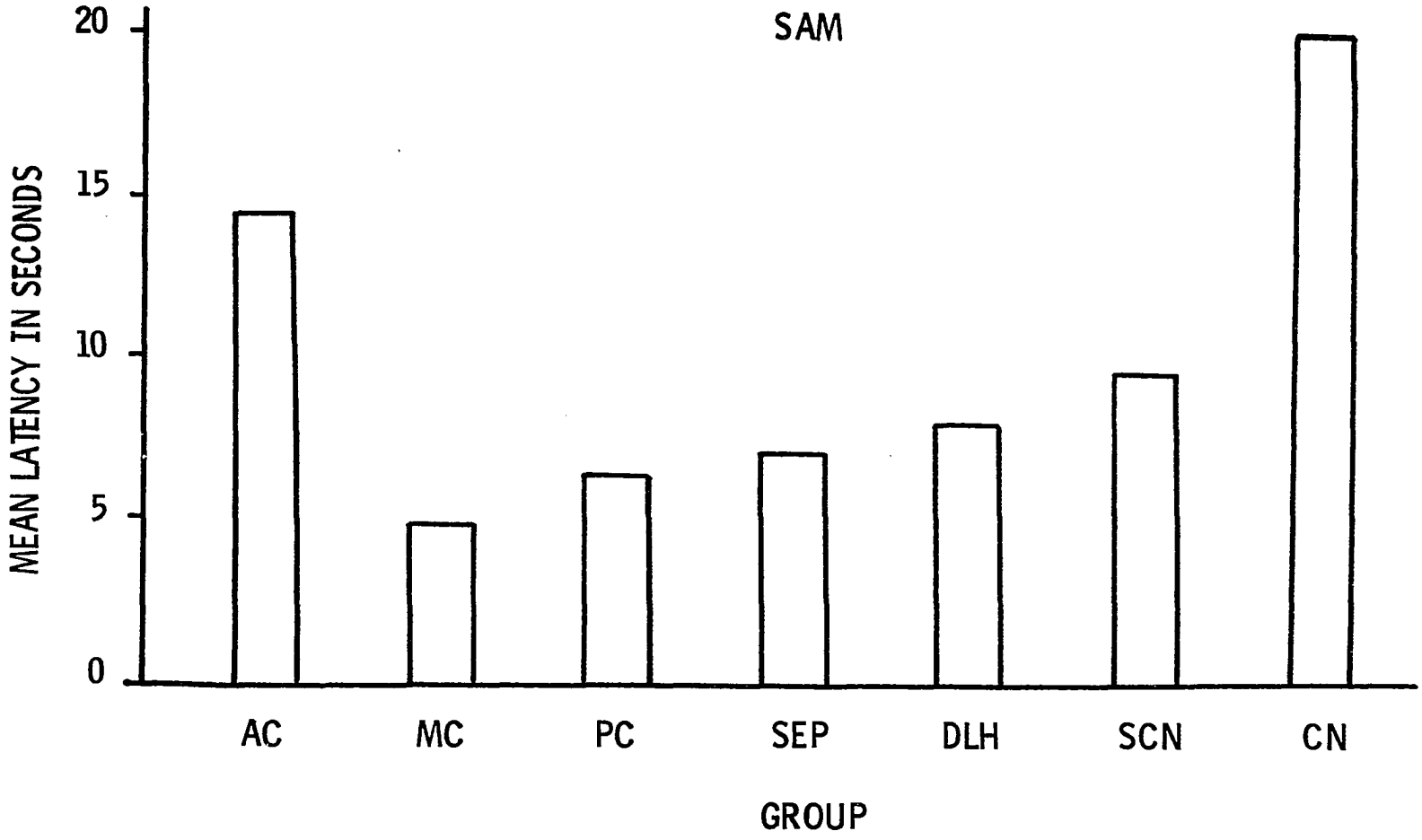

Figure 25. Mean straight alley runway extinction latency per group

for the extinction phase of the SAM testing. Mann-Whitney U comparisons of the groups in terms of extinction latencies revealed no significant differences.

TABLE 10

MANN-WHITNEY U COMPARISONS OF TOTAL LATENCIES ON DEM FOR ALL GROUPS

	AC	MC	PC	SEP	DLH	SCN
CN	*	*	*	*	*	*
AC		*	*	.032	*	*
MC			*	.004	*	*
PC				.007	*	*
SEP					*	.017
DLH						*

A comparison of the mean for trials nine plus ten for acquisition minus the mean of trials nine plus ten during extinction (Wilcoxon matched-pairs signed-ranks test; Siegel, 1956) indicated that all groups did show significant extinction. The AC and SEP groups did not have enough Ss in each group for an adequate statistical analysis of this data (N = 5 per group) but they appeared to show appreciable extinction.

Activity

Cortical effects. A comparison of the group means of the activity scores (total photocell interruptions) which combined the S's activity scores in Experiment I with the cortically lesioned Ss activity score for day one of Experiment II indicated that the MC group was significantly more active than the AC ($t = +2.32$, $df = 20$, $p < .05$) but not

the PC group ($t = +1.63$, $df = 22$, NS). There was also no significant activity differences between the AC and PC groups ($t = -0.97$, $df = 20$, NS).

The percentage of cortex removed also failed to show a significant correlation with activity across groups (34 cases, $R = 0.172$). There were also no significant correlations between amount of cortical destruction and activity within any of the three cortical groups (AC, $R = +.224$; MC, $R = +.532$; PC, $R = -.170$). The number of cases in each group were ten, twelve and twelve respectively.

The correlation between activity and door errors, across groups (34 cases), was significant ($R = +.458$, $p < .01$) but this was not the case for the alternation error ($R = +.267$). There were no significant correlations between activity and either error within any cortical group.

The activity phase of Experiment II can be seen in Figure 26 which shows the three days activity scores for each of the seven groups. On day one (Table 11) the CN group was significantly less active than every group except the AC and the SCN groups. The AC group was significantly less active than the MC, the SEP and the DLH groups. The MC group was significantly more active than the SCN group. The PC group was significantly less active than the SEP group. Both the SEP and DLH groups were significantly more active than the SCN group. There was a tendency for the subcortically lesioned Ss to be more active than the other groups followed by the cortically lesioned and control Ss in that order.

