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Chapter 6

The Role of Prefrontal Control in the Programming of Motor Behavior

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I. General Considerations

Almost all studies on conditioning and learning begin with establishing the programs of training to which the animals are subjected. Thus, in classical conditioning we pair in various ways two variables, conditioned stimuli (CSs) and unconditioned stimuli (USs). According to the program of the experiment, we may establish various excitatory conditioned reflexes (CRs), or transform them into inhibitory CRs, or introduce differentiations, and so on. In instrumental conditioning the scope of possible programs is much larger, because here we deal with three variables, namely the CSs, the USs, and the instrumental responses. Whereas in humans such programs can be (at least in instrumental conditioning) carried over to the subject by oral instruction, in animals they must be trained by repeating the appropriate combinations of stimuli and

provoking, by various techniques, the required instrumental responses. When a subject succeeds in mastering the task, this means that we have "inscribed" that program in his brain, so that he is able to accomplish it correctly. Of course we do not assume that the design of the program in our own brains and in those of the experimental animals is identical. It is certain, however, that the animal's program of fulfilling a given task corresponds to that which we have established for him, since the responses of the animal are predictable. It happens, however, that the subject may establish a quite different program from that which we tried to teach him; in that case the subject's responses will become unpredictable for us, until we find out the program which he has developed.

Studies concerned with the actual designs of programs inscribed in the animal's brain can be carried out in various ways. First, we can reach conclusions about these designs by varying the stimuli impinging upon the animal (and/or his internal environment) and observing changes which occur in his responses to these modified stimuli. Second, we can influence directly the animal's brain by ablation or stimulation techniques and observe the effects of these operations on the fulfillment of the program.

In this paper we shall narrow the scope of our discussion by dealing exclusively with instrumental conditioning based on alimentary (food) reinforcements. This means that we shall leave out classical conditioning, as well as conditioning based on noxious reinforcements.

We can roughly classify programs used in instrumental alimentary conditioning into the following groups:

1. The most elementary programming occurs when the animal is required to perform a given movement (R) to obtain continuous or intermittent reinforcement. No specific CSs are present.

2. A more complex program results when an instrumental response is linked to one or more controlling discriminative stimuli. The response is thereafter emitted only in the presence of these CSs.

3. R–no R Pavlovian differentiation: R–no R programming is defined by a situation in which the animal is differentially trained to perform an instrumental movement (R) to a CS_1, while in the presence of a CS_2 (a discriminative stimulus somewhat similar to CS_1) the instrumental act is not reinforced. This is analogous to an S^D–S^Δ situation. The stimulus controlling instrumental responding is called the positive CS (CS^+) and that presented without reinforcement is the negative CS (CS^-).

4. R–no R, both reinforced differentiation: In this program the CS_1 is followed by food if the animal does perform the movement R, whereas in the presence of a CS_2 (similar to CS_1) reinforcement occurs only if the animal does not perform this movement.

5. R_1–R_2 differentiation: In this program the animal is trained to perform movement R_1 in response to CS_1 and movement R_2 in response to CS_2.

6. R_1-R_2 delayed response program: This may be regarded as a variation of the R_1-R_2 program, in that the animal is allowed to react not to the actual CSs but to their traces.

It is easy to notice that programs 3 through 6 are parallel programs, since they are concerned with the formation of various responses to be performed to various stimuli, respectively. We have, however, left out sequential programs in which the subject is trained to perform a sequence of movements in a definite order. Since experiments on sequential programs have been not very numerous, they are not yet suitable to our analysis.

Many experimental studies (to be quoted further in the text) have shown that parallel programs are largely under the control of the prefrontal region of the cerebral cortex. Moreover, recent experiments (to be quoted below) have established that the prefrontal cortex is not functionally "equipotential," since particular parts of this region are related to particular programs. The aim of this article is to present experimental evidence demonstrating the functional heterogeneity of the prefrontal cortex and to draw conclusions about the functional organization of this region.

Generally, the behavioral experiments dealing with the above programs were systematically carried out on dogs and monkeys. Since only these materials are comparable, we shall limit our discussion mainly to these two species.

Since we shall base our discussion largely on the data obtained on dogs in our own laboratory, we present, for the reader's convenience, two experimental situations in which the corresponding experiments have been conducted. One situation is a Pavlovian soundproof chamber with the dog placed on a stand and the experimenter controlling the course of the experimental session from the prechamber (Fig. 1). The animal is usually trained to lift his left or right foreleg and place it on the feeder, or press a pedal situated nearby. The second situation is a compartment with three feeders and a starting platform situated in front of the experimenter's table (Fig. 2).

