NLP-53

Reprinted from EXPERIMENTAL NEUROLOGY, Volume 20, Number 2, February 1968

EXPERIMENTAL NEUROLOGY 20, 197-202 (1968)

Effect of Amygdalectomy on Stimulus Threshold of the Monkey

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Received June 22; Revision Received September 25, 1967

To determine whether the apparent loss of fear of amygdalectomized monkeys is due to a loss of sensitivity, stimulus-threshold measurements were made using the galvanic skin response. Amygdalectomized subjects were found to have a *lower* threshold than normal monkeys. In addition they failed to respond differentially to different intensities of stimulus.

Introduction

A great number of investigators have attributed the defective performance of amygdalectomized monkeys primarily to a loss of fear based on the possibility that these animals are less sensitive to painful stimulation. This view stems from the obvious increase in tameness following amygdalectomy. Experiments to test this hypothesis have included tests of amygdalectomized subjects for stimulus- avoidance threshold (8), stimulus generalization with shock as a negative reinforcer for an operant response (5) unexpected stimulus (4) and stimulus avoidance conditioning (3). All of these workers concluded that most likely amygdalectomized subjects were normally sensitive to electrical stimulation, but a nagging doubt remained. Each of the experimental paradigms left open the possibility that the results obtained were specific to the particular task used in each experiment and that, indeed, elevated pain threshold might not be entirely ruled out.

The present study was undertaken to allay this doubt. We believed that the use of physiological measure such as the galvanic skin response (GSR), which in other circumstances was shown to be sensitive to amygdalectomy (2) would provide a reliable and sensitive indicator of threshold and at the same time settle the question as to whether the indicator itself was directly (i.e., peripherally) affected by the lesion. We had hope of success in this venture since the GSR had already been reported as effective in determining auditory thresholds in monkeys (7).

[1] This research was supported by NIMH grant MH, 12970.

Method

Subjects. Ten mature rhesus monkeys were used. All had been subjects in the three previous experiments (visual discrimination, habituation to tone, and a differential classical conditioning experiment). Six of the monkeys (three males, three females) had received single-stage bilateral ablations of the amygdala, group AM; the remaining four monkeys, constituting group N (two males, two females) had sham operations (1).

Apparatus. Skin resistance was continuously monitored with a Fels Dermohmeter (model 22A) and recorded on an Esterline-Angus graphic ammeter (model AW). Beckman silver–silver chloride electrodes filled with Offner electrode jelly were used. The electric stimuli were generated by a reliable constant-current d-c generator constructed in the laboratory and were administered via standard Grass silver-disc EEG electrodes half filled with Offner electrode jelly. Actual stimulus given on each trial was determined by monitoring the voltage passing through a 1000-ohm resistor mounted in series in the shock circuit. A foot switch which activated an event marker on the ammeter was used to record body movements.

Procedure. Each animal was restrained in a Foringer primate chair with ankles and wrists comfortably secured to the chair. One GSR electrode was attached to the palm of the right hind paw and the other to a shaved area on the right lower leg after thorough cleaning of the skin with Phisohex and acetone. Two stimulating electrodes were secured to the dorsum of the right forepaw less than 0.5 cm apart. Finally the eyes were covered with a simple-lined Elastoplast blindfold which could be molded to the skin. These preparations required approximately 30 min and allowed a sufficient period for hydration of the GSR electrodes. Finally the monkey was moved into a lighted, sound attenuated laboratory room where it was observed via a one-way window from the adjoining room which contained all recording equipment. Three series of stimuli, varying from 0.1 to 10 mamp, were administered in one session. In each series the stimulus intensity was increased in 0.2–0.5 mamp steps up to 2.0 mamp, and in 1.0 mamp steps to a maximum of 10.0 mamp and back down to 0.1 in the same stepwise fashion. Before the third series the blindfold was removed. Intertrial interval was randomized around 60 sec. Blank trials were given at the beginning and end of each run.

Scoring. A response was scored when there occurred a drop in skin resistance of 500 ohms or more with a latency of not less than 0.8 sec and not more than 5.0 sec from the onset of the stimulus. Amplitude of response was measured in kohms from the onset of the deflection to the peak.

Amplitude resistance values of each response were corrected for baseline resistance on each trial; $(R/BR) \times 100$. Responses contaminated by the animals' movement within 1 sec of the onset of the GSR response were

eliminated. Thus only responses occuring without movement were considered in the analysis.

Percentage GSR response within different stimulus intensity ranges was determined using 0.4-mamp divisions at the lower levels (0.01–2.0) and again using 2.0-mamp divisions for the entire stimulus-intensity series. Blank trials were scored for percentage response and amplitude of response.

Movement as a response was scored regardless of the occurrence of a GSR response if the response latency was not less than 0.8 sec and not more than 5.0 sec from the onset of the stimulus.

Results

Rate of response is shown in Fig. 1 as percentage GSR response for each group as a function of ascending and descending stimulus intensities.