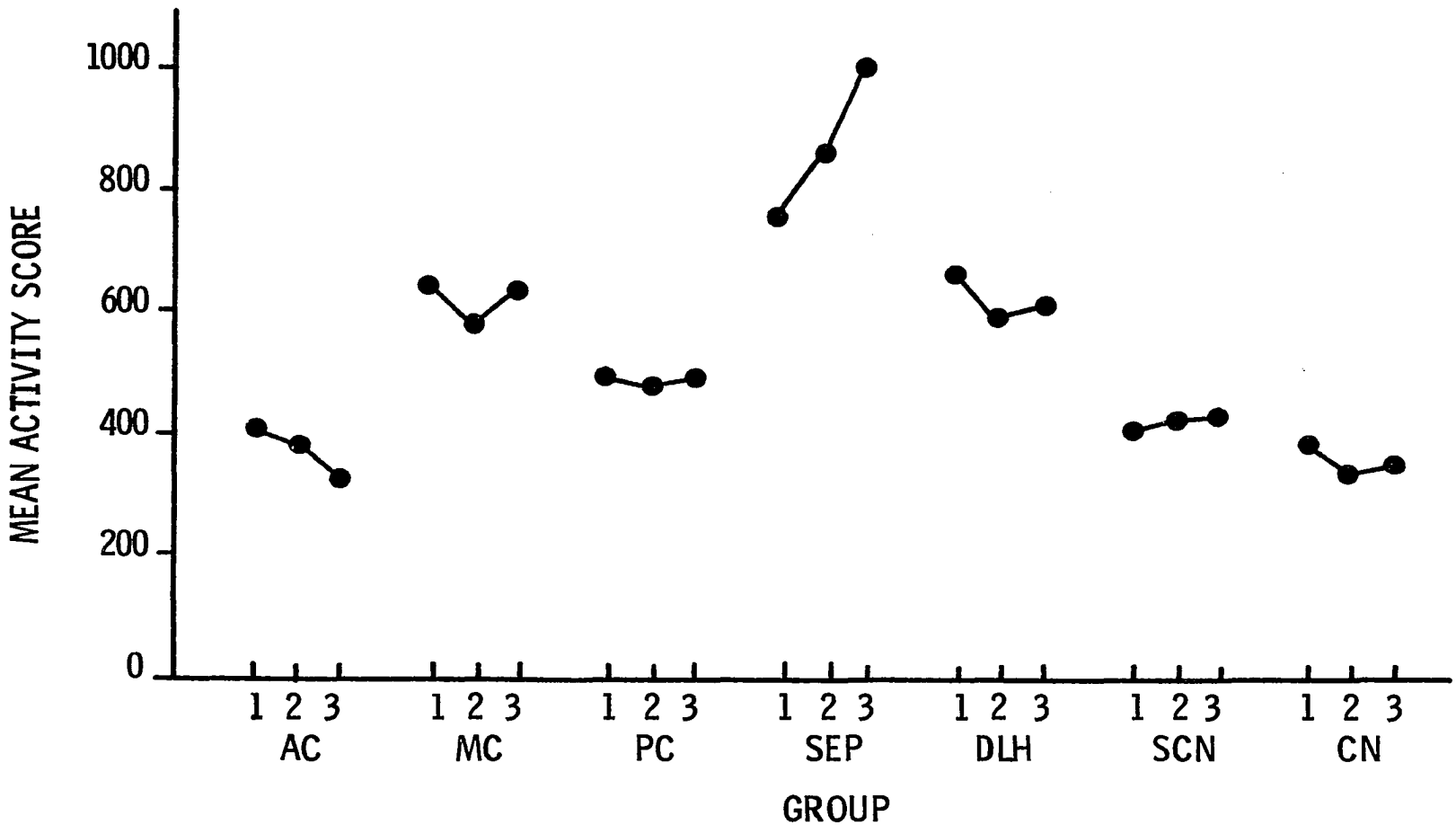

Figure 26. Mean activity score per group for the three activity sessions

TABLE 11

MANN-WHITNEY U COMPARISONS OF DAY ONE
ACTIVITY SCORES FOR ALL GROUPS

	AC	MC	PC	SEP	DLH	SCN
CN	*	.000	.05	.002	.000	*
AC		.006	*	.008	.002	*
MC			*	*	*	.002
PC				.008	.038	*
SEP					*	.006
DLH						.000

By day two (Table 12) the main differences were between the subcortically lesioned Ss (SEP and DLH Ss) and the other groups with the subcortically lesioned Ss being the most active.

TABLE 12

MANN-WHITNEY U COMPARISONS OF DAY TWO
ACTIVITY SCORES FOR ALL GROUPS

	AC	MC	PC	SEP	DLH	SCN
CN	*	.028	*	.000	.002	*
AC		.030	*	.008	.006	*
MC			*	.02	*	*
PC				.008	*	*
SEP					.01	.002
DLH						.02

On day three (Table 13) the CN group was significantly less active than all groups except the AC and SCN groups. The AC group was also significantly less active than the other cortical or subcortical

groups. The MC group was significantly more active than the CN, AC and SCN groups but was significantly less active than the SEP group. The PC group was significantly more active than the CN and AC groups but was significantly less active than the SEP group. The SEP Ss were significantly more active than all other groups. The DLH Ss were significantly more active than the CN, AC and SCN groups but were significantly less active than the SEP group. The SCN group was significantly less active than the MC, SEP and DLH groups. On day three, as on day one, the order of activity, from most to least active, was roughly subcortical groups first, cortical groups second and control groups were the least active.

TABLE 13

MANN-WHITNEY U COMPARISONS OF DAY THREE ACTIVITY SCORES FOR ALL GROUPS

	AC	MC	PC	SEP	DLH	SCN
CN	*	.004	.01	.000	.000	*
AC		.01	.004	.004	.002	*
MC			*	.008	*	.028
PC				.002	*	*
SEP					.004	.002
DLH						.054

CHAPTER III

DISCUSSION

The finding that the MC group made significantly more door errors than either of the other two cortical groups suggests that the rat neocortex is not equipotential with respect to maze mastery. This conclusion is further strengthened by the finding that there were no significant differences between the groups in terms of amount of cortex destroyed. Thus the principle of "mass action" cannot explain the differences in performance between the lesion groups.

The finding that all three cortically lesioned groups performed nearly identically with respect to the alternation error would suggest that the rat neocortex is equipotential with respect to maze mastery. The finding that there were no significant differences between the groups in terms of amount of cortex destroyed would simply strengthen this conclusion as the law of mass action would also appear to be satisfied.

Thus one finding of this study was that while a given neural mass may be equipotential with respect to some behaviors that same neural mass may not be equipotential with respect to another set of behaviors. The apparent inconsistency of this statement is underscored by the fact that it is based upon observations of the same individuals

following a single nervous system lesion.

Any inconsistency in the above statement cannot be due to simple inaccuracies of lesion measurement because the two behaviors being measured were in the same individuals at approximately the same time and therefore size of lesion was controlled. If the increase in door errors in the MC group was due to a greater amount of mass being removed (but inaccurately measured) why was there not a corresponding increase in alternation errors in that group?

These findings suggest the possibility of a site by behavior interaction as far as a deficit in learning the III maze habit is concerned. That is, the effect of a lesion is dependent upon the cortical region destroyed and the behavior under study. Furthermore, since there was no significant correlation between alterations of these behaviors and amount of brain damage, the concept of mass action is also challenged. Finally, the percentage of cortex removed failed to relate to other behaviors such as general activity.

These findings would suggest that some caution should be exercised when proposing "equipotentiality" tempered with "mass action" as determinants of neurobehavioral relationships. As important as these concepts are, they must be considered in terms of particular behaviors. That is, behavioral definition is as important as neuroanatomical specification when attempting to state neurobehavioral relationships.

The finding that both III maze errors were significantly elevated in nearly all surgical groups indicates that these behaviors are affected by brain damage. Only the PC group failed to show a significant departure from control values for door errors (even this was

significant for one-tailed comparisons) while only the SCN group failed to significantly differ from the CN group in terms of alternation errors. Here it should be noted that the SCN should not be considered to be non-brain-damaged. That is to say, their brains were not normal, having filled the cavity created by the skull removal (Figure 18). While the complete effects of this situation are not known it would not be surprising to find that these Ss differed significantly from controls in several behaviors.