II. Simple S–R Conditioning

We cannot enter here into a full discussion of the central mechanism of instrumental conditioning either to the environment as a whole (ΣS), or to sporadic CSs. This problem was discussed in great detail in the author's recent book (Konorski, 1970, Chapters 8–10). Briefly, we have good reason to believe that the instrumental CR is based on two lines of connections (Fig. 3). One line goes "directly" from the central representation of the CS (further denoted as the "CS center") to the central representation of kinesthesis produced by instrumental movement (further denoted as the "movement center"). The other line goes from the CS center to the movement center through the central representation of drive—in our discussion, of hunger (further denoted as the

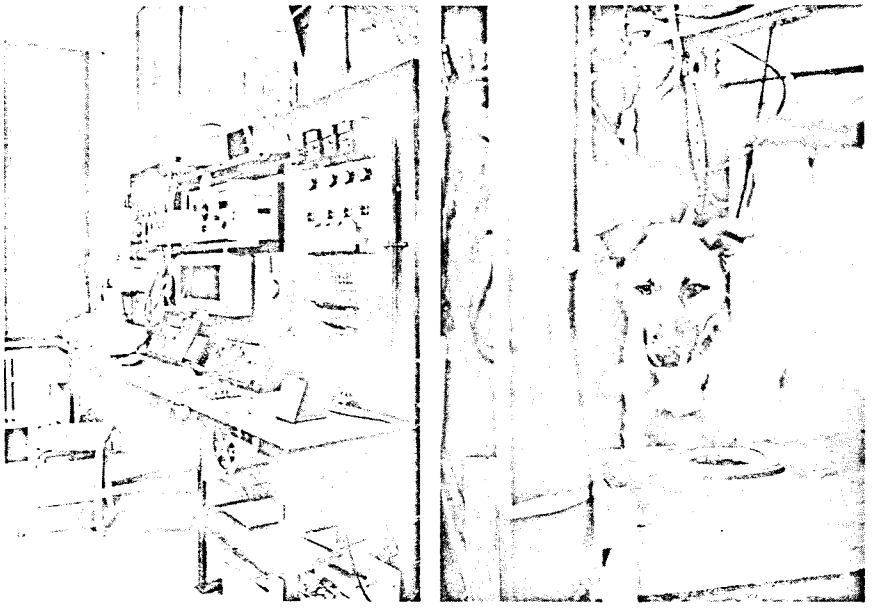

Fig. 1. The CR chamber. Left side: pre-chamber. Right side: the soundproof chamber with a dog on the stand.

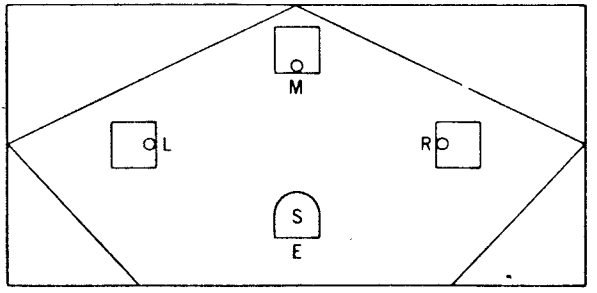

Fig. 2. Experimental room for locomotor CRs. S, starting platform; L, M, R, feeders; E, experimenter's seat. Reprinted by permission of McGraw-Hill, New York.

"hunger center"). Both of these lines must be jointly activated to produce the instrumental response. Thus, when the hunger center is inhibited by full satiation, or when in the presence of hunger the CS is not given, the instrumental response will not be produced. The indispensable condition for the formation and performance of an instrumental alimentary CR is that the motor response be reinforced by presentation of food, provoking the consummatory reaction and partially inhibiting the hunger drive.

It should be noted that in the first stage of CR training the animal performs the instrumental movement not only to the CS but also during the intertrial intervals. This means that the CR is first formed to the environmental compound stimulus (ΣS). Only in the next stage of training are the intertrial movements extinguished because of lack of reinforcement, while the CS acting within that environment instigates the instrumental response. In other words, the instrumental CR is established to $\Sigma S + CS$, while the CR to the ΣS operating alone is inhibited. The significance of this fact will be seen in the next section.

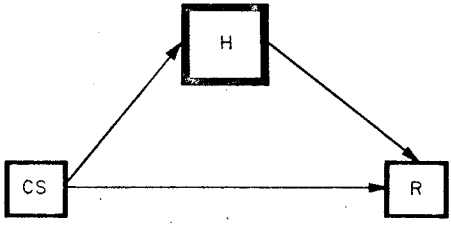

Fig. 3. Model of the instrumental CR. CS, R, and H denote the centers of CS, kinesthesis of instrumental response, and hunger drive, respectively. (Based on the model of W. Wyrwicka, *Acta Biol. Exp.*, 1952, **16**, 131-137.)

If our model of the instrumental CR represented in Fig. 3 is correct, then it must be predicted that by destroying particular parts of this model the CR should be impaired or abolished.

