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Effects of Septal and Amygdaloid Lesions on Pain-Elicited Fighting

John H. Bryant
Western Michigan University

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EFFECTS OF SEPTAL AND AMYGDALOID LESIONS
ON PAIN-ELICITED FIGHTING

by

John H. Bryant

A Thesis
Submitted to the
Faculty of the School of Graduate
Studies in partial fulfillment
of the
Degree of Master of Arts

Western Michigan University
Kalamazoo, Michigan
August, 1970
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John H. Bryant
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INTRODUCTION

Early investigations of the behavioral functions of the amygdala reported that bilateral lesions of this nucleus resulted in rage responses characterized by hyperirritability and increased arousal (Speigel, 1940). Bard and Mountcastle (1948) and Bard (1950) reported that after massive bilateral removal of the amygdala and pyriform cortex, their subjects showed an increase in "emotional behavior." 1

Contradictory evidence concerning behavioral changes subsequent to amygdalectomy is plentiful. Following discrete lesions of the amygdala, increased tameness has resulted in the rat (Reynolds, 1965; Anand and Brobeck, 1952), lynx and agoutie (Schreiner and Kling, 1953, 1956), monkey (Schreiner and Kling, 1956, Rosvold, Mirski and Pribram, 1954; Weiskrantz, 1956), and cat (Shealy and Peele, 1957; King and Hutt, 1958).

Galef (1969) has found that such lesions also inhibit the mouse killing response of feral Norway rats. Schwartzbaum, Thompson and Kellicutt (1964) and Kellicutt and Schwartzbaum (1963) also reported deficits in affective responses following changes in stimuli previously learned to be the nonreinforced components of a discrimination.

While bilateral amygdalectomy produced a quiescent animal, bilateral lesions in the septal area produced behavioral changes in the opposite direction. Brady and Nauta (1953, 1955) found that septal lesions consistently resulted in a postopera-
tively irritable, vicious and hyperemotional rat which was difficult to capture and handle. This savageness does not last long (Brady and Nauta, 1953, 1955; Yutzey, Meyer and Meyer, 1964; Reynolds, 1963) and declines in a period from one to four weeks (Wetzel, Conner and Levine, 1967). Another consistent component in the septal syndrome is an exaggerated startle response to preoperatively neutral stimuli (Brady and Nauta, 1953, 1955). Attempts at replication of this syndrome have not always been successful, as reported by Kling, Orbach, Schwartz and Towne (1960). After lesioning the posterior septum, fornix and anterior commissure of adult cats, no changes in irritability or arousal were observed, although one might attribute this result to the considerable anatomical differences between the rat and cat.

With such contradictory evidence being reported for emotional behavior, it would seem that a major problem in such investigations concerns finding a measure sensitive both to increases and decreases in the emotion to be observed. Fuller, et al. (1957), found that a variety of measures yielded very different results in attempting to quantify the effects of pyriform and hippocampal lesions in addition to amygdaloid lesions. King (1958) and King and Meyer (1958) concluded that the reason massive amygdaloid lesions failed to produce changes in their subjects was that the animals initially may not have been emotional enough for taming to be noticeable.

It is evident that there is a great need for the quanti-
fication of the behavior of animals in terms of an index of emotionality. The majority of the studies concerned with investigating emotional behavior have used the rating scale, or variations of it, that Brady and Nauta (1953, 1955) employed to measure their dependent variables (Yutzey, et. al., 1964; Wetzel, et. al., 1967; Reynolds, 1963). Reynolds (1963) points out that this scale is not a true interval scale and that some basic statistical assumptions in the data analysis are not met.

Pain-elicited fighting (O'Kelly and Steckle, 1939; Ulrich and Azrín, 1962) would seem to provide an excellent behavioral baseline upon which the effects of both septal and amygdaloid lesions could be assessed. Unlike situations in which the behavioral response studied is almost exclusively peculiar to the laboratory, i.e., the manipulation of a lever, intraspecies fighting is frequently observed in the natural environment. In Scott and Fredericson's analysis of fighting behavior in mice and rats (1951), the point is made that rating such behavior is unnecessary. They feel that "...the presence or absence of certain behavioral traits may be considered a more objective and accurate measure of behavior than using scales or grading impressions." A binary event recording system, as opposed to a rating scale, would provide a more reliable standard with which to measure changes in emotional behavior. This idea is consistent with the objections and the solutions to the point raised by Reynolds (1963).

