ME cannot be attributed to any G1-G2 relational magnitude effects, it could reflect either the effect of a relationship between G1 reward on the measurement trial and previous G1 reward amounts or a simple, nonrelational effect of G1 reward. In common theoretical terms, the faster speeds following S as compared with L could reflect the occurrence of a frustration-drive increment on S trials (the relational interpretation) or a larger demotivation (hunger reduction) on L trials as compared with S trials (the nonrelational or absolute interpretation). Certain aspects of the present data as well as related previous literature, however, invite the conclusion that this ME is an absolute-magnitude effect rather than a relational effect of the type relevant to frustration theory. Thus, the appearance of the ME in the early stages of training in the present data argues against a frustration interpretation of the effect since conventional assumptions (cf. Amsel & Ward, 1965) regarding the growth of reward expectancy and the relation of frustration to expectancy preclude the occurrence of frustration early in training. Secondly, the within-Ss FE, a specific case of the ME in which the smaller reward is zero, would appear to be independent of G1 reward history prior to experience (within-Ss) with different G1 reward events (McCain & McVean, 1967). This result, discussed elsewhere (McHose, 1969), implies that the FE is completely independent of any relational effects, i.e., that the FE depends solely on absolute-magnitude effects.

The present data, in conjunction with previous findings, strongly suggest the presence of a ubiquitous absolute G1 magnitude effect on A2 speeds in double-alley studies such that A2 performance is inversely related to the amount of reward received in G1 on the measurement trial. The importance of this tentative conclusion lies in the fact that, given this effect, a wide spectrum of double-alley phenomena, including observations previously taken as indicative of frustration phenomena, may be seen as discrimination-learning effects (McHose, 1969).

The second finding of major interest, that the A2 speeds of Groups R and S in A2 were directly related to amount of reward received in G2 is consistent with previously reported A2 magnitude effects (Hamm, 1967). In the present data, however, the occurrence of a G2 magnitude effect indicates that Ss learned a discrimination based upon G1 magnitude of reward. Thus the hypothesis that differential (nonzero) reinforcements give rise to differential stimulus aftereffects which may serve as discriminative stimuli (Capaldi, 1967) is unequivocally supported by the present data. Finally, the observation that discrimination groups (Groups S and R) eventually ran more slowly to an S– reward of two pellets than did a nondiscrimination control condition (Group ND) demonstrates that a negative S– contrast effect occurs when reward events serve as discriminative stimuli. This finding suggests that the recent failure (Capaldi & Lynch, 1968) to obtain stable negative contrast (depression) effects with repeated shifts from large to small reward in simple instrumental conditioning is a function of the pattern of large- and small-reward trials rather than any peculiar stimulus properties of reward magnitudes as a discriminative stimulus.

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Aphagia and adipsia following lesions of the amygdala

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Lesions of the medial nucleus of the amygdaloid complex in rats produces aphagia and adipsia.

Grossman & Grossman (1963) report that for feeding behavior, "a very diffuse inhibitory mechanism may be located throughout the ventral amygdala." Lesions of this area resulted in increased food intake, while electrical stimulation inhibited feeding behavior. Additionally, lesions in the anteroventral portion of the amygdala decreased water consumption. The time course of food and water intake change is separate, indicating possible independent control mechanisms for each. Studies by Morgane & Kosman (1959) and Wood (1958) support these observations.

Opposite results were obtained with rhinencephalic lesions in cats by Green et al (1957) who found that the animals as a rule failed to eat voluntarily and so lost weight. Koikegami (1964) reports that bilateral amygdalectomy involving medial and basal nuclei in rats produced "hypophagia to some extent or perhaps a marked loss of appetite." Kling & Schwartz (1961) found profound and persistent aphagia which only a few animals will survive without forced feeding, resulting from total destruction of the amygdala. In contrast, Anand & Brobeck (1952) reported no changes in food and water intake of amygdalectomized animals.

The present study was undertaken to investigate changes in food and water intake with relatively restricted amygdala lesion sites.

METHOD
Ten male Sprague-Dawley (Holtzman) rats were bilaterally lesioned electrolytically

Fig. 1. Histological composite of the maximum (stippled) and minimum (black) extent of the lesions. The black area was damaged in all Ss.

for 30 sec at 2.0 mA using stereotaxic procedures under pentobarbital anesthesia. Three anodal lesions were made in each hemisphere 2.0, 4.0, and 6.0 mm anterior from lambda with a lateral distance of 3.5 to 4.0 from the midline and at a depth of 9.5 mm from the cortical surface. The lesioning electrode was made from 26-ga stainless steel hypodermic tubing with 0.5 mm of exposed tip.