This in fact occurs. The destruction of the hunger system on both its levels (lateral hypothalamic lesion, Rozkowska and Fonberg, 1970, or medial amygdalar lesion, Fonberg, 1969) abolishes the instrumental CR for shorter or longer periods.

Similarly, lesions sustained in the motor, or rather kinesthetic, centers also affect the instrumental CRs to various degrees and in different ways (Konorski, 1970, Chapter 11). Without going deeper into this subject we should stress only that the "movement center" in our model represents that area of the brain in which the kinesthetic pattern of the trained movement is being formed. In fact, we have good evidence to show that lesions in the premotor area have detrimental effects on the performance of manipulatory instrumental movements (Stepien, Stepien, and Konorski, 1960, Stepien, Stepien, and Kreiner, 1963; Gerbner, and Pásster, 1965, and others).

To end this discussion on simple instrumental conditioning programming, Stepien and Stepien (1965) and Stepien, Stepien, and Sychowa (1966) have shown that after the removal of a small area in the posteromedial part of the precruciate area [Fig. 4, according to the myeloarchitectonic map of Kreiner (1966)], a curious symptom may be observed: in experiments with locomotor CRs in which the source of the CS is noncontiguous to the feeder, the lesioned

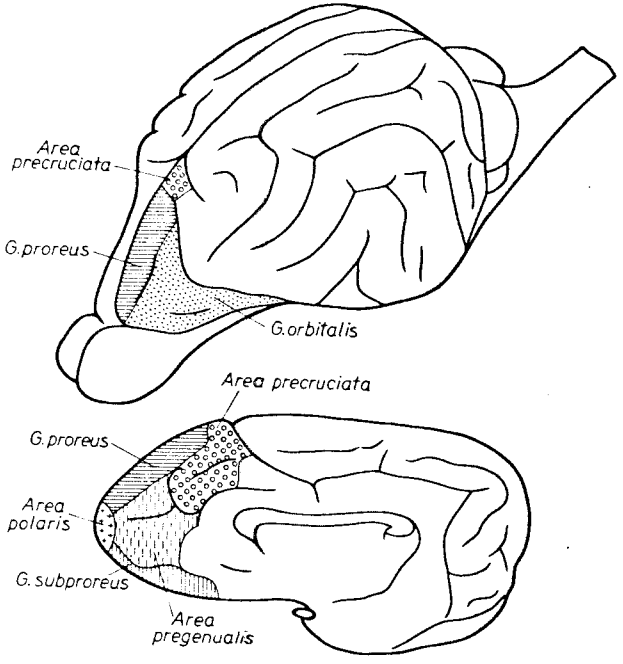

Fig. 4. The surface of the dorsolateral and medial aspects of the cerebral cortex of the dog, indicating those areas which are dealt with in this chapter.

dogs, in response to the CS, approach the source of the CS and not the feeder. The authors have called this symptom "magneto-reaction." It is interesting to note that this reaction is elicited only by positive alimentary CSs, but not by neutral stimuli, nor by the negative (nonreinforced) CSs.

The phenomenon of magneto-reaction may be understood if we realize that in natural life the signals of food (such as its sight, smell, or sounds produced by a prey) operate from the very place of food and only in our "artificial" experimental situations are they separated. Accordingly, in our CR training the animal must inhibit the normal tendency to approach the signal of food and learn to go straight to a feeder. Consequently, we are confronted here again with some inhibitory process which is included in the learned program and impaired after the appropriate cortical lesion. In other words, the magneto-reaction may be regarded as an early established natural CR, which may be suppressed by special training during the formation of an instrumental CR to noncontiguous CS.

To sum up, we may see that this "simple" program of instrumental conditioning to sporadic stimuli noncontiguous to the place of feeding is in the

animal's brain more complex than in the experimenter's brain: quite unexpectedly for us it includes the act of suppressing a natural tendency to approach the source of the CS, as well as the tendency to perform the trained movement in response to the whole environment.

III. Pavlovian R–no R Differentiation

We began to study the effects of prefrontal lesions on Pavlovian differentiation in dogs in the early 1950's (Konorski, Stepien, Brutkowski, Lawicka, and Stepien, 1952; Brutkowski, Konorski, Lawicka, Stepien, and Stepien, 1956). We used as positive CSs auditory stimuli–metronomes, bells, tones, etc. As negative CSs, either stimuli similar to the positive CSs were presented, or an inhibitory compound composed of a stimulus quite different from the CS+ (the so-called conditioned inhibitor, CI) followed by the positive CS. The CI-CS+ interval was usually protracted to 5 sec or more. The instrumental response was lifting the right foreleg and placing it on a feeder situated in front of the animal. The food was presented by using remote control to move into position a bowl in the feeder. In some experiments vocal CRs were used, the dog being required to bark in response to the CS (Lawicka, 1957b).