It is the purpose of the present study to investigate the
apparent opposing function of septal and amygdaloid complex lesions in rats using pain-elicited fighting as a behavioral baseline measure.
METHOD

Subjects

Subjects for this experiment were 22 experimentally naive, 95-100 day old male albino rats obtained from the Upjohn Laboratories, weighing preoperatively from 239-418 grams. The day before the first experimental session all subjects were shaven around haunches, tail, scrotum, and dorsal and lateral back. Subjects were individually housed with food and water available at all times.

Apparatus

Sessions were run in a chamber whose effective fighting area was 11" x 9" x 13". The chamber was positioned approximately 3" from a one-way mirror through which the experimenter observed the subjects. Three walls of the chamber were constructed of 1/2" plywood, painted flat black on the inside to maximize contrast for photography. The other wall was constructed of clear plexiglas and covered on the outside with a thin sheet of cheese cloth in order to reduce the amount of reflection from the one-way mirror. The floor of the chamber was constructed of 21 parallel 3/32" stainless steel rods approximately 1/2" apart at the centers. Chamber illumination was provided by a single shaded GE 210 high intensity light bulb placed centrally outside the chamber on the 3/16" plexiglas ceiling. A forced air fan provided ventilation and masked extraneous noises from outside the chamber room.
The various stimulus conditions used in the experiment were programmed through electromechanical switching circuits with running time meters and digital counters located in the observation room. Shocks of various intensities were delivered by a Grason Stadler E6070B shock generator. A shock scrambler was used to change the pattern of polarities so that any two of the floor grids would be of opposite polarity during part of each shock duration. One-hundred and twenty 0.5 second shocks were delivered with a 15.0 second intertrial interval. The observer in the observation room recorded responses and response duration using microswitches. Fighting duration and shock presentation were recorded on a Gerbrands cumulative recorder. A duplicate recording system was used for sessions in which reliability checks were made.
PROCEDURE

Surgery

All surgery was performed under clean but not aseptic conditions using sodium pentobarbital (40 mg/Kg Intraperitoneal) anesthetic. Atropine sulfate was used to reduce respiratory secretions. Subjects were placed in a Kopf stereotaxic instrument with the tooth bar set 2.4 mm. below the interaural line. A midline incision was made, the periostium retracted and small burr holes made at the appropriate coordinates for the specific operation. Bilateral lesions were produced electrolytically using stereotaxically placed electrodes (insulated insect pins, Clay Adams #00) passing 2.0 ma anodal DC for 30 seconds through 0.5 mm. of the uninsulated tip of the electrode. The lesion circuit was completed by a reference electrode clipped to the wound edge.

For septal operations, electrodes were inserted using an angular approach to the coordinates anterior 7.1 mm., lateral 0.6 mm., and +0.8 above zero according to the atlas of Konig and Klipple (1963). For all amygdaloid operations, a vertical approach was used with coordinates of A-P +3.1 mm., lateral 4.0 mm., and -3.1 below horizontal zero (Konig and Klipple, 1963). For the sham operates of Group S, electrodes were placed in the appropriate structure without passing current and then withdrawn. After surgery all subjects were given 0.25 cc of Bicilin² intramuscularly. In all cases, both animals in a pair were surgically operated upon the day after

²Bicilin
stabilization and run again approximately 24 hours after the operation.

**Behavioral Methods**

The dependent measures recorded throughout all procedures were:

1. The presence or absence of a single fight response per shock presentation for 120 shocks.
2. The cumulative duration of fighting for the 120 trials per session.

Throughout the different group treatments, Pearson product moment correlations between the two dependent variables were compiled. In addition, other differences in fighting behavior not directly measured by the dependent variables were noted and recorded each day.

