THE SYNDROME OF SEPTAL DAMAGE

THE BEHAVIORAL SYNDROME OF SEPTAL DAMAGE IN LABORATORY RATS

by

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SCOPE AND CONTENTS:

Experiment 1 included a study of the following behaviors in normal and in septal-damaged rats: drinking; hyperemotionality (rated with a new scale); escape from electric shock; movement inhibition (MI) shaping and performance; jump avoidance conditioning with no handling and a 15 sec. CS-US interval (JA-15); and weight gain.

Experiment 2 included jump avoidance conditioning with a 5 sec. CS-US interval (JA-5) and MI (without shaping) tested in counterbalanced order. Seven groups received either sham, unilateral septal, bilateral septal, preoptic, septal-preoptic, caudate-septal, and frontal cortex-septal lesions.

Experiment 3 included JA-5 with below-the-grid lighting tested in septal damaged and control rats.

Septal lesions disrupted directed movements made during exploration, escape, MI and JA. This disruption correlated with hyperemotionality when tested after prior handling but not when tested before handling and not with weight gain. A new hyperemotionality scale was developed which correlated with Brady and Nauta's (1953) scale but was more reliable, especially for the less emotional rats (in control rats reliability for the new scale was .95 and for the old it was .77).

For the septal damaged animals prior experience with JA improved MI performance but prior experience with MI impaired JA performance. These effects were mitigated by adding preoptic damage to the septal damage.

Below-the-grid lighting had no detectable effect on the JA-5 impairment caused by septal damage.

Generalizations about behavior after septal damage were discussed with emphasis on a generalized fragmentation of voluntary responses into isolated, component acts.

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CHAPTER 1

HISTORY

Introduction

The septal nuclei are situated centrally in the limbic system between the lateral ventricles and at the junction of the rhinencephalon and the diencephalon. They are bounded dorsally by the corpus callosum and ventrally by the anterior commissure. With diffuse interconnections with other parts of the rhinencephalon and with the diencephalon (see the sections on anatomy and autonomic functions below) they would seem to play a central role in interrelating the functions of these two sections of the brain. Although theories of the behavioral functions of the septal nuclei of the brain have existed for many decades (e.g. see Smith, 1895a, b; Papez, 1937) empirical studies with only a few exceptions (e.g. Ransom, 1895) are a development of only the last 20 years. Since the early 1950's, however, the somewhat surprising combination of effects of septal damage has generated considerable interest and a number of effects have been established. In addition at least two attempts at general theories have been made (Mc Cleary, 1966; Thomas, Hostetter

and Barker, 1968) and one general review was done several years ago (Andy and Stephan, 1964). Nevertheless, there is still no widespread agreement on the general functions of these nuclei.

Although the present review is concerned primarily with the behavioral syndrome of septal dysfunction, that portion of the review will be prefaced by reviews of the nuclear structure, the anatomical connections and the autonomic and somatic functions of the septal area. The distinction between autonomic and somatic functions on the one hand and behavioral functions on the other is based on a narrower than usual definition of behavior. Rather than including "anything that an animal does or experiences" (which taken literally would include such processes as cell division), only processes that are not of the most automatic sort are included in the term behavioral functions (e.g. glandular responses, heart rate, respiration, and simple, isolated reflexes are not described as behavior).

The behavioral functions will be described in two sections: one on spontaneously occurring behaviors (i.e. behaviors that appear in all normal members of at least the species being considered) and one on learned behaviors. Following the description of the behavioral effects of septal dysfunction, the present study will be outlined.

Nuclear Structure of the Septal Area

The septal nuclei are situated between the lateral ventricles and below (ventral to) the corpus callosum in the anterior portion of the brain. Volumetric studies reveal a definite increase in absolute size (and in relative size compared with the brain stem) with an increase in phylogenetic level from the lowest insectivores to the higher primates (Stephan and Andy, 1962). In man and in some other animals with large cerebral cortices (e.g. the bear and the monkey), the area appears continuous with an elongated thin vertical strip of neural tissue. In man this strip is termed the septum pellucidum. As Andy and Stephan (1968) have pointed out, the major portion of the septal area in man lies ventral to this strip and therefore, the septum pellucidum should not be confused with the septal area. Early studies of the nuclear structure of the septal area of rats distinguished at least three areas in what are now considered the septal nuclei (Johnston, 1913; Gurdjian, 1925; Kappers, 1920). These included a nucleus parolfactorius lateralis (Johnston's terminology; Gurdjian considered this synonymous with the lateral septal nucleus of Kappers; it is synonymous with the nucleus accumbens of König and Klippel, 1963), a nucleus parolfactorius medialis (medial septal nucleus according to Gurdjian and nucleus of the diagonal band of Broca according to König and Klippel), and a third area termed the hippocampal primordium (including the medial, lateral and fimbrial nuclei

of König and Klippel). As Gurdjian (1925) pointed out there was still debate as to whether all these were properly termed "septal" structures. Apparently there have been no recent studies devoted to the nuclear structure of the septal area in rats. However, Stephan and Andy have done such studies in the insectivore, Soricidae (1959), the galago (1961), the cat (Andy and Stephan, 1964), the monkey (1964b), and the human (Andy and Stephan, 1968). The results were similar except that the cells were somewhat more differentiated in the animals that were higher on the phylogenetic scale (1964b). Thus, it would seem to be most appropriate to base the description of the septal nuclei on the very detailed analyses of Stephan and Andy even though they have not per se analyzed the septal area of the most commonly used animal in the analysis of behavioral functions (the rat).

Stephan and Andy differentiate four major groups of septal nuclei: (1) dorsal, (2) ventral, (3) medial and (4) caudal. The first two were further differentiated into four parts and two parts respectively. The third (the medial group) consisted of two nuclei: n. septalis medialis, which was situated both dorsal and lateral to the second, the nucleus of the diagonal band of Broca. The fourth consisted of four nuclei: n. septalis fimbrialis, n. septalis triangularis, and the bed nuclei of the anterior commissure and the stria terminalis.

Anatomical Connections of the Septal Nuclei

Concerning the anatomical connections of the septal nuclei of the rat, an excellent study and review has been provided by Raisman (1966). The present summary is based on that review. In general, all the connections of the septal area (with the exception of some afferents from the midbrain) are with structures in the diencephalon and the older portion of the telencephalon. The principal telencephalic input to the septal area is from the hippocampus via the fornix and includes projections to all parts of the septal area except the bed nucleus of the stria terminalis. This projection is part of a relatively complicated system of reciprocal interconnections (see Raisman, 1966, for details). Telencephalic afferents also project from the pyriform cortex to the diagonal band nucleus and from the amygdala via the stria terminalis to the bed nucleus of the stria terminalis. Subcortical afferents have been analyzed by both Guillery (1957) and Raisman (1966). These arise in both the hypothalamus and the midbrain and ascend via the medial forebrain bundle. Damage to the hypothalamic portion results in degeneration in both the medial and lateral septal nuclei while midbrain damage results in degeneration only of the medial nucleus. Telencephalic efferents from the septal nuclei are primarily involved in the reciprocal septo-hippocampal system. They arise in the medial septal and diagonal band nuclei and ascend to the hippocampus

probably via the fimbria. Diencephalic efferents, which form a large part of septal outflow, have two components. The dorsal component arises probably from the septofimbrial nucleus and projects primarily to the medial habenular nucleus. The ventral component arises in the medial and lateral septal nuclei and projects to the medial forebrain bundle from which it diverges to the lateral preoptic area, the lateral hypothalamus, and the anterior amygdala. Although the intrinsic connections have not been worked out in detail an important relay from the lateral to the medial portions of the septal area is known. Considering the non-septal as well as the septal connections of the structures mentioned above, Raisman concludes that the septal area "is an important cell mass located at the junction of the fibre systems linking the rhinencephalic parts of the cerebral hemispheres and the diencephalon." (p. 344)

Autonomic and Somatic Functions of the Septal Nuclei

Ban (1966) has described a septo-preoptico-hypothalamic system which mediates autonomic responses. The septal area along with the lateral and periventricular hypothalamus and preoptic area (e.g. the areas involved in the ventral component of the diencephalic efferents from the septal area described by Raisman, 1966) comprise the parasympathetic component whereas the periventricular stratum of the hypothalamus and preoptic area comprise the sympathetic

component. When stimulated* the parasympathetic portion produces a fall in blood pressure and heart rate, inhibition of ovulation (also lesions produced premature parturition in the last stage of pregnancy), an increase in renal volume, bladder contraction, respiratory inhibition with a shift toward expiration, a decrease in gaseous metabolism, penile erection (Mac Lean, 1958; Mac Lean, Ploog and Robinson, 1960), ovulation (in rabbits; Harterius, 1937), sneezing and sniffing, (Hess, 1957) and sleep. Stimulation of the sympathetic portion produces opposite effects (a rise in blood pressure, ovulation, premature parturition--in the last stage of pregnancy--milk ejection, a decrease in renal volume, bladder relaxation, respiratory acceleration along with a shift toward inspiration, an increase in gaseous metabolism, and sham attack behavior).

Regarding somatic functions septal stimulation causes a decrease in the frequency of spontaneous movements during light, chloralose anesthesia (see Kaada, 1960). Some points also inhibit cortically induced limb movements and reflexive knee jerks (Hodes, Peacock and Heath, 1951). On the other hand Peacock and Hodes (1951) actually found a greater number of points in the septal area that facilitate cortically induced movements than of those that inhibit them.

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*Some of these responses have also been described by Covian (1967), Gloor (1954), Hess (1957), Kaada (1960), Kabat (1936), Kabat, Magoun and Ranson (1935, 1936), Malmo (1961, 1963), Mukawa and Andy (1962), Ranson, Kabat and Magoun (1935), and Torii and Kawamura (1960).

In addition, Hess (1957) found stimulus specific tongue and mouth movements and Hopkins (1967) found shaking (resembling the response produced by wetting the fur) with septal stimulation (also observed by Bland, Wishart, Altman and Vanderwolf, personal communication, 1970).

Behavioral Functions of the Septal Nuclei

Spontaneously Occurring Behaviors

Spontaneously occurring behaviors that have been studied in relation to septal area functions include freezing, sleep, thermoregulation, grooming, sexual behavior, drinking, feeding, maternal behavior, nest building, hoarding, social investigatory behavior, aggressive behavior and exploratory behavior. These behaviors are distinguished from learned behaviors such as bar pressing, running from a light (as a conditioned stimulus), etc. that do not normally occur in all members of a species.

Freezing and Lying. Findings concerning freezing and lying (resting) behaviors following septal damage vary depending on the stimulus conditions under which the behavior was observed. In an open field Corman, Meyer, and Meyer (1967) found that animals with septal lesions show a much longer latency to move away from the center of an open field than control Ss. Septal damaged rats also lie motionless as much as control rats in a four-arm + maze (Dirlam, 1969). In addition Bunnel, Bemporad and Flescher (1966) found that

animals with septal lesions tend to sit passively in a doorway and wait for an opponent to approach from the other direction when the two rats are competing to use the door to gain access to food.

Sleep. Findings concerning the role of the septal area in sleep are conflicting. Electrical stimulation of Ban's (1966) parasympathetic system produces sleep. Cholinergic (carbachol) stimulation of the septal area produces sleep according to Hernandez-Peon and Chavez-Ibarra (1963) and "definitely" does not produce it according to Mac Phail and Miller (1968). In addition, electrical stimulation in man reliably awakened a patient with narcolepsy (Heath, 1963) and septal damage in the rat makes them more susceptible to the effects of barbiturates (Heller, Harbey, Hunt and Roth, 1960; Hunt, 1957). Perhaps these discrepent results can be explained by the proximity of the septal area to the preoptic area. There seems to be general agreement that preoptic stimulation produces sleep (Hernandez-Peon and Chavez-Ibarra, 1963; Hess, 1944; Roberts, Bergquist and Robinson, 1969; Roberts and Robinson, 1969; Sterman and Clemente, 1962a, 1962b; Clemente and Sterman, 1967) and preoptic lesions block it (Mc Ginty and Sterman, 1968). Therefore, the positive effects of stimulation of the septal area on sleep could have been due to spread of excitation to the preoptic area.

Thermoregulation. There seems to be some evidence indicating that the septal nuclei are somewhat involved in

the regulation of temperature. Septal lesions may cause transient effects on the ability to maintain body temperature in the cold either spontaneously or by behavioral means (lever pressing; Carlisle, 1969) but changes in heat loss in the cold following septal lesions were not found by others (Heller et al., 1960; Stuart, Kawamura, Hemingway and Price, 1962). Also septal stimulation was found to induce shivering (Akert and Kesselring, 1951; Andersson, 1957; Hemingway, 1963) and the septal area does contain cells that are mildly thermosensitive (Eisenman and Jackson, 1967).

Grooming. Dirlam (1969) found that septal rats groom more than control rats after exploring a novel maze. This behavior appeared within about 10 minutes after the first exposure to a small, enclosed but well-lit portion of a four-arm plus (+) maze. In addition Hess (1957) reported self-licking from septal stimulation in cats. Since this is the same effect as with lesions, the stimulation may have disrupted normal functioning.

Sexual Behavior. A relation between electrical stimulation of the septal area and sexual behavior has been noted in man (Heath, 1963), in the monkey (Mac Lean and Ploog, 1962) and in the rabbit (Harterius, 1937). However, septal lesions appear to have no effect on this behavior at least in the rat (Goodman, Bunnell, Dewsbury and Boland, 1969; Heimer and Larsson, 1966/67). Some authors (Goodman et al., 1969) suggest that this may indicate a disruptive effect of septal stimulation such as that found by Goldstein

(1966) and by Kasper (1964, 1965), but if this were the case, septal damage should lead to excessive sexual behavior like stimulation does. That possibility has not been noted. At least the findings involving septal-area destruction strongly suggest that it does not play a crucial role in the control of sexual behavior.

Drinking Behavior. Cholinergic stimulation of the septal area produces increases in drinking (Fisher and Coury, 1964; Grossman, 1964; Greene, 1968) and cholinergic blocking agents depress it (Grossman, 1964). However, findings involving drinking in animals with septal dysfunction are mixed. Some authors report increases (Beatty and Schwarzbaum, 1967; Besch and Van Dyne, 1969; Carey, 1967b, 1969; Donovick and Burright, 1968; Donovick, Burright and Gittelson, 1968; Harvey and Hunt, 1965; Harvey, Lints, Jacobson and Hunt, 1965; Lubar, Boyce and Schaefer, 1968; Pizzi and Lorens, 1967; Singh and Meyer, 1968; Wishart, 1970) while others report no change (Dirlam, 1969; Kaada, Rasmussen and Kviem, 1962; Kasper, 1965; Stevenson, 1967; Wishart, 1970). Apparently animals with septal damage drink more than normal in response to saccharine (Beatty and Schwarzbaum, 1967, 1968a), press levers faster for sucrose reward (Beatty and Schwarzbaum, 1968b; Buckland and Schwarzbaum, 1970; Pubols, 1966) and run faster and more directly for sucrose (Clody and Carlton, 1969). In addition, they drink less water than control Ss when it is adulterated with quinine (Beatty and

Schwarzbaum, 1967). Possibly, the discordant results can be explained in part by daily and/or regional difference in the taste qualities of water. However, Wishart (1970) found increased drinking in some septal rats and not in others when all Ss were tested at the same time and place. Another possibility is that lesion variation might be responsible for the effects. However different experimenters have found different loci to be effective. Therefore, if the latter explanation is correct the relation must be complicated (e.g. increased drinking is found only if one part of the septal or surrounding areas is damaged and another is not).

Feeding Behavior. Animals (both rats and cats) with septal damage seem to eat more than controls (Simmons and Thomas, 1961; Singh and Meyer, 1968). They also seem to be more finicky (Singh and Meyer, 1968) and to lose a slight but reliable amount of weight (Beatty and Schwarzbaum, 1967; Clody and Carlson, 1969). These findings suggest that animals with septal-area damage tend to have less efficient food absorption than normal animals or have greater food requirements. In addition electrical stimulation has been found to depress feeding in monkeys (Rubenstein and Delgado, 1963) and in cats (Fonberg and Delgado, 1961). On the other hand termination of stimulation results in "rebound" feeding (Bland et al., 1970) and adrenergic stimulation elicits feeding in satiated rats (Booth, 1967; Coury, 1967).

Maternal Behavior. Maternal behavior in animals with septal damage has been studied in mice (Carlson and Thomas, 1968) and in rats (Slotnick, 1967). Although Slotnick found no major disruption of this behavior, his lesions were restricted to the posterior portion of the septal area. Carlson and Thomas (1968), however, found a very marked disruption of this behavior in the mouse. An interesting point of their study is that although mice with septal damage average 10 times as many retrieval responses, they took more than 20 times as long to effectively retrieve the first pup and most of them never retrieved all the pups in the 15 min. test (it took control Ss about 1 min.). Carlson and Thomas estimate that it took mice with septal damage 15 times as many responses to retrieve each pup as it took control mice. In addition "although individual species-typical component acts of maternal behavior were observed, these Ss did not put many responses together in a proper sequence as did Ss in other groups." (p. 735). Carlson and Thomas suggest that a loss of response inhibition explains these findings. However, such a loss could be the effect rather than the cause of the disruption. The brain damaged mice could have the same drive as normal mice to retrieve pups but since their behavior has become fragmented or disintegrated the probability that they will produce an effective sequence of behaviors (one that satisfies the drive) is very markedly reduced. Consequently, they must produce many more responses

in order to be effective. In any case it is evident that septal damage markedly increases the frequency of appearance of the component acts involved in pup retrieval but just as markedly decreases their effectiveness.

Nest building. Carlson and Thomas also studied nest building in mice with septal damage. No specific time was allotted or behavioral observations taken. However, the effectiveness of septal damaged mice was markedly reduced. Most animals with cingulate, neocortical, thalamic or no damage had shredded 25 pieces of twine and placed it all in a cup to form a well-shaped nest within a five-day prepartum period. Septal damaged mice had only shredded some twine and only placed some of that in a nest. That septal damaged hamsters also do not make nests was observed by Matalka (1967). In this light it is interesting that humans with tumors of the septum pellucidum are also unable to keep house (Zeman and King, 1958).

