Table 2. Scores of pre- and postoperative inhibitory trials and errors, including the criterion, in group 2 animals (1-minute intertrial interval). *T*, trials; *E*, errors.

Dog No.	Preoperative		Postoperative	
	T	E	T	E
Dorsolateral prefrontal lesions				
38	305	163	50	3
39	135	75	60	8
40	150	81	50	5
Medial prefrontal lesions				
41	100	48	105	36
42	170	63	130	57
43	220	76	95	35

of disinhibition were scored if the animal responded positively to the presentation of an inhibitory conditioned stimulus. Fifteen positive and 15 inhibitory trials were presented daily to each animal. In group 1 ($N = 13$), trials were separated by 15 seconds, while in group 2 ($N = 6$) they were separated by 1 minute. Training continued until the animals attained the criterion of 50 correct positive responses and 45 correct inhibitory responses in 100 consecutive trials. Upon completion of training, two animals in group 1 were retested after a 10-day interval; these served as unoperated controls. The remaining dogs were retested 7 days after the following cortical areas were ablated: (i) the dorsolateral portion of the proreal gyrus and the anterior part of the orbital gyrus, which, taken together, constitute the dorsolateral aspect of the dog's prefrontal cortex; (ii) the cortex of the upper medial wall of the prefrontal lobe, that is, sparing the subgenual and subproreal regions; (iii) the posterior cingulate cortex.

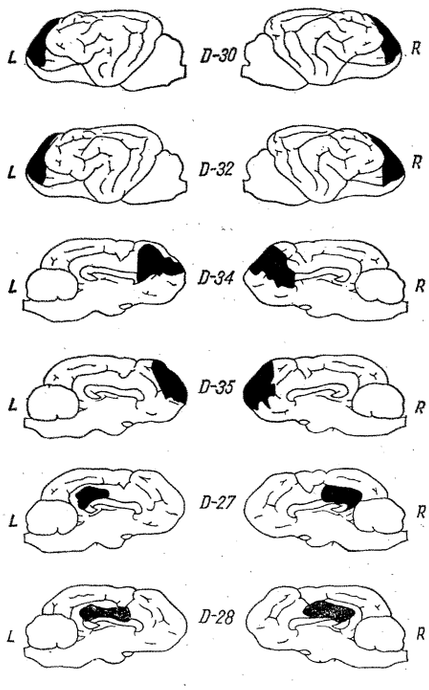

Fig. 1. Representative reconstructions of the prefrontal lesions described in the text. (Top two) Lateral reconstructions of bilateral ablation of the proreal and anterior orbital gyri in dogs Nos. 30 and 32. (Middle two) Medial reconstructions of bilateral ablation of the medial prefrontal cortex in dogs Nos. 34 and 35. (Bottom two) medial reconstructions of bilateral ablation of the posterior cingulate cortex in dogs Nos. 27 and 28.

Three months after operation the animals were killed and their brains were fixed in formalin, sectioned at 20 μ, and stained with thionine. Representative reconstructions of each type of lesion are shown in Fig. 1.

Since responses on positive trials were generally unaffected, the results are shown for all the dogs on the inhibitory trials only. In Table 1 data are given for dogs in group 1. It is seen that both the unoperated animals and the animals in which the posterior cingulate area was removed were not impaired, whereas lesions of both the dorsolateral and medial prefrontal cortex produced temporary disinhibition of inhibitory CR's. In contrast, in dogs in group 2 the impairment on inhibitory trials followed only the medial lesions. Ablation of the dorsolateral prefrontal cortex was without effect (Table 2).

Examination of other aspects of the behavior indicates that, in the initial postoperative period, the dogs with medial lesions displayed marked food-oriented behavior characterized by sniffing, licking, and gazing at the cup throughout the testing session. In addition, they scratched the food tray and performed many intertrial CR's. On the other hand, the dogs with dorsolateral prefrontal lesions were not affected by a similar increased "drive" for food. Their impairment on the 15-second-interval schedule seems to be a reflection of somatoperseverative tendencies which have been described previously in animals with prefrontal lesions (*3*). Short intervals between trials appear to produce an increase of this type of error in animals in which the dorsolateral prefrontal cortex is ablated.

The findings in the present study add support to the conclusion derived earlier from studies on monkeys (*4*) that different "inhibitory" defects follow selective lesions restricted to the orbital and dorsolateral frontal cortex. Future research in this area must involve testing procedures other than instrumental conditioning in order to permit a more precise evaluation of the view that different types of disinhibition result from different prefrontal lesions (*5*).

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References and Notes

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Xenon Hydroxide: An Experimental Hazard

An ~ 0.39-g sample of xenon tetrafluoride in an evacuated silica bulb equipped with a Hoke valve was dissolved in 1.5 ml of distilled water. The solid reacted vigorously and the fumes of hydrogen fluoride were pumped off immediately. Evaporation of the resulting clear solution at ambient temperature left a white solid. Previous runs in nickel weighing bottles indicated that this residue had a composition of $Xe(OH)_4$ or $XeO_2{,}2H_2O$. This solid detonated when warmed under vacuum above 30° or 40°C and completely shattered the enclosing vessel (*1*).

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