Either frozen or celloidin-embedded sections of 30 microns were stained with cresyl violet to reconstruct the lesions.

Rats were housed individually with ad lib access to lab chow and water. Daily measurements were taken of weight and food and water consumption.

RESULTS

Figure 1 shows the histological evidence for the rat with the most extensive damage and the rat with the least extensive damage. For both of these rats and all other rats, major destruction is confined to the
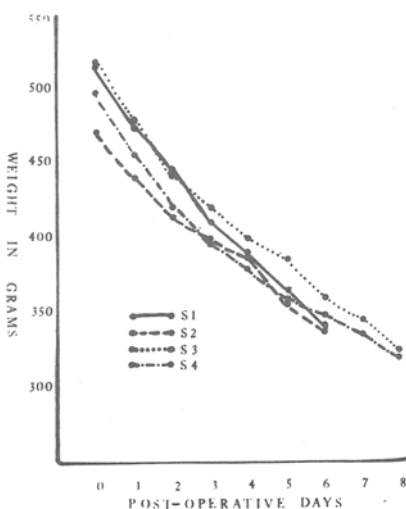

Fig. 1. Daily body weights for four representative Ss.

amygdaloid complex and the pyriform cortex. Note that the lesions in Fig. 1 do not infringe upon the lateral hypothalamic region.

In all 10 lesioned rats, histological evidence confirmed the lesion site to be in the amygdala, and in all 10, complete aphagia and adipsia resulted. Figure 2 shows daily weights for four representative Ss. Both food and water consumption immediately fell to zero in the postoperative period and remained there until the animal was sacrificed for histology. If the Ss were not sacrificed (pilot study preceding this study), they continued to lose weight until death occurred 8 to 14 days after the operation, with Ss at approximately 60% of preoperative body weight.

Ten control animals had either unilateral damage to the amygdala, bilateral lesions in the hippocampus, or insertion of the electrodes into all six bilateral amygdalectomy sites, but no lesioning current. In all cases, the rats suffered a sharp weight loss on the first postoperative day, but then stabilized their weight.

The animals were, in some cases, presented with a highly preferred food, chocolate. Amygdala lesioned animals generally slowed their weight loss when such food was available, but there appeared to be no deliberate eating involved. It appeared that Ss would get the chocolate mixture on their fur and inadvertently ingest some in the process of grooming. These animals actively resisted being fed chocolate and water mixture with an eyedropper, a procedure that normal rats readily accept.

DISCUSSION

The most striking characteristic of these results for amygdala lesions is their close resemblance to the aphasia and adipsia caused by lateral hypothalamic lesions. Because of the results of hypothalamic lesioning, this region has been considered to have a powerful influence on food and water motivation. Perhaps, in view of the present results, more consideration should be given to the role of amygdala in this kind of behavior.

These results are consistent with data reported by Koikegami (1964) and by Kling & Schwartz (1961). The aphagia and adipsia reported here appears to be more complete than that which Koikegami reported, which suggests that the medial nuclei, which, as the only amygdaloid nucleus totally destroyed in all of our animals, is more closely correlated with the food and water intake than the basal nuclei which was also lesioned in Koikegami's study.

Previous investigators have reported seemingly incompatible results of amygdalectomy producing either hyperphagia or hypophagia. However, a few factors can be pointed out which may

differentiate the results of this study from amygdalectomy-induced hyperphagia. Notice that three relatively large lesions are made in both nuclei. This suggests that destruction of the amygdala is fairly complete in the anterior-posterior dimension. In addition, there is extensive destruction of ventral structures due to the electrode tip being deeply placed. This results in the consistent total destruction of the cortical nucleus of the amygdala and a large portion of the pyriform cortex along with frequent damage to other nuclei of the ventral amygdala. The consistent destruction of such ventral structures in both the anterior-posterior and ventral-dorsal directions may well be the prime factor for differentiating hypophagia from hyperphagia following amygdalectomy. Although destruction of the pyriform cortex may be the crucial variable, it should be pointed out that experiments using the open suction technique, such as the work of Morgane & Kosman (1959), have caused extensive damage to the pyriform cortex but have obtained hyperphagic animals.

Taking an overview of research on the amygdala, we find data suggesting inhibitory and excitatory mechanisms for both food and water intake in the amygdaloid complex. Unfortunately, Grossman and Grossman's "diffuse inhibitory mechanism" and the lesion sites for this study both lie in the ventral amygdala and make localization of neural elements very difficult. However, data now available appear to assign a powerful and complex role to the amygdala in controlling hunger and thirst.

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