Hoarding. Both Matalka (1967) and Sodetz and Bunnell (1970) have noted that hamsters with septal-area damage do not hoard, but only moderate (Wishart, Brohman and Mogenson, 1969) or no (Vanderwolf, 1966) effects on hoarding in laboratory rats were found. For both hoarding and nest building, however, it would be valuable to have observations on the actual details of behavior such as those Carlson and Thomas provided on maternal behavior.

Social Investigatory Behavior. Social investigatory behavior--initiating contact with another animal--is seen more often in animals with septal damage than in control animals (Sodetz and Bunnell, 1969; Sodetz and Smith, 1966). The surprising finding with regard to this behavior is that although it might be considered inappropriate for submissive animals (and normal submissive animals show a very low frequency of this behavior), submissive hamsters with septal damage show as exaggerated an amount of this behavior as do dominant hamsters with septal damage. In fact Bunnell and Smith (1966) have noted that septal-damaged, wild cotton rats persist in this approach behavior toward aggressive opponents to the point of being killed.

Social Aggressive Behavior. Two sorts of social aggressive behavior have been observed following septal damage: spontaneous social aggression and shock-induced social aggression.

Spontaneous social aggression following damage to the septal area has been observed in the hooded rat (Bunnell, et al., 1966) the cotton rat (Bunnell and Smith, 1966), the hamster (Sodetz and Bunnell, 1970) and in humans (Zeman and King, 1958). All these studies show disruption of some type. The mildest disruption appeared in the rat. These animals were tested in a situation which required them to compete from different sides, for which should pass through a doorway only large enough for one (the Robin Hood-Little John problem).

After septal damage, rats showed more wins than before. However, this was accomplished in a peculiar manner. Instead of approaching the doorway, the rat would sit just behind it and wait for the opponent to approach. When it did some rats with septal damage would "slash out" with their teeth bared while others would freeze and refuse to move until the opponent finally backed out.

In hamsters, septal damage had an effect that was intermediate between the effect on laboratory rats and the effect on wild rats (Sodetz and Bunnell, 1970). Septal damaged hamsters showed an increased amount of aggressiveness following the operation but would become submissive in the face of a dominant opponent. Nevertheless, these submissive animals would persistently approach even the dominant opponents, so that regardless of whether the animals were submissive, cages with septal damaged hamsters showed more fighting than those with normal hamsters.

In wild cotton rats the normal defense against a dominant opponent is not submission, but flight (Bunnell and Smith, 1966). However, septal damaged cotton rats attack more frequently than control animals even if they had just previously been injured. If an opponent ran away after an attack by a septal damaged cotton rat the latter would switch to some irrelevant behavior. However, if the opponent fought back and even injured the brain damaged rat, the latter would run frantically around and around the cage and often

stop only a few inches from the opponent. If the opponent did not renew the attack, the brain damaged rat would do so, even if it had just been injured. Such behavior at times led to the death of a septal damaged rat. In addition, Bunnell and Smith note that unlike normal wild rats the brain damaged animals would break off the aggression after attacking and they were never observed to bite an opponent.

In humans, Zeman and King (1958) noted that septal tumors resulted in violent anger and threats which were at times followed by prolonged disorientation. They were made in the presence of both submissive (children, wives) and dominant (husbands) persons.

In conclusion, there are striking species differences in the effects of septal damage on aggressiveness. In general all the species tested attack, or at least investigate, opponents more frequently. Also, the wilder the species, the less effective the aggression is (i.e. the more likely it is that the brain damaged animal will be harmed). However, the general ineffectiveness of laboratory animals in fighting is well known (e.g. See Barnett, 1963). Perhaps, aggressive behavior is disrupted in all species but the disruption is noticeable only with an opponent which provides a strong stimulus for aggression. Such a notion is supported by the fact that although animals with ventromedial hypothalamic (VMH), olfactory bulb (OF) or septal damage all

attack a provoking object (i.e. a pencil moved toward the animal) more than control rats both those with VMH and OB lesions also kill mice more than control rats but those with septal damage do not (Malik, 1970).

Septal damage leads to an increase in the amount of fighting following footshock (Ahmand and Harvey, 1968; Blanchard and Blanchard, 1968; Wetzel, Connor and Levine, 1967). Social housing eliminates this effect as it does hyperemotionality (to be discussed in the next section) but increased shock-induced aggression is independent of changes in emotionality.

Hyperemotionality. Hyperemotionality or hyper-irritability refers to such behaviors as an exaggerated response to a puff of air or loud noise, unprovoked or provoked attack of an inanimate object (e.g. a stick or pencil), and great resistance to capture or handling in a normally tame variety of animals. Several early studies (Fulton and Ingram, 1929; Ransom, 1895; Spiegel, Miller and Oppenheimer, 1940; Wheatley, 1944) suggested the involvement of the septal area in such behaviors, but these studies did not use standardized lesions or behavioral testing procedures and the crucial involvement of the septal area remained relatively obscure. However, since the original controlled study of Brady and Nauta (1953) in which damage was confined to the septal area and systematic observations in standard situations were used, the phenomenon has been subjected to a

large number of studies. It is greater in hooded rats than in albino (Wistar) rats (King, 1959) and it is produced with either irritative or non-irritative lesions (Reynolds, 1964). It gradually disappears with normal housing (Heller, Harvey and Moore, 1962; Seggie, 1968; Singh, 1969; Wetzel et al., 1967) and this decline is facilitated by handling (Brady and Nauta, 1953, 1955; Seggie, 1968), paired housing (Brady and Nauta, 1953, 1955) or shaking (Seggie, 1968). Amygdaloid damage has been found to prevent it by some authors (King and Meyer, 1958; Schwarzbaum and Gay, 1966) but not by others (Kleiner, Meyer, and Meyer, 1967). On the other hand neocortical lesions (located almost anywhere but fairly sizable) prevent the usual abatement (Clark, Meyer, Meyer and Yutzey, 1967; Yutzey, Meyer and Meyer, 1964; Yutzey, Meyer and Meyer, 1967) and cortical spreading depression causes it to reappear after abatement (Cytawa and Teitelbaum, 1967).

Hyperemotionality appears to be an effect of tumors in the area of the septum pellucidum of humans (Zeman and King, 1958) and it is probably an effect of lesions in the rabbit (Green and Arduini, 1954, reported heightened fearfulness after septal lesions) but it does not appear to any great extent after septal lesions in rhesus monkeys (Votaw, 1960), squirrel monkeys (Buddington, King and Roberts, 1967), cats (Bond, Randt, Bidder and Rowland, 1957; Heath, 1954; Moore, 1964; Wheatley, 1944; but Spiegel, Miller and Oppenheimer,

1940, did report a sham rage type reaction after septal lesions in cats), guinea pigs (Nauta, reported in Sodetz et al., 1967), cotton rats (Bunnell, reported in Sodetz et al., 1967) or hamsters (Sodetz et al., 1967). In addition it does not appear in rats with unilateral septal damage (Green and Schwarzbaum, 1968) and medial septal damage produces placidity (Clody and Carlton, 1969). In fact, after a study of the neural structures involved in hyperemotionality Turner (1970) concluded that its appearance was not associated with septal area damage per se, but rather it was associated with damage to the stria terminalis. Furthermore, although many authors have looked for relations between hyperemotionality and other behavioral effects of septal damage, to my knowledge no one has reported such a relation in the literature. This fact lends some plausibility to Turner's conclusion, especially since it is difficult to confine brain lesions to a precisely defined anatomical locus. Nevertheless, more evidence is needed to substantiate Turner's conclusion. In addition, more precise measurements of emotionality and further studies of its relation to other behaviors would be helpful.

Exploratory Behavior and Activity. Detailed observations of the sequential pattern of exploratory behavior in septal damaged animals have not been made. However, much is known about the overall level of activity and the frequency of specific acts. In a 20 minute test in an enclosed (no

pair of walls were more than 6 inches apart), brightly lit maze, septal damaged rats rear less and groom more than normal rats (Dirlam, 1969). In shorter tests (5 minutes) in an open field they also rear less than normal animals but in this situation they sniff more (Novak and Pihl, 1969). Also, in an open field septal damaged rats show a longer latency to begin exploration, spend less time in the center after they begin exploring, investigate a novel object less than controls (Corman et al., 1967) and show less overall activity (Corman et al., 1967; Gotsick, 1969; Schwarzbaum and Gay, 1966) as do septal damaged hamsters (Sodetz and Bunnell, 1970). On the other hand Donovick and Wakeman (1969) report enhanced open-field activity in either bright or dim light following septal damage. In support of the findings showing a decrease in activity due to septal damage, such rats show less activity in running wheels whether tested over a fraction of a day (Kenyon, 1962; Douglas and Raphelson, 1966b) or over several days (Dirlam, 1969) and they show less activity when tested by a device which is sensitive to the changes produced in an electromagnetic field by movements of the animals (Pihl and Greenberg, 1969). Clody and Carlson (1969) also found that septal damaged rats were less active, but on the other hand they were faster to climb out of an enclosure. These rats also are more active during short tests in photocell cages (Douglas and Raphelson, 1968) and as active in photocell

cages as normal animals when tested for several days (Dirlam, 1969). In addition Nielson, Mc Iver and Boswell, (1965) and Schwarzbaum, Green, Beatty and Thompson (1967) found that septal damaged rats explore more than control rats in a dim, enclosed apparatus.

Beginning attempts to determine the relation between the effects of septal lesions on the responses to novelty and on various other behaviors have been made recently. In that study it was found that rearing during 2, 20-minute tests of exploratory behavior was significantly positively correlated with the total amount of wheel running in a continuous 2 week test and both were significantly negatively correlated with grooming during the exploration test (Dirlam, 1969). This suggests that changes in these three behaviors with brain damage may all be due to one common cause.

Given the rather mixed effects of septal lesions on exploratory behavior, it appears that some new approaches are needed. One possibility would be to do an analysis of the sequential pattern of the actual details of behavior (such as that done by Hopkins, 1969, on grooming in the rat) to determine whether the activity of septal damaged animals appears more sterectyped than that of control animals. In addition, some attempt to determine the effectiveness of exploratory behavior should be made (e.g. latent learning tests or times to find hidden escapes, food or water). Finally, much more work on the relation between the effects

of septal damage on exploratory behavior and those on other behaviors is needed.

Learned Behaviors

There are a large number of studies on the effects of septal damage on various learning tasks. Since the natural patterns of behavior serving consummatory goals (e.g. eating or drinking) differ considerably from those serving escape or avoidance of pain (see Bolles, 1969; Breland and Breland, 1966), behavior serving these different goals will be considered separately. A further distinction will be made between the motor requirements of the tasks--whether they require locomotor or manipulative responses (bar pressing or other responses requiring primarily the use of the forepaws).

Learning with Consummatory Goals: Locomotor Responses. The first experimental findings on the effects of septal lesions on the learning of locomotor responses for consummatory goals were those of Thomas, Moore, Harvey and Hunt (1959). According to their findings rats with septal damage make more errors in learning a complex (Lashley III) maze but also run faster than control rats. In addition Carey (1968b) found an almost complete retention loss of a preoperatively learned, four-choice T maze. In later studies (Barker, 1965; also see Thomas et al., 1968), Thomas and his colleagues found that septal damaged rats would not alternate their running speeds in a straight alley as normal

rats do when reinforcement is withheld on alternate trials. Also, unlike normal rats that alternate arms of a maze when all choices are reinforced, rats with septal damage do not alternate (Dalland, 1970; Winocur and Mills, 1969). However, this effect appears to be a perseveration of stimuli rather than of responses because if the starting point is reversed (in a + maze similar to that used by Tolman, Ritchie and Kalish, 1946) septal-damaged rats will perseverate stimuli (arms of the maze) rather than responses (direction of turn; Dalland, 1970). The perseveration of stimuli may be due to an exaggerated aversion toward light that was noted in animals with septal lesions (Ellen and Bates, 1970; Schwarzbaum, et al., 1967). Although Dalland (1970) used a maze with black and white arms he did not mention which was chosen by his animals with septal damage. In addition to deficits in spontaneous alternation it has also been demonstrated that septal damage results in an inability to learn a spatial alternation (Schwarzbaum and Donovick, 1968). However in agreement with the original findings of Thomas et al. (1959) septal damaged rats perform responses faster than normal rats in the alternation task. On the other hand, the increase in errors and faster running speeds were not found in a recent test using the Hebb-Williams maze (Ain, Lubar, Moon and Kulig, 1969). However, their lesions appeared to be somewhat smaller than those of Thomas et al. (1959) and no hyperemotionality was found.

On several other locomotor tasks there seems to be no effect of septal damage on original learning, but deficits in the reversal of this learning have frequently been found (Donovick and Schwarzbaum, 1966, 1968; Gittelson and Donovick, 1968; Hamilton, Kelsey and Grossman, 1970; Kasper, 1965; Schwarzbaum and Donovick, 1968). The one exception that has been noted is that there is no deficit in the reversal of a response requiring the rat to shuttle between compartments and press one of two bars (Schwarzbaum and Donovick, 1968). Even in this case there was no effect of the lesions in only one task, a brightness discrimination, while there was a deleterious effect on a position discrimination. The former problem required an average of 150 or more trials for the control rats to learn or to reverse, while the latter required only 18 to learn and 68 to reverse. Although septal damage appears to have no effect on the original learning of these simpler tasks, if the operations are performed after learning is complete they mildly impair retention (Kleiner, Meyer and Meyer, 1967).

A recent finding about which there is still some confusion is that septal damage results in an enhanced tendency to avoid open places (sometimes called "thigmotaxis"). Green and Levinthal (1967) noted that all rats would take a long route toward a goal that involved staying near a wall rather than a shorter one which involved entering the center of a field. However, normal rats quickly learn to take the

shorter path while septal damaged rats stick to the longer one. This finding was confirmed in a study using the Dashiell maze (Ellen and Bate, 1969). On the other hand, Clody and Carlson (1969) found that septal-damaged rats would take a direct route across the center of an open field (in order to drink sucrose) more often than normal rats would.

In conclusion, septal lesions appear to have little or no effect on the original learning of locomotor responses, but they do affect the reversal of such learning. However, more information on the ability of septal damaged animals to learn other sorts of locomotor tasks such as the delayed response (Hunter, 1917), umweg (Tolman and Honzik, 1930) and reasoning problems (Maier, 1929, 1932) would be helpful. In addition attempts should be made to pick tasks that are "natural" for normal animals--e.g. teaching a cow to run for food (Breland and Breland, 1966) or a rat to rear in order to avoid shock (Bolles, 1969) are not "natural" tasks.

Manipulatory Responses. Rats with septal damage bar-press more than control rats when there is no reinforcement (Schwarzbaum, Kellicutt, Spieth and Thompson, 1964) as well as when food or sucrose solutions are used as a reinforcer (Beatty and Schwarzbaum, 1968; Buckland and Schwarzbaum, 1970; Ehrlich, 1963; Ellen and Powell, 1962; Ellen Wilson and Powell, 1964; Holdstock and Edelson, 1969; Kenyon, 1962; Schwarzbaum and Gay, 1966). They also show greater resistance

to extinction (Butters and Rosvold, 1968a, b; Carey, 1967a) but only if the septal damage is made before training. If the damage is made after training resistance to extinction is less (Carey, 1967a), which probably indicates that there is some retention loss as Kleiner et al. (1967) found.

As mentioned above rats with septal damage have no difficulties with the original learning of brightness or position discriminations. This holds true for the learning of a position discrimination in the WGTA by cats with septal lesions (Zucker and Mc Cleary, 1964) and for learning to discriminate how much the wall holding a lever is tilted (Holdstock and Edelson, 1969). But the rats mentioned above did have difficulty with reversal. Similarly, cats with septal damage have difficulty reversing a position discrimination (Zucker and Mc Cleary, 1964).

One of the most striking effects of septal damage on learned manipulative responses is the effect on DRL performance. Both learning to produce the low rates of responding and retaining them when they were preoperatively learned is disrupted by septal area damage (Agnew and Meyer, 1969; Burkett and Bunnell, 1966; Caplan and Stamm, 1967; Carey, 1967b, c, 1968; Ellen and Butters, 1969; Mac Dougall, Van Hoessen and Mitchell, 1969a, 1969b). The deficit cannot be simply a disruption of time estimation because animals with septal damage "scallop" normally on a FI schedule (Beatty and Schwarzbaum, 1968). Also it is apparently not

due to increased motivation, which increases the number of responses per reinforcement (i.e. decreases the efficiency) of normal rats, because rats with septal damage perform worse than normal rats with even 72 hours of water deprivation. Caplan and Stamm (1967), however, have made considerable progress in elucidating the disability by showing how the brain damaged animals can be taught to perform the low rates of responding. If training is begun with short (4 sec.) delays which are increased by 2 sec. each time the animals begin performing at criterion level (reinforcement to response ratio of 50%), then septal-damaged rats perform as well as control rats. It would be interesting to know if other learning deficits (such as those on the Lashley III maze) could be overcome by the use of gradual training.

Escape or Avoidance Learning: Locomotor Responses. There are three kinds of avoidance learning that involve locomotor responses. One involves having the animal move away from an unsafe place to a safe one; the second involves punishing the animal for spontaneous locomotion (movement inhibition training); and the third involves training animals to make a locomotor response and then punishing it for making the response (passive avoidance). These three kinds of avoidance will be considered separately.

Septal damage results in a marked impairment in the ability to learn to move from an unsafe place to a safe one when the two locations maintain a constant position

in space. This is true for tasks requiring running of rats (Kenyon, 1962; Kenyon and Krieckhaus, 1965, Mc New and Thompson, 1966; Vanderwolf, 1964, 1967) or of hamsters (Babbini and Davis, 1967) and of those requiring jumping of cats (Hamilton, 1969; Hamilton and Grossman, 1969). Attempts have been made to explain away these defects as being caused by an aversion on the part of septal damaged animals to the handling that takes place in the safe part of the apparatus (Zucker, 1965; Mc Cleary, 1966). In fact Zucker (1965) using a shuttle box, each end of which rotated 180° after each trial, found an improvement in learning following septal area damage. He considered that this apparatus was the same as other one-way apparatus except that no handling was required. However, Krieckhaus (1965) has argued that the cats in this situation were probably taking their cues from inside Zucker's apparatus and that in effect this made it more similar to a shuttle box (see next section). A method of returning septal damaged animals to the start box that is not aversive to them would help resolve this conflict.

