ACTA NEUROBIOL. EXP. 1972, 32: 417–429

Lecture delivered at Symposium "The frontal granular cortex and behavior"
held in Jabłonna near Warszawa
August 1971

A FURTHER STUDY ON THE EFFECT OF MEDIAL PREFRONTAL ABLATION UPON CLASSICAL DEFENSIVE CONDITIONED REFLEXES IN DOGS

Stefan SOŁTYSIK, Krystyna JAWORSKA and Anna SZAFRAŃSKA-KOSMAL

Department of Physiology of the Nervous System,
Institute of Psychoneurology, Pruszków
and
Department of Neurophysiology, Nencki Institute of Experimental Biology
Warszawa, Poland

Abstract. In 8 dogs with removed medial prefrontal cortex, the positive and differential classically conditioned leg flexion responses and accompanying heart rate reactions were not changed or slightly reduced. In some animals a transient increase in cardiac responses to shock was also observed.

Although clinical observations strongly suggest that removal of the prefrontal cortex or severing the connections between it and other cerebral structures reduces fear and anxiety (Moore et al. 1948, Heath 1949, Landis 1949), the experimental evidence is still controversial. Typically, the effect of prefrontal cortical ablation on defensive behavior in animals has been studied by using three basic types of learning procedures: (i) retention or acquisition of avoidance conditioned responses, (ii) conflict between approach and avoidance learned behaviors, and (iii) classical defensive conditioning.

The results of studies using the first procedure showed decrease in avoidance performance after prefrontal ablation in dogs (Afanasev 1913) [1], cats (Zieliński et al. 1962, Zieliński 1966) and monkeys (Water-

[1] Afanasev's doctoral thesis was misquoted by several authors (Auleytner and Brutkowski 1960, Brutkowski 1965, and Sołtysik and Jaworska 1967). Careful reading of this monograph reveals that the author used an avoidance rather than

house 1957). Furthermore, in rats trained after prefrontal ablation the latencies of avoidance responses tended to be longer than in normal animals (Dąbrowska and Jarvik 1970). These findings do not necessarily indicate that the fear reactions are attenuated and, in fact, alternative explanations like hyperactivity (Maher et al. 1962) or instrumental response impairment (Brutkowski 1965) have been proposed.

The issue was not further clarified by the data obtained from experiments in which a conflict between food rewarded approach responses and avoidance was created (Lichtenstein 1950, Streb and Smith 1955). In these instances the diminution of avoidance could have been interpreted as an increase in hunger drive with no or only insignificant changes of fear in either direction.

In this context, an experimental analysis of classically conditioned defensive reflexes may help to elucidate the physiological significance of the prefrontal lobes for acquired behavior to aversive stimuli. There are, however, surprisingly few publications in which changes in classically conditioned reactions were studied in prefrontal animals. Auleytner and Brutkowski (1960) reported enhancement of classical conditioned leg flexion reflexes and concomitant disinhibition of these responses to inhibitory stimuli in 3 dogs. In the same paper the authors described similar effects of prefrontal ablation in 3 other dogs trained in defensive salivary reflexes reinforced by acid injection into the mouth. Sołtysik and Jaworska (1967) were unable to replicate these findings using leg flexion response in 4 dogs. Except for slight shortening of the latency of the conditioned motor response, there was no evidence that the prefrontal lobectomy increased reactivity to either conditioned or unconditioned aversive stimuli. In fact, they observed no disinhibition to differ-

---

classical defensive training procedure. The shock US was used only to maintain fading conditioned responses or to facilitate differentiation. It is worth mentioning that in his 4 prefrontal dogs a weakening of positive CRs was observed and that conditioned inhibition in one dog was *improved* after the operation. Afanasev also notices some "temporary decrease of differentiation" but again, by the present standards it was rather extremely good retention of discrimination. Two of his dogs were trained to differentiate the tones of harmonium and after prolonged training were able to respond to the CS tone c' but did not react to any other tone, even close tones as d' or b' flat. One of the dogs could even differentiate d' from d' sharp, i.e., half tone difference in pitch. Immediately after the operation the differentiation was preserved. A few days later, after applying the US because the CR to c' was unstable, one of the dogs showed generalization to the tones d' and b' flat, i.e., the tone immediately higher and lower from the CS tone c'. This "impairment" in discrimination lasted for two and a half weeks. Afterwards, the differentiation improved to the pre-operative level. This fantastic performance was observed in spite of the increased motility and "impulsiveness".

ential stimuli but rather some diminution of heart rate responses to conditioned stimuli and to the shock.