Although the differences between cortical groups in terms of door errors was significant when the Ss for Experiments I and II were combined, this was not the case for either set of data analyzed separately. Since there were no significant differences between the groups for either error measure the two groups were considered to be statistically from the same population. The significant effect observed from pooling the groups indicates that this was a trend of the population that was not easily detected in small samples. Therefore it is not surprising to find no significant differences between cortical groups for door errors in the analysis of the data from Experiment II (Table 5). It was surprising to find that the SCN group and the DLH lesioned Ss differed significantly from the MC group in terms of door errors. Apparently the swelling of the brain into the bone cavity in the same area as the MC lesions did not produce effects similar to the lesion.

The seven group, alternation error, analysis for the III maze found that all brain-damage groups differed significantly from the CN group. This indicates that the alternation error is also affected by brain damage. It was surprising to find that this error was so highly

elevated in the DLH lesioned Ss which differed significantly from the CN and the SEP group. The MC group also made significantly more alternation errors than the SEP group or the SCN group. Thus it appears that the alternation error is more sensitive to a variety of lesions than is the door error which is expressed mainly in terms of cortical effects.

The trial latencies for the III maze failed to uncover any specific lesion groups with deviant latencies. However the finding that the lowest latencies were made in row six and that this row also had the greatest number of alternation errors would indicate that the alternation error is not due to a simple slowing of behaviors.

The door error maze (DEM) analysis indicated that it too could detect brain damage in a manner highly similar to the III maze. With few exceptions Table 5 and Table 8 appear to be almost identical. The tasks themselves would appear to have basic similarities. The III maze door task can be reduced roughly to "going through an 'opening' in a wall". For the DEM the task is more like "going down a 'hall' every time you come to one". The latency analysis for the DEM indicated that the SEP group was the fastest in spite of the fact that they were not the best performers in terms of errors (Table 9). This type of error may also be somewhat independent of latency.

The finding that the alternation error maze (AEM) failed to distinguish between groups would indicate that the AEM may not adequately reflect the "alternation error" component of the III maze. The alternation component of the III maze is probably determined by the sequencing of alternatives while the AEM used in this study required only that the S learned to turn in a particular direction and required

no sequence of turns.

The failure to find any significant differences between groups in terms of either the straight alley maze (SAM) acquisition or extinction behaviors would suggest that the brain-damage related behaviors seen in this study were not due to unusual motivational or motoric factors. The extinction behaviors of all groups would also argue against a simple "response perseveration" hypothesis to explain the results of certain lesions. Thus there appear to be no basic failures of either "maze excitation" or "maze inhibition" in any of the experimental groups. If elevations in either error behavior for the III maze had been due to a simple lack of either general motor facilitation or inhibition this deficit was not seen in the studies dealing with straight runway acquisition or extinction.

The findings that the cortical groups which made the most door errors was also the most active and that there was a significant correlation between door errors and activity across groups suggest that activity is an important dimension of the door error behavior. This does not seem to be the case for the alternation error as the correlation between these behaviors and activity were nonsignificant. The activity phase of Experiment II found that large changes in activity were produced by both cortical and subcortical lesions. The groups that were the most active (Figure 26) were also the ones which made more door errors (Figure 19).

The two primary behaviors under study, door and alternation errors in the III maze, while having some apparent face validity in terms of their distinctions, are not completely understood in terms of

their respective behavioral significance. Although the two behaviors were significantly correlated with each other this correlation was less than perfect ($R = +.473$, $p < .01$) and the two behaviors were affected differently by the various lesions. Furthermore the dynamics of the two behaviors are distinguishable in the maze. The door error shows a goal-gradient across rows with the row furtherest from the goal (row one) showing the most errors and the row nearest the goal (row six) being the least difficult. This was not the case for the alternation error as row six was one of the most difficult rows for this behzvior. The two behaviors also differed with respect to their level of recovery following brain damage. The door error, although being strongly exaggerated by the cortical lesions, had recovered to a near normal level by trial twelve (Figure 12) but the alternation error showed only slight recovery by trial twenty (Figure 14).

The failure of the AEM to detect any group differences while the alternation error behavior in the III maze did (Table 7) would indicate that the alternation error in the III maze is not analogous to a simple T-maze alternation task. However, the door error behavior does appear to be related to whatever is measured in the DEM and in that respect may not be as dependent upon the structure of the III maze for its occurrence as is the alternation error.

III Maze Behaviors

Door Errors

Observations of brain-damaged and normal Ss in the III maze gives one the impression that the rat which makes a large number of

door errors is one which has become disoriented in the maze. That is, quite often that S will explore the wall of the row which does not have an opening (the door is shut once the S enters a row) and will not attend to the wall of the row with the door opening into the next row. This behavior can persist for many minutes in one or several rows. Then suddenly the S will enter the door into the next row, and may or may not repeat the performance of the preceeding row.

Failure to go through a door is not due to a sensory deficit alone as the rat will often orient toward a door, momentarily, and then pass up the opportunity to enter by turning 180° and examining the other wall of the row. Quite often the S will actually peer into the opening but refuse to enter. This behavior is especially exasperating to Es. A normal S may occasionally refuse to enter a door and continue to explore the row but this behavior is usually abandoned after very few trials. However the normal S seldom exhibits the severe disorientation which is easily inferred from observations of the brain-damaged S.

Alternation Errors

The alternation error is difficult to analyze in terms of either related activities or normal and brain-damaged behaviors. The alternation error is usually committed with less loss of time than is the door error, especially in the case of exaggerated door errors and is quite often committed several times in a trial which has a shorter total latency than an earlier trial on which no errors were committed. This error simply consists of turning into the blind cul-de-sac immediately upon entering a row. As the direction of the correct turn varies

from row to row (r l r l r l) the efficient (no error) execution of this behavior requires some type of sequencing mechanism. This could very likely be accomplished by utilizing extra-maze cues although other cues, such as just having turned left (or right), could be utilized. Very possibly the "alternation" behaviors in the III maze involve a more complex interaction between extra-maze, intra-maze and organismic variables than do the "door" behaviors which involve the simpler task of only going forward through the available openings.

A unique feature of the alternation error is that it is always committed prior to a door error. That is, the only opportunity to execute an alternation error is removed once the S turns in the correct direction for that row; door errors are committed at some later point in time. The execution of an alternation error could disorient some brain-damaged Ss and contribute to their door error behavior. If the probability of a door error is higher following the execution (and resulting confusion) of an alternation error, this could explain the significant correlation between the two errors, in the cortically damaged Ss. That these behaviors were not significantly correlated in the CN group ($R = +.304$, $N = 8$) would be consistent with this explanation. However these behaviors were also significantly correlated ($R = +.858$, $N = 8$, $p < .05$) in the DLH group which was low in door errors but high in alternation error behaviors.