Considering movement inhibition training, Blanchard and Fial (1968) found no effect of septal lesions on the amount of crouching or on the number of passive avoidance errors in a runway with an 8" square island in the center. On the other hand, septal lesions do retard learning to stay on a safe perch (Novak and Pihl, 1969; Winocur and Mills, 1969). The perches used for the latter tests were smaller

than the 8" square and therefore, the negative findings of Blanchard and Fial may be due to the large safe area used in their study.

Mowrer (1960) and Bolles (1970) have pointed out that the shuttle box procedure of training locomotor avoidance responses requires animals to return to a place where they were previously shocked and therefore, is likely to produce the same kind of inhibition that passive avoidance training produces. This argument may explain why it takes roughly 10 times as many trials for normal animals to learn the locomotor response in a shuttle box as it takes them to learn the same response in a one-way (running-to-safety) apparatus (e.g. see Bolles, 1970). It was an early and often repeated finding that septal dysfunction facilitates learning the shuttle-box avoidance response (Buddington et al., 1967; Donovick, 1968; Fox, Kimble, and Lickey, 1964; Green, Beatty and Schwarzbaum, 1967; Green and Schwarzbaum, 1968; Hamilton, Mc Cleary and Grossman, 1968; Hamilton, 1969; Kenyon, 1962; Kenyon and Krieckhaus, 1965; King, 1958; Krieckhaus, Simmons, Thomas and Kenyon, 1964; La Vacque, 1966; Lown, Hayes and Schaub, 1969; Matalka, 1968; Mc Cleary, 1961; Meyer, Johnson and Vaughan, 1970; Novak and Pihl, 1969; Schwarzbaum, et al., 1967; Trafton, 1967; Van Hoessen, Mac Dougall and Mitchell, 1969). The effect is found in the squirrel monkey (Buddington et al., 1967), guinea pig (Lown et al., 1969) and hamster (Matalka, 1968) as well as

in cats and rats and it persists when septal lesions are combined with total neodecorticaton which normally severely impairs leaning (Meyer et al., 1970). It is often accompanied by a greater amount of intertrial responding (Donovick, 1968; Green et al., 1967), but this is not necessary since middle septal lesions (i.e. at the middle of the septal area along the anterior-posterior dimension) produce the facilitation of learning without greater intertrial responding (Donovick, 1968). In addition the amount of performance improvement is significantly correlated with the disruption of the CER (a measure of freezing) but this correlation explains only 11% of the variation (Trafton, 1967). It is also accompanied by greater resistance to extinction (Hamilton et al., 1968; La Vacque, 1966). That improvement in the performance of this problem is an indication of dysfunction is strongly suggested by the findings of Novak and Pihl (1969) which show that large doses of amphetamine (3 or 9 mg/kg) which can lead to complete disorganization of behavior, improve performance on this task in animals without brain damage. That both the lesion effect and poor performance of normal animals is due to the punishment of effective responses is strongly suggested by the fact that both septal damaged and normal rats learn to shuttle for food in less than twenty trials (Schwarzbaum and Donovick, 1966) but it takes several times as many trials for them to learn to shuttle under shock inducement. It is interesting in this context that

it takes approximately as long for septal damaged animals to learn the shuttle avoidance as it takes them to learn the one-way active avoidance, even though they are better than normal animals on the former and worse on the latter. This suggests that the shock for returning to the previously safe compartment has little effect on these animals.

The conclusion that punishing learned locomotor responses has little effect on animals with septal damage is supported by tests in situations other than the shuttle box. Whether the original response was food motivated (Fried, 1969; Ursin, Linck and Mc Cleary, 1969; Van Hoessen et al. 1969; Wishart and Mogenson, 1970), water motivated (Fox et al., 1964; Hamilton et al., 1970; Mc Cleary, Jones and Ursin, 1964; Middaugh and Lubar, 1970) or shock motivated (Mc New and Thompson, 1966), animals with septal dysfunction are more persistent in making the response after receiving shock for making it than normal animals are. However, if the approach response is water motivated and they receive quinine for making the response, they are <u>less</u> persistant than normal animals (Gittelson, Donovick and Burright, 1969). This last finding suggests that septal damaged animals can be flexible only when both the reward and the punishment are part of the same closely related system (i.e. the drinking system--water reward and taste punishment are related but water reward and foot-shock are not).

Manipulatory Responses. Findings concerning the acquisition of the CER, which involves manipulatory-response suppression are somewhat mixed. Brady and Nauta (1953) found no effect of septal damage but Trafton (1967) found an impairment. Harvey, Jacobson and Hunt (1961) and Tracy and Harrison (1956) found retention deficits and the former authors noted that if the animals were tested at least 16 weeks after the operation, there was also a learning deficit. In addition either contingent (Brady and Conrad, 1960) or non-contingent (Goldstein, 1966) septal stimulation retards acquisition of the CER (suggesting a disruptive effect of the stimulation on normal septal functioning).

Morgan and Mitchell (1969) have found that septal damaged animals learn to press a bar in order to postpone shock faster than normal animals do. The brain damaged rats also press less frequently than normal rats and therefore, are much more efficient. However, it took normal and septal damaged rats four 4-hour sessions to reach an optimal rate of responding and Bolles (1969) has argued that this response is not really effective from the animals point of view because it does not permit escape from the situation. In addition Bolles found that normal animals could learn the bar-press avoidance response only by first freezing on the bar. Thus, it would be helpful to know the latency to freeze near the bar of septal damaged and control rats.

Conclusions for Avoidance Learning. In the learning of avoidance responses septal damaged animals appear to be impaired on easy tasks and actually better than normals on difficult tasks (easy and difficult are defined in terms of the amount of time required for normal animals to learn the task). However, according to Bolles (1969, p. 35) "For an avoidance response to be rapidly learned in a given situation the response must be an effective SSDR [species specific defense reaction] in that situation." By "effective" Bolles means functionally effective--i.e. actually making flight possible. But the difficult avoidance situations described above do not make flight possible. Therefore, it appears that septal damaged rats are better than normal rats at learning functionally ineffective responses and at the same time they are much poorer at learning functionally effective ones. In future studies it would probably aid our understanding if more attention were paid to the actual details of behavior in the learning situation.

General Summary and Conclusions of the Behavioral Syndrome of Septal Damage.

The mechanisms for the production of freezing, thermoregulation sleeping and sexual behavior are only mildly if at all disrupted by septal damage. Also the main effect on grooming, feeding and drinking appears to

be only an enhanced probability of occurrence of some components of these behaviors. In animals with genetic disruption of aggressive behavior (laboratory rats), there is an increased frequency of some components of the behaviors while in animals in which effective aggressive responses have survival value (wild rats) there is a severe disruption combined with an increase of some component acts. Maternal behavior, nest building and possibly hoarding are almost totally blocked by septal lesions but again some components of at least maternal behavior increase in frequency. Findings concerning exploratory behavior seem to depend very much on stimulus conditions but much work is needed before these effects are thoroughly understood. Septal damage has little effect on the original learning of tasks which use consummatory rewards, but it does affect the reversal of this learning. However, little is known of the effects of septal damage on learning which requires complex processing such as umweg, delayed responses and reasoning tasks. Septal damage does result in marked deficits in learning to produce low rates of bar pressing unless gradual training methods are used. Regarding avoidance responses, septal damage appears to disrupt the learning of effective responses (those that make species specific defense responses, SSDR, possible), but improves the ability to learn ineffective responses (i.e. bar pressing, a task

which does not permit SSDR). However, this conclusion is still being questioned because of the effects of intertrial handling on septal damaged rats. Finally, although attempts have been made to generalize about the syndrome of septal dysfunction (e.g. Mc Cleary, 1961, 1966; Thomas et al., 1968), there is no widespread agreement.

In general, where there is most disagreement about results and interpretations, detailed observations of behavior and interpretation in terms of the response effectiveness or value to the survival of the animal have not been made. In addition, studies that show interrelations between the effects of a number of measures and several tasks in the examination of each animal would be quite helpful in deciding not only which generalization would be appropriate but also whether a single generalization about the syndrome of septal dysfunction should be sought.

The Present Study

The present study examines the effects of damage to the septal and/or surrounding areas on a variety of behaviors. The behaviors include drinking, hyperemotionality, exploratory behavior, escape behavior, passive avoidance behavior, learning and performance of movement inhibition, and jump avoidance behavior. Drinking was measured daily for approximately one month. Hyperemotionality was rated several

times by four raters using both a new and a previously used rating scale. Components of exploration in an open field were sampled and recorded every 12 sec. for 5 or 3 min. tests on 4 different days. Escape to a safe, 36 in.^{2} island in the middle of an open field with an electrified grid floor was tested by continuous shocking on alternate 6 sec. periods for 20 min. Passive avoidance was measured by the latency to descend from 36 in.^{2} and $6\frac{1}{4}$ in.^{2} islands in the center of the open field. The criterion latency was 60 sec. Movement inhibition was trained by shaping Ss to escape first to the large island and then to the small island in the center of the open field, and it was tested by placing Ss on the grid with the small island in the center and leaving them there for 60 min. on two different days. Jump avoidance was tested in a 1 cu. ft. box with a safe shelf around the top and a grid floor. A 15 sec. shock latency was used in one experiment and a 5 sec. latency in another. Later experiments tested for order effects involving the tests of jump avoidance learning and movement inhibition, and for the effects of additional brain damage to areas surrounding the septal area, which included the preoptic area, the caudate nucleus and the medial frontal cortex. For this study the safe island was at the periphery of the field and no shaping was used. A final experiment tested the effect of below-the-grid rather than overhead lighting on jump avoidance learning of septal damaged rats.

CHAPTER 2

EXPERIMENT 1

Method

Subjects

The Ss were 32 male albino rats (Sprague Dawley) from Woodlyn Farms weighing 265-325 g. (mean = 285 g.) at the time of the operations. All Ss were housed singly in constant light at room temperature. All Ss were weighed immediately before the operation and again immediately before they were sacrificed. The time span between these dates ranged from 106-117 da. Because of this variability, measures of weight gain over this period were stated in terms of gain per day.

Surgery and Histology

Nembutal anesthesia was used. Electrodes were 0.010 in. diameter insulated nichrome wire with bare tips of 1.0-2.0 mm. They were positioned in the medial portion of the septal area by means of a Krieg stereotaxic instrument, with the electrodes entering 1.5 mm. anterior to

bregma and 1.5 mm. lateral to the midline, and lowered to 5.5 mm. ventral to the dura at an angle of 14° in order to avoid puncture of the superior sagittal sinus. Electrolytic lesions were made by passing 3 ma. DC for 20 sec. through the electrodes. In order to prevent excessive pitting of the electrodes, they were replaced after being used during electrolysis for a total of approximately 1.5 min. Thermocoagulation lesions were made by heating the electrodes by means of a Grass radio-frequency (2 megacycles) lesion maker. The current was adjusted to a value slightly less than sufficient to cause the audible "pop" or steam bubble that can be generated by heating the electrode tip (e.g. see Hoebel, 1965) and for a total of 7 sec. Sham operations were produced in control animals by the same method as the normal operation except that no current was used. Originally, there were 12 rats in each of the 2 lesion groups and 8 rats in the control group. However, 1 rat with a sham operation and 1 with a heat induced lesion died during the experiment and histological study showed that 1 rat with an electrolytic lesion had unusually little damage and 1 rat with a heat-induced lesion had extensive damage in the diencephalon. This left 11 animals in the DC lesion group, 10 animals in the RF group and 7 animals in the control group.

At the end of the experiment, all animals were sacrificed, perfused with normal saline and 10% formalin, and their brains were extracted for later sectioning.

Sections from all operated animals and 2 control animals were taken at 15/4 and stained with haematoxylin and eosin for histological examination. When extent-of-damage measures were used, these were done by ranking the extent of damage in different animals on the basis of apparent differences. Those with small or non-obvious differences were given tied ranks.

Water Consumption

Water consumption was measured daily from the time of the operation until 36-40 da. afterwards. Tap water was used and measurements were taken by weighing the bottles at the same time every day. Water and bottles were changed weekly at the time of weighing.

Emotionality Rating

Emotionality ratings were done 14, 16 and 29-33 da. after the surgical operation. Two rating scales were used. One was identical to that designed by Brady and Nauta (1953) and the other scale was similar except that behavior indicative of each value on the scale was specified, and in addition to the situations described by Brady and Nauta, the reaction of the animals to a puff of air was observed. The behavior which defined each scale value was based on descriptions of emotional behavior of rats. Thus, there were four situations: a puff of air was directed at S when the cage was still closed; a pencil was presented one in. from S's snout; S was prodded with the pencil; and

S was captured and handled. For each of these situations, 2 points were given for vigorous attack with biting or for vigorous, jumping escape, 1 point was given for either threat behavior (rearing with forelimbs extended and mouth open without falling back against a wall for the 15 sec. duration of the situation) or running escape (all four legs move at least once in quick succession), and 0 points were given for any other behavior including freezing, submissive threat (rearing with forelimbs extended, back against a wall and thus, adopting a poor posture for defense) or slow withdrawal. In addition 1 point was added to the score for each situation if the animal vocalized. Finally, 1 point was added to the total score of an animal for showing each of the following responses at any time during the test: urination, defecation and piloerection. There was a minimum score of 0 and a maximum score of 15 on the test. Two raters each using 1 of the 2 scales rated emotional behavior on day 14 and this was repeated by 2 different raters on da. 16. Approximately 2 weeks later all 4 raters simultaneously rated the Ss using the same scale they previously used. All 4 raters were experienced in handling animals, but had not previously used these scales.

Exploratory Behavior

Behavior of each S in the open field was observed for 5 min. on the 15th and 16th da. after the operation and for 3 min. on the 24th and 38th da. after the operation.

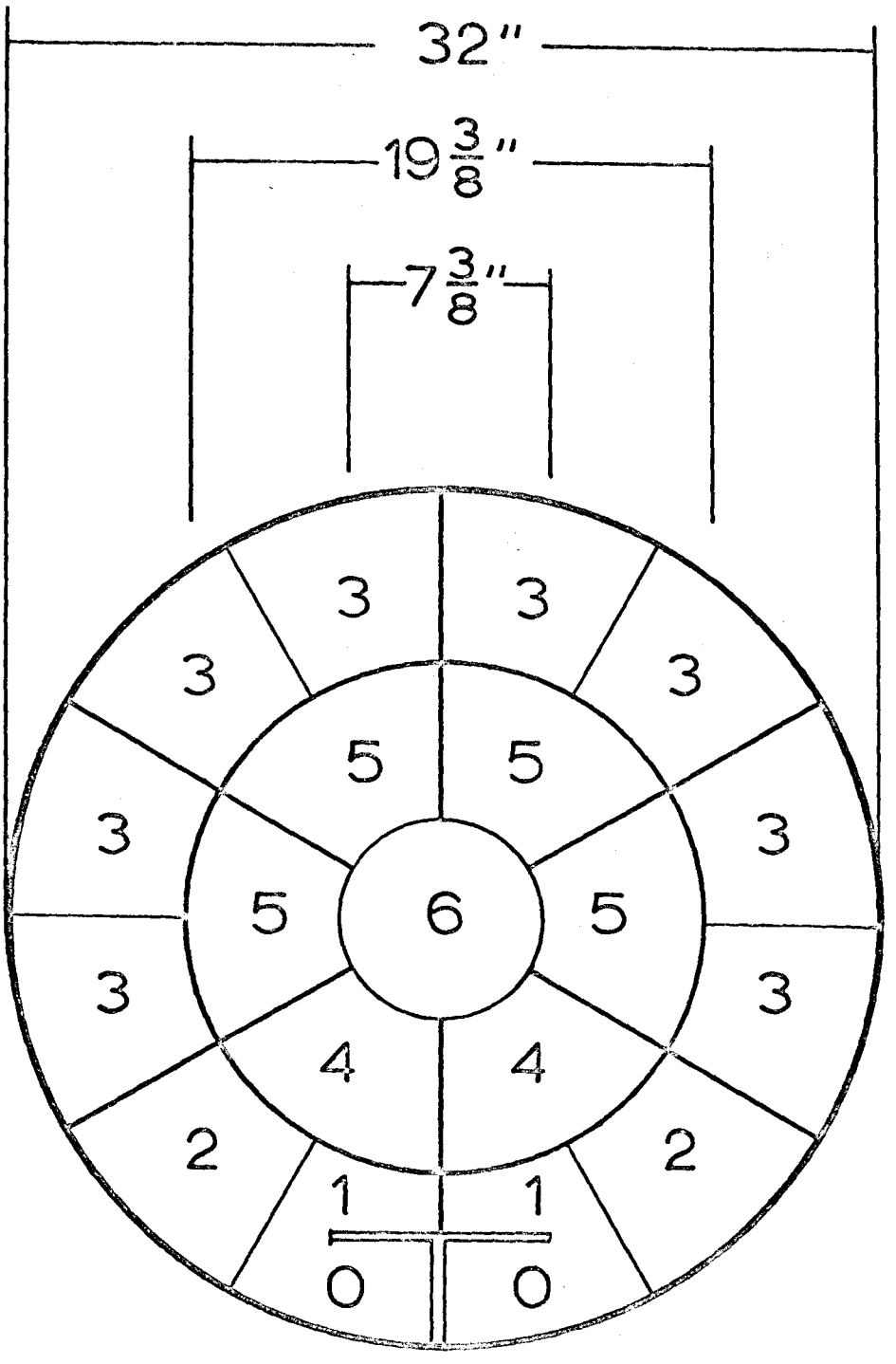

FIG.1 Floor pattern of open field
(scale: $\frac{1}{10}$)

The open field was identical to that used by Broadhurst (1961) except that a small enclosure was placed near the wall (see Fig. 1). The field was a round wooden enclosure 32 in. in diameter with 12 in. high walls. The floor was light colored linoleum and the walls were white except those of the enclosure which were natural wood. The field was marked off into 19 areas of equal size (approximately 42 sq. in.) and 2 of these areas were divided by the enclosure. The areas were assigned a number from 0 to 6 according to their distance from the enclosure and from the wall (see Fig. 1). The only light was provided by an ordinary desk lamp with a 60 W bulb and was placed opposite the enclosure (which, therefore, was only indirectly lighted) approximately 6 in. from the wall and 18 in. above the field. The observer sat behind the enclosure and was divided from the field by a one-way screen. Sound was kept to a minimum except for a time marker which clicked every 12 sec.