A fighting response was defined as contact made by the paws of one subject on the other subject inclusive of an area defined by a line between and just behind the ears and running down diagonally and posteriorly to a point where the abused rat's elbow would rest when down and drawn in toward its body. Contacts were not counted if the defined response area was contacted with the hind feet of the other subject or if the subject making the attack had one of its front paws on the grid, even though proper contact was being made with the other paw. Preoperation baseline fighting behavior above 40% yet below 70% was criterion for rejection on the grounds that it was unclear as to whether the particular pair was a high or low probability fighter. After the first operation there were no restrictions
on the level of fighting behavior. Following stabilization after the last treatment, subjects were sacrificed.

Group A (N=3) was run at shock intensities that produced stabilized fighting on more than 70% of the shock presentations. After preoperative stabilization of their fighting, they received amygdaloid lesions. The period following the first lesion is referred to as the postoperative period I (PPI) and the period following the second lesion is referred to as postoperative period II (PPII). After stabilization of their responding during PPI, they were given septal lesions and allowed to stabilize during PPII before being sacrificed.

Two pairs (#10 and #14) in Group B (N=5) were run under shock conditions which produced fighting on less than 40% of the shock presentations during the preoperation baselining. After stabilization of their rates of fighting, they received septal lesions. Following stabilization of responding during PPI, they received amygdaloid lesions, were allowed to stabilize their fighting during PPII and then sacrificed. The fifth pair (#17) of subjects in Group B were run at a shock intensity which resulted in less than 40% fighting to shock presentation. After the preoperative stabilization they were given septal lesions. This pair was not given any other lesions in the hope of ascertaining the persistence of any changes consequent to septal lesions.

Two pairs (#13 and #6) in Group B fought on more than 70% of the shock presentations during the preoperation period. After stabilization of their fighting, the current intensity for each pair
was lowered in order to reduce the amount of fighting below 40%. In each case, it took two changes in intensity before the experimenter decided that the particular current level was low enough to reduce the amount of fighting but not so low as to eliminate fighting completely. After stabilization at the lowered preoperative level, each subject received septal lesions. Amygdaloid lesions were made in a second operation after stabilization during PPI. In the final phase after PPII stabilization, these two pairs had the current changed back to its original preoperative intensity and changes in fighting behavior were recorded.

Group S (N=3) were sham operated pairs and used as a control for the effects of the operations. Because preliminary data suggested that the effects of infection or cortical damage tended to reduce the probability of fighting, all subjects in the group were high probability fighters and received an amygdaloid operation first and the septal operation second.

Histology

All lesions were verified histologically at the conclusion of the experiment using the method of Guzman et. al. (1963). Each subject was perfused intracardially with normal saline followed by a formol-saline solution. Frozen sections were cut at approximately 75 micra intervals. Selected sections were then photographed in order to determine the extent of lesion damage.
RESULTS

Starting with the first day of the preoperative period, the subjects explored and groomed during the short period before the first shock presentation. On subsequent sessions this initial exploratory and grooming behavior declined and the animals assumed a huddling motionless posture in the center of the chamber until the first shock presentation. Following shock onset, the subjects usually fought or began to move away from each other. By the second or third shock, they began fighting on a regular basis if they were "high" rate fighters (fighting to over 70% of the shock presentations) and on a less frequent basis if they were "low" rate fighters (fighting to under 40% of the shock presentations). It can be concluded that the behavior measured actually was pain-elicited fighting as it was observed that preoperative rats with several days shock-induced fighting experience never fought prior to the onset of the first shock.

Subjects who had been fighting at a low level and who received a septal lesion first occasionally began fighting when the second subject was placed in the chamber prior to the start of the session. More frequently, there was an increase in exploratory behavior at the beginning of each session. Unlike the amygdaloid lesioned animals, those with septal lesions were never observed to groom themselves or their partner at either the beginning of the session or during the intertrial intervals (ITI).
Those pairs first receiving amygdaloid nucleus operations engaged in extensive exploratory and grooming behavior. This occurred in two pairs (04 and 08) throughout the first two or three sessions during PPI. Later in this period, the grooming and exploratory behavior declined and was replaced with the "frozen" posture or fighting behavior.