The Maze Habit

The importance of the door and alternation errors may not be in some intrinsic property of either but in the fact they demonstrate

that the term "maze habit" may not be a sufficiently detailed description of maze behaviors. The successful maze habit may consist of more meaningful subdivisions than just the door and alternation error behaviors. Highly informative behavioral analyses may focus on some yet undefined behavioral complex. The door and alternation errors can be conceived of as one type of deviation about the true path. That is if an animal alternates incorrectly and runs past the door into a cul, it has left the extreme boundaries of the true path and has committed one alternation and one door error. The S can still fail to enter the next row by leaving the cul and moving back out into the middle of the row, thereby executing a second door error which may reflect a different behavioral substrate from the first door error as the S is back on the true path, only reversed. A yet unexamined class of behaviors could be deviations (failures of orientation) within the true path, such as turning 180° and reversing directions within a row. This could happen after entering the row but prior to encountering the next door and in other situations.

There are many examples of behavior which can be studied in the III maze. The choice of any is somewhat arbitrary. The approach employed here was to analyze the behavioral demands of the test situation. These demands are determined by the physical environment and in the case of the III maze vary from moment to moment depending upon the Ss position in the maze. The problem is one of trying to fractionize these behaviors into meaningful units. Some behaviors, such as tail twitching, are probably non-related to maze mastery while other behaviors, such as going through a door at every opportunity, and turning

left or right in the proper sequence once having entered a door, are obviously intimately involved in the "maze habit". Thus the maze habit can be considered to consist of at least two "units" of behavior which can be defined in terms of the various behavioral demands of the test situation. An examination of the response requirements for the two errors reveals that one, the alternation error, may require a temporal sequencing ability that is not seen in the door error. The organization required of the alternation behaviors would hardly appear to be the same as that seen in door behaviors which do not involve the same level of response sequencing.

Perhaps one dimension along which behaviors may be evaluated is in terms of "temporal" as opposed to "spatial" environmental demands which occasion the behaviors. Behaviors which have a temporal nature may represent a more highly organized set of behaviors than those behaviors which are expressed in terms of immediate, more spatial, environmental demands (such as door entry). Behavior was viewed as expressing varying degrees of such organization by the founder of the Behavioristic movement--John B. Watson. Describing behavior in terms of the "stimulus and response" model, Watson stated

> By response we mean anything the animal does--such as turning toward or away from a light, jumping at a sound, and more highly organized activities such as building a skyscraper, drawing plans, having babies, writing books, and the like. (Watson, 1963, p. 6).

Thus even Watson implied a type of behavioral organization which is amenable to a spatio-temporal analysis. Some behaviors deal with immediate physical environmental demands while others incorporate a level of organization that is not expressed in any one response

execution. Other investigators have also proposed an analysis of behavior in terms similar to this.

> In summary, then, a working distinction can apparently be made between two types of behavioral process. One type occurs in response to simple repetitions of events. The other takes place when the temporal patterns of redundancy with which events occur are more complex. (Pribram, Lim, Poppen & Bagshaw, 1966).

The behavioral analysis of the III maze may afford new insights into neurobehavioral relationships. For instance, is there a point of behavioral fractionation beyond which no reasonable neurobehavioral relationship can be established? This question can only be answered by future research. The present data do indicate, however, that all components of the "maze habit" may not be equally affected by brain lesions.

A Neurobehavioral Analysis

Any proposed neural model, based on III maze behaviors, must take into account the inconsistencies between brain lesions and behaviors, in terms of equipotentiality, seen in this study. There is probably no clear division between the various boundaries, neurologically, as lesions in both the anterior and posterior zones would occasionally inflate the door error score while some lesions covering most of the middle zone failed to produce an increase in door errors. Thus the localization of the door error would not appear to be a precise, static type of neurobehavioral arrangement but instead a more "dynamic" or "graded" localization similar to that proposed by Luria (1966).

Perhaps a neural model which incorporates both the remarkable equipotentiality of the alternation error and the elusive localization

of the door error for the same neural mass is not presently available. This may not be due as much to failings of neuroanatomical sophistication as to the tendency to view behavior as a unitary concept rather than as a behavioral complex. The term "behavioral complex" will refer to a behavior state which includes two or more behaviors in its expression. The term "simple(r) behavior" will refer to those behaviors which compose the more complex behavior. Thus the successful maze "habit" will be designated "complex" when it includes successful "door" and "alternation" behaviors. The concept of a "behavioral complex" allows for a continual analysis of the components of that complex whereas the concept of a "habit", "reflex" or "behavior" implies a definiteness that discourages further analysis. This could greatly influence neurobehavioral theory (and investigations). Granted the concept of a "behavioral complex", as opposed to a "habit", the argument could be made that the more complex the behavior in question (i.e., maze mastery as opposed to a righting response), the higher the probability that several brain systems contribute to that behavior. Therefore, in the absence of an examination of the components of that behavioral product, the greater the probability that "equipotentiality" holds for that behavior being measured. As the total behavioral picture can be reduced to its components then the probability of "specificity" may be increased for the various behavior components.

This argument, while being similar to that of Hunter (1930) emphasizes the neurobehavioral units rather than the neurosensory components of a behavioral complex. Hunter argued that the rat neocortex appeared to be equipotential because "...there are many sensory

projection areas involved in maze control and the lesions do not affect all of them." (1930, p. 466). The following quotations from Hunter's 1930 article explain his position more fully.

> In the maze habit we are not yet wise enough to specify what precise stimuli are involved and when and where they function in the maze. (p. 495).
> . . . . . .
> The maze act and the learning process are much more complicated phenomena than the conclusions of some previous investigators would indicate. (p. 461).
> . . . . . .
> ...the maze habit is controlled by a multiplicity of sensory cues. (p. 464).
> . . . . . .
> If the maze response were controlled by a single sensory field, as is the case with brightness discrimination, then one would expect the results on cortical lesions to reveal some fairly definite localization of the neural control. If, however, many different stimuli are essentially equally effective in the control of the response, then no one cortical lesion should destroy the response, and the ability to form and retain the habit should be decreased roughly in proportion to the amount of cerebral tissue destroyed. As more and more cortical projection areas are removed, and the total integrative function of the cortex is increasingly hampered. (p. 465).

These formulations obviously represent an era of psychology which was still strongly concerned with questions of behavior which were expressed in terms of the reflex arc doctrine. Thus "stimuli" and "response" were considered to be the principal characters in the expression of behavior. If, however, emphasis is placed upon the <u>response</u> side of the S-R formula for behavior then the statements by Hunter (quoted above) might be written as follows:

> In the maze habit we are not yet wise enough to specify what precise behaviors are involved and when and where they function in the maze.
> . . . . . .
> The maze act and the learning process are much more complicated phenomena than the conclusions of some

> previous investigators would indicate.
> . . . . . .
> ...the maze habit is composed of a multiplicity of behavioral units.
> . . . . . .
> If the maze habit were controlled by a single response, as is the case with brightness discrimination, then one would expect the results on cortical lesions to reveal some fairly definite localization of the neural control. If, however, many different behaviors are essentially equally effective in the control of the habit being measured, then no one cortical lesion should destroy the response, and the ability to form and retain the habit should be decreased roughly in proportion to the amount of cerebral tissue destroyed. As more and more tissue is eliminated, more and more neurobehavioral units will be removed, and the total behavioral integration controlled by that neural mass will be increasingly hampered.