During each test period, the S was placed in the center of the field. The area number was recorded whenever the S moved all four legs from 1 of the 21 areas to another. Behavior (walking, turning, rearing, chewing, grooming, sniffing, lying or freezing) which was in progress at each click of the 12 sec. time marker and the total number of fecal boluses during the test were recorded. The latency to move, to reach the wall after moving and to reach the enclosure after finding the wall, the time spent in the

enclosure, the frequency of entering the enclosure, the number of times leaving the wall, the total distance traversed, the frequency of different behaviors, and the change of behavior during and between tests were determined from these observations.

Escape Training

The escape training was conducted in the movement inhibition apparatus which was a square open field 24 in. x 24 in. x 18 in. high with a grid floor and a 6 in. square wooden island in the center. Escape from the grid to the island proved to be a difficult response to establish in this apparatus. Other research has established that the response will usually occur spontaneously if the grid is narrow (Teitelbaum and Milner, 1963) or if the island is placed adjacent to the wall (Experiment II of this report). Nevertheless, in the open field with the island in the center, the first attempt to "train" the animals by making escape from shock contingent upon climbing onto the island was unsuccessful except in a few cases. Therefore, this attempt (which will be called "escape testing") was followed by passive avoidance tests and shaping of the escape response (the term "escape training" will subsequently be reserved for the shaping of the escape response).

For the escape test the Ss were placed on the grid and allowed 15 min. exploration before testing. During the test, scrambled, 1 ma. electric shock (from a 28 VDC power

supply) was applied through the grid continuously for 6 out of every 12 sec. This part of the procedure was terminated after 20 min. or after the S had remained on the island continuously for 60 sec. (of the 28 Ss only 8 including 4 control Ss had learned to find the island within the 20 min. period). The number and latency of escape responses and freezing responses were recorded as well as the number showing threat responses and the number of Ss reaching the island.

After the original escape testing all Ss (whether or not they had previously been on the island) were given passive avoidance training. For this, the animals were placed on the large island with the grid electrified so that they were shocked immediately if they descended from the island. Once they left the island, they were allowed 30 sec. to return to it before the trial was terminated. If the animals remained on the island for 60 sec., training was terminated. The number of trials to the criterion of 60 sec. for each S was recorded.

After the passive avoidance response was established, escape training was begun. For this the animal was placed on the electrified grid and allowed 30 sec. to find the island. If the island had not been reached during this period, shaping was begun (i.e. successive approximations toward the island were rewarded by temporary termination of the shock). Shaping was continued until the animal climbed on the island

and remained there for 60 sec. This procedure was repeated until the animal had found the island with the 30 sec. period of continuous shock (i.e. without shaping) on five successive trials.

After the animals had learned to escape to the large island, the passive avoidance and escape training were repeated with the small (2.5 in. x 2.5 in. square) island. The number of trials with shaping and the mean escape latency on the last criterion trials were recorded.

Movement Inhibition Test

The day after the final escape training and again 2-3 wks. later, the Ss were given 1-hr. tests of movement inhibition. This was done by placing the Ss on a grid electrified with 1 ma. scrambled shock. The $2\frac{1}{2}$ in. island in the center of the field was mounted on a micro switch which advanced an electromagnetic (Mercury) counter when changing from a down to an up position and activated a Rustrak recorder when it changed position. The time required to get on the island and the frequency of descending from the island during each of the two, one-hour tests were recorded.

Active Avoidance Learning

One to two weeks after the second movement inhibition test (11-12 wks. after the operation), the Ss were trained on a simple active avoidance learning problem. The apparatus which was similar to that described by Vanderwolf (1969) was a 12 x 12 x 12 in. plywood box with a grid floor

and a 2-½ in. wide shelf surrounding the box, ½ in. below the upper edge. A small carrying box measuring 6 x 6 x 4½ in. housed the rats during the intertrial interval of 30 sec. Animals with septal lesions are highly resistant to handling especially in avoidance situations, a factor which might affect scores on behavioral tests. A method was devised for handling such animals without disturbance. When animals jumped to the shelf the carrying box was placed in front of them. If the animal did not enter the box spontaneously it was tapped on the back lightly with the lid. After the animals entered the box, the lid was placed on it for the duration of the trial.

After allowing 15 min. exploration, the Ss were put on the grid and left for 15 sec. If they had not jumped from the grid to the ledge in this period, they were given 2 ma. shocks of short duration every 2-½ sec. until they escaped. The latency of the response was recorded and training was terminated when the S had avoided the shock on 9 out of 10 trials.

Statistical Methods

For a large number of the analyses in this experiment, the Mann-Whitney U was used. In the case of a large number of tied scores, the correction suggested by Siegel (1956) was used. In cases where it was likely for a difference to be in either direction (as in comparisons between the two brain-damaged groups) significance levels were based

on a two-tailed test; otherwise (as in comparisons between brain-damaged and control Ss) one-tailed tests were used.

The interrelations between behaviors were analyzed by means of rank-order correlation coefficients (Spearman's, corrected for ties) which detect variability about positively or negatively sloped lines regardless of curvature (except for lines with multiple and/or U shaped curves). These were computed by an IBM 1130 computer. Only 16 behavioral variables were used in this analysis since in the others there was too little variability for the correlations to be meaningful, or not all the Ss in some group(s) were measured, or there was no significant relation to any of the other measures (only two variables were rejected for this last reason; they were boluses in the open field and weight gain). This left 120 correlation coefficients for each of the three groups.

The remaining correlations were analyzed primarily by inspection. The traditional methods of handling large numbers of correlations were inappropriate because the Ns were too small for factor analysis and cluster analysis produces varying results depending on minor variations in the criteria used. The reasoning behind the particular inspections made is as follows. The main point of concern in examining the correlations is whether given measures of behavior measure the same thing in different groups of animals. For example inactivity could be due to either freezing or

relaxation. If it were due to both, the correlation between a pure measure of freezing and inactivity would not be perfect. One indication of whether the behaviors underlying a measure are the same in both groups is to observe whether the measure is correlated with other measures in the same way in both groups. This was done by inspection but as a purely descriptive index of the similarity, the rank order correlation of the two patterns of correlations was computed. If the patterns of correlations are similar, then it is very likely that they are measuring the same factor in both groups. For example, if inactivity in one group of rats is highly positively correlated with three other measures, highly negatively correlated with six measures and only moderately correlated with five measures and if the same relations are true for a second group of rats, it is very unlikely that the behavior being measured is not very similar in some important respect (in addition the rho between these two patterns of correlations will be very high). On the other hand if the correlations of inactivity with other behaviors are different in the two groups (if the correlation of the correlations is low), there are two main possibilities. The measure could be measuring different things in the two groups or it could be an inadequate measure (not precise enough to distinguish accurately between animals) in one or both of the groups. However, a measure that has several very high correlations with other measures on the same animals, clearly

must be able to distinguish accurately between animals. Therefore, a measure that correlates highly with other measures in different ways in different groups must be measuring different things. Of course these different "things" can either be different behaviors or different causes or both, but that is a problem for further experimental analysis.

In summary, the method used to analyze the correlations was first to look for measures that correlated in different ways in different groups (e.g. there was a 0 or a - correlation between the correlations of the two groups). Secondly, the correlations were examined for measures that showed highly similar patterns in the three groups. Finally, the remaining correlations were discussed in terms of those with different or similar patterns for the three groups.

Results

Water Consumption

Table 1 shows the mean and standard deviations of water consumption for Ss with radio-frequency (RF), direct-current (DC) or sham operations of the septal area. There was no detectable effect of the operations on water consumption. Data for the first week after the operation were used in the analysis because during the second or third week of measurement a gradual increase in water consumption

Table 1. Mean water consumption during first week after operation (cc.).

	RF	DC	SHAM
Mean	196	204	201
Standard Deviation	41	44	30
N	10	11	7

followed several days later by a gradual decrease to the original baseline was observed in every animal except for one with a sham operation. Possibly, this was caused by a mild illness. There was no noticeable relation between the location or size of the lesion and the amount or time of onset of this change.

Emotionality Ratings

Table 2 shows the means and standard deviations of the emotionality ratings using the scale developed in this study. Table 3 shows the unweighted means analysis of variance and post-hoc comparisons for the data presented in Table 2. The results of these analyses show that the animals with RF lesions were more emotional as defined by the scale than those with DC lesions ($p < .05$). The animals with sham operations were less emotional than those with either RF ($p < .001$) or DC ($p < .05$) lesions. Animals in all three groups were less emotional during the second measurement

Table 2. Emotionality ratings using scale developed in this study.

Group	Test 1		Test 2		Test 3		3 tests combined	
	M	SD	M	SD	M	SD	M	SD
DC	5.7	2.7	2.1	1.5	4.2	2.5	4.0	1.6
RF	7.4	2.3	6.3	2.5	7.2	2.6	7.0	1.8
SHAM	3.3	2.1	1.6	1.3	2.3	1.2	2.4	1.3

M - mean, SD - standard deviation

Table 3a. Analysis of variance of emotionality ratings.

SOURCE	DF	MS	F	p
Between Ss				
Group	2	28.3	3.41	.05
Sw.g.	26	8.3		
Within Ss				
Tests	2	164	23.7	.001
Group X Tests	4	1.67	0.24	NS
Tests X Sw.g.	52	6.90		

Table 3b. Post-hoc comparisons.

GROUP	TEST	SUM	COMPARISONS					
DC	1	63	1	1		1	1	
	2	23	1	1		-1		1
	3	46	1	1			-1	-1
RF	1	81	-1		1	1	1	
	2	69	-1		1	-1		1
	3	79	-1		1		-1	-1
SHAM	1	23		-1	-1	1	1	
	2	11		-1	-1	-1		1
	3	17		-1	-1		-1	-1
p			.05	.05	.001	.001	NS	.05

than during the first ($p < .001$) or the third ($p = .05$) tests. In other words, two days after the first measurement there was a sharp decline in the emotionality of all three groups of animals but two weeks later the emotionality had nearly returned to its previous intensity (there was no significant difference between the 1st and 3rd test). Although the ratings using the present scale were quite highly correlated (.90) with those using the Brady and Nauta (B-N) emotionality scale, the latter scale was less reliable than the scale developed in this study. The ratings made by different raters based on the B-N scale differed by more than 20% of the mean scale value 69% of the time while those based on the present scale did so only 10% of the time ($X^2 = 20.9$, $p < .001$). These differences were evenly distributed over the three groups and therefore, they must reflect a disagreement over

what is meant by a non-emotional reaction (a base-line reaction) to specific situations of the B-N scale. The correlations between the pairs of ratings for each scale are presented in Table 4. The disagreements between the ratings

Table 4. Correlations between ratings on third tests.

SCALE	DC	RF	SHAM	ALL Ss
B-N	.85	.95	.77	.95
New scale	.95	.95	.95	.96

based on the B-N scale were positively related to the variation within the groups. However, the correlations were uniformly very high when the new scale was used. Since the control Ss show the least variation, the differences between the emotionality of "normal" rats are not as reliably detected with the B-N scale. This also reflects the difficulty mentioned above of determining the baseline reaction when using the B-N scale.

Open Field Behavior

The analysis of behavior in the open field is presented in Table 5a and Table 5b. Both the tables and the figure show medians and interquartile ranges for most of the measures.

Table 5a. Frequency of open-field behaviors (4-day totals).

Group	Measure	Exploration	Grooming	Inactivity	Defecation (fecal boli)	Squares traversed	Leaving wall	Entering compartment
RF	MD IQR	4½ 4-15	1½ 1-3	67½ 63-71	8 7-12	17 12-25	0 0	3 1-4
DC	MD IQR	17 6-23	7 3-9	56 49-67	8 4-10	29 14-55	0 0-1	6 4-7
Sh	MD IQR	34 27-40	7 3-9	39 39-44	4 1-11	86 60-150	5 3-9	11 7-15
Comparisons								
RF X DC	U P	NS	24 <.05	22 <.05	NS	NS	NS	21 <.05
RF X Sh	U P	1 <.001	11 <.01	0 <.001	NS	8 <.01	5½ <.005	1 <.001
DC X Sh	U P	4 <.001	NS	2½ <.001	NS	10 <.01	8½ <.005	11½ <.01

MD - median, IQR - interquartile range, U - Mann-Whitney U (corrected for ties), p - probability

Table 5b. Open-field latencies (in number of 12 sec. intervals).

Group	Measure	Movement latency				Latency to find wall after first movement				Latency to enter compartment after finding wall				Average time in compartment per entry
		Day 1	Day 2	Day 3	Day 4	Day 1	Day 2	Day 3	Day 4	Day 1	Day 2	Day 3	Day 4	
RF	MD	1½	3	2	½	2	1	1	1	6	2	10	Max	13
	IQR	1-4	2-5	0-2	0-4	0-4	0-6	0-1	0-4	2-Max	0-18	3-Max	3-Max	5-14
DC	MD	2	0	0	0	1	0	0	1	6	3	1	3½	8
	IQR	1-4	0-2	0-1	0-2	0-3	0-1	0	0-1	4-13	1-6	1-6	1-9	7-14
Sh	MD	0	0	0	0	1	0	0	0	2	0	1	0	4
		0-1	0	0	0	0-1	0	0	0	1-4	0-1	0-1	0	3-7
Comparisons														
RFxDC	U		8½	28			25½	20					21½	
	P	NS	<.005	<.05	NS	NS	<.05	<.01	NS	NS	NS	NS	<.05	NS
RFxSH	U	8½	0	10½	17½	12	10½	7	10½	13	9½	10½	3½	14½
	P	<.005	<.0005	<.005	<.01	<.05	<.005	<.005	<.005	<.05	<.01	<.05	<.001	=.06
DCxSH	U	7½	21		21	17½			21	7½	11½	20	3½	20
	P	<.005	<.01	NS	<.05	<.05	NS	NS	<.05	<.01	<.01	=.06	<.001	=.06

Md - Median, IQR - inter-quartile range, U - Mann-Whitney U corrected for ties (reported only where P $\leq$ .06), p - probability

The control group showed more exploratory behavior than either of the experimental groups. The time samples showed more walking, rearing and turning ("exploration" in the table) in the control group ($p < .001$) and this group traversed a greater distance than the brain-damaged groups ($p < .01$). The brain-damaged groups showed more inactivity ($p < .001$) and a longer latency of movement especially on the first two days ($p < .01$). But even after they began to explore, the brain damaged rats took longer to approach the wall and the compartment. The usual pattern was first to approach the wall and then to enter the enclosure. On the first test-day both brain-damaged groups spent a longer time between moving and reaching the wall ($p < .05$) but by the second day the DC group performed more like the control group (though only one animal found the wall in < 12 sec. on all three last days while all 7 of the control animals did, $p = .0002$), while the RF group still averaged a longer time to approach the wall ($p < .005$ on the last three days). After reaching the wall, the control animals were also faster to find the enclosure on all four days (p values varied but $p \leq .06$ on all days for comparisons between the control and both experimental groups). Even though the brain damaged animals were slower to reach the wall and the compartment, they were slower to leave the wall after reaching it ($p < .005$) and they remained in the compartment for a longer time after entering it than the control animals ($p = .06$).

Another interesting feature of the exploratory behavior of the control and brain-damaged rats is that the former went in and out of the compartment much more frequently than the latter ($p < .001$ for the RF group and $p < .01$ for the DC group).

There were also some differences in exploration between the two experimental groups. The RF group showed a longer latency than the DC group to reach the wall on the second and third days ($p < .05$, $p < .01$ respectively). They also showed a greater latency to enter the compartment on the fourth day ($p < .05$). Finally they went in and out of the compartment even less frequently than the animals with DC damage. In conclusion rats with septal damage show a greater amount of initial immobility before exploring and walk about less frequently and travel a shorter distance with each continuous move than the animals with DC damage.

The brain damaged animals showed more sitting and sniffing, and lying than control rats did ($p < .001$). They also showed a greater latency to move. Both the DC and sham groups showed more grooming than the Rf group ($p < .05$ and $p < .01$, respectively). There was no difference in the number of fecal boli of the three groups.

Escape Performance

During the 20 min. of escape testing behavioral observations were made on all but two animals from the DC group. These included observations of running, jumping,

crouching, threatening and climbing on the island. The threat responses were either the threat or the submissive threat response described in the section on emotionality rating above. The results are shown in Table 6. There were no significant differences between the two groups with brain damage. However, the combined experimental groups made an average of four times as many escape responses (running or jumping) as the control group during the 20 min. test. This was true for the total number of escape responses made by animals that did not find the island ($p < .005$) and for the number of escape responses in the first two min. made by all animals ($p < .001$), but for those that did learn only the DC group made significantly more responses than the control animals ($p = .06$; there was only one animal with RF damage that learned so comparisons of this subgroup with control rats are not very meaningful. However, this single rat made more escape responses than all but one of the four control animals that learned).

The brain damaged rats also made fewer freezing responses than the control rats ($p < .02$) and they showed a longer latency to make freezing responses ($p < .01$). It might be assumed that running or jumping would facilitate finding the island while freezing would interfere with it. Nevertheless, control rats (which froze more and ran or jumped less) were somewhat more likely to find the island than brain

Table 6. Behavior during the escape test.