**Fighting Rate**

In Group A, three pairs of subjects (04, 07 and 08) were run under shock conditions that produced fighting to more than 70% of the shock presentations. After stabilization of the fighting rate, each pair received amygdaloid lesions. Subsequently, all three pairs showed a marked drop in fighting rate. Pair 07, however, demonstrated a less extreme decrement in fighting rate than did Pairs 04 and 08. While Pair 07's initial decrease in the fighting rate was more than could be accounted for by chance, the rate slowly returned to near-preoperative level before stabilizing. Since Pair 07's behavior obviously departed from the behavior of the other two pairs in this respect, Pair 07 did not receive another lesion.

Pairs 04 and 08 stabilized their respective fighting rates before they received septal lesions. Pair 04 changed relatively little following the initial decline and kept the same low rate of fighting to about 15% of the shock presentations. In comparison, Pair 08's rate of fighting rose slightly before stabilizing.

In Group B, three pairs (010, 014 and 017) were run under conditions that produced fighting on less than 40% of the shock
Figure 1

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #7. The arrow indicates the session following which the pair received an amygdaloid (A) lesion.
Figure 2

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #4. The arrows indicate the sessions following which the pair received an amygdaloid (A) lesion and a septal (S) lesion.
Pair 4

Percent Fighting

Duration of Fighting (in seconds)

Sessions

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Figure 3

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #8. The arrows indicate the sessions following which the pair received an amygdaloid (A) lesion and a septal (S) lesion.
presentations. Each pair was given septal lesions after preoperative shock; radical and dramatic increases in rate of fighting occurred in each pair. Typically in all septal pairs, the high fighting rate was maintained until the session was over and a few minutes had passed without shock delivery.

Following rate stabilization during PPI, Pairs #10 and #14 were given amygdaloid lesions. Figure 4 shows the remarkable drop in the rate of fighting for Pair #10 from approximately 85% to nearly 0% before slowly rising to almost preoperation level.

For Pair #17 it was decided to extend the PPI in expectation of finding the stabilization limits of the septal lesioned fighting rate and the extent of the duration of fighting decline. On PPI day-1, the pair was run and it was later noticed that one of the animals had an infection. The subject was given 0.25 cc Bicillin\textsuperscript{2} and allowed two days to recover, at which time the infection had disappeared. Continuation of the fighting on PPI day-4 resulted in fighting at a rate equivalent to the first day of PPI fighting. Not until a week after the continuation of the sessions did the fighting rate show the expected decrement in responsiveness to the foot shock. Thirteen days after the septal operation, the fighting rate had declined to the preoperative level. However, this decline proved transient and was followed by a much more variable fighting rate which approximated that rate prior to the decline.

In a second part of Group B, two pairs of rats (#6 and #13) responding to 70% of the shock presentations were run until their fighting rate appeared to be stable. At this point the shock

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Figure 4

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #10. The arrows indicate the session following which the pair received an amygdaloid (A) lesion and a septal (S) lesion.
Figure 5

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #17. The arrow indicates the session following which the pair received a septal (S) lesion. The pair was not run during sessions 9 and 10 due to postoperative infection.
intensity was lowered in order to reduce the rate of fighting. For Pair #6, the intensity was first dropped to 0.6 ma and then raised to 0.8 ma where it remained until after stabilization of PPII. For Pair #13, the first reduction was to 0.8 ma and then lowered further to 0.6 ma until the end of PPII.

Stabilization of Pair #6 following the current reduction was followed by septal lesions. These resulted in dramatic changes in behavior. Fighting rate increased, with the rate approaching a maximum ceiling during PPI. Following rapid stabilization, both subjects in this pair were given amygdalectomies. Consequently, fighting rate dropped radically, stabilizing at about their second preoperative level. It can be seen in Figure 6 that the changes due to returning to the original current level resulted in only a slight increase in fighting rate but a moderate increase in rate variability.

Following a lengthy stabilization leading to current change, Pair #13's fighting rate fell in a similar fashion to that of Pair #6. Stabilization and the septal lesioning that followed resulted in a marked increment in the fighting rate, again similar in most respects to the increment which occurred during PPI in Pair #6.

Ultimate levels of fighting rate for this group were approximately the same as for Pair #6, e.g., 96%-97%.