Hunter's formulations would lead one to believe all that was necessary to demonstrate localization of function within the rat cortex, for the III maze habit, was to identify the necessary stimuli which occasioned the expression of the habit and look for response failures to each stimulus following discrete brain damage to the neural area concerned with the reception of that stimulus. Cannot the same logic be applied using the concept of a "behavioral" as opposed to a "stimulus" analysis of the III maze habit.

Employing a behavioral analysis of the maze habit much in the manner which Hunter advocated a sensory (stimulus) analysis, leads to the following conclusions: (a) the more complex a behavior, the greater the probability it will exhibit an equipotential neurobehavioral relationship; (b) the simpler behaviors will show a higher degree of localization; and (c) "mass action" should hold for the more complex behaviors. The only conclusion which is challenged by the data from this study is the last conclusion (c). Neither door nor alternation errors correlated

with amount of cortical damage significantly, as a function of location. This could be seen in comparisons of door and alternation error behaviors in the same animals. The alternation error also failed to correlate with amount of cortical damage. But again this could be due to the small range of lesion sizes in this study. One would certainly expect that large lesions (say 80% of the cortex) would generally produce a greater deficit in behaviors which were not highly localized. However, it may take quite large differences in lesion size to produce a mass effect. There could of course be, for some behaviors, a deficit level beyond which further increases in neural damage no longer produce increasing behavioral loss.

Thus the concept of an analysis of a "behavioral complex" by incorporating a Hunter-like model but emphasizing "behavioral unit" instead of "stimulus" can explain both equipotential and localized neurobehavioral relationships as a function of response complexity. This model would interpret the alternation error as being more complex than the door error as the alternation error involved a type of response sequencing that the door error did not. This is somewhat supported by the finding that the DEM apparently presented about the same behavioral demands as the III maze in terms of door behaviors while the III maze alternation error was not equivalent to the simple alternation problem presented in the AEM. Also, the alternation error failed to show an appreciable recovery while the door error exhibited marked recovery. Intuitively it would seem that a "simpler" behavior would show a faster and perhaps a greater amount of recovery than a more complex one. Normal Ss also master the door behaviors more readily than the

alternation behaviors on the III maze.

General Considerations

The possibility of either a stimulus or response analysis of various habits has certainly been recognized by previous researchers, including K. S. Lashley. Writing in 1930 (Beach, Hebb, Morgan & Nissen, 1960) Lashley discounted both of these possible modes of analysis as being inadequate to the task of establishing information of a neuro-behavioral nature. The reasoning for this was based on two conclusions: (1) various stimuli can occasion the same response and (2) the same motor elements are not necessarily used in the learning and performance of motor habits. Thus integrated behavior consisted of more than stimulus--response units and shows a "unity of action" even in the presence of brain lesions. These conditions, being granted, still do not invalidate the concept of an analysis of the behavior side of the neurobehavioral problem in terms of defined behavioral units.

The criticism that "the same motor elements are not necessarily used in the learning and performance of motor habits" is irrelevant to the observations that a correct sequence of alternations is required to reach a door which must be entered to eventually obtain food. A left turn is not equivalent to a right turn nor is failure to go through a door equivalent to entering that door. The execution of these behaviors is demanded by specific task requirements. The behavioral requirement, at least, can be held constant. This is one method of controlled behavioral observation and allows for the fractionation of a complex behavior into its component parts. This method of observation enables us to see that although behavior may have an integrated "unity"

it can also be viewed as a multitude of units which may not all be controlled in the same manner by a given neural mass. While a given neural mass may exhibit equipotentiality with respect to some behaviors, other behaviors, perhaps less complex, may be more localized.

What is the significance of finding that some behaviors may be more localized than others?

> Specialization of functions in the cerebral cortex is an indisputable fact, but we have yet to find an adequate interpretation of it. We have asked, where are psychological functions localized in the brain? and have gained a meaningless answer. We should ask, How do specialized areas produce the details of behavior with which they are associated: what are the functional relationships between the different parts and how are they maintained? (Lashley, 1930).

This statement referred to sensory localization but suggests that questions concerning psychological localization are meaningless. In this statement "psychological functions" is equated to "the maze habit". This must be the case as Hunter states

> It has been necessary in evaluating Lashley's theory of the equipotentiality of cerebral action to offer a detailed analysis of the sensory control of the maze habit because the theory rests almost solely upon data gathered on this habit. (Hunter, 1930, p. 465).

Thus conclusions concerning psychological processes are derived from rat maze data. This immediately raises the question, Can some of the psychological processes of Man be inferred from the maze behavior of the rat? Lashley apparently thought so.

> Analysis of the maze habit indicates that its formation involves processes which are characteristic of intelligent behavior. Hence the results for the rat are generalized for cerebral function in intelligence. Data on dementia in man are suggestive of conditions similar to those found after cerebral injury in the rat. (Lashley, 1929, p. 176).

Both man and rat can be seen as organisms expressing varying degrees of behavioral organization.

> The question, 'What is the mental state of an animal?' means then: What is the level of organization of its activities? The question can have no other meaning because no other conception of mind can be derived from experience.
> . . . . . .
> The evolution of mind is the evolution of nervous mechanisms, but only the simpler of these can as yet be analyzed directly. (Lashley, 1949).

Thus the study of rat maze behavior is a legitimate approach to the study of mind. This is the argument of the comparative psychologist and is either accepted or rejected basically as it stands. Assuming that neurobehavioral relationships, observed in rat maze behaviors, are accepted as valid observations concerning the relationship between the brain, the mind and behavior, what now? The theory of equipotentiality has supported the concept of mind as a unitary state of the organism with assertations of a similar nervous system action.

> In short, current brain theory encourages us to try to correlate our subjective psychic experience with the activity of relatively homogeneous nerve-cell units conducting essentially homogeneous impulses through roughly homogeneous cerebral tissue. (Sperry, 1964, p. 406).

Sperry suggests that to search for psychical events in neural terms is a naive form of <u>psychoneural isomorphism</u> because the cerebral processes apparently do not duplicate, even remotely, the patterns of subjective experience. Sperry reiterates the validity of Sherrington's now twenty-odd year old remark that "We have to regard the relation of mind to brain as still not merely unsolved, but still devoid of a basis for its very beginning."

Sperry, in the 1964 article, is of the opinion that in order to advance our thinking of brain functioning "...our present one-sided preoccupation with the sensory avenues to the study of mental processes will need to be supplemented by increased attention to the motor patterns."

> Utilization of this motor approach immediately helps us to view the brain objectively for what it is, namely, a mechanism for governing motor activity. Its primary function is essentially the transforming of sensory patterns into patterns of motor coordination.... In man as in the salamander the primary business of the brain continues to be the governing, directly or indirectly, of overt behavior.
> . . . . . .
> ...the entire activity of the brain, so far as science can determine, yields nothing but motor adjustment. The only significant energy outlet and the only means of expression are over the motor pathways.... In both its phylogenetic and ontogenectic histories, mental activity develops out of, and in reference to, overt action.... Any separation of mental and motor processes in the brain would seem to be arbitrary and indefinite. (Sperry, 1964).