Because of this disagreement and because of the small number of animals used in the two studies mentioned, a new series of experiments was carried out on 8 dogs using the same leg flexion conditioning procedure. The data obtained from these animals before and after the medial prefrontal ablation are presented in this paper. The main goal of this experiment was to find out, first, whether the prefrontal dogs do have exaggerated defensive responses, and second, whether the prefrontal dogs are disinhibited in their responses to differential conditioned stimuli.

MATERIAL AND METHODS

Eight mongrel dogs were trained during a 5–7 month period in a standard semi-soundproof room using classical leg flexion conditioning procedure. Both positive and differential conditioned stimuli were used. Each daily session consisted of 10 positive and 10 inhibitory trials randomly distributed. As a conditioned stimulus (CS) a rhythmic click was delivered via the loudspeaker situated in front of the animal. This stimulus was always followed by the shock from the DISA constant current stimulator applied to the right forepaw. This nociceptive unconditioned stimulus (US) lasted for 0.5 sec and consisted of 25 pulses of 2 msec duration and 18 msec interpulse intervals. The intensity of shock was 2 ma above the threshold stimulation which elicited the flexion of the paw. The threshold was tested at the beginning of every session. This US elicited a vivid flexion response but no vocal or generalized reactions were observed during its application. Duration of the CS was 5 sec and since the US onset coincided with the CS termination, the CS-US interval was also 5 sec. The differential stimulus (DS) was the same rhythmic click sound delivered from the loudspeaker placed on the right wall of the room. This DS was never paired with shock and most of the dogs ceased reacting to it, with the exception of the dog, Bob, who never learned to differentiate between the two loudspeakers.

The stimulating electrodes were firmly attached to the shaved distal part of the right foreleg. The stimulated leg was attached by a string to a switch which closed a cirruit when the leg was raised to a certain pre-set height. All the dogs were prepared with carotid artery loops in order to facilitate the recording of heart rate changes elicited by CS, DS and US. Heart rate and leg flexions were recorded on a kymograph. Data was analyzed by reading the latencies of motor responses and calculating the pulse rate from the inter-beat intervals.