Amygdaloid lesions followed the stabilization during the PPI for Pair #13 and resulted in extreme suppression of the fighting rate to approximately the level during the second preoperative period. Returning the current level during the second postoperative period failed to show an increase in the fighting rate. In neither
Figure 6

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #6. The arrows indicate the sessions following which the pair received an amygdaloid (A) lesion and a septal (S) lesion. The two additional arrows labeled (C) indicate on which sessions the current intensity was changed.
Pair 6

Percent Fighting

Duration of Fighting (in seconds)

Sessions

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Figure 7

Shows the duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #13. The arrows indicate the sessions following which the pair received an amygdaloid (A) lesion and a septal (S) lesion. The two additional arrows labeled (C) indicate on which sessions the current intensity was changed.
Pair #6 nor #13 did a return to the original current level after PPII stabilization result in changes remotely approximating the degree of change resulting from current reduction prior to the first lesion.

Three pairs (#2, #3 and #11) in Group S were run under shock conditions producing fighting on more than 70% of the shock presentations. Order of the operations for all three pairs were the amygdaloid sham first and the septal sham second.

As Figures 8, 9 and 10 indicate, in no instance did a pair in this group show changes in the dependent measures resembling the extreme changes incurred following the amygdaloid and septal lesions in the experimental animals. Additional behavioral observations support the contention that the changes in fighting rate were, in fact, caused by lesions in the specified area.

**Duration of Fighting**

Comparisons of the duration of fighting with the fighting rate, subsequent to either lesion, show changes in the same direction.

The three pairs in Group A (#4, #7 and #8) were run under conditions that yielded fighting to over 70% of the shock presentations. Following the first (amygdaloid) lesion, the duration of fighting and the fighting rate provide relatively good correlations. Pair #7, as shown in Figure 1, displayed a good deal more variability in duration of fighting when the fighting rate returned to its pre-operative level, causing the correlations, indicated in Table I, to be lower during PPI than they are for Pairs #4 and #8 (See Figures 8, 9 and 10).
Figure 8

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials, (solid lines) for Pair #2. The arrows indicate the sessions following which the pair received sham amygdaloid operations (AS) and sham septal operations (SS).
Pair 2

Percent Fighting

Duration of Fighting (in seconds)

Sessions

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Figure 9

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #3. The arrows indicate the sessions following which the pair received sham operations of the amygdala (AS) and sham septal operations (SS).
Figure 10

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for Pair #11. The arrows indicate the session following which the pair received sham amygdaloid operations (AS) and sham septal operations (SS).
TABLE I

The Pearson Product Moment Correlations Between the Fight Response Per Shock For 120 Trials and the Duration of Fighting for the 120 Trials Per Session for Each Pair. The Current Intensity in Milliamps for Each Pair is also Shown

<table>
<thead>
<tr>
<th>Group</th>
<th>Pair #</th>
<th>Current Intensity</th>
<th>Preoperative I</th>
<th>Preoperative II</th>
<th>PPI</th>
<th>PPII</th>
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<td>14</td>
<td>1.0 ma</td>
<td>.914</td>
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<tr>
<td></td>
<td>17</td>
<td>0.8 ma</td>
<td>.749</td>
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<tr>
<td></td>
<td>6</td>
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<td>.713</td>
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<tr>
<td>S</td>
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<tr>
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Changes in the duration following the second (septal) lesion varied relatively little during PPII. However, inspection of the graphs for each pair show that the durations of fighting were more stable and changes less abrupt than the fighting rate measure. In the case of Pairs #4 and #8, it appears that the duration would have been a better measure of stabilized behavior, and thus manipulation of an independent variable, than fighting rate.

Subjects in Group B (Pairs #10, #14 and #17) showed postoperative increases in the duration of fighting following septal lesions to match the fighting rate changes. Correlations for both Pair #10 and Pair #14 show a breakdown of the preoperative correlation following septal lesions caused in part by the restriction of the fighting rate measure. Thus, duration of fighting appeared to be more sensitive to septal lesioning in that it was not restricted by a "ceiling effect." In Pair #10, which prior to the operations had fought approximately 15 seconds per session, fought 140+ seconds on PPI day-1. A similar increase (approximately 15 seconds to 130 seconds) was seen in Pair #14's cumulative duration fighting rate. Figure 11 indicates that pair #14's initial duration of fighting was less sharp than the others in the group. Post-infection durations were more in line with the rates of the other two pairs in the group. In all pairs, the fighting rates remained stable while the duration rates declined rapidly after reaching an apex on the first postoperative day of PPI, or in the case of Pair #17, after the fourth post-operative day.
Figure 11