Thus to study mind one must study overt behavior or pre-motor activity. The obvious complexity of subjective psychical phenomena is overwhelming as are the obvious complexities of behavior. However, granting Sperry's argument, the only fruitful approach to the study of these psychological processes is through an analysis of behavior, regardless of the awesome nature of the task. The question now is "Is mind (and therefore behavior or vice versa) unitary or can it be fractionated into its component parts?

Attempts to analyze "mind" into its components have not been particularly successful. At the same time, to approach mind as solely a unitary phenomenon is to deny its obvious complexity. Behavior, on the other hand, can be subjected to an ever increasingly more

microscopic analysis, as the maze habit has shown.

If the brain's primary function is to organize behavior, either for present or future activity, then an analysis of the structure of behavior is the only way in which a valid understanding of neurobehavioral relationships can be reached. Logical derived elements of behavior, defined by environmental demands, can then be studied as they relate to the activity of a given neural substrate. The relationship between various behavioral units may be as important in determining neurobehavioral relationships as the structure of the nervous system itself. Behavior must be studied in terms of its logically derived complexity before the laws of neurobehavioral control can themselves be derived.

Conclusions

An analysis of the maze habit into other logically derived units of behavior has shown that previously unsuspected neurobehavioral relationships may exist such as the localization of some but not other behavioral components of a larger behavioral complex. The determinants of localization versus non-localization, in terms of behavioral units are not clearly understood but might be related to the spatio-temporal properties of the behavioral complex under investigation. This possibility has been recognized by other investigators, including K. S. Lashley, but was never utilized as an experimental approach to neurobehavioral mechanisms.

A technique for a precise behavioral analysis would allow for a taxonomy of behaviors in terms of spatio-temporal demands that will

begin to allow us to decipher the phenomenon of the serial order of behavior (Lashley, 1951). Temporal behaviors, which may incorporate spatial requirements relevant to a multitude of other behaviors, may be less localized than their more spatial counterparts in a behavioral complex. The alternation error in the III maze is obviously involved with the temporal execution of either of two behaviors, turning left or right in a proper sequence, while the door error is possibly concerned more with the execution of one set of behaviors, entering a break in the row wall, every time the proper conditions are spatially available. The successful maze habit consists of the "set" of both door and alternation behaviors.

Although the present study found that the rat cortex is not equipotential with respect to all components of the III maze habit, the basic conclusions of this study, that neurobehavioral relationships can only be understood when behavior is viewed in terms of its complexity, is in close agreement with conclusions about neurobehavioral relationships which Lashley reached nearly twenty years ago, dealing with "The Problem of Serial Order in Behavior". (Lashley, 1951).

In that 1951 article Lashley expressed the opinion that many forms of behavior, from speech to maze mastery, could be viewed as a "complex" or "set" of other behaviors and that this behavioral complex was the interaction of temporal and spatial systems. This set of behaviors was not viewed as being controlled by either central associative chains or peripheral sensory-motor reactions but instead was controlled by "...some central nervous mechanism which fires with predetermined intensity and duration or activates different muscles in predetermined

order."

The central nervous mechanism which integrates temporal and spatial components of a behavioral set was thought to reside in the networks of cells of short axons. Moreover, "...the nets active in rhythmic and spatial organization are apparently almost coextensive with the nervous system." This is basically a re-statement of the laws of equipotentiality and mass action which were based upon the view that the maze habit is unitary.

> The available evidence seems to justify the conclusion that the most important features of the maze habit are a generalization of direction from the specific turns of the maze and the development of some central organization by which the sense of general direction can be maintained in spite of great variations of posture and specific directions in running. (Lashley, 1929, p. 138).

This conclusion is consistent with the data concerning the effects of brain lesions upon alternation errors but does not agree with the findings concerning brain damage and door errors. The nerve nets active in these two behaviors do not appear to be simply coextensive with the nervous system as the door error demonstrates localization of function. Since the behaviors may differ in terms of spatio-temporal requirements it is quite possible that spatio-temporal systems may not be equally integrated by all neural masses. An analysis of behaviors in terms of their temporal order and spatial restrictions might result in new ideas of neurobehavioral relationships.

The maze habit is probably as valid a picture of behavioral integration as is available and should be a fruitful area of research concerning both the temporal and spatial characteristcs of behavior.

> Not only speech, but all skilled acts seem to involve the same problems of serial ordering, even down to the temporal coordination of muscular contractions in such a movement as reaching and grasping. Analysis of the nervous mechanisms underlying order in the more primitive acts may contribute ultimately to the solution even of the physiology of logic. (Lashley, 1951, p. 489).

Efforts toward a more complete understanding of the neurophysiological aspect of the behavioral syntax of the III maze habit could be a logical step toward an understanding of the physiology of the organization of behavior.

CHAPTER IV

SUMMARY

Much of the history of the inquiry into the nature of brain-behavior relationships has dealt with the study of the localization of functions and asks "Is the control of any behavior(s) the principle responsibility of a given neural locus?" Each side of the question of localization of function has received the support of scientific opinion at some time in history and the issue is presently not resolved. The issues, opinions and answers regarding this question have been strongly influenced by the existing views of neuroanatomy and behavior. While neuroanatomical specificity is granted for many sensory and motor functions, current neurobehavioral theories accept the proposition that no part of the rat neocortex is more essential to the acquisition of a complex maze habit than is any other part. Rat cortical neurobehavioral relationships are assumed to be governed by the law of "mass action". That is, the assumed determinant of a behavioral deficit following cortical damage is amount of damage, not site of damage.

This position is based in part on the view that the maze habit (Lashley's III maze) is a unitary class of behaviors although the efficient maze habit can be expressed in terms of at least two components: one involves entering a door into a new compartment at every opportunity

and the other involves alternating in a prescribed temporal sequence once a compartment is entered. The maze habit can thus be operationally defined as consisting of at least two logically distinct units of behavior. The evaluation of performance in terms of door and alternation errors represents a quantitative and a qualitative improvement in maze behavior measurement over the traditional cul-de-sac error. The alternation behaviors require the learning of a temporal response which is not required in the execution of door behaviors. To consider the III maze habit unitary is to deny the moment to moment changes in response requirements during a successful maze performance.

Earlier studies of the effects of cortical lesions upon maze mastery have suffered from the use of either a non-systematic selection of destruction sites or the use of only two destroyed zones. The systematic examination of at least three cortical zones increases the possibility of isolating specific corticobehavioral mechanisms. The selective destruction of various subcortical structures was necessary to determine whether or not elevations in errors following brain damage was primarily a cortical effect. The present investigations examined the effects of the bilateral destruction of three cortical zones (AC, MC and PC) and two subcortical sites (SEP and DLH) upon the acquisition of the two logically derived and operationally distinct components of the III maze habit--door and alternation behaviors.

The finding that the MC group made significantly more door errors than either of the other two cortical groups suggests that the rat neocortex is not equipotential with respect to maze mastery. This conclusion is further strengthened by the finding that there were no

significant differences between the groups in terms of amount of cortex destroyed. Thus the principle of "mass action" cannot explain the differences in performance between the lesion groups.

The finding that all three cortically lesioned groups performed nearly identically with respect to the alternation error would suggest that the rat neocortex is equipotential with respect to maze mastery. The finding that there were no significant differences between the groups in terms of amount of cortex destroyed would simply strengthen this conclusion as the law of mass action would also appear to be satisfied.