Surgery and verification of lesions. The prefrontal ablation was per-

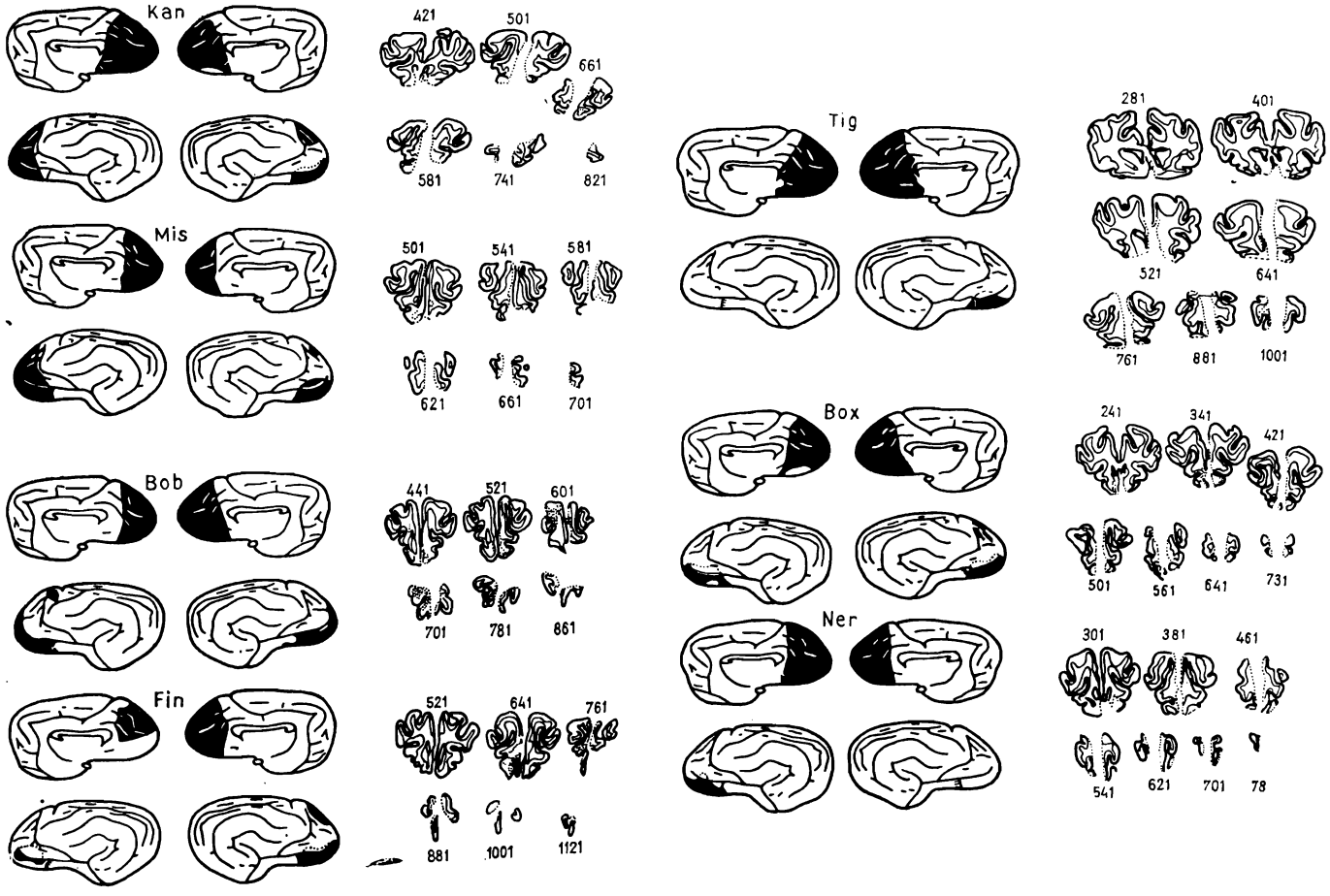

Fig. 1. Reconstruction of lesions in medial and dorsolateral aspects of frontal lobes. The numbers refer to the sections which were chosen to exemplify the extent and location of the lesion in the deeper portions of the lobe.

formed aseptically and 10–15 days were given after the operation for full recovery. The operation consisted of bilateral removal, by suction, of the medial prefrontal cortex. After the complection of the experiment the animals were sacrificed, their heads perfused first with saline solution followed by 10% formalin. The brains were then removed and subjected to histological examination [2]. Frontal lobes were sectioned coronally at 20 μ and every tenth section was alternately stained by the Klüver and Nissl method. The detailed analysis was carried out in order to obtain the reconstruction of the lesion, as shown in Fig. 1. Table I presents the detailed account of the cortex removed. To facilitate reading of this Table, Fig. 2 shows the areation of the canine frontal cortex according to Kreiner (1961).