Shows duration of fighting per session (dashed lines) and the percent of a single fight response per shock per 120 trials (solid lines) for pair #14. The arrows indicate the session following which the pair received amygdaloid (A) lesions and septal (S) lesions.
Pair #14's initial duration of fighting rate dropped following septal lesions and then became erratic just prior to the amygdaloid lesions. As was the case for Group A, the changes in duration of fighting were less abrupt in Pairs #10 and #14 following amygdalectomy than they were following septal lesions.

Pair #17 received only a septal lesion but the effects were obvious. Following a 2-day "high", duration of fighting began to drop in a decrement approximating the other pairs in this group. Figure 5 shows that not until a week later did the fighting rate show the effects of the seven days of decreasing fighting duration. Thirteen days after the septal operation, and eleven days of PPI, the duration reached its preoperative level. The duration of fighting did rise briefly before beginning to fluctuate in a consistent manner prior to the termination of this part of the experiment.

In the second part of Group B, two pairs (#6 and #13) were run above the 70% level and had their current lowered prior to receiving septal lesions. The duration changes for Pair #6 were difficult to measure accurately because of the rapid striking movement when animals made contact. Duration of fighting reached its highest levels during this pair's PPI (See Figures 6 and 7). The duration of fighting did start to decline after five days of exceptionally high response rates. Following amygdala lesions, changes in the duration of fighting dropped radically in a fashion analogous to Pairs #10 and #14. Stabilization during PPII was remarkable and again the duration measure of fighting was more stable and changed
less abruptly than did the fighting rate. Succeeding the change back to the original current level, no changes in the duration occurred except for the increase in response variability. Except for the higher increment of the duration of fighting following septal lesions in Pair #6, the duration measurements are extremely similar to their stability during PPI and PPII. Changes following PPII to the original current level seemed to have more effect on Pair #13 than on Pair #6, but in neither case did the change remotely resemble the changes that took place following lowering of the current to its experimental level.

For Group S, Pairs #2, #3 and #11's fighting varied in a rather independent manner in terms of the two dependent variables (See Figures 8, 9 and 10). In no way did the changes in these subjects match corresponding changes in animals that received a lesion rather than an electrode placement.

On three occasions, the reliability of fight recording was simultaneously monitored by two observers. Observers were isolated from each other and watched the fighting from different angles. Figure 12 is a good example of the agreement between observers with respect to fight responses and changes in fighting rate.
Figure 12

Cumulative record depicting two-observer agreement in the simultaneous recording of fighting responses for a high-rate fighting pair.
HISTOLOGICAL VERIFICATION

Amygdaloid lesions were centered in the posterior lateral portion of the complex. Destruction common to all such lesions, unless otherwise specified, included the posterior cortical, medial and basolateral portions of the complex. Amygdala lesions also invaded the ventral lateral portions of the hippocampus, sometimes extensively and bilaterally. Less frequently involved structures were the frimbria of the hippocampus, posterior ventral lateral geniculate area, claustrum and minimal portions of the internal capsule.

Septal lesion destruction was usually complete, showing bilateral destruction of the lateral, and less extensively medial portions. Damage to the postcommissural septum and columns of the fornix varied from moderate to severe. Damage of other structures was limited, but occasionally extensive to the corpus callosum and stria terminalis.