Thus one finding of this study was that while a given neural mass may be equipotential with respect to some behaviors that same neural mass may not be equipotential with respect to another set of behaviors. The apparent inconsistency of this statement is underscored by the fact that it is based upon observations of the same individuals following a single nervous system lesion.

Any inconsistency in these findings cannot be due to simple inaccuracies of lesion measurement as the two behaviors being measured were in the same individuals at approximately the same time, size of lesion was controlled. Therefore if the increase in door errors in the MC group was due to a greater amount of mass being removed (but inaccurately measured) why was there not a corresponding increase in alternation errors in that group?

These findings would suggest the possibility of a site by behavior interaction as far as a deficit in learning the III maze habit is concerned. That is, the effect of a lesion is dependent upon the

cortical region destroyed and the behavior under study. Furthermore, since there was no significant correlation between alterations in these behaviors and amount of brain damage, the concept of mass action is also challenged. Finally, the percentage of cortex removed also failed to relate to other behaviors such as general activity.

These findings indicate that some caution should be exercised when proposing "equipotentiality" tempered with "mass action" as determinants of neurobehavioral relationships. As important as these concepts are, they must be considered in terms of particular behaviors. That is, behavioral definition is as important as neuroanatomical specification when attempting to state neurobehavioral relationships.

Both errors were also significantly elevated in the subcortically damaged Ss indicating that these behaviors are not under exclusive cortical control.

A model of the III maze habit which includes door and alternation type behaviors acknowledges the complexity of the successful maze habit which requires the ability to initiate and inhibit locomotor activity, the ability to recognize and enter openings or breaks in a wall surface and the ability to learn a temporal order of turns which varies from choice point to choice point.

In an effort to more fully understand the significance of the door and alternation errors, tasks were included in one experiment which were designed to test specific behavioral dimensions which relate to door and alternation behavior. One task, the door error maze, DEM, simply required the S to detect and enter a break (door) in a runway wall surface. This is one description of successful door behavior in

the III maze.

Another task, the alternation error maze (AEM), was a T-maze and required the S to learn a simple alternation response that never changed. If III maze alternation errors were due to the inability to master a simple alternation problem then the results from this instrument should correspond to the alternation error behavior seen in the III maze.

Another task measured the ability to acquire and extinguish a simple straight runway locomotor response. A general measure of spontaneous activity was also taken to further examine the role of this behavioral substrate in the III maze behaviors.

The DEM analysis indicated that it too could detect brain damage in a manner highly similar to the III maze. With few exceptions the results from the two tasks appear to be almost identical in terms of group effects. The tasks themselves would appear to have basic similarities.

The finding that the AEM failed to distinguish between groups would indicate that the AEM may not adequately reflect the "alternation error" component of the III maze. The alternation component of the III maze is probably determined by the sequencing of alternatives while the AEM used in this study required only the learning of a simple alternation response that never varied.

The failure to find any significant differences between groups in terms of either the straight alley maze (SAM) acquisition or extinction behaviors would suggest that the brain damage related behaviors seen in this study were not due to unusual motivational or motoric

factors. The extinction behaviors of all groups would also argue against a simple "response perseveration" hypothesis to explain the results of certain lesions. Thus there appear to be no basic failures of either "maze excitation" or "maze inhibition" in any of the experimental groups.

The findings that the cortical group which made the most door errors was also the most active and that there was a significant correlation between door errors and activity across groups suggest that activity is an important dimension of the door error behavior. This does not seem to be the case for the alternation error as the correlation between these behaviors and activity were nonsignificant. The activity phase of Experiment II found that large changes in activity were produced by both cortical and subcortical lesions. The groups that were the most active were also the ones which made more door errors.

The two primary behaviors under study, door and alternation errors in the III maze, while having some apparent face validity in terms of their distinctions, are not completely understood in terms of their respective behavioral significance. Although the two behaviors were significantly correlated with each other they were affected differently by the various lesions. Also the dynamics of the two behaviors are distinguishable in the maze. The door error shows a goal-gradient across rows with the row furtherest from the goal (row one) showing the most errors and the row nearest the goal (row six) being the least difficult. This was not the case for the alternation error as row six was one of the most difficult rows for this behavior. The two behaviors also differed with respect to their level of recovery

following brain damage. The door error, although being strongly exaggerated by the cortical lesions, had recovered to a near normal level by trial twelve but the alternation error showed only slight recovery by trial twenty. Previous studies have also found the rate of acquisition of the two behaviors to differ in normal S with the door error being more quickly eliminated than the alternation error over trials.

Perhaps a neurobehavioral model which incorporates both the remarkable equipotentiality of the alternation error and the elusive localization of the door error, for the same neural mass, is not presently available. This may not be due as much to failings of neuroanatomical sophistication as to the failure to view behavior in terms of its obvious complexity.

While environmental demands and responses vary from moment to moment the integrated behavior of the organism retains a unity of expression that sometimes masks the complexity of the organization of the various moment to moment behaviors. There are certainly levels of behavioral organization which include other behaviors in their expression. There may even exist two types of behavioral processes--one involving responses to simple repetitions of events and the other occurring when the temporal requirements of response execution are more complex. The expression of these various behaviors can be viewed as a "behavioral complex" where the total integrated behavior may be composed of other distinguishable behavioral states. Thus the successful maze habit is complex when it includes successful door and alternation behaviors in its expression. The concept of a "behavioral complex" allows for a continual analysis of the components of that complex

whereas the concept of "behavior" or "reflex" or "habit" implies a finiteness that discourages further analysis. This could greatly influence neurobehavioral theory (and investigations).

Granted the concept of a "behavioral complex", as opposed to "habit", the argument can be made that the more complex the behavior in question (i.e., maze mastery as opposed to a righting response), the higher the probability that several brain systems contribute to that behavior. Damage to any one of the several brain systems involved in a highly complex behavior would result in a deficit in that behavior. Therefore the greater the probability that "equipotentiality" holds for that behavior. As the total behavioral picture can be reduced to its component parts then the probability of localized neural control for any component is increased.

Thus the concept of an analysis of a "behavioral complex" by incorporating a Hunter-like model but emphasing "behavioral unit" instead of stimulus can explain both equipotential and localized neurobehavioral relationships as a function of response complexity. The problem thus becomes one of evaluating the complexity of a given set of behaviors. One dimension of response complexity is spatio-temporal in nature. Responses which require the learning of a temporal sequence involve a level of organization not seen in responses executed to simple repetitions of events.

The alternation behaviors, which require a temporal sequencing, should therefore represent a more complex behavioral expression than the door behaviors which afford a more limited response requirement. There are other lines of evidence to suggest that the alternation behavior is

more complex than the door behavior: (1) the door behaviors are easier to master than are the alternation behaviors in normal Ss and (2) the door behaviors show recovery following brain damage while the alternation errors do not.

Intuitively it would appear that a less complex habit would be easier to master initially and re-learn following brain damage. Thus the equipotentiality of the door behavior could be due to its temporal complexity. Temporal behaviors, which may incorporate spatial requirements relevant to a multitude of other behaviors, may be less localized than their more spatial counterparts in a behavioral complex.