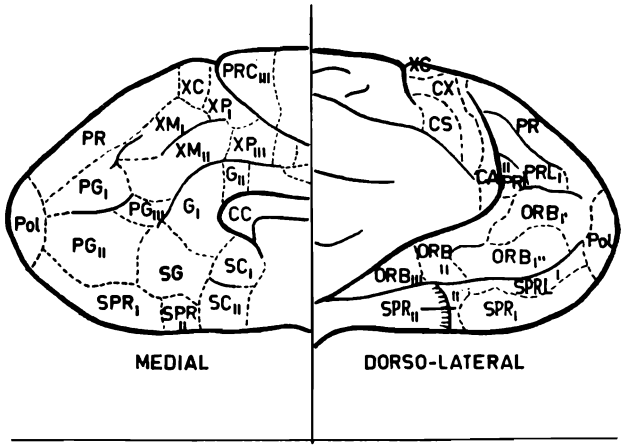

Fig. 2. Myeloarchitectonic areation of the frontal cortex in dog by J. Kreiner (1961). Abbreviations: CA, area composita anterior; CC, corpus callosum; CS, area composita sigmoidea; CX, area composita precruciata; G, area genualis; ORB, area orbitalis; PG, area pregenualis; POL, area polaris; PR, area prorea; PRL, area prorea lateralis; SC, area subcallosa; SG, area subgenualis; SPR, area subprorea; SPRL, area subprorea lateralis; XC, area precruciata centralis; XM, area precruciata medialis; XP, area precruciata posterior.

RESULTS

The results of the comparison of 100 reactions of the dogs before and 100 reactions after the ablation are shown in six Tables. Table II shows how many dogs significantly changed their cardiac or somatic responses

[2] The dog War developed a cerebral abscess and died two months after the operation. His brain was not used for histological examination. The data from this dog are not included in Fig. 3, and Table I.

TABLE I

Detailed account of the cortex removed. (Abbreviations the same as in Fig. 2.)

Dog		Medial surface											Lateral surface					
		Pol	PR	PG_I PG_{II}	PG	XM_I XM_{II}	XC	G_I G_{II}	SG	SC_I SC_{II}	SPR	Genu CC	Pol	PR	ORB	SPR_I	SPR_{II}	CX CS
Bob	R	xxx	xx	xxx	xxx	x	o	x	xx	x	xxx	o	xx	x	x	xxx	x	o
	L	xxx	xx	xxx	xxx	x	o	o	x	o	xx	o	xxx	xx	o	xxx	x	x
Box	R	xxx	xx	xxx	xxx	xx	o	x	xxx	xx	xxx	o	x	x	x	xxx	x	o
	L	xxx	xx	xxx	xxx	x	o	x	xx	o	x	o	x	o	x	xx	x	o
Fin	R	xxx	xx	xxx	xxx	x	o	x	xx	o	x	o	x	xx	x	xxx	o	o
	L	x	xx	xx	x	xx	o	x	x	o	o	o	x	x	x	x	o	o
Kan	R	xx	x	xxx	xxx	xx	o	xx	xxx	x	xx	xxx	x	xx	x	xx	x	o
	L	xxx	xx	xxx	xxx	xx	o	xx	xxx	xx	xxx	xxx	xxx	xxx	x	xxx	x	o
Mis	R	xxx	x	x	x	o	o	o	o	o	xx	o	x	x	x	xxx	x	o
	L	xxx	x	xxx	xxx	x	o	o	xxx	o	xx	o	x	xx	x	xxx	x	o
Ner	R	xxx	xxx	xxx	xxx	x	o	x	xx	o	xxx	o	o	o	o	o	o	o
	L	xxx	xxx	xxx	xxx	xx	o	x	xx	x	xxx	o	o	x	x	xx	x	o
Tig	R	xxx	xxx	xxx	xxx	xx	xx	xx	xxx	xx	xxx	xxx	o	x	x	xx	xxx	o
	L	xxx	xx	xxx	xxx	xx	o	xx	xxx	xx	xxx	xxx	o	o	o	o	o	o

Symbols: R, right hemisphere; L, left hemisphere; xxx, total ablation; xx, ablation of more than 50%; o, intact.

TABLE II

Changes in heart rate responses to US, CS and DS after prefrontal ablation

Stimulus	Number of dogs whose responses			Number of dogs whose responses		
	did not change	increased	decreased	did not change	increased	decreased
Unconditioned	2	3	3	5	0	2
Conditioned	6	1	1	7	0	0
Differential	5	0	3	2	1	4
Period of testing	2 weeks after ablation			2 months after ablation		

Changes in conditioned leg flexion responses after prefrontal ablation

Stimulus	Number of dogs in which latency of leg flexion response			Number of dogs in which percentage of leg flexion response		
	did not change	increased	decreased	did not change	increased	decreased
Conditioned	5	2	1	5	1	2
Differential	—	—	—	5	1	2