Group	Measure	Escape responses: Frequency during test (Ss with test >18 min.)	Escape responses: Frequency in first 2 min. — All Ss	Escape responses: Frequency in first 2 min. — Ss that escaped	Escape responses: Latency (in 12 sec. intervals)	Freezing responses: Frequency during test (Ss with >18 min. of test)	Freezing responses: Latency (no. of 12 sec. intervals)	No. of Ss finding island
RF	Md	101	31½	25	0	29	12	
	IQR	87-151	24-37		0	13-47	9-16	
	N	9	10	1	10	9	10	1 of 10
DC	Md	125	93	43	0	29	10	
	IQR	63-146	28-44	24-54	0	23-73	8-19	
	N	7	9	3	9	7	9	3 of 11
Sh	Md	27½	16	18	0	58½	7	
	IQR	22-65	11-23	8-35	0	45-84	6-9	
	N	4	7	4	7	4	6	4 of 7
Comparisons								
RFxDC	U							
	P	NS	NS	NS	NS	NS	NS	NS
RFxSh	U	0	9½			2	6½	
	P	<.01	<.02	NS	NS	<.01	<.01	=.06
DCxSh	U	2	4	1			9	
	P	<.05	<.01	=.06	NS	NS	<.05	NS
(RF+DC) X Sh	U	2	13½			7	15½	
	P	<.005	<.001	NS	NS	<.02	<.01	=.08

Md - median, IQR - Interquartile range, U - Mann-Whitney U corrected for ties, p - probability.

damaged rats ($p = .08$; when only the RF group is considered, $p = .06$).

Results on passive avoidance and escape training are shown in Table 7. The brain damaged rats needed a greater number of trials with shaping (e.g. trials on which the animals took more than 30 sec. to find an island and was then shaped) before learning (defined as 5 successive trials with a latency less than 30 sec.) to find the island than did control rats ($p < .005$). There was considerable variability between animals which was influenced by the prior escape test during which a few animals learned spontaneously to find the island. However, if only those animals which did not learn to find the island during the prior escape test are considered, the ones with brain damage still required more trials than control animals ($p < .02$ for both brain damaged groups combined). In addition to requiring more trials to reach criterion, the brain damaged rats were slower to escape on the criterion trials ($p < .005$). However, there was no difference in the number of trials required to learn to freeze when put on either the large (6 in.) or the small (2½ in.) island.

Movement Inhibition Test

On the two one-hour tests of movement inhibition, the brain damaged rats were only slightly inferior to the control rats (see Table 8). Both groups were slightly inferior on the first test ($p < .05$); on the second test the

Table 7. Passive avoidance and escape training.

Group	Measure	No. shaping trials to criterion of escape training			Mean escape latency (sec.) on criterion trials			Trials to criterion on passive avoidance		
		6 in. island	2½ in. island	Total	6 in. island: last 3 trials	2½ in. island: last 3 trials	Total: all 10 trials	6 in. island	2½ in. island	Total
RF	Md	7	0	9	8½	11½	10	½	1	9½
	IQR	4-7	0-8	7-10	6-11	6-18	9-13	0-3	0-3	1-5
DC	Md	2	2	9	11	9	10	1	1	2
	IQR	1-9	0-4	3-18	6-14	3-11	7-13	1-2	1-2	1-4
Sh	Md	1	1	3	2	3	5	0	1	4
	IQR	0-2	1-2	2-3	2-6	2-6	4-6	0-3	0-4	1-6
Comparison										
RFxDC	U									
	P	NS	NS	NS	NS	NS	NS	NS	NS	NS
RFxSh	U	7		6	8½	7	1			
	P	<.005	NS	<.005	<.01	<.005	<.0005	NS	NS	NS
DCxSh	U			19	9½	16	7			
	P	NS	NS	<.05	<.01	<.05	<.005	NS	NS	NS
(RF+DC) X Sh	U	29½		25	18	23	8			
	P	<.01	NS	<.005	<.005	<.005	<.0005	NS	NS	NS

Md - median, IQR - interquartile range, U - Mann-Whitney U corrected for ties, p - probability

Table 8. Movement inhibition performance.

Group	Measure	Day 1	Day 2	Total
RF	Md	3	3.5	7.5
	IQR	1=7	2-6	4-17
DC	Md	4	1	4
	IQR	3-6	0-1	3-12
Sh	Md	1	1	4
	IQR	1-2	0-4	2-5
Comparisons				
RF X DC		NS	23, $p < .05$	NS
RF X Sh		19.5, $p = .06$	18.5, $p < .05$	19, $p = .05$
DC X Sh		15, $p < .05$	NS	NS
(RF+DC) X Sh		34.5, $p < .05$	NS	NS

Md - median, IQR - interquartile range,
p - probability (based on Mann-Whitney U)

Rf group was inferior to both the DC and the control groups ($p < .05$) but there was no significant difference between the last two groups.

Jump Avoidance Conditioning

As Table 9 shows, septal damaged rats make more errors in learning to avoid shock by jumping ($p < .0005$) and the mean latency they eventually reach on the ten criterion trials is also longer ($p < .001$). In addition they are somewhat less consistent (e.g. they make more errors after the first avoidance response, $p < .05$) but they do not

Table 9. Jump avoidance performance.

Group	Measure	Errors	Latency on criterion trials	Errors after first avoidance	Trial 1 escape latency (in sec.)
RF	Md	5.5	4.5	.5	18
	IQR	1-15	3-6	0-1	9-36
DC	Md	5	5	1	57
	IQR	4-12	4-7	0-3	12-104
Sh	Md	1	2	0	43
	IQR	0-2	2-3	0	16-61
Comparisons					
RF X DC		NS	NS	NS	NS
RF X Sh		9.5, $p < .01$	10.5, $p < .01$	17.5, $p^* < .05$	NS
DC X Sh		0.5, $p < .0005$	6, $p < .005$	17.5, $p^* < .05$	NS
(RF+DC) X Sh		10, $p < .0005$	16.5, $p < .001$	35, $p^* < .05$	NS

Md - median, IQR - interquartile range, p - probability (based on Mann-Whitney U)

differ significantly in the latency to escape on the first trial. There was also no meaningful difference in the latency to escape on later trials (the only difference was in the number of escapes requiring more than 2 sec., but since the control rats made so few escapes, this is not a meaningful difference).

Weight Gain

Control rats gained about 1.8 g. per day compared with a gain of 1.4 g. in the operated rats ($p < .05$). The variation is shown in Table 10.

Table 10. Weight gain per day.

Group	Measure	Gain (in gm.)
RF	Md	1.5
	IQR	1.4-1.7
DC	Md	1.4
	IQR	1.3-1.8
Sh	Md	1.8
	IQR	1.6-2.3

Comparisons

RF X DC	NS
RF X Sh	16, $p < .05$
DC X Sh	21, $p = .06$
(RF+DC) X Sh	37, $p < .05$

Md - median, IQR - interquartile range,
$\bar{p}$ - probability (based on Mann-Whitney U)

Interrelations Between Behaviors

The correlations between specific behaviors for each of the three groups are listed in Table 11. Considering the patterns of intercorrelations of one measure with all the others, the measures with the most dissimilar patterns in the two brain damaged groups were the two measures of movement inhibition (MI1 and MI2). As a descriptive indication of this similarity, the rhos between the two groups (correlating the correlations) were -.18 and -.26 respectively. This difference was most striking in the

Table 11. Correlations between 16 different measures for control, RF, and DC groups (correlations multiplied by 100).

Abbreviations	Measure
E1	Emotionality ratings--Day 14
E2	Emotionality ratings--Day 16
G	Grooming during exploration test
EX	Walking, turning, and rearing during exploration
DIS	Distance traveled during exploration
IN	Inactivity (sniffing, lying and freezing) during exploration
OFQ	Frequency of entering compartment during exploration
OFL	Latency to reach compartment during exploration
SH6	Escape shaping trials--large island
SH2.5	Escape shaping trials--small island
ESL6	Escape latency--large island
ESL2.5	Escape latency--small island
MI1	Movement inhibition errors--Day 1
MI2	Movement inhibition errors--Day 2
JAE	Jump avoidance errors (trials with shock)
JAL	Latency of JA response on criterion trials
MVL	Movement latency during exploration (RF and DC groups only)

Table 11. (Contd.)

Sh Group

	E1	E2	G	EX	DIS	IN	OFQ	OFL	SH 6	SH 2.5	ESL 6	ESL 2.5	MI1	MI2	JAE	JAL
E2	-23															
G	36	22														
EX	-01	-23	-30													
DIS	-36	15	-41	71												
IN	01	31	16	-96	-75											
OFQ	-48	-46	-53	79	71	-82										
OFL	21	-16	-16	37	68	-45	40									
SH6	-38	43	23	-76	-68	77	-61	77								
SH2.5	-33	-59	-25	35	-08	34	-62	34	-16							
ESL6	-34	68	48	09	14	04	-05	04	29	-33						
ESL2.5	-03	50	-36	-07	44	12	-18	12	-15	77	48					
MI1	21	38	11	-69	-16	70	-69	70	24	-79	03	70				
MI2	22	-52	63	-02	23	-11	05	-11	04	43	-80	-81	-37			
JAE	-82	-18	-20	-07	18	-01	40	-01	40	33	09	-27	-32	25		
JAL	-29	63	-34	-25	16	34	-32	34	39	-71	80	78	52	-74	13	

Table 11. (Contd.)

RF Group

	E1	E2	G	EX	DIS	IN	OFQ	OFL	SH 6	SH 2.5	ESL 6	ESL 2.5	MI1	MI2	JAE	JAL
E2	52															
G	-63	-78														
EX	-31	-37	44													
DIS	-01	-10	32	82												
IN	45	46	-64	-93	-72											
OFQ	-45	-74	48	65	43	-64										
OFL	48	80	-64	-56	-35	64	-94									
SH6	-10	57	-34	-16	04	21	-27	36								
SH2.5	10	15	-18	-39	-28	22	-21	25	-21							
ESL6	14	37	-11	06	12	03	-32	25	11	27						
ESL2.5	26	63	-55	-08	07	24	-38	53	59	-43	-30					
MI1	68	32	-40	18	15	-01	-24	-53	-30	-30	-05	40				
MI2	60	-14	-17	35	36	-20	23	-06	35	-15	-04	-08	63			
JAE	-40	20	-15	27	-13	31	00	13	46	24	06	17	-66	-73		
JAL	10	-16	06	03	09	-18	13	-01	-57	78	-05	40	-41	15	-04	
MVL	-18	52	-36	-51	-39	52	-63	73	47	23	04	50	-26	-67	65	-08

Table 11. (Contd.)

DC Group

	E1	E2	G	EX	DIS	IN	OFQ	OFL	SH 6	SH 2.5	ESL 6	ESL 2.5	MI1	MI2	JAE	JAL
E2	64															
G	16	-86														
EX	-12	04	-38													
DIS	-12	-40	38	32												
IN	-10	55	-32	-70	-56											
OFQ	-20	-40	39	28	97	-59										
OFL	05	09	28	-60	-27	55	-30									
SH6	-02	04	12	-30	-34	13	-22	-15								
SH2.5	-22	-10	07	24	21	-48	33	-47	-10							
ESL6	-48	-22	12	-16	-38	12	-33	48	-01	-24						
ESL2.5	-23	32	57	-36	01	07	04	66	20	-22	32					
MI1	-19	13	-10	-42	-65	56	-66	-11	18	-59	77	28				
MI2	-47	47	-66	41	-44	17	-45	-17	19	-26	27	-24	33			
JAE	-30	40	-44	03	-73	39	-72	21	05	-24	59	17	74	68		
JAL	-65	-54	53	-03	30	-29	42	15	39	-06	40	57	06	01	-08	
MVL	-23	41	-11	-64	-65	75	-58	63	15	-32	50	44	63	19	56	-01

relations between MI and the number of errors on the jump-avoidance task (JA). For the RF group the correlations were approximately -.70 and for the DC group they were approximately .70 (both correlations are significant at close to the .01 level). This means that in the RF group the faster a rat learned JA, the more errors it made on MI. In the DC group the faster a rat learned the JA, the less errors it made on MI.

The measures with the most similar correlations in the two brain damaged groups were inactivity (IN) and movement latency on the second, third and fourth test days (MVL) in the open field. As a descriptive indication of this similarity, if the two MI measures are excluded, the rhos were .88 and .86, respectively (including the MI tests they were .67 and .61 respectively. Thus, the MI tests were related to the other tests in a way that was different in the RF group from what it was in the DC group, while IN and MVL were related to the other measures in similar ways in both groups.

When comparing these patterns to those of the control animals there was little similarity between the interrelations of the MI tests of either brain damaged group and those of control animals. There were also only low positive relations between the IN pattern of the brain damaged groups and that of the control group (rho .34 for the RF group and .41 for the DC group; excluding the MI tests the rhos are

.31 and .35, respectively). There was no variation in MVL of the control group, so it was meaningless to correlate it with the other measures. Somewhat surprisingly, however, the pattern of correlations for the IN and MVL tests of the brain damaged groups were both highly related to the pattern of correlations for the number of shaping trials needed to escape to the large island (SH6) needed in the control group. In fact when MI1 and MI2 are excluded, the pattern of correlations of SH6 in the control group is correlated .90 and .85 with the pattern of correlations of MVL in the DC and RF groups, respectively. Thus, IN and MVL during exploration in the brain damaged groups are more similar to SH6 in the control group than they are to IN and MVL during exploration in the control group. In addition the SH6 pattern is dissimilar in all three groups.

Observations of actual behavior suggest an interpretation of the similarity between SH6 in the control group and MVL in the operated groups. Since there is piloerection and diarrhea during the initial immobile period before exploration in the operated groups, MVL measures freezing. Control animals did not show this pattern of behavior during exploration but did when shock was present. Further evidence that open-field immobility in septal damaged and SH6 in normal rats activate similar processes is that backwards circling appeared as the first movement in the open field in several operated animals (circling was the first movement

recorded in 3 rats from the DC group and 4 from the RF group) but was not observed in the normal animals except when footshock was present. Both freezing and backwards circling suggest a defective ability to make directed or voluntary locomotor movements. Thus, the subsequent description of results will be based on the interpretation that SH6 in control rats and MVL in septal damaged rats are measures of an inability to make directed locomotor movements.

The inability to make directed locomotor movements was associated with the second test of emotionality (E2), the five measures of exploratory behavior and JA. The correlations with E2 in the brain damaged groups accounted for only about 20% of the variance and were not quite significant. However, with both brain damaged groups combined to produce a larger sample the correlation was .64 (accounting for about 40% of the variance and significant with $p < .002$). In addition considering that the scale was not based on equal units,* such a correlation strongly suggests a definite relationship. The inability to make directed locomotor movements was correlated negatively with MI in the RF group and positively with MI in the DC group. In the brain damaged groups the pattern of relations of MI were rather similar

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*There is no reason to suspect that vocalization three different times during the test is an indication of exactly the same amount of emotionality as jumping to a puff of air--to achieve even an indication of equality of units a much larger N would be needed.

on both days but the two patterns were quite different in the control group. On the first day MI was highly positively correlated with the latencies to escape to the small island during criterion trials (ESL2½) and latency of the jump response during criterion trials of jump avoidance conditioning (JACL) and on the second day MI was highly negatively related to ESL2½ and JACL. On neither day did MI show a high correlation with SH6 (the measure of the ability to make directed movements).

The first test of emotionality (E1) showed only moderate relations with the other variables except in the RF group where it was negatively related to grooming during exploration (GR) and positively related to MI. The amount of GR was closely related (but negatively) to E2 in both brain damaged groups (rho=-.78 and -.86 in the RF and DC groups, respectively). Also ESL was closely related to the time to find the compartment during exploration (OFL) in both groups with brain damage.

Summary of Behavioral Results

There was no detectable difference in water consumption. The emotionality of the rats with RF lesions was rated higher than that of rats with DC lesions which in turn was rated higher than that of the control rats. The emotionality results were based on a new scale that was correlated with but more reliable than the one (by Brady and Nauta, 1953) commonly used. In the open field rats with septal

damage show a greater amount of initial freezing and walk about more slowly and less frequently after they begin exploring. In addition rats with RF lesions are more seriously affected on these measures than those with DC lesions. They also show less grooming than rats with DC lesions but there was no detectable difference between the grooming of the DC group and that of the control group.

Even though brain damaged rats made escape responses at more than twice the rate of control rats during an escape test, a somewhat greater percentage of control rats learned spontaneously to escape to the island. In addition during subsequent escape training (which was preceded by passive avoidance training in which there were no detectable differences between groups in the number of training trials needed before an animal climbed and froze on an island for 60 sec.), the septal damaged animals needed more trials to learn to escape to the island than did the control rats. Furthermore, even after this extra training the septal damaged rats made more errors during two one-hour tests of movement inhibition (staying on the island) than did control rats. The brain damaged rats were much worse than the control rats at learning to avoid shock by jumping to a platform. Finally there was a moderate but significant deficit in the amount of weight gained per day in the brain damaged groups.

In an analysis of the interrelations between 16 of the measures for each of the three groups it was found that the most dissimilar patterns of interrelations involved the movement inhibition test. In the RF group it was highly negatively related to jump avoidance errors while in the DC group the two measures were positively correlated. Movement latency on the last three days of exploratory testing in the two brain damaged groups and the number of shaping trials needed to learn to escape from shock to a safe island in an open field in the control group showed similar patterns of relation to other tests. Based on behavioral observations as well as the pattern of correlations, it was concluded that these measures with similar patterns were measures of the ability to make directed locomotor movements. This ability was rather highly associated with more than half of the measures analyzed in the three groups. Even though emotionality after prior handling was only moderately related to the ability to make directed locomotor movements this finding is notable because there have not been previous reports of correlations between hyperemotionality. In addition there were very high correlations between emotionality and grooming in the brain damaged groups.

Histological Study with Behavior Correlates

All animals in the DC group had bilateral damage to both the medial and lateral septal nuclei and to both the anterior and posterior portions of the septum, except one

animal which had only unilateral damage to the lateral nuclei. All animals in this group also showed some expansion of the lateral ventricles at and posterior to the level of the hippocampal commissure (hydrocephalus). The animal with unilateral damage to the lateral nucleus received a relatively high emotionality rating especially on the first test, but also made few errors on MI, many exploratory responses in the open field, and had low latencies to move, to find the wall, and to find the compartment in the open field. Its performance on other measures was about average for the group. Only one animal had damage in the medial portion of the anterior caudate nucleus and this was unilateral. Its behavior was not noticeably different from other members of the DC group. Four animals received some damage to the preoptic area although this damage was unilateral in two of these and confined to the most anterior portions in a third. The remaining animal had damage to the preoptic area on both sides of the midline. This animal showed the best JA performance of the DC group and was tied with three others for the lowest MVL of that group.

