NLP-52

Bagshaw + Coppock

Galvanic Skin Response Conditioning Deficit in Amygdalectomized Monkeys

MURIEL H. BAGSHAW AND HAROLD W. COPPOCK[1]

Neuropsychology Laboratories, Stanford University, Palo Alto, California 93404

Amygdalectomized monkeys have depressed galvanic skin responses during tests of the orienting reaction to repeated presentation of simple tones. An effort to condition the galvanic skin response (GSR) with electric stimulation of skin was instituted. No evidence of conditioning of a weak conditional stimulus (light off) was obtained from the amygdalectomized monkeys in a differential classical situation, despite normal GSR to skin stimulation. Normal monkeys were found conditionable.

Introduction

Two previous studies (1, 2) demonstrated that there is a marked depression in the galvanic skin response (GSR) component of the orienting reaction of monkeys after amygdalectomy. In addition, respiratory and cardiac components of orienting were shown to be absent while electroencephalographic low-voltage fast-wave onset and ear movements were nearly normal. These results supported the hypothesis that the orienting reaction is composed of at least two phases and that one phase, labeled "registration" because of its relationship to habituation, was dependent on orderly autonomic reactivity.

Classical conditioning provides a paradigm for further investigation of the role and mechanism of this registration function in behavior. If registration is impaired, conditioning, which provides an over-ride on habituation, might also be expected to be impaired. The following experiment was therefore undertaken. Normal and amygdalectomized monkeys were tested for immediate and 3-sec trace conditioning to a light-offset stimulus with light onset as the neutral stimulus, and the rate of GSR as the response measure.

Method

Subjects. Two groups of immature monkeys (*M. mulatta*) were used.

A control group (group N) consisted of three unoperated and three sham-operated animals. Group AM consisted of six bilaterally amygdalectomized monkeys in which lesions had been made 1 year previously by subpial suction resection under direct vision via a transtemporal approach in a single operation. The sham procedure included all details of the same surgical procedure short of suctioning tissue. Reconstructions of the lesions have been published in a previous paper (1). All animals had been used in a study involving habituation to tone and had been trained in various visual discrimination tasks.

Apparatus. The light stimulus was supplied by a 40-w lumiline bulb which was cycled on and off by a 15-sec timer. A constant current d-c stimulator constructed in the laboratory supplied the electric stimulus.

A Fels Dermohmeter was used to measure the skin resistance which was recorded on an Esterline-Angus recorder. A simple phonograph pickup device was mounted on the monkey chair and allowed body movement to be recorded on one of the event markers of the recorder.

Procedure. Each animal was run for one 30-min session. He was restrained in a Foringer chair, fitted with GSR and stimulating electrodes. One GSR electrode[2] was securely taped to the cleaned plantar surface of each hind paw. Stimulating electrodes were made from nails which were placed in the closed fist of each forepaw which was taped shut. Sanborn electrode jelly was used for skin contact. There was a ground electrode strapped to the skin over the sternum to prevent passage of current across the heart. Stimulation did not cause severe pain.

The chair was placed in a sound-deadened, light-proof room. An initial stablization period of 15 min was allowed. Then a series of eighty trials was instituted. Each trial consisted of a cycle of 15-sec light on and 15-sec light off. A stimulus of 9mamp was administered after light offset on 50% of the occasions of light offset (diagram, Fig. 5) according to the 20-trial sequence OOOSOSSOSSSOSSSSOOOO. Four blocks of this sequence were presented. In 20-trial blocks 1 and 4, the CS (light off)-US (electric shock) interval was 0.5 sec. In 20-trial blocks 2 and 3, it was 3.0 sec (trace condition). No time intervals were inserted between blocks.

Scoring. A drop in skin resistance occurring between 0.8 and 5 sec after light offset (CS) on test trials was accepted as a CR if there was no movement and if it measured 500 ohms or more resistance drop from onset to peak. All interstimulus noncontaminated responses were also measured.

[2] One cm of the battery base was cut off and capped with a disc of saline-moistened cellulose sponge. Resistance between electrodes was <1000 ohms and comparative tests with Zn–ZnSO₄ electrodes showed no differences in basal resistance.

BAGSHAW AND COPPOCK

Amplitude, basal resistance level, and latency measures for each response were obtained. Amplitude was tallied both as raw kohms response and as

$$\frac{\text{kohms response} \times 100}{\text{basal resistance}} .$$

Results

Conditioned responses are shown in Fig. 1 (upper left) as percentage response to S+ (light off) on 36 trials for both groups across the four periods of conditioning. The control group showed an increasing incidence of responses across trials while group AM showed a decreasing incidence. These and all subsequent curves presented were found by linear regression analysis to show no significant deviations for linearity, allowing comparison of slopes (Table 1). Note that S+ test condition for the controls showed a positive (increasing) slope as trials progressed, which was significantly different from that of group AM. Group-AM data showed a negative slope suggestive of simple adaptation to the CS.