Period of testing: 2 weeks after prefrontal ablation. In each animal the performance during 100 trials before and 100 trials after the operation was compared; difference in mean latency was estimated by the *t*-test, difference in fraquency of responses was evaluated by Chi square.

after the operation. It is evident that there were no systematic changes either two weeks or two months following the prefrontal decortication. Most of the dogs did not change their performance and some of them, especially 2 months after the operation, had their responses reduced. Figure 3 presents the results from 7 dogs (excluding War). Arithmetic means and standard errors are plotted for cardioacceleratory responses to US, CS and DS before and after surgery. This graphic representation of the data visualizes the lack of any tendency to augment the emotional reactivity to aversive stimuli following prefrontal ablation; instead, rather an opposite effect, i.e., a reduction of responses is seen for the heart rate reaction to the DS immediately after the operation and to the US 2 months later.

The Tables III through VII need little comment. Responses to the shock US (Table III) increased in three dogs soon after the ablation. However, 2 months later none of the dogs displayed significantly greater cardiac responses than before the operation, whereas 2 dogs displayed significantly reduced responses. Even fewer changes are seen in responses to CS (Table IV). Only one dog increased the conditioned heart rate response and one decreased it. Two months later, all the cardiac responses of the dogs to the CS were at the pre-operative level. The heart

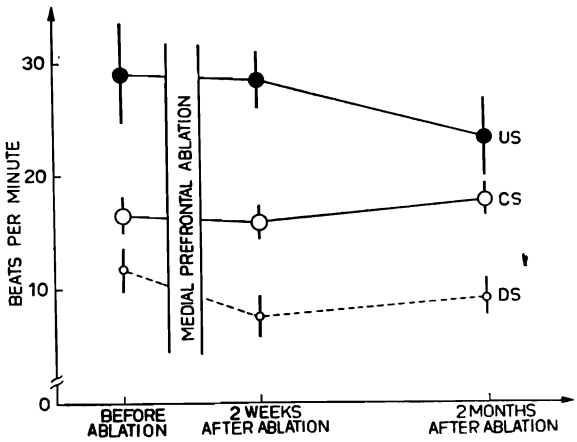

Fig. 3. The average amplitude of cardiac acceleratory response to unconditioned (US), conditioned (CS) and differential (DS) stimuli in a group of seven dogs. Vertical bars show the standard errors of the means. Note that the cardiac responses to the CS did not change after the operation while the responses to the DS diminished slightly immediately after the ablation. Also the responses to the shock US were reduced 2 months after the operation. The amplitude of cardiac response is defined as a difference in heart rate measured during and before presenting of a stimulus.

TABLE III

The effect of prefrontal medial ablation upon the heart rate (H. R.) response to the US

Dog	H. R. response before ablation	Change in H. R. response 2 weeks after ablation	Change in H. R. response 2 months after ablation
Bob	33.71	— 7.79*	— 6.23°
Box	23.81	— 0.26°	— 0.49°
Fin	27.58	+ 3.49°	—10.24***
Kan	17.02	+13.23***	+ 1.04°
Mis	49.34	—10.05***	— 4.54°
Ner	11.48	+11.69***	+ 6.48°
Tig	39.96	—16.04***	—30.08***
War	24.74	+11.68**	?

The H. R. response to US was defined as a difference of the heart rate during the 2nd second after the application of shock US and the heart rate during the 5 sec period immediately preceding the onset of CS. Each score is calculated from 100 responses.

° non significant; * significant at the 0.05 risk level; ** significant at the 0.01 risk level; *** significant at the 0.001 risk level.

rate responses to the differential stimulus (Table V) were significantly diminished in 3 dogs. In the 4 other dogs, they were slightly reduced but the difference did not reach the 0.05 level of confidence; in the eighth dog, there was a negligible and insignificant increase. In a group of 8

TABLE IV

The effect of prefrontal medial ablation upon the heart rate (H. R.) response to the CS

Dog	H. R. response before ablation	Change in H. R. response 2 weeks after ablation	Change in H. R. response 2 months after albation
Bob	20.27	—12.26***	—4.67°
Box	18.16	— 1.68°	+1.76°
Fin	22.57	+ 0.40°	—2.45°
Kan	11.17	+ 4.22**	+1.25°
Mis	16.06	— 0.12°	+3.10°
Ner	10.93	+ 3.62°	+3.57°
Tig	16.59	+ 0.97°	+0.77°
War	20.06	+ 5.65°	?