There was bilateral damage to the medial, lateral, anterior and posterior portions of the septal area in all animals in the RF group. All had expanded lateral ventricles at and posterior to the level of the hippocampal commissure and some damage to the anterior part of the caudate nucleus, although this damage was unilateral in four animals,

confined to the dorsomedial portion bilaterally in two others and confined to the medial portions bilaterally in the remaining four. In addition one of these animals with bilateral damage had damage to the medial frontal cortex just anterior to the genu of the corpus callosum. This animal showed the least exploratory behavior and the worst performance on JA of the group. However, the overall correlation of caudate damage with JA was only .08 and with MI it was only .18. One effect that might be expected to result from damage to the caudate nucleus or internal capsule fibres is the backwards circling found during exploration. However, since this was present in nearly as large a percentage of the DC group as of the RF group, it cannot be accounted for by such damage. Seven of the animals (excluding the one with frontal cortical damage and two others) had damage in the preoptic area. This damage was bilateral and fairly extensive (though not symmetrical) in six cases. though it was confined to the dorsal portion in two of these. The seventh had damage to the anterior portion only. There was a rank-order correlation (Spearman's, corrected for ties) of -.76 ($p < .01$ with a two-tailed test) between the extent of preoptic damage and JA errors. In addition preoptic damage was correlated -.45 with MVL (the three animals without preoptic damage had the greatest MVLs in the group but the three with damage confined to the dorsal or anterior

portions had lower MVLs than those with more extensive damage), and +.52 with MI1 and MI2 combined.

In conclusion, both groups showed total damage to the septal area in nearly all Ss. However, there was considerably more damage outside of the septal area in the RF group. This extra-septal damage involved the anterior caudate nucleus, the preoptic area and in one S the medial frontal cortex. Relations between the histological and behavioral evidence suggests that the differences between the RF and DC groups were due to the preoptic damage in the RF group. In particular the more preoptic damage in combination with septal damage an animal had, the fewer errors it made on JA and the more errors it made on MI.

CHAPTER 3

EXPERIMENT 2

Experiment 1 found a surprising high negative correlation between the amount of preoptic area, brain damage added to septal area damage and errors during jump avoidance conditioning, but there was a small positive correlation between additional preoptic damage and errors on a movement inhibition task. The present experiment was designed to provide an independent replication of these effects as well as to include controls for experimenter and order effects and for variability in brain damage. Thus, even though no correlation between caudate or cortical damage and the behaviors measured was found in the last study, groups with damage in either one of those areas without preoptic damage were included in this study.

Method

Subjects

The Ss were 93* male albino (Sprague-Dawley) rats

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* The actual number was considerably larger before the Ns were reduced by deaths (up to 60% in one group) and lesion variability.

from Woodlyn Farms, Ontario, weighing 250-350g. at the time of the operations. Testing was done between 10 and 20 da. after the operations.

Surgery and Histological Study

Surgical procedures were similar to those used in Experiment 1 except for electrode placements and for the use of electrolysis in all cases (with 2 ma. DC for 15 sec.). The coordinates for the septal lesions were the same as those in the previous experiment except that the electrode was lowered from 2.5 mm. anterior to bregma and at an angle of 7° to the coronal plane (as well as at an angle of 14° to the sagittal plane). This resulted in the same tip position as in the previous experiment. Preoptic lesions were made by the use of the same coordinates as the septal lesions except that the electrodes were lowered an additional 3 mm. The change in coordinates for the septal lesions was made so that combined preoptic-septal lesions could be made with only one drill hole and dura puncture on each side of the midline. The septal-frontal cortical lesions were made by adding a lesion 2 mm. anterior to the septal lesion and the caudate-septal lesions were made by adding a lesion 1 mm. lateral and $2\frac{1}{2}$ mm. dorsal to each side of the septal lesion.

Histological procedures were the same as the previous experiment except that the tissue was stained with thionin.

Movement Inhibition Test

The movement inhibition (MI) apparatus was the same as that used in the previous experiment except that the small ($2\frac{1}{2}$ in.) island was placed adjacent to the center of one wall. Under these conditions the majority of the brain damaged animals could learn to escape to the island within 1 hr. without being shaped by the experimenter. Therefore, testing in this apparatus was the same as in the previous experiment except that no escape testing or training was used.

Active Avoidance Learning

The method used for jump avoidance (JA) conditioning was the same as in the previous experiment except that the animals were allowed only 5 sec. to avoid the shock. In some animals JA preceded MI and in others MI preceded JA. In both cases JA was preceded by 15 min. exploration of the JA apparatus but MI was not preceded by exploration. Table 12 presents the number of animals per group and the order of testing.

Statistical Methods

The principal results were analyzed by computing the % time off the island in the MI tests and the number of JA errors divided by 30 (which is the same as the % of JA errors if it is assumed that all trials after reaching the criterion of 9 out of 10 errorless trials would have been errorless if tested to 30 trials). The following groups

Table 12. Groups, Ns and testing order.

Group	N	Group	N
C1	10	U2	4
C2	6	FS2	6
P1	10	CDS2	4
P2	6	CDS1	4
S1	10		
S2	10		
PS1	9		
PS2	14		

C - sham operated controls
P - preoptic damage
S - septal damage
F - frontal cortical damage
CD - caudate nucleus damage
U - unilateral septal damage
1 - JA presented first
2 - JA presented second
combined letters - damage in both structures indicated

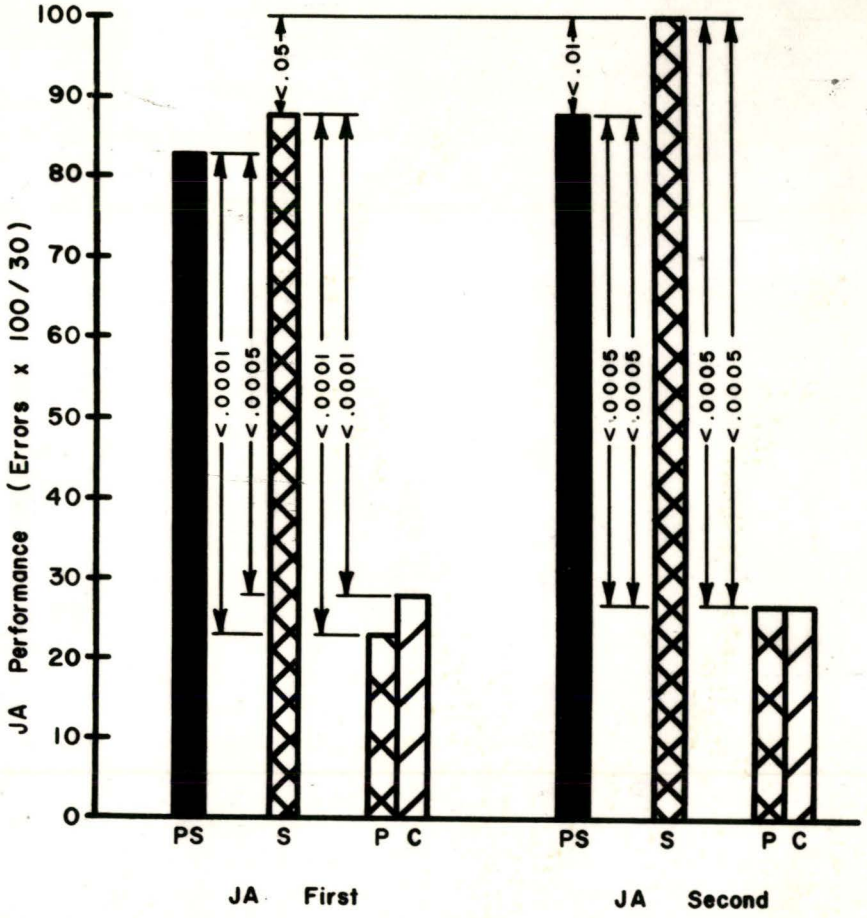
were excluded from this analysis either because both orders were not tested or because the Ns were small: FS2, CDS2, CDS1, and US2. These groups were compared with the others by means of the Mann-Whitney U. The analysis was also done with the scores normalized by means of the arcsine transformation. The latter analysis gave the same probability levels as the former indicating that the analysis of variance is robust with respect to the particular deviations from normality found in this study. As a second check, paired comparisons were based on the Mann-Whitney U and the probability levels determined from that test were also reported.

Results

Movement Inhibition and Jump Avoidance Performance

Table 13 and Fig. 2 present the means of the JA and MI scores used for the principal analysis. Table 14 presents the analysis of variance based on these means. The analysis of variance revealed large ($p < .001$) group X test and order X test interactions and a moderate triple interaction ($P = .06$). As in the previous experiment, S2 showed poorer JA performance than PS2 ($p < .005$), and both operated groups were worse than C2 ($p < .0005$). In addition both of the septal damaged groups were worse than P2 ($p < .0005$). The difference between the groups with septal damage (PS2 and S2) was apparently due to the detrimental effect ($p < .05$) of prior experience with MI on the JA performance of septal damaged animals. This order effect was not present in the P or C groups and was obliterated by adding preoptic damage to the septal damage. Although prior experience with MI hindered or did not affect JA performance, prior experience with JA improved MI performance in all three brain damaged groups ($p < .05$ or less). Testing order had much less effect on the MI performance of the controls than it did on the brain damaged groups and it was somewhat less effective for the PS group than for the S or P group. The PS group was also the only group that scored higher ($P = .001$) than control Ss on MI when both testing orders were combined.

Fig. 2. Jump avoidance (JA) and movement inhibition (MI) performance for groups of animals with preoptic and septal (PS), septal (S), preoptic (P) or sham (C) brain damage with all significant differences (based on Mann-Whitney U) shown.


JA Performance (Errors x 100/30)
100
90
80
70
60
50
40
30
20
10
0
p<.0001
p<.0005
p<.05
p<.0001
p<.0001
p<.01
p<.0005
p<.0005
p<.0005
p<.0005
PS
S
P
C
PS
S
P
C
JA First
JA Second

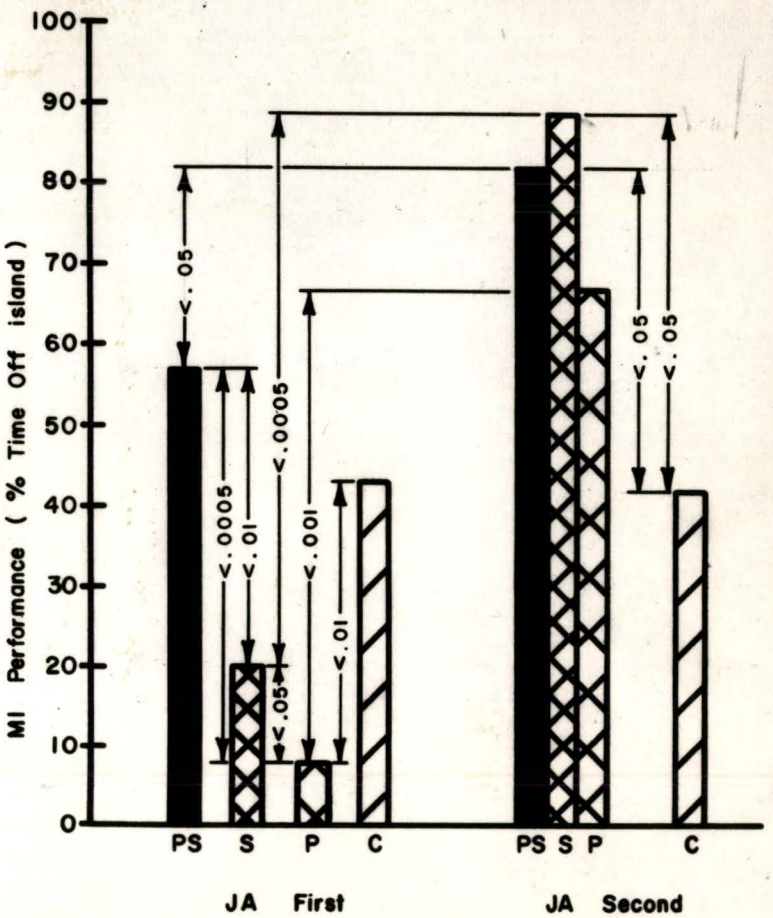

MI Performance (% Time Off island)
100
90
80
70
60
50
40
30
20
10
0
p<.05
p<.0005
p<.01
p<.05
p<.0005
p<.001
p<.01
p<.05
p<.05
PS
S
P
C
PS
S
P
C
JA First
JA Second

Table 13.

Mean JA and MI performance.

Group	JA (errors X $\frac{100}{30}$)		MI (% time off the island)	
	JA First	JA Second	JA First	JA Second
C	31	28	43	42
P	26	29	8	67
S	82	96	20	89
PS	79	82	57	82

See Table 12 for group designations.

Table 14.

Analysis of variance of JA and MI performance.

SOURCE	DF	MS	F	P
Between Ss				
Groups (G)	3	17876	35.47	.001
Order (O)	1	15556	30.87	.001
G X O	3	367	1.00	NS
Error	67	504		
Within Ss				
Test (T)	1	1131	1.95	NS
G X T	3	4240	7.32	.001
O X T	1	10292	17.78	.001
G X O X T	3	1494	2.58	.06
Error	67	579		

Table 15 presents the means and medians of JA and MI performance for U2, FS2, CDS2, and CDS1 and shows the comparisons of these with other groups that were tested in the same order. The unilateral septal damage produced MI and JA performance that was very similar to that of the C and P groups. The FS2, CDS2 and CDS1 groups were not detectibly different from the other groups with bilateral septal damage (S and PS), except that like the S2 group, FS2 and CDS2 performed worse on JA than PS2.

In summary, prior MI experience is detrimental to JA performance in animals with septal lesions (group S) but not animals with combined septal and preoptic lesions (group PS). In addition, lesions of the preoptic area only (P) produced no detectable effect and both groups (S and PS) with septal damage were much worse than both the P group and the control (C) group. On the other hand, MI performance was improved by prior JA experience in all the brain damaged groups but not in control rats. In addition when MI was presented first the groups with septal damage performed worse than the control group. Finally, it should be noted that except for the refinement of the testing procedures and the lesion accuracy, the S2, PS2 and C2 groups were a replication of Experiment 1. The comparisons between these three groups produced differences that were in total agreement between the earlier experiment and this one.

Table 15.

JA and MI performance of U2, FS2, CDS1 and CDS2 groups compared with other groups tested in the same order (see Table 12 for group designations and Table 13 for means not listed here). P (based on Mann-Whitney U) less than value given unless otherwise stated.

Group	JA (Errors x $\frac{100}{30}$)				MI (% time off island)			
	U2	FS2	CDS2	CDS1	U2	FS2	CDS2	CDS1
C	NS	.01	.05	.001	NS	.05	NS	NS
P	NS	.0005	.005	.001	NS	NS	NS	.01
S	.001	NS	NS	.05	.05	NS	NS	NS
PS	.001	.005	.005	.05	=.06	NS	NS	NS
U2		=.005	=.01			=.06	NS	
CDS2		NS		NS		NS		NS
Median	13	100	100	100	63	99	47	97
Mean	20	100	100	100	63	87	49	84

Histological Study

All animals with septal damage (except the US group) received nearly total damage to both the medial and lateral septal nuclei at the middle and anterior levels (at and anterior to the level of the anterior commissure) and in all but 9 of these animals (3 in the S group, 4 in the PS group, 2 in the CS group and 2 in the FS group), there was extensive damage to the posterior septal area as well. In only one of the animals in the S group was there any preoptic

damage and this was very slight. The only additional damage in the S group was some slight (usually unilateral) damage to the cingulate cortex overlying the septal area in 7 of the animals. In addition hydrocephalus was either very slight or non-existent in all Ss.* A typical septal lesion is shown in Fig. 3.

In contrast to the septal lesions the preoptic lesions did not show bilateral damage except in a few cases where the lesions were quite small. Very likely bilateral preoptic damage accounted for the high mortality rate (above 50%) of animals with preoptic damage (in both the P and PS groups) and this acted to select the animals with unilateral damage. In addition to preoptic damage, in some cases the anterior hypothalamic nucleus was also partially damaged on one side of the midline. An example of a preoptic lesion is shown in Fig. 4. The animals in the PS group showed the same pattern of damage as those in the P and those in the S groups combined.

The CS lesions were confined to septal damage plus damage to a small, dorsomedial portion of that structure and to some of the overlying cortex. The FS lesions damaged the medial portions of the cortex anterior to the genu of the corpus callosum and in most cases showed some damage to the medial cingulate cortex overlying the septal area. The

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*Therefore, since the findings of the first and second experiments were compatible, there was no detectable effect of the hydrocephalus.

Fig. 3. A typical septal lesion.