Percentage response to the neutral stimulus, S— (light on) on test trials (upper right of Fig. 1) shows that control animals were not simply being sensitized to all stimuli by the shock experience or by the temporal pattern
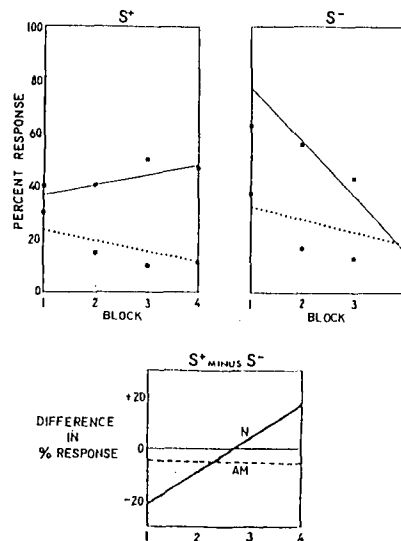

Fig. 1. Mean percentage GSR response on test trials across the four conditioning blocks for control and amygdalectomized groups: Upper left: CR (responses to light offset; upper right: responses to the neutral stimulus (light onset). Below: difference function between the responses to light offset and to light onset (S+ minus S—) for each group; □—□ represents group N; ⊙ - - - O represents group AM.

TABLE 1

Slope ± SD of Slope Estimate

Trials	(N)	Group N	Group AM	p Value
S + test	6	.0259 (± .030)	—.0390 (± .027)	.06
S + shock	6	—.0538 (± .144)	—.0133 (± .162)	NS
S— test	6	—.1010 (± .196)	—.0460 (± .113)	NS
S— shock	6	—.0797 (± .179)	—.0110 (± .120)	NS
S + test	3[a]	.0703 (± .046)	—.0474 (± .023)	.02
S + shock	3[a]	—1.04 (± .121)	—.0040 (± .048)	NS

[a] Subjects matched for shock responsivity.

of stimulus presentation since here there was a decreasing incidence of response to the unshocked stimulus (Table 1). Group AM reacted in a similar fashion to the S— but at a clearly lower rate of response.

Extinction in the control group is evident in Fig. 2, the composite distribution of responses across test trials within the 20-trial conditioning block. There were significantly more mean responses by the controls on two trial blocks 2 and 3 which followed a cluster of seven shock trials than on trial block 1 which followed only three shock trials or than on trial blocks 4 and 5 which followed four unshocked trials (Walsh test, $p < .05$). Group AM showed no such differences and responded at the same low level across the conditioning block.
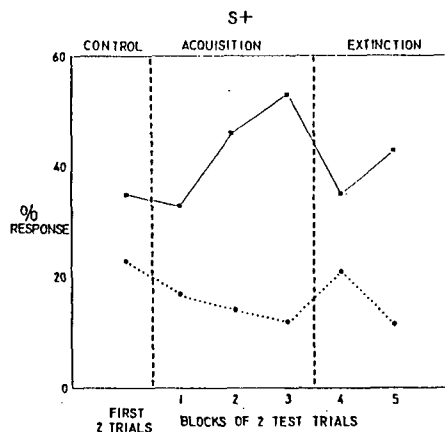

FIG. 2. Composite distribution of group mean percentage GSR response to test trials across the 20-trial conditioning block. Note that for the control group response rates increase on trials interspersed with shock (acquisition) and decrease with cessation of shock (extinction); ■—■ represents group N; ● - - - ● represents group AM.

Responsitivity to S+ and S— on shock trials is shown in Fig. 3. There was a significantly higher rate of responses to the electrical stimulus on shock trials in the control group than in group AM (Table 1). Yet the curve for UCRS decreases in slope as the CR curve is increasing. Responses to S— on shock trials also showed no evidence of sensitization as both groups have decreasing rates of response.
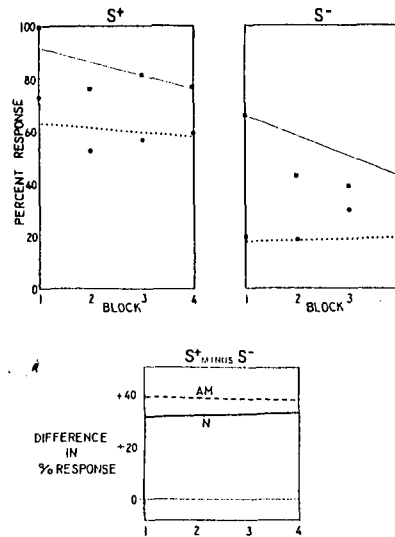

FIG. 3. Mean percentage GSR response on shock trials across the four conditioning blocks for control and amygdalectomized groups: Upper left: UCR (response to light offset plus shock); upper right: responses to the neutral stimulus (light onset). Below: difference function between the responses to light offset and to light onset (S+ minus S—) for each group; □—□ represents group N; O – – – O represents group AM.