The H. R. response to CS was defined as a difference between the heart rate during the 5 sec of the CS and the heart rate during the 5 sec period immediately preceding the onset of CS. Each mean is calculated from 100 responses. Statistical significance as in Table III.

TABLE V

The effect of prefrontal medial ablation upon the heart rate (H. R.) response to the DS

Dog	H. R. response before ablation	Change in H. R. response 2 weeks after ablation	Change in H. R. response 2 months after ablation
Bob	20.10	—6.20**	—5.92*
Box	6.52	—7.99***	—0.92°
Fin	18.88	—9.20***	—7.68***
Kan	4.93	—1.97°	+0.07°
Mis	10.15	—2.57°	—3.73*
Ner	10.92	+0.94°	+5.56
Tig	10.70	—2.73°	—6.20*
War	11.87	—0.74°	?

The heart rate response to the differential stimulus was defined as a difference between the heart rate during the 5 sec of the DS and the heart rate during the 5 sec period immediately preceding the onset of the DS. Each mean is calculated from 100 responses. Statistical significance as in Table III.

dogs this result (2 weeks after ablation) is significant at 0.02 level by the two-tailed Wilcoxon matched pairs signed ranks test. However, it seems unfair to use this test since in 5 of 8 animals the differences were very small and statistically not significant.

Latency and percentage of somatic conditioned leg flexion responses (Table VI and VII) also showed no changes in 5 animals and no systematic changes in the remaining three. Even the dogs exhibiting extreme scores, like Bob, unable to inhibit the leg flexion CR to the DS, and Kan, who had extremely good inhibition, did not change their performance after the operation at all. The well established post-operative differentia-

TABLE VI

The effect of prefrontal medial ablation upon the latencies of conditioned leg flexion

Dog	Mean latency and number of responses in 100 trials before ablation		Mean latency and number of responses in 100 trials after ablation		Difference	t	p
Bob	0.910	$n = 100$	0.951	$n = 100^{o}$	+0.041	0.41	0.682
Box	2.717	$n = 91$	3.404	$n = 79^{*}$	+0.687	4.35	0.001
Fin	2.653	$n = 93$	2.491	$n = 93^{o}$	—0.162	1.08	0.282
Kan	2.424	$n = 84$	1.627	$n = 97^{**}$	—0.797	5.93	0.001
Mis	1.927	$n = 97$	1.859	$n = 100^{o}$	—0.135	0.68	0.497
Ner	1.646	$n = 92$	1.946	$n = 90^{o}$	+0.300	2.24	0.026
Tig	2.081	$n = 90$	2.307	$n = 68^{***}$	+0.226	0.73	0.466
War	2.689	$n = 74$	2.937	$n = 70^{o}$	+0.248	1.58	0.116

Note that, since each dog was tested in 100 trials before and in 100 trials after the operation, the number of responses (n) used for calculation of means represents also the percentage of correct responding. Statistical significance of differences in the number of correct responses, before and after ablation was computed by using the Chi square and is shown in the third columm.

° not significant; * significant at 0.05 level of confidence; ** significant at 0.01 level of confidence; *** significant at 0.001 level of confidence.

TABLE VII

Percentage of correct responses (inhibition of leg flexion CR) to differential stimulus before and after prefrontal ablation

Dog	Bob	Box	Fin	Kan	Mis	Ner	Tig	War
Before ablation	4	88	72	100	73	78	83	85
After ablation	4°	100***	76°	99°	66°	62*	76°	94*

Statistical significance of the differences by Chi-square: ° not significant; * significant at 0.05 level of confidence; *** significant at 0.001 level of confidence.

tion in Kan was even more impressive if we notice (in Table VI) that he improved his CR scores by increasing the percentage of correct responses to the CS from 87 to 97 and shortened the leg flexion CR latencies by nearly 0.8 sec.