If the brain's primary function is to control or organize behavior, either in the present or for some future action, then an analysis of the organization of behavior is the only way in which a valid understanding of neurobehavioral relationships can be reached. An analysis of behaviors in terms of their temporal order and spatial restrictions might result in new ideas of neurobehavioral relationship. Logical derived elements of behavior, defined by environmental demands, can then be studied as they relate to the activity of a given neural substrate. The relationship between various behavioral units may be as important in determining neurobehavioral relationships as the structure of the nervous system itself. Behavior must be studied in terms of its logically derived complexity before the laws of neurobehavioral control can themselves be deduced.

REFERENCES

Bender, R. M., Hostetter, G., & Thomas, G. J. Effects of lesions in hippocampus-entorhinal cortex on maze performance and activity in rats. Psychonomic Science, 1968, 10, 13-14.

Boring, E. G. A history of experimental psychology. (2nd ed.) New York: Appleton-Century-Crofts, Inc., 1957.

Chow, K. L. Effects of ablation. In G. C. Quatron, T. Melnechuk, F. O. Schmitt (Eds.), The Neurosciences. New York: The Rockefeller University Press, 1967, Pp. 705-713.

Deese, J. General psychology. Boston: Allyn & Bacon, 1967.

Head, H. Aphasia and kindred disorders of speech. Vol. 1. Cambridge: University of Cambridge Press, 1926.

Herrnstein, R. J., & Boring, E. G. A source book in the history of psychology. Cambridge: Harvard University Press, 1966.

Hunter, W. S. A consideration of Lashley's theory of the equipotentiality of cerebral action. Journal of General Psychology, 1930, 3, 455-468.

Jackson, W. J., & Strong, P. N., Jr. Differential effects of hippocampal lesions upon sequential tasks and maze learning by the rat. Journal of Comparative and Physiological Psychology, 1969, 68, 442-450.

John, E. R. Mechanisms of memory. New York: Academic Press, 1967.

Kirk, R. E. Experimental design: Procedures for the behavioral sciences. Belmont, California: Brooks/Cole Co., 1968.

Krech, D. Cortical localization of function. In L. Postman (Ed.), Psychology in the making: Histories of selected research problems. New York: Alfred A. Knopf, 1963, Pp. 31-72.

Lashley, K. S. Brain mechanisms and intelligence. Chicago: University of Chicago Press, 1929.

Lashley, K. S. Brain mechanisms and intelligence. New York: Dover Publications, Inc., 1963.

Lashley, K. S. Persistent problems in the evolution of mind. (1949). In F. A. Beach, D. O. Hebb, C. T. Morgan, & H. W. Nissen (Eds.), The Neuropsychology of Lashley. New York: McGraw-Hill Book Company, Inc., 1960, Pp. 455-477.

Lashley, K. S. Basic neural mechanisms in behavior. (1930). In F. A. Beach, D. O. Hebb, C. T. Morgan, & H. W. Nissen (Eds.), The Neuropsychology of Lashley. New York: McGraw-Hill Book Company, Inc., 1960, Pp. 191-209.

Lashley, K. S. The problem of serial order in behavior. (1951). In T. K. Landauer (Ed.), Readings in physiological psychology: The bodily bases of behavior. New York: McGraw-Hill Book Company, Inc., 1967, Pp. 480-501.

Lashley, K. S., & Franz, S. I. The effects of cerebral destruction upon habit formation and retention in the albino rat. Psychobiology, 1917, 1, 71-140.

Luria, A. R. Higher cortical functions in man. New York: Basic Books, 1966.

Luria, A. R. The functional organization of the brain. Sci. American, 1970, 222, 66-73.

Pellegrino, L. J., & Cushman, A. J. A stereotaxic atlas of the rat brain. New York: Meredith Publishing Co., 1967.

Penfield, W., & Jasper, H. Epilepsy and the functional anatomy of the human brain. Boston: Little, Brown and Company, 1954.

Pickett, J. M. Non-equipotential cortical function in maze learning. American Journal of Psychology, 1952, 65, 177-195.

Pribram, K. H., Lim, H., Poppen, R., & Bagshaw, M. Limbic lesions and the temporal structure of redundancy. Journal of Comparative and Physiological Psychology, 1966, 61, (3), 368-373.

Saavedra, M. A., Pinto-Hamuy, T., & Oberti, C. Auditory avoidance behavior after extensive and restricted neocortical lesions in the rat. Journal of Comparative and Physiological Psychology, 1965, 60, 41-45.

Siegel, S. Nonparametric statistics for the behavioral sciences. New York: McGraw-Hill Book Company, Inc., 1956.

Sperry, R. W. Neurology and the mind-brain problem. In R. L. Isaacson (Ed.), Basic readings in neuropsychology. New York: Harper & Row, 1964, Pp. 403-429.

Thomas, G. J., Moore, R. Y., John, A. H., & Hunt, H. F. Relations between the behavioral syndrome produced by lesions in the septal region of the forebrain and maze learning of the rat. Journal of Comparative and Physiological Psychology, 1959, 52, 527-532.

Thompson, R. Learning and retentiveness in brain-damaged rats. Journal of Comparative and Physiological Psychology, 1959, 52, 501-505. (a)

Thompson, R. The comparative effects of anterior and posterior cortical lesions on maze retention. Journal of Comparative and Physiological Psychology, 1959, 52, 506-508. (b)

Vygotsky, L. S. Psychology and localization of functions. Neuropsychologia, 1965, 3, 381-386.

Watson, J. B. Behaviorism. Chicago: University of Chicago Press, 1963.

White, R. K., & McGee, S. A. The effect of two goal box variables upon errors and latency in the Lashley III maze. Atomic Energy Commission Contract #8T(40-1)-1982. R. K. White, Principal Investigator, 1964.

White, R. K., & Vardiman, D. R. Some dimensions of learning in 400-day old rats: I - straightaway and Lashley III maze behavior. Atomic Energy Commission Contract #8T(40-1)-1982. R. K. White, Principal Investigator, 1964.

Winer, B. J. Statistical principles in experimental design. New York: McGraw-Hill Book Company, 1962.

APPENDIX A

TOTAL DOOR (De) AND ALTERNATION (Ae) ERRORS AND PERCENTAGE OF CORTICAL DESTRUCTION (%), BY SUBJECT, FOR THE COMBINED CORTICAL GROUPS OF EXPERIMENT I AND II

Anterior			Middle			Posterior		
%	De	Ae	%	De	Ae	%	De	Ae
28.3	20	40	21.8	60	54	25.3	45	52
32.6	66	52	19.9	81	70	19.1	19	35
18.6	36	54	18.2	70	75	24.2	136	73
31.1	74	62	25.7	64	70	22.1	83	68
33.9	30	47	25.5	187	47	11.4	20	26
10.7	24	42	28.1	97	69	22.1	62	49
12.2	58	53	29.4	41	45	18.2	49	56
11.5	42	56	23.2	57	48	28.5	36	37
20.2	90	41	10.3	31	37	17.8	21	43
24.9	63	67	22.9	32	44	15.6	53	53
			19.7	135	63	31.5	42	44
			25.3	132	48	28.7	33	49
22.4	50.3	51.4	22.5	82.3	55.8	22.0	49.9	48.8
9.0	23.3	9.0	5.1	47.6	12.8	5.9	32.9	13.3