Difference functions (S+) — (S—) for both groups on test trials (Fig. 1) and shock trials (Fig. 3) emphasize the major finding, i.e., the only difference function with a positive slope was that seen for the control group on test trials. All three other comparisons showed no group differences in slope of response to S+ vs. S—.

The possibility that the amygdalectomized monkeys failed to condition because 'of a lower level of reactivity to the electric stimulus itself was tested by comparing pairs matched for shock response. This was possible in three instances. Figure 4 shows the CR and UCR curves for these three pairs. Note that though UCR-response levels are comparable, CR curves diverge significantly like the larger group curves (Table 1).

Anticipatory responses were examined as a second measure of the effect of amygdalectomy in the conditioning situation. Responses in the 10 sec of
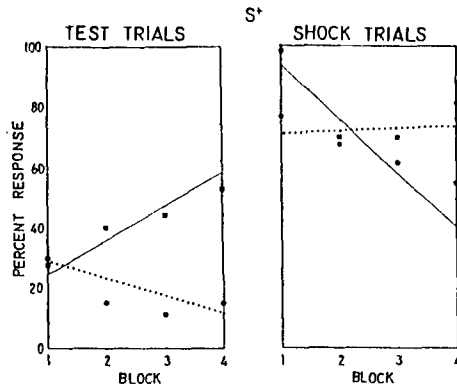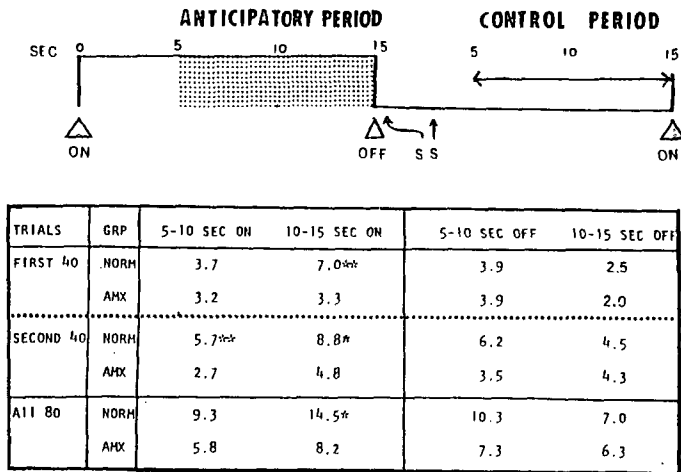
Fig. 4. Mean percentage GSR response to the S+ on test trials (left) and on shock trials (right) for three pairs matched for response to shock. (Three monkeys from each group); □—⊟ represents group N; O - - - ⊙ represents group AM.

S— just before S+ onset are shown in Fig. 5. In the last 5 sec of light on (S—) there were more responses per animal in the control group ($p < .05$, U test). This difference occurred in both halves of the experiment. In a control period (last 5 sec of dark) there were no differences between the groups. In addition during the middle 5 sec of light the normal animals increased the total number of responses from the first half (3.7) of the experiment to the second half (5.7) while there was no increase in group AM.

TRIALS	GRP	5-10 SEC ON	10-15 SEC ON	5-10 SEC OFF	10-15 SEC OFF
FIRST 40	NORM	3.7	7.0**	3.9	2.5
	AMX	3.2	3.3	3.9	2.0
SECOND 40	NORM	5.7**	8.8*	6.2	4.5
	AMX	2.7	4.8	3.5	4.3
All 80	NORM	9.3	14.5*	10.3	7.0
	AMX	5.8	8.2	7.3	6.3

* - p < .08

** - p < .05

Fig. 5. Mean number of GSR occurring in 10-sec period of light on just preceding light offset (CS) in the first 40 and in the second 40 trials for each group.