General behavior. This lack of systematic changes in the defensive conditioned reflexes contrasted with the altered general behavior of the dogs. They appeared more aggressive, particularly towards the other dogs and also seemed more voracious. No specific attempts were made to measure these changes, however.

Differences in lesion locus did not appear to be correlated with the behavior of the dogs. For example, two dogs (Fin and Tig), who displayed markedly reduced cardiac responses to US after the ablation, had evidently different lesions: Tig's lesion included removal of genu corporis callosi and subcallosal and subproreal gyri, whereas in Fin, these structures were spared in the left hemisphere. On the other hand, Box and Ner, two dogs with almost identical lesions and who displayed increased latencies of leg flexion after the operation, differed in their cardiac responses to the US. The responses of Box did not change from the preoperative level, whereas Ner's responses were significantly increased. Also Kan and Tig had similar extensive medial ablation reaching or involving the genu corporis callosi, yet they displayed opposite changes in heart rate response, increased in Kan, and reduced in Tig. The motor leg flexion CRs showed similar changes, being stronger in Kan and weaker in Tig.

DISCUSSION

Typically, disinhibition and increased reactivity to positive CSs and USs have been described in dogs and cats trained in classical and instrumental CRs reinforced by food or water (cf. Brutkowski 1965, Sołtysik and Jaworska 1967). These effects were not observed in animals trained in defensive instrumental reflexes (Afanasev 1913, Waterhouse 1957, Zieliński et al. 1962). Conflicting data have been published on the classical defensive reflexes in dogs (Auleytner and Brutkowski 1960, Sołtysik and Jaworska 1967).

The present experiment was devoted to further analysis of the role of prefrontal medial cortex in defensive behavior. Two main questions were asked: (i) Does the prefrontal ablation result in disinhibition of inhibitory conditioned defensive responses? and (ii) Does the prefrontal ablation change the reactivity to aversive conditioned and/or unconditioned stimuli?

Undoubtedly, the first question poses no problems in the light of the present evidence. The differentiation of two identical auditory stimuli presented from two different loudspeakers was not disturbed in our prefrontal dogs. Also Afanasev's (1913) results, whose dogs, although trained by a quasi-instrumental method, were subjected to a fair amount of shock stimulation, provide a strong support for the conclusion that prefrontal dogs, despite their increased reactivity to many "appetitive" stimuli and their "impulsiveness" are quite capable of inhibiting conditioned leg flexion responses.

The problem of reactivity changes to aversive conditioned and uncon-

ditioned stimuli is less clear. True, in most dogs in our study, as well as in 4 dogs in our previous experiment, the reactions were not changed or reduced. However, some dogs displayed transient increases in cardiac and/or somatic responses to CSs or USs. We cannot, thus, with full confidence, restate our conclusion from our previous study that "the fear responses are attenuated in operated animals trained in classical defensive CR situation." In fact, there appeared to be no consistent changes in reactivity to noxious stimuli. The majority of animals showed no change or a decrease in reactivity. Only a small number of animals displayed an increase.

In the previous study, we observed shorter latencies of the leg flexion CRs after the prefrontal ablation. In the present group of 8 dogs, only one had significantly shorter latencies after the operation; 5 remaining dogs did not change and 2 animals displayed longer latencies. This discrepancy between the two experiments might be related to the fact that the dogs in the previous study had been trained for over 2 years and had longer latencies in spite of a much shorter CS-US interval: their average latency was 2.5 sec with a 3 sec CS-US interval. Our present dogs had a mean latency of 2.1 sec before and 2.2 sec after the ablation in the training situation in which the CS-US interval was 5 sec. On the other hand, it is possible that the larger lesions involving the dorso-medial prefrontal cortex in the previous experiment might have contributed to the difference in the post-operative leg flexion latency.

The question of whether prefrontal ablation changes defensive conditioned and differential reactions in the course of acquisition or shorter after an asymptote is reached remains unanswered. The fact that Auleytner and Brutkowski's dogs, trained for only 3 months, did show disinhibition and enhancement of conditioned responses suggests that there may be a differential effect of prefrontal ablation upon recently acquired, as compared to overtrained, defensive classical conditioned reflexes.