Plate I shows typical damage resulting from both septal and amygdaloid lesions, and Figure 13 shows typical cumulative records for both amygdaloid-septal and septal-amygdaloid lesions.
Plate I

Photographs of 75 micra sections of both septal (A) and amygdala (D) target areas. (B) and (C) present typical damage of septal lesion, while (E) and (F) indicate extent of amygdaloid lesions.
Amygdala

Septal

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Figure 13

Typical cumulative records for baseline, postoperative period I and postoperative period II. (A), (B) and (C) represent these phases respectively for the septal-amygdaloid lesioned subjects, while (X), (Y) and (Z) are representative for those subjects receiving amygdaloid lesions first and septal lesions second.
DISCUSSION

There were two major advantages for using shock-induced aggression as a baseline measure of changes in emotional behavior. First, shock is an unconditioned stimulus that reliably elicits reflexive fighting. Secondly, pain-elicited fighting behavior is stereotyped in rats as well as other animals, and this emotional behavior is similar to the behaviors resulting from lesions of the septum in rats (Brady and Nauta, 1953, 1955). "Septal" rats revert to a defensive posture (Scott and Fredericson, 1951) and often overrespond to objects intruding into the environment in a manner similar to shock-induced fighting in normal rats. It is this aspect of pain-elicited fighting that makes it a valuable baseline measure in the study of changes resulting from septal and amygdaloid lesions.

The present experiment demonstrates the sensitivity of pain-elicited fighting in measuring behavioral changes resulting from septal and amygdaloid lesions or combined septal-amygdaloid, amygdaloid-septal lesions in one animal. In two early studies, it was found that septal irritability or hyperemotionality declined over a period of three weeks, as measured by a 7-point rating scale (Brady and Nauta, 1953, 1955). Results of the present study repeatedly showed that using a binary measure of fighting per shock presentation, the fighting rate stabilized and remained at a high level, while the cumulative duration of fighting per session declined in a function
similar to the decline in emotion as measured by a rating scale (Reynolds, 1963). With respect to the stability of the changes in fighting due to septal lesions, this tends to support the findings of Ahmad and Harvey (1968), who found that the elimination of septal irritability or emotionality after 45 postoperative days did not eliminate the increased pain-elicited fighting in rats. Schwartzbaum and Gay (1966) provide further support for this contention and show that there are residual effects that remain following dissipation of the septal syndrome.

A case in point is Pair #17 in the present study. Although the extent of the damage caused by the infection cannot be determined, their behavior during PPI suggests two possible analyses: (1) pain-elicited fighting is a more sensitive measure of emotional behavior than rating scales; and (2) duration of fighting is a measure of sensitization, while the shock-induced fighting rate measures changes in the pain-threshold. The stability of fighting rate, typified by Pair #17, represents the persistence of the change in threshold, as opposed to sensitization.

One method of testing for sensory threshold change is to establish a high fighting rate, e.g., minimal of 70%, and by reducing the current, lower the fighting rate to a maximum of 40% before giving septal lesions. It is speculated that the duration of fighting would decline, and the response rate remain at a relatively high and stable level, consistent with the results obtained from Pair #17. Following a lengthy postoperative period, the shock intensity would then be returned to its original level. A specific
requirement for this investigation is the use of a dependent variable not restricted by a ceiling effect, to measure changes which occur following the increased shock intensity.

The bizarre behavior of Pair #7 following amygdalectomy was found to be due to unilateral destruction of the amygdala and posterior damage to the lateral reticular formation. These data support Gloor (1960) that for lesions to be effective, they must be bilateral.

While it is difficult to ascertain if there are sensory changes following amygdaloid lesions, it should be noted that subsequent to septal lesions, Pairs #6 and #13's duration increased enormously and the fighting rate stabilized at a very high level. This indicates a possible decrease of the pain-threshold, resulting in increased levels of fighting. Following the stabilization of the rates in PPII, the current was returned to its original level. No significant increases in fighting rate or duration resulted subsequent to this condition change. The amygdaloid lesions may have also altered the level of sensory threshold to shock. However, this analysis becomes strained when one considers that lesions of the ventromedial hypothalamus override the amygdala unresponsiveness and produce rage behavior. Therefore, in this case hypothesizing that the changes are due to central sensory modification is unsatisfactory with regard to what is known about the functions of the hypothalamic nuclei.
FOOTNOTES

1 Emotion, emotional behavior or affective behavior, as used here are defined as the overt bodily changes in re­sponding mobility, contact with other animals or environmental stimuli expressed in terms of increased or decreased states of arousal.


King, F.A. Effects of septal and amygdaloid lesions on emotional behavior and conditioned avoidance responses in the rat. Journal of Nervous and Mental Disease, 1958, 126(1), 55-63.