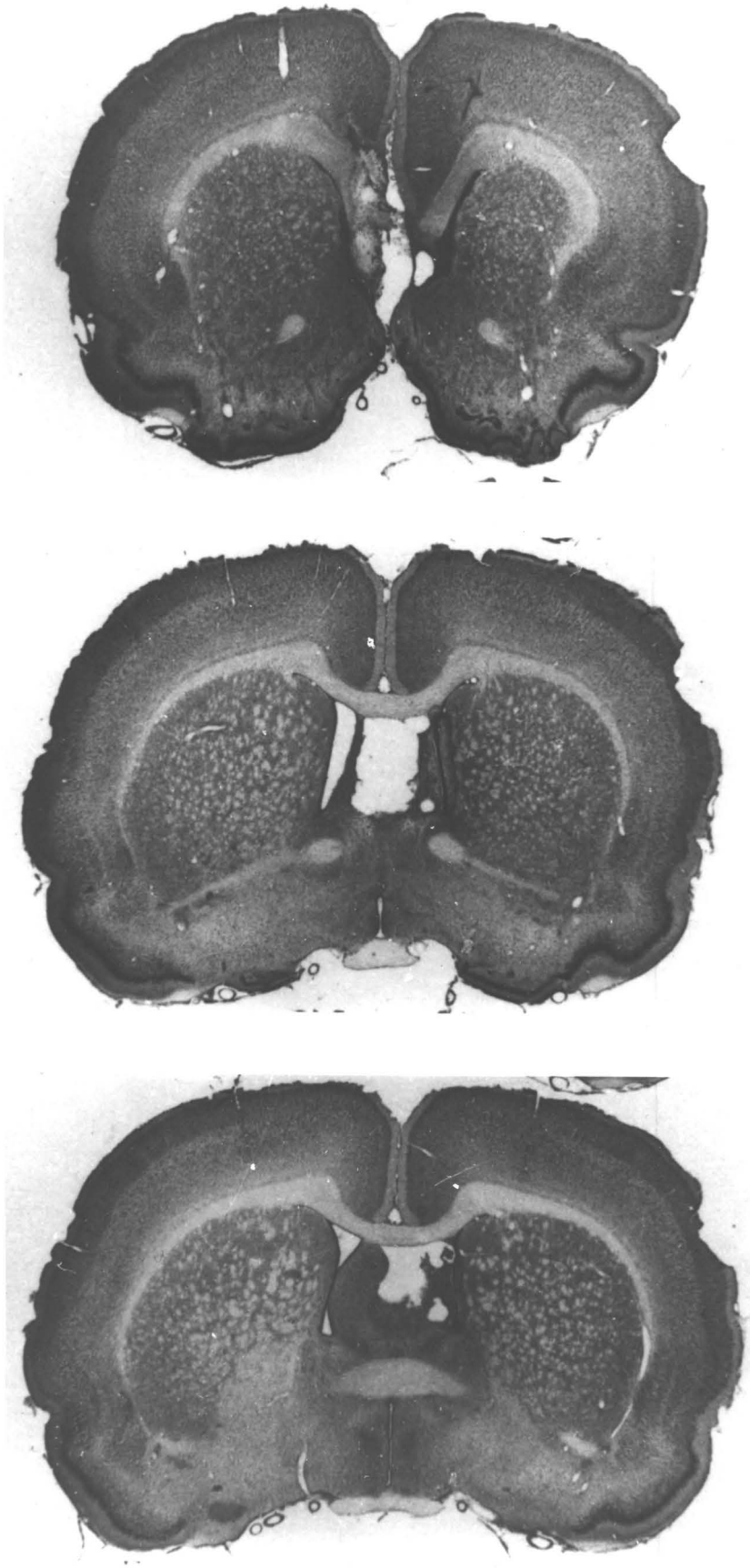
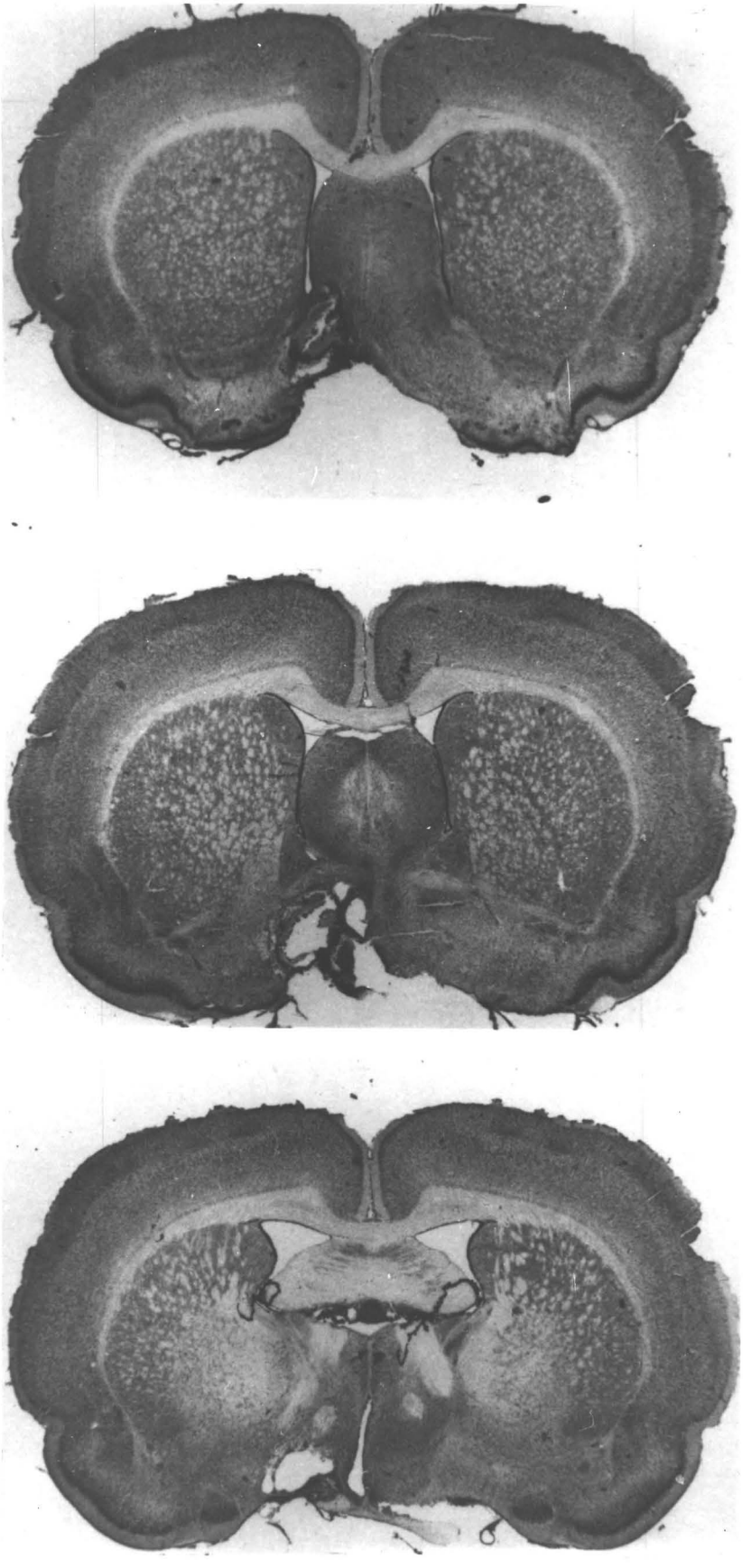


Fig. 4. A typical preoptic lesion.



unilateral septal lesions produced damage only to part of the lateral septal nucleus on one side of the midline.

CHAPTER 4

EXPERIMENT 3

The third experiment was designed to test whether the impaired JA performance due to septal damage in rats could be due to an aversion to light such as that found by Schwarzbaum et al. (1967). The rationale is that since overhead lighting was used in the previous experiments the JA deficit could be due to a conflict on the part of septal damaged rats between avoiding the shock and avoiding (jumping toward) the light. If this were the case, lighting the JA apparatus from below should improve the performance of these animals.

Method

The Ss were 13 naive albino rats similar to those in the second experiment. Five of these made up the control group and eight the experimental group. The surgical and histological procedures were the same as those for the appropriate groups in the previous experiment except that the tissue was stained with cresyl violet. The JA training technique was also the same as that used in the second

experiment except that testing was conducted in a sound-proof and light-proof cubicle with the only source of light placed beneath the JA apparatus. It illuminated the grid with approximately 7 FC. To permit comparisons with the previous experiment, performance was analyzed in the same way as in that experiment (performance = errors X $\frac{100}{30}$.

Results

Jump Avoidance Performance

The mean JA performance for the present experiment are shown in comparison with the C1 and S. groups of experiment 2 in Table 16. There was no apparent difference between the C groups of the two experiments or between the S groups. But like experiment 2, in this experiment there was also a significant difference between the C and S groups (Mann-Whitney U=1, p=.002).

Table 16. Mean JA performance of C and S groups of experiments 2 and 3.

Experiment	Control (C) Group	N	Septal (S) Group	N
3	30.8	5	79.1	8
2	30.9	6	82.4	10

Histological Study

The septal damage in this experiment was very similar to that found in Experiment 2. The only notable exception was one animal with a fairly large hemorrhage in the area of the septal nuclei that caused extension of one of the lateral ventricles posterior to the level of the hippocampal commissure. The performance of this S was the second poorest of the group (without this S, the group mean JA performance would be 77.1 instead of 79.1).

CHAPTER 5

DISCUSSION

In general the results indicate that in a variety of tasks the behavioral effectiveness of rats with septal damage is markedly reduced. This is true for exploratory behavior as well as for learning to escape or avoid shock. This lack of effectiveness appeared to be due to a marked deficiency in the ability to make appropriately directed movements. Thus, during exploration rats with septal damage take longer than normal rats to find the wall of an open field even though they showed a much greater tendency to remain at the wall once they found it. They also took a longer than normal time after approaching the wall to find a small compartment adjacent to the wall, even though they also showed a much greater tendency to remain in the compartment than normal rats. During escape learning rats with septal damage ran or jumped four times as often as normal rats, but fewer of the brain damaged animals actually escaped shock by running to a safe "island" on the grid. Even after the appropriate escape response was learned by the septal damaged animals, they made more errors (left

the island more frequently) than control animals in two one-hour tests of movement inhibition. Furthermore, although rats with septal damage escaped shock as readily as normal rats by jumping out of a 1-ft. deep box, they were much slower to learn to avoid the shock. In fact if the latency of the shock (CS-US interval) was reduced from 15 to 5 sec. (the latter was used in experiments 2 and 3), several septal-damaged rats were unable to make any avoidance responses during the 30 trial test. The effect on avoidance learning could not be due to such post-trial effects as handling because instead of returning the animals to the grid by hand they were returned by means of a small box which the septal-damaged rats entered readily. It was also not due to an aversion to light because lighting the apparatus from beneath the grid did not improve the performance of the rats with septal damage (Experiment 3). In addition the effect was exaggerated by prior movement inhibition experience (i.e. there was negative transfer) as long as there was no preoptic damage combined with the septal damage. The control rats and the rats with preoptic damage also did not show the negative transfer.

The results also indicate that a single generalization concerning the effects of septal lesions may be appropriate for a wide variety of behavioral effects but would not explain all the effects of these lesions. For example, although the inability to make directed locomotor movements

was related to hyperemotionality after handling, exploration, and escape and avoidance learning, it was not related to the amount of weight gained per day or to the amount of hyperemotionality without prior handling.

Three possible reasons for the inability to make directed movements will be considered. First, Mc Cleary (1961) has suggested that septal damaged rats may not be able to inhibit responses. This generalization was previously discussed by me in some detail (Dirlam, 1967, pp. 2-9 and 58-65). Briefly, the generalization was found to be appealing because it is consistent with the following findings: (1) that some nerve cells may inhibit others, (2) that stimulation of some portions of the septal area inhibits some reflexes (Hodes et al., 1951; Kaada, 1960), and (3) that according to Mc Cleary's own well-conceived study of septal damage (1961), lesions may impair learning to inhibit a passive response and facilitate learning to make an active response. On the other hand, the generalization was found to be inadequate because it is inconsistent with the following findings: (1) that some active responses are incompatible with others (e.g. see Bindra, 1961) and therefore all active responses could not simultaneously be facilitated, and consequently it would be impossible to predict how the loss of response inhibition would affect specific behaviors in given situations, (2) that septal stimulation facilitates some reflexes (Peacock and Hodes, 1951), and (3) that some active behaviors are impaired by septal damage (Dirlam,

1967; Kenyon, 1962). The present findings provide additional support for the third inadequacy of the generalization that the septal area is inhibitory. Damage to an inhibitory structure would not be expected to cause the long movement latencies before exploring the open field that were found in the septal-damaged animals of the present study. In addition damage to such a structure would not be expected to impair the ability of an animal to move to a safe place as septal damage did in the present study.

In a later article by Mc Cleary (1966), the generalization about septal functions was revised. According to the revision, the septal area inhibited only the most probable response in a given situation. Thus, septal damage would result in the facilitation only of the most probable response in the situation and a less probable response, that was incompatible with the first, might actually be suppressed by septal damage. Therefore, the first inadequacy of the loss-of-response-inhibition generalization does not apply to this revision. Furthermore, with additional evidence (see Mc Cleary, 1966), the other inadequacies of the earlier generalization were mitigated. However, the revision produced new difficulties. First, the simplicity of the earlier generalization was lost: although the neural organization necessary for a structure to inhibit active responding is clear (all connections between septal cells and any others involved in the production of active behavior would be inhibitory), the organization necessary for a structure to

inhibit only those cells involved in the production of highly probable responses is not clear. Secondly, although there is much evidence about septal functions that is consistent with the revised generalization (see Mc Cleary, 1966), there is some evidence that is definitely inconsistent with it. For example, grooming by rats within the first 20 min. of the first exposure to a four-arm, plus (+) maze is definitely not the most probable response for normal rats but it is the most probable response for septal-damaged rats (Dirlam, 1969). Furthermore, in the present study the first response of normal animals when placed in the center of an open field was to move toward the wall. An animal that has lost the ability to inhibit probable responses would not be expected to take a much longer time to make this response than control animals. Nevertheless, the septal-damaged rats of the present study did take a much longer time than the control rats.

Although it may well be that no unitary generalization about septal dysfunction is possible, it may still be of value to search for a new conceptualization of the effects of septal dysfunction. It would be of definite advantage if a new conceptualization could (1) account for the tendency of septal-damaged animals to perseverate in a number of situations as well as (2) provide a different view of the properties of septal cells than that suggested by Mc Cleary's revised generalization. The remainder of

this chapter is devoted to a third generalization that accounts for a number of the perseverative effects that were outlined by Mc Cleary (1966) without suggesting that septal cells are inhibitory only with respect to cells involved in the production of highly probable responses. This interpretation has not previously been offered as a generalization concerning septal dysfunction but has been used as a description of the effects of septal damage on one particular type of behavior. This concerns the fragmentation of behavior that Carlson and Thomas (1968) reported as an effect of septal damage on maternal behavior. According to them, all the species-specific components of maternal behavior were present in such animals but they were not put together in an appropriate (effective) sequence. That description fits a number of the present findings as well. After the first test of exploration the normal rats would go from the center of the open field to the enclosed compartment in one smooth sequence. But even on the fourth day several brain-damaged animals first circled backwards from the center, then approached the wall and finally entered the compartment as a third separate movement. During the escape test, the septal damaged rats made more escape responses but with less probability of escaping than the control rats.

The fragmentation was even more clear in the escape training and avoidance learning situations. During

escape shaping of the septal-damaged rats, maximum shock had to be applied first for running to or freezing in the corners of the field, then for any peripheral location, then for not putting the forelegs on the island and finally for not bringing the fourth leg onto the island, whereas in the control rats shaping was rarely needed before the animals could run from the corner of the grid to the center and climb on the island in one smooth sequence (after extensive shaping of the brain damaged rats their response durations were still much longer than those of the control rats). Also, in the jump-avoidance-learning situation the septal damaged animals were able to approach the wall within the 5 sec. interval before shock and from time to time an animal would jump straight in the air and land back on the grid before it was shocked, but it took much longer than the control rats for these animals to combine the approach to the wall with the jump to the top in one smooth sequence. Thus, if an integrated response is defined as the longest sequence of movements that an animal can make in smooth succession, then for every integrated response that a normal rat learns, a septal-damaged rat must learn a chain of responses.

One difficulty with the generalization that septal lesions cause response fragmentation is that an added preoptic lesion tended to improve performance on JA when MI testing was done prior to JA testing--it might be questioned

how damage in one part of the brain could destroy the integration of a response and how additional damage in another part could even partially put the response back together. However, the examination of the results of Experiments 1 and 2 suggests that the preoptic damage only improved performance in situations where there was negative transfer from previous experience and then it never improved performance beyond the level which obtained when there was no previous interfering experience. There were three major findings which support this interpretation. First, previous MI experience (which was mostly experience with shock in septal-damaged rats) made JA performance worse in the S2 group of Experiment 2 while it had no effect on the performance of the PS2 group. On the other hand when there was positive transfer, preoptic damage tended to interfere. Thus, in Experiment 1, the DC group (with septal damage only) showed shorter latencies to move, to find the wall after moving and to enter the compartment after finding the wall on later days than on the first day, but the RF group (which contained several animals with preoptic as well as septal damage) did not. In this respect, preoptic damage appears to have effects on exploratory behavior that are similar to those of amygdaloid lesions. Schwarzbaum and Gay (1966) have shown that with amygdaloid damage there is not the typical between-days decline in exploratory behavior.

They also reported the typical within-day decline even in animals with amygdaloid damage. This was not found in the RF group of Experiment 1. However, in contrast to the lack of between-days decline, the lack of the within-day decline appeared in both the DC group (with relatively little preoptic damage) and in the RF group. Therefore, the lack of between-days decline appears to be a function of preoptic damage while the lack of within-day decline appears to be a function of septal damage. In this context it is interesting that there are extensive, direct connections between the amygdala and preoptic area via the stria terminalis (Gloor, 1960). A second finding that supports the view that preoptic damage affects transfer is that there was less decline in emotionality over tests in the RF group than in the DC group. A third finding is that in Experiment 2 prior JA training improved learning to escape to the island in the MI apparatus much more in the S1 group than in the PS1 group. One difficulty is that preoptic-only lesions did not diminish MI-JA or JA-MI transfer compared with control lesions. However, this difficulty is mitigated by the total lack of apparent JA-MI or MI-JA transfer in sham operated control animals. Thus, the preoptic lesions appeared to cause a definite deficit in transfer in several different types of tasks when added to septal lesions. Also, the fact that the preoptic-septal groups performed in a way very similar to that of the septal group when there was no prior

experience, suggests that the preoptic damage had little effect on response fragmentation per se.

Some other effects of septal lesions may be interpretable as side effects of response fragmentation as well. For example, the enhanced probability of some stereotyped component acts may be a compensation for decreased efficiency. Thus, if motivation to retrieve pups is the same as in normal animals and the effectiveness of each response is markedly reduced, then it would be expected that septal animals would produce more responses. This could account for the perseverative effects of septal lesions on maternal behavior (Carlson and Thomas, 1968), grooming (Dirlam, 1969), aggressive behavior in wild rats (Bunnell and Smith, 1966) and other behaviors such as those discussed by Mc Cleary (1966).