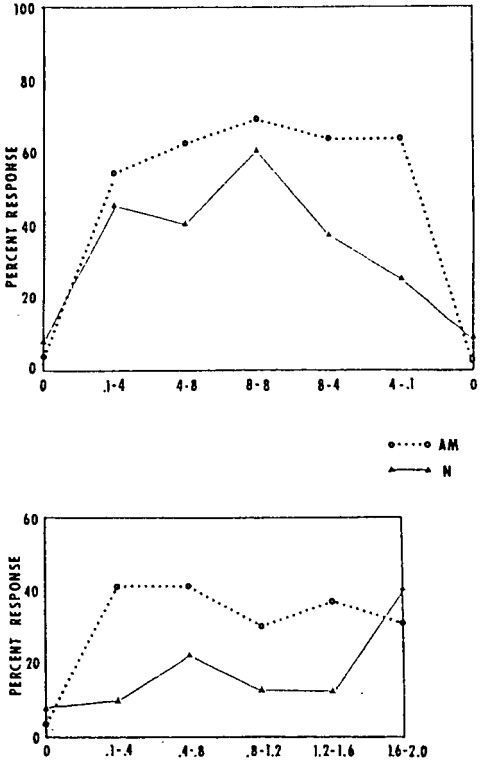

FIG. 1. Above: Curves of percentage GSR generated by three runs of stimuli of ascending and descending intensity (in mamp) by the amygdalectomized (AM) and control (N) groups. Below: A finer breakdown of stimulus values from .1 to 1.0 mamp, pooled ascending and descending values.

The amygdalectomized monkeys were more responsive to lower intensities (threshold of 0.1–0.4 mamp), whereas the control group's threshold was 1.6–2.0. (threshold" was considered to be 50% above nonstimulus response level).

The amygdalectomized group thereafter had a relatively flat curve across all intensities. There were no significant increases or decreases in the percentage of responses with change in stimulus intensity, ($\chi^2 = 2.75$, $p = .09$). A finer analysis of stimulus levels (Fig. 1, lower graph) between 0.1 and 2.0 mamp failed to show any differences with intensity within the amygdalectomized group. The controls, on the other hand, showed clearly lower response rates up to 1.6 mamp ($p = .05$, U test) and thereafter gradually increased responses as a fraction of shock level, ($\chi^2 = 9$, $p < .02$, $df = 1$) more marked in the descending side of the curve.

Amplitude of response is graphed in Fig. 2. (No zero-response trials were included since this would confound the amplitude variable with the rate of response variable). Again the amygdalectomized animals shows generally higher mean amplitude of response than the controls across all blocks. The difference is statistically significant only at the lower intensity levels, 0.1–4 mamp, whether ascending or descending ($p = .03$, U test). Finer analysis of this block (Fig. 1) shows that the control's threshold was approximately 2.0 mamp and, again, for the amygdalectomized monkeys this value lay at the very low level of 0.1 mamp.

The shape of the curves suggest that control animals produced increased amplitude of GSR with increase in stimulus intensity, but the small num-

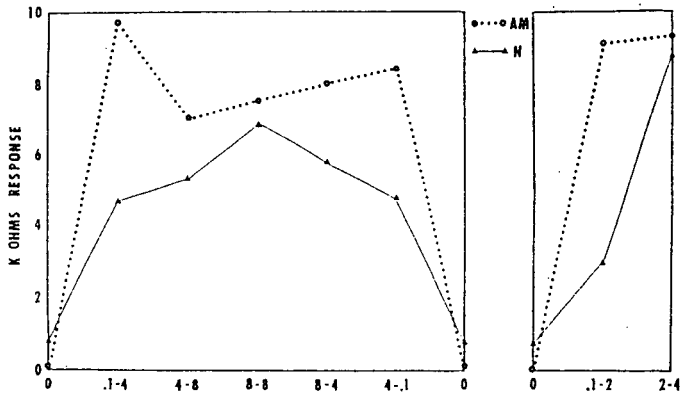

FIG. 2. Left: Curves of mean amplitude of response generated by three runs of stimuli of ascending and descending intensity (in mamp) by the amygdalectomized (AM) and control (N) groups. Right: A finer breakdown of the .1–4 mamp block of trials, pooled ascending and descending group values.

ber in the group made a good statistical test of the effect difficult since some blocks contained only one or two responses for some monkeys.

There were no significant differences between the groups in mean total trials given, mean total percentage response, mean latency of GSR response, mean intertrial interval, or in percentage movement responses (Table 1). Series 3 (eyes uncovered) elicited a twofold increase in movement, 5 sec post stimulus in both groups. Distribution of these responses were homogeneous across stimulus intensities for both groups.

TABLE 1

	Group N	Group AM
Total trials	62.0	68.3
Total % response	27.8	39.0
Latency GSR (sec)	2.9	2.6
Mean ITI	56.5	61.6
% Movement response	41.2	36.8

Discussion

These results clearly indicate that amygdalectomized monkeys do *not* have a higher than normal threshold to stimuli. Both rate and amplitude of response measures show a *lower* threshold. In addition, the rate of response measure for the control monkeys shows a lower threshold on the ascending than on the descending side of the scale; this is probably an effect of the novelty of the situation, an effect not shown by the amygdalectomized animals. This finding is consistent with the failure of amygdalectomized monkeys to show GSR orienting responses.

A bonus finding here was the dependence of response rate on stimulus level in the normal group. Again, no such smooth differentiation occurred in the amygdalectomized group. Individual curves showed that only one amygdalectomized monkey generated a centrally peaked curve. All four controls had typical centrally peaked curves.

These two results, reduced threshold of GSR to stimuli and indiscriminate autonomic response to varying stimulus intensities, give us some new evidence in interpreting the effects of amygdalectomy. The suggestion has been made that the amygdala ordinarily acts as a higher order control on the operation of the organism's servomechanisms (6). Removal of this control would be expected to leave the organism less sensitive to nuances in stimulus characteristics. This expectation is confirmed in the present study. From the results of other studies, however, it is clear that this

blunting of sensitivity can be overcome by differential reinforcement (5). Thus it is likely that the failure of amygdalectomized animals to show the "registrational" components of the orienting reaction is but another manifestation of the absence of this higher order control mechanism.

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