SUMMARY

Eight dogs trained using classical leg flexion conditioning procedure were subjected to medial prefrontal ablation. Tested on retention two weeks and two months after the operation, their responses to conditioned stimuli were for the most part unchanged; heart rate responses to the shock were in some dogs temporarily increased or decreased, whereas the somatic and cardiac responses to differential stimuli were slightly reduced. These data do not provide any direct support for the hypothesis of reduced fear reactivity after prefrontal ablation, but are not incompatible with such an hypothesis.

The authors are indebted to Dr. M. Levine for reading and correcting the manuscript and to Dr. J. Villablanca for his valuable suggestions. Thanks are also due to Mrs. B. Rubinstein for her help in preparing the typescript. This investigation was partially supported by Foreign Research Agreement No. 05-275-2 of the US. Department of Health, Education and Welfare under PL 480.

REFERENCES

AFANASEV, N. I. 1913. Data concerning the functions of the frontal lobes (in Russian). Ph. D. Thesis. Petersburg.

AULEYTNER, B. and BRUTKOWSKI, S. 1960. Effects of bilateral prefrontal lobectomy on the classical (type I) defensive conditioned reflexes and some other responses related to defensive behaviour in dogs. Acta Biol. Exp. 20: 243–262.

BRUTKOWSKI, S. 1965. Functions of prefrontal cortex in animals. Physiol. Rev. 45: 721–746.

DĄBROWSKA, J. and JARVIK, M. E. 1970. Black-white discrimination performance in frontal rats. Acta Neurobiol. Exp. 30: 19–32.

HEATH, R. G. 1949. Psychiatry. *In* F. E. Mettler (ed.), Selective partial ablation of the frontal cortex. P. B. Hoeder, New York, p. 497–502.

KREINER, J. 1961. The myeloarchitectonics of the frontal cortex of the dog. J. Comp. Neurol. 116: 117–133.

LANDIS, C. 1949. Psychological changes following topectomy. *In* F. A. Mettler (ed.), Selective partial ablation of the frontal cortex. P. B. Hoeder, New York, p. 306–312.

LICHTENSTEIN, P. E. 1950. Studies of anxiety. II. The effects of lobotomy on a feeding inhibition in dogs. J. Comp. Physiol. Psychol. 43: 419–427.

MAHER, B. A., ELDER, S. D. and NOBLIN, C. D. 1962. A differential investigation of avoidance reduction versus hypermotility following frontal ablation. J. Comp. Physiol. Psychol. 55: 449–454.

MOORE, B. F., FRIEDMAN, S., SIMON, B. and FARMER, J. 1948. A co-operative clinical study of lobotomy. *In* The frontal lobes. Williams and Wilkins Co., Baltimore, p. 769–794.

SOŁTYSIK, S. and JAWORSKA, K. 1967 Prefrontal cortex and fear-motivated behaviour. Acta Biol. Exp. 27: 429–448.

STREB, J. M. and SMITH, K. 1955. Frontal lobotomy and the elimination of conditioned anxiety in the rat. J. Comp. Physiol. Psychol. 48: 126–129.

WATERHOUSE, I. K. 1957. Effects of prefrontal lobotomy on conditioned fear and food responses in monkeys. J. Comp. Physiol. Psychol. 50: 81–88.

ZIELIŃSKI, K. 1966. Retention of the avoidance reflex after prefrontal lobectomy in cats. Acta Biol. Exp. 26: 167–181.

ZIELIŃSKI, K. SOŁTYSIK, S. and ROUGEUL, A. 1962. Effect of prefrontal lobectomy upon the retention of the positive and inhibitory avoidance reflexes in cats. Proc. XXII Int. Congr. Physiol. (Leyden). Abstr. of free commun., Vol. II, No. 1178.

Received 6 August 1971

Stefan SOŁTYSIK and Krystyna JAWORSKA, Department of Physiology of the Nervous System, Institute of Psychoneurology, Pruszków-Tworki, Partyzantów 3/4, Poland.

Anna SZAFRAŃSKA-KOSMAL, Department of Neurophysiology, Nencki Institute of Experimental Biology, Pasteura 3, Warszawa 22, Poland.