That aggressive behavior is associated with response fragmentation in laboratory rats as well as wild rats is suggested by the correlation of the second test of aggression with the measures of the inability to make directed movements. To my knowledge there has been no other report of hyperemotionality correlating with other effects of septal lesions. It is likely that this is due to the facts that (1) the animals are relatively tame by the time later tests are made and (2) the less emotional animals are, the less differentiation previously used rating scales show.

Regarding maze learning and other findings involving learning with consummatory rewards, more attention to the details of the behavior is needed to test the adequacy of the response-fragmentation generalization. Thus, it would appear that there should be greater deficits in learning than those reported, if the generalization is not to be qualified. For example, perhaps fragmentation is apparent only when the animal is threatened or when there are stringent requirements for fast responses (as during active avoidance learning) or when there is no such requirement perhaps it is apparent only when the behavior sequences are relatively long or complex (as in umweg or "reasoning" tests).

An additional difficulty was mentioned earlier: in each of the behaviors described above, there is a problem in deciding what are component acts and what are whole acts. As a preliminary step the criteria used by Piaget (1952) to differentiate voluntary from involuntary behavior in infants could be used: voluntary acts are (1) not centered on the body, (2) novel adaptations rather than stereotyped or repetitive acts and (3) composed of many intervening acts rather than few. However, these criteria are rather abstract. The work of Vanderwolf (1964, 1969) on the hippocampal EEG and voluntary behavior suggests an alternative criterion and provides additional evidence on the role of the septal area in the integration of stereotyped component

acts. It is well known that septal damage prevents the appearance of rhythmical slow activity (RSA) in the hippocampal EEG (Green and Arduini, 1954). Vanderwolf's studies indicate that such activity is correlated with the initiation of voluntary movements such as jumping to avoid shock or walking during exploration. It is not correlated with components of such stereotyped behaviors as grooming and mating and only appears in these behaviors when postural adjustments are made during the transition from one component act to the next. Since septal damage disrupts hippocampal RSA it might be expected that those behaviors with which RSA is associated would also be disrupted. This conclusion is consistent with the findings of the present study in which rats with septal lesions were found to be slow to initiate walking during exploration and jumping during avoidance training, but were more normal when making grooming, feeding, drinking or undirected escape responses (running, jumping, struggling, attacking, freezing). It is also consistent with other studies of septal damaged animals which found a disruption of integrated, effective sequences of stereotyped acts but no effect on or an enhancement of the probability of the isolated acts.

In an experiment, which like the present study involved the observation of a variety of behaviors in each of a number of brain-damaged animals, Glickman, Higgins and Isaacson (1970) also found a correlation between the behaviors

correlated with hippocampal RSA and those affected by limbic system damage. In their study, hippocampal damage in gerbils produced an increase in the frequency of those behaviors correlated with RSA. Whether or not this increase implies a disruption of the behaviors is difficult to determine because the behaviors were not analyzed in terms of effectiveness. For example, the frequency of locomotion, rearing and sniffing were analyzed, but not the latency to find a reinforcing stimulus, and the frequency of attack or fighting was analyzed but not whether the attack led to the eventual flight or submission of either the attacker or the opponent. Some findings, however, suggest that these increases may reflect a disruption. A major example is that although hippocampal lesions produced more locomotion in an open field (a definite difference from the effects of septal lesions in the rat), there was very little decline compared with control animals in locomotion over the 10 min. test period. This latter result suggests that the locomotion was not as effective in the hippocampal-damaged gerbils as in the controls. This conclusion is based on an analogy between exploration and consummatory behaviors such as eating, mating, etc. If consummatory acts are effective, then over time they are self-terminating (e.g. over time, eating tends to terminate eating). It is well-known that this is true of the exploration of normal animals. Since it is not true of the exploration of animals with hippocampal damage, this

suggests that the behavior is not effective. A latent learning study would be needed to test this suggestion, but at least it is consistent with the finding that hippocampal damage results in excessive but ineffective locomotion in other situations (e.g. where escape from shock is dependent on inhibiting locomotion--Teitelbaum and Milner, 1963).

The study by Glickman et al. illustrates some of the considerations that must be accounted for in analyzing behaviors in terms of voluntariness. Vanderwolf (1969) describes two major criteria: (1) "The type of movement said to be 'voluntary' can easily by controlled by any one of a number of different motive states." and (2) "The sequence of movements is not fixed." The concept of fragmentation (into fixed sequences of behaviors), discussed above, is primarily concerned with the second criterion. Since the concept of effectiveness is concerned with the goal of the behavior from which the motives must be inferred, it is concerned with the first criterion. In short, to analyze behavior in terms of voluntariness, the goal as well as the pattern of the behavior must be considered.

In conclusion, the present findings support the generalization that septal damage causes a fragmentation of voluntary behaviors. Thus, integrated sequences of such behaviors have a decreased probability of appearance but isolated component-acts are either unaffected by the damage or made more probable. The generalization does not preclude

the possibility that such voluntary behaviors could be learned, but it should take septal damaged animals longer than normal animals because for every effective response a normal animal learned the septal damaged animal would be learning a chain of responses. Finally, the response-fragmentation generalization may be restricted to situations which normally are threatening or fear-inducing to the species being considered.

CONCLUSIONS

1. An historical review of the study of septal damage suggested that detailed, within-subjects observations of animals in several situations and interpretation of these observations in terms of the effectiveness of the behavior for the survival of the animals are needed for a more complete understanding of the effects of septal damage.

2. Three experiments, reported in this study showed the following effects of septal damage:

(a) Septal lesions disrupt directed movements made during the testing of exploration, escape, movement inhibition, and jump avoidance learning;

(b) The disruption of directed movements is related to hyperemotionality when it is measured by a newly developed scale, which indicated the emotionality of previously handled (tame) animals more reliably than previously used scales;

(c) When added to septal lesions, preoptic damage disrupts both positive and negative transfer;

(d) The disruption of directed movements during jump avoidance learning is not due to intertrial handling or to a tendency on the part of septal-damaged rats to avoid light; and

(e) Although a wide variety of effects of septal damage are interrelated, some effects (like the decrease in weight-gain) are not related to others.

3. Interpretation of this and previous experiments suggests the following generalization and two corollaries:

(a) For a wide variety of the effects of septal damage, there is a fragmentation of voluntary behaviors, such that integrated sequences of such behaviors have a decreased probability of appearance but isolated component-acts are either unaffected or made more probable; and

(b) The fragmentation of voluntary behaviors results in both (i) the perseveration of isolated acts and (ii) the slower learning of effective responses, because for every effective response that a normal animal makes, a septal-damaged animal must make a chain of isolated acts.

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APPENDIX:

Performance of individual animals.

Table A1.

Water consumption during first week after recovery (3 da.) from operation (cc.).

DC		RF		Sh	
Ss	Consumption	Ss	Consumption	Ss	Consumption
1	243	1	280	1	161
3	179	2	204	2	187
4	175	4	224	3	217
6	164	5	196	5	214
7	210	6	166	6	186
8	200	7	138	7	181
9	147	8	153	8	168
10	194	9	163		
11	175	11	123		
12	214	12	139		
13	165				

Table A2.

Emotionality ratings for individual animals.

Ss	Day 1	Day 2	Day 3	Total
DC 1	5	6	6	17
3	4	3	5	12
4	4	2	3	9
6	7	0	5	12
7	10	2	7	19
8	2	2	4	8
9	8	2	2	12
10	9	2	7	18
11	4	0	0	4
12	3	2	0	5
13	7	2	7	16
RF 1	9	9	10	28
2	5	7	4	16
4	5	1	6	12
5	7	7	11	25
6	9	6	10	25
7	8	10	8	26
8	5	7	6	18
9	5	5	6	16
11	13	8	9	30
12	9	7	5	21
Sh 1	7	1	3	11
2	2	0	4	6
3	5	1	2	8
5	2	3	2	7
6	0	3	1	4
7	4	3	4	11
8	3	0	1	3

Table A3.

Frequency of open-field behavior of individual animals (4-day totals).

Ss	Behaviors						
	Exploration	Grooming	Inactivity	Defecation (foecal boli)	Squares traversed	Leaving wall	Entering compartment
DC 1	23	0	57	2	38	1	5
3	6	1	73	4	14	0	4
4	16	8	55	8	55	0	13
6	16	11	53	6	39	1	7
7	28	5	47	5	24	0	4
8	19	3	58	8	26	0	3
9	6	7	67	14	13	0	2
10	17	7	56	10	69	3	7
11	18	14	48	3	69	3	12
12	25	6	49	9	29	0	6
13	3	9	68	10	14	0	3
RF 1	17	1	62	3	74	1	1
2	15	2	63	12	25	0	4
4	25	4	51	17	17	0	5
5	13	3	64	1	28	0	4
6	10	1	69	7	23	1	5
7	0	0	80	15	0	0	0
8	9	1	70	7	16	0	3
9	4	10	66	8	12	0	3
11	3	0	77	9	10	0	2
12	6	2	72	8	17	0	0
Sh 1	34	7	39	20	150	6	10
2	40	2	38	1	136	9	18
3	42	9	29	4	86	5	12
5	22	14	44	11	60	3	7
6	41	3	35	6	163	9	15
7	27	4	49	3	15	0	5
8	31	7	42	0	79	4	11

Table A4.

Open-field latencies (in 12 sec. intervals)

Ss	Movement latency				Latency to find wall after first movement			
	Day 1	Day 2	Day 3	Day 4	Day 1	Day 2	Day 3	Day 4
DC 1	2	1	0	0	1	0	0	0
3	6	2	0	1	2	0	0	1
4	4	2	1	2	14	1	0	1
6	2	0	0	0	3	0	0	1
7	0	0	0	0	1	0	0	0
8	1	2	0	0	1	0	0	0
9	2	2	1	4	0	0	1	10
10	2	0	0	0	0	0	0	0
11	2	0	0	0	0	2	0	0
12	5	0	0	1	10	1	0	0
13	0	0	6	7	0	0	1	3
RF 1	3	5	1	0	6	0	0	1
2	1	3	2	4	3	0	1	0
4	1	1	0	0	1	1	0	0
5	1	2	0	0	2	0	1	1
6	0	2	0	0	0	2	1	0
7	25	25	15	15	0	0	0	0
8	3	5	2	4	4	1	1	1
9	4	3	3	3	0	7	1	4
11	1	3	2	2	0	1	1	1
12	2	3	2	2	1	6	1	6
Sh 1	1	0	0	0	2	0	0	0
2	0	0	0	0	1	0	0	0
3	0	0	0	0	1	0	0	0
5	1	0	0	0	1	0	0	0
6	0	0	0	0	1	0	0	0
7	0	0	0	0	0	0	0	0
8	0	0	0	0	0	0	0	0

Table 4 (cont'd).

Ss	Latency to enter compartment after finding wall*			
	Day 1	Day 2	Day 3	Day 4
DC 1	15	2	1	11
3	5	1	1	5
4	13	17	3	6
6	4	6	1	2
7	4	0	1	0
8	13	2	Max	1
9	5	7	Max	NFW
10	9	4	1	1
11	6	5	3	9
12	3	0	1	1
13	9	3	6	Max
RF 1	Max	18	Max	Max
2	2	6	10	Max
4	1	0	1	0
5	6	1	4	3
6	2	2	3	2
7	NFW	NFW	NFW	NFW
8	4	2	0	Max
9	14	9	10	Max
11	Max	0	Max	Max
12	Max	Max	Max	Max
Sh 1	4	2	4	0
2	2	1	1	0
3	0	0	1	0
5	6	0	1	0
6	2	0	1	0
7	1	0	0	0
8	1	0	0	0

*Max-did not enter compartment on this test day;
NFW-did not find wall on this test day.

Table A5.

Behavior of individual animals during the escape test.

Ss	Escape responses		Freezing responses		Ss finding island (X= S found island)
	Number during test	Number in first 2 min.	Number	Latency (in 12 sec. periods)	
DC 4	125	43	(29)	16	X in 19 min.
6	188	43	23	19	
7	50	33	79	10	
8	(61)	54	(6)	13	X in 5 min.
9	146	43	27	44	
10	(29)	24	(8)	5	X in 4 min.
11	71	27	54	8	
12	63	44	73	10	
13	142	28	4	10	
RF 1	89	33	47	16	
2	101	17	29	7	
4	(27)	25	(32)	8	X in 10 min.
5	98	44	50	12	
6	87	23	11	21	
7	151	34	13	12	
8	78	30	42	11	
9	122	37	37	9	
11	108	24	28	15	
12	205	58	20	18	
Sh 1	40	8	45	5	X in 21 min.
2	(22)	13	(25)	9	X in 15 min.
3	(35)	35	(0)	--	X in 2 min.
5	22	11	64	10	
6	(30)	23	(18)	7	X in 8 min.
7	65	16	53	7	
8	25	21	84	6	

Table A6.

Performance of individual animals on escape training and passive avoidance.

Ss	Number of shaping trials during escape training			Mean escape latency (sec.) on last 3 trials			Number of trials during passive avoidance test		
	Large Isl.	Small Isl.	Total	Large Isl.	Small Isl.	All Cr* trials	Large Isl.	Small Isl.	Total
DC 1	2	0	2	2	9	8	2	2	4
2	15	4	19	10	3	8	1	0	1
4	1	4	5	12	10	11	0	1	1
6	9	0	9	11	7	10	3	1	4
7	2	76	78	6	1	5	2	2	4
8	0	0	0	14	4	7	0	2	2
9	1	1	2	14	11	13	2	1	3
10	0	3	3	5	3	5	1	1	2
11	2	8	10	6	14	13	2	4	6
12	8	2	10	15	10	13	1	0	1
13	18	0	18	11	14	12	1	1	2
RF 1	7	3	10	11	17	15	4	1	5
2	7	0	7	4	22	10	1	1	2
4	0	0	0	9	4	10	0	5	5
5	8	0	8	11	7	11	3	1	4
6	2	8	10	7	6	8	2	3	5
7	7	64	71	22	11	14	3	2	5
8	10	0	10	8	18	13	0	0	0
9	6	21	27	6	4	6	1	0	1
11	7	0	7	5	19	10	7	0	7
12	4	0	4	9	12	9	0	0	0
Sh 1	0	0	0	2	7	4	0	1	1
2	0	3	3	2	3	5	0	7	7
3	0	2	2	2	2	6	0	0	0
5	2	1	3	2	3	4	4	0	4
6	1	1	2	9	6	7	0	4	4
7	2	1	3	6	4	6	2	1	3
8	2	2	4	2	2	4	3	3	6

*Cr. - Criterion (last 5 trials were criterion trials for each island).

Table A7.

Movement inhibition performance of individual animals.

Ss	Errors		
	Day 1	Day 2	Total
DC 1	3	1	4
3	4	1	5
4	3	0	3
6	4	0	4
7	1	1	2
8	5	7	12
9	16	0	16
10	3	0	3
11	0	0	0
12	10	5	15
13	6	1	7
RF 1	13	12	25
2	3	1	4
4	6	5	11
5	1	3	4
6	3	14	17
7	2	0	2
8	1	3	4
9	0	2	2
11	22	6	28
12	7	4	11
Sh 1	4	1	5
2	1	1	2
3	0	4	4
5	2	2	5
6	1	0	1
7	2	0	2
8	1	5	6

Table A8.

Jump avoidance performance of individual animals.

Ss	Errors	Mean latency on criterion trials (sec)	Errors after first avoidance	Trial 1 escape latency (sec.)
DC 1	5	4.0	1	30
3	8	4.4	1	1
4	4	6.8	0	210
6	3	7.2	0	12
7	5	3.3	0	190
8	12	5.1	0	17
9	13	3.8	9	1
10	4	1.3	1	67
11	4	9.1	0	104
12	20	8.9	9	47
13	10	6.1	3	103
RF 1	1	5.9	0	9
2	17	4.6	1	10
4	1	4.0	0	56
5	7	2.4	0	09
6	6	10.6	0	18
7	18	5.8	9	36
8	15	2.3	5	1
9	5	6.2	1	96
11	1	2.7	0	8
12	3	3.7	1	23
Sh 1	0	2.6	0	54
2	1	1.6	0	61
3	0	1.4	0	16
5	1	1.7	0	10
6	2	2.3	0	105
7	0	2.8	0	42
8	3	1.6	0	43

Table A9.

Weight gain (in gm.) per day of individual animals.

DC		RF		Sh	
Ss	Weight gain	Ss	Weight gain	Ss	Weight gain
1	1.7	1	1.6	1	1.9
3	1.1	2	1.9	2	1.6
4	1.3	4	1.7	3	2.4
6	1.4	5	1.3	5	1.7
7	1.3	6	1.1	6	1.8
8	1.6	7	1.7	7	2.3
9	1.1	8	1.4	8	1.3
10	2.4	9	1.8		
11	1.9	11	1.4		
12	1.8	12	1.7		
13	1.3				

Table A10.

Mean JA and MI performance of individual animals.

Ss	JA (errors X 100/30)	MI (% time off island)	Ss	JA (errors X 100/30)	MI (% time off island)
C1 1	13	7	C2 1	33	3
2	50	100	2	23	100
3	30	93	3	27	41
4	63	2	4	40	44
5	47	5	5	17	7
6	13	100	6	27	48
7	13	8			
8	27	98			
9	13	8			
10	40	10			
S1 1	80	3	S2 1	100	100
2	77	30	2	100	100
3	53	21	3	100	100
4	97	5	4	100	90
5	87	58	5	100	88
6	83	10	6	77	100
7	100	5	7	100	98
8	47	52	8	100	100
9	100	13	9	87	97
10	100	2	10	93	20
P1 1	37	3	P2 1	30	82
2	33	2	2	20	25
3	30	2	3	23	72
4	33	45	4	30	27
5	37	7	5	20	97
6	20	8	6	53	100
7	23	2			
8	17	3			
9	17	3			
10	13	2			

Table A10 (cont'd).

Ss	JA (errors X 100/30)	MI (% time off island)	Ss	JA (errors X 100/30)	MI (% time off island)
PS1 1	100	52	PS2 1	73	23
2	77	50	2	100	67
3	70	98	3	90	95
4	83	52	4	93	80
5	57	48	5	80	100
6	100	92	6	67	42
7	100	17	7	63	100
8	40	38	8	87	97
9		65	9	90	100
			10	60	100
			11	90	42
			12	60	100
			13	100	100
			14	97	97