Analysis of the response-inhibition concept of septal functions in "passive-avoidance" behavior¹

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Abstract

The effects of septal lesions in rats on "passive avoidance" of a food-reinforced lever response were studied in relation to strength of response tendency and intensity of shock. The lesion impairment related initially to strength of response. But, unlike control performance, increased shock failed to deter responding by septal Ss.

Introduction

Recent studies of the septal area of the brain by McCleary (1961) and Kaada et al., (1962) have led to a revision in conceptualization of its functional role. In line with electrophysiological data, they propose that the septal area acts as part of a more diffusely organized system in the inhibitory control of somatomotor activity. The evidence that such regulation might extend to the expression of complexly organized instrumental behavior is based upon the contrasting pattern of effects produced by septal lesions in what Mowrer (1960) characterizes as "passive" and "active" shock-avoidance behavior. While septal Ss have difficulty in inhibiting some established consummatory response to avoid punishment (passive avoidance), they tend to show more rapid development of avoidance responses in the conventional shuttlebox situation (King, 1958; Krieckhaus et al., 1964). These findings led McCleary and Kaada et al. to rule out possible disturbances in "emotionality" or learning.

In our previous work (Schwartzbaum et al., 1964), we examined some implications of the response-inhibition concept of septal functions in food-reinforced discriminative behavior, and demonstrated the applicability of the concept to nonshock conditions. The present experiment extends this analysis to passive-avoidance behavior. Taken at face value, the response-inhibition concept implies a direct relationship between septal-induced deficiencies in suppression of a punished response and the "strength" of the response. One would expect Ss to have less difficulty in inhibiting weak response tendencies compared to well established response tendencies. It also seemed of interest to examine the role of intensity of shock used as punishment. Although one might expect on the basis of the response-inhibition concept an inverse relationship between impairment in response suppression and intensity of shock, the possibility of disturbances in somatic reactivity to shock itself (Kaada et al., 1962; Krieckhaus et al., 1964) clouds any prediction.

Method

The Ss were 48 male Sprague-Dawley rats about

300 gm in body-weight. They were randomly assigned to test conditions formed by a 2 x 2 x 2 design in which operative treatment, strength of response and intensity of shock were varied. Half of the Ss received bilateral septal lesions electrolytically (2.0 ma DC anodal current for 20 sec.). The lesions produced the initial "hyper-emotionality" characteristic of such preparations and were comparable histologically with those described previously (Schwartzbaum et al., 1964). The other half of the Ss served as unoperated or sham-operated controls; some received small neocortical lesions as a brain-damage control.

Tests were conducted in a conventional lever box containing a lever, food hopper, and grid floor-bars through which scrambled shock could be administered. Ss were placed on a 1-hr. daily feeding schedule supplemented by a 5-7 gm pellet. After 1 session of "magazine training," shaping of the lever-response began. Responses were reinforced on a 100% schedule with 45 mg food pellets (P. J. Noyes). Half the Ss within each operative group were allowed a total of 25 self-initiated lever-presses for 1 session. The other half of the Ss were allowed 50 reinforced responses per session for 5 consecutive daily sessions. This was followed by 5 sessions of response suppression in which lever-presses were punished with shock. The schedule was arranged so that all punishment tests began on the 8th or 9th day postoperatively immediately after lever-training was completed. The first 10 responses in each of these sessions were never punished; S was allowed 20 min. in which to make these responses. The 11th response and every response thereafter during a 20-min. shock test was followed by shock through the grid bars (the lever itself was not electrified). Half the Ss in each group received 0.5 ma (peak current) and the other half 1.0 ma; shock duration was fixed at 0.5 sec. Each cell of the experimental design, thus, contained an N of 6.

Results and Discussion

Results are summarized in Table 1. Analysis of variance of the session-1 data disclosed overall Lesion effects ($F = 11.42$, $df = 1/40$, $P < .01$) and a Lesion x Shock-level interaction ($F = 6.90$, $df = 1/40$, $P < .05$). Septal Ss tended to take more shocks than control Ss. However, orthogonal comparisons of the treatment sums indicated that nearly all of the group differences occurred with high shock combined with a strongly established response ($F = 22.56$, $df = 1/40$, $P < .001$). Here septal Ss averaged 4.5 shocks compared to 1.3 for control Ss. The effects were evident within the first 5 min. and tended to persist during the remainder of

Table 1. Mean Number of Shocks*

No. Reinforced Responses	Shock Intensity ma	Septal		Control	
		Session-1	Total	Session-1	Total
25	0.5	2.3 (1-4)	4.7 (2-8)	2.2 (1-4)	2.7 (1-5)
	1.0	2.3 (1-4)	4.7 (1-10)	1.7 (1-2)	1.8 (1-3)
250	0.5	3.0 (1-5)	6.3 (1-11)	2.7 (1-4)	4.5 (2-7)
	1.0	4.5 (3-8)	6.3 (3-13)	1.3 (1-2)	2.0 (1-4)

*Excluding first shock in session-1.
Values in parentheses indicate range.

the session; occasional instances in which Ss received multiple shocks because of reflexive response to shock were scored only as a single shock.

Thus, a septal impairment in passive avoidance was not manifested initially when the response to be suppressed was not well established or, if it were well established, when shock intensity was relatively low. Septal Ss, unlike control Ss, took significantly more of the intense shock with a well established lever response compared to poorly established response ($F = 7.37$, $df = 1/20$, $P < .05$ using the pooled error variance of the septal Ss alone since it was somewhat higher than that of control Ss). Unlike the effects obtained with control Ss where increased shock intensity reduced the number of punished responses ($F = 8.00$, $df = 1/20$, $P < .05$), increased shock did not deter responding by the septal Ss ($F = 1.77$, $df = 1/20$).

Over the entire 5 sessions, septal Ss tended to take more shocks than did control Ss under *all* conditions. Although variability in performance of septal Ss was significantly greater, they still differed from control Ss in terms of the nonparametric median sign test ($\chi^2 = 4.85$, $df = 1$, $P < .05$). Performance by both groups was independent of the differential training on the lever, i. e., 25 vs. 250 reinforced responses. But while control Ss took significantly fewer shocks with the higher shock intensity ($F = 8.01$, $df = 1/20$, $P < .05$), septal Ss again were not deterred by increased shock. They showed absolutely no difference in performance in relation to shock intensity.

Any conclusions from this study must of course be

constrained by the limited values of each behavioral parameter examined. However, two points may be noted. First, the initial relationship of septal impairment to strength of punished response is in keeping with the response inhibition concept of McCleary (1961) and Kaada et al., (1962). Difficulties in response suppression would seem to depend in part upon the strength of the punished response. The absence of any relationship over the long term is not serious since none was found in control Ss.

However, the failure of increased shock to act as a deterrent for septal Ss and the general behavior of Ss during the punishment tests suggest an alternative explanation. A good deal of time was spent by Ss in approach-avoidance toward the lever and in exploration of the food hopper. Punished responses emerged out of this matrix of conflict and "agitation." The septal impairment in passive avoidance may, therefore, actually represent a *disinhibition* of suppressed responses generated by events associated with the food reward and perhaps with the shock itself. In other words, the difficulty in response inhibition may, at least in part, reflect greater susceptibility to disinhibiting influences.

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