Another condition where anticipatory responses were sought was in those trials where the CS–US interval was 3.0 sec. It was reasoned that when the CS–US interval was lengthened from 0.5 to 3.0 sec in blocks 2 and 3 the delay of the shock would provide a 3-sec period of dark on shock trials where additional increased incidence of GSR in conditioned monkeys might appear. Figure 6 shows that the controls generated a mean of 22.5% responses on these 20 shock trials compared with only 1% in group AM ($p = .01$, U test). A control period (the last 3 sec of dark) showed 11.5% response for both groups. The mean latency of these conditioned anticipatory responses became progressively shorter from block 2 to block 3 (Table 2), i.e., closer in time to the CS. This is good evidence that they were being generated in relation to the shock reinforcement and were different from simple orienting responses which are known to increase in latency with repetition of a stimulus.
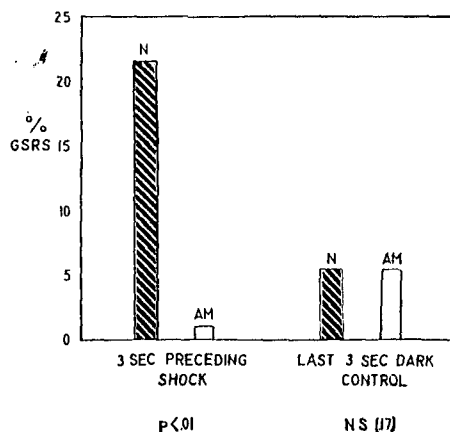

FIG. 6. Mean percentage CSR responses occurring in the CS–US interval (3.0 sec) in the second and third conditioning blocks for each group.

TABLE 2

LATENCY OF SHOCK ANTICIPATORY RESPONSES IN GROUP N

Period	Trial	Latency (sec)
2	1–5	2.40
	6–10	1.93
3	1–5	1.60
	6–10	1.45

Note.—$\chi^2 = 4.16$; $p < .05$.

Discussion

The data clearly show a defect in the ability of the amygdalectomized monkeys to show classically conditioned responses to a weak stimulus. Their complete lack of conditioned GSR on test trials is supported by their low incidence of anticipatory GSR in the later periods of the S— (just preceding S+) and in the prolonged preshock period in blocks 2 and 3.

Despite a moderate defect in the operated animals in the response incidence to electric stimuli, the data on matched pairs show that this is not the crucial variable. Similarly, response curves to the S—, although different in level, follow the same adaptive slope in both groups. If the amygdalectomized group were simply less "responsive" to shock but were conditionable one would expect to find a curve for the CR which was at a lower level but characterized by a positive slope. In addition, stimulus thresholds, as measured in these same animals by the GSR indicator, have been found actually *lower* in this group (3).

Gale and Stern (4) recently published a GSR-conditioning curve for human subjects strikingly similar to the one obtained for the normal monkeys in our experiment. They presented electric stimuli during the last 0.2 sec of an 8.0-sec tone. Adaptive curves shown for a neutral tone stimulus were also similar in slope to those reported here.

Two other studies in man, these obtained during learning, have shown a build-up in GSR reaction similar to conditioning until criterion was reached. In a paired-associate experiment by Kintsch (5) the GSR occurred whenever errors were made; in that of Grings (6) they were anticipatory responses during a selection task.

Our experiment confirms and extends the results of earlier ones which assayed the occurrence of a GSR in amygdalectomized animals in a novel situation. In the present experiment the outstanding finding is the absence of any normally occurring increment in GSR, either conditioned or anticipatory. This finding suggests that the amygdala is intimately involved in the temporal (both pre- and post-) extension of the occurrence of GSR with the repetition of "significant" events. In the normal group it is almost as if some sort of "rehersal" were taking place, a suggestion which is in support of the hypothesis that the GSR is crucially involved in the process of "registering" the events experienced by the organism.

References

1. BAGSHAW, M. H., and S. BENZIES. 1968. Multiple measures of the orienting reaction and their dissociation after amygdalectomy in monkeys. *Exptl. Neurol.* **20**: 175–187.
2. BAGSHAW, M. H., D. P. KIMBLE, and K. H. PRIBRAM. 1965. The GSR of monkeys during orienting and habituation and after ablation of the amygdala, hippocampus and inferotemporal cortex. *Neuropsychologia* **3**: 111–119.

3. BAGSHAW, M. H., and J. PRIBRAM. 1968. Effect of amygdalectomy on stimulus threshold of the monkey. *Exptl. Neurol.* **20** : 197–202.

4. GALE, E. N., and J. A. STERN. 1967. Conditioning of the electrodermeal orienting response. *Psychophysiology* **3**: 291–301.

5. GRINGS, W. W., and R. A. LOCKHART. 1966. Galvanic skin response during avoidance learning. *Psychophysiology* **3**: 29–34.

6. KINTSCH, W. 1965. Habituation of the GSR component of the orienting reflex during paired-associate learning before and after learning has taken place. *J. Math. Psychol.* **2**: 330–341.