When the training of both excitatory and inhibitory CRs was completed [as seen in Fig. 5(A)], the prefrontal regions of the cerebral cortex up to the presylvian sulcus were bilaterally removed. One week after surgery the experiments were resumed, and the following picture was observed. The positive CRs were completely preserved. As to the no–R responses to the CS–, they were strongly disinhibited. Moreover, the animals performed many instrumental movements in the intertrial intervals, thus manifesting that the instrumental CR established originally to the environment was also disinhibited (cf. Section II). The no–R response to the inhibitory CI–CS+ compound was also completely disinhibited, but the no-response to the conditioned inhibitor was unaffected. It should be recalled that CI, usually being quite distinct from the CS+, never evoked the instrumental response preoperatively. The dog's performance after prefrontal ablation is represented in Fig. 5(B).

When the animals were retrained, their instrumental responding gradually improved. At first the intertrial responses ceased to occur, then the dogs stopped responding to the (differentiated) CS–. Next the response to the CS+ immediately following the CI became inhibited, and only much later was the inhibitory response to the CI-CS compound with the 5-sec interval restored [Fig. 5(C)]. Usually the number of trials required for inhibitory responses to the (differentiated) CS– were equal to or less than that for original training, while the retraining of inhibition to the CS following CI by 5 sec was much more protracted.

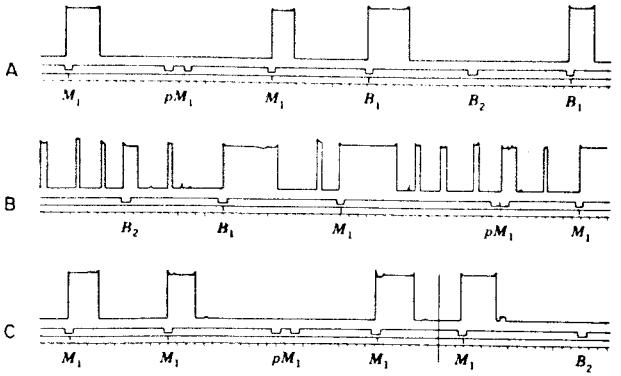

Fig. 5. Records of the experimental sessions with R–no R Pavlovian differentiations. Each record from top to bottom: lifting the foreleg and placing it on the feeder, CSs, presentation of food, time marker (in 5 secs). M_1, B_1, positive CSs; B_2, negative CS; pM_1, M_1 preceded by CI. (A) Correct responding. (B) Responding after prefrontal lobectomy. (C) Responding after recovery. Note that in (A) pM_1 with 5 sec CI-CS interval as well as B_2 elicits inhibitory response; in (B) many intertrial movements are seen; the responses to B_2 and M_1 preceded immediately by CI, but not CI itself, are disinhibited; (C) again completely normal responding. Note that the placing of the foreleg on the feeder is always prolonged in positive trials; this is due to the fact that the dog keeps his leg on the feeder during the act of eating and puts it down only after the portion of food is consumed. (After Brutkowski *et al.*, 1956.) Reprinted by permission of Polish Scientific Publishers, Warsaw.

In order to see whether the disinhibitory syndrome appears selectively after prefrontal ablations, a parietal area of the same size was removed. The animal's instrumental responding was completely normal after the lesion, and no disinhibition was ever observed.

The next task was to see whether the prefrontal cortex is "equipotential" with regard to the Pavlovian R–no R differentiation, or whether there are some crucial areas responsible for the performance of this task. Experiments performed by Szwejkowska, Kreiner, and Sychowa (1963) and Brutkowski and Dabrowska (1963, 1966) have shown that lesions limited to the medial part of the prefrontal area (pregenual area and medial precruciate area, Fig. 4) give rise to the clear disinhibitory syndrome. On the other hand, lesions sustained in the dorsal part (proreal area) or the lateral part (so-called orbital area, not to be confused with orbital area in monkeys) failed to produce this syndrome, provided that the intertrial intervals are about 1 min or more. Lesions sustained in the subproreal area situated in the basal part of the prefrontal region also fail to produce disinhibition (Szwejkowska, Stepien, and Kreiner, 1965). Finally, it should be mentioned that when the intertrial intervals are shortened to 15 sec, disinhibition of inhibitory responses is also obtained after dorsal prefrontal lesions (Brutkowski and Dabrowska, 1963, 1966). This fact will be commented upon further in the text.

Now we should turn to the problem of whether there are other regions in the brain where damage produces a disinhibitory syndrome. First, as found by Brutkowski and Mempel (1961), the genual portion of the anterior cingulate gyrus (but not the posterior cingulate gyrus) also produces disinhibition of inhibitory responses in R–no R differentiation. This shows that the medial frontal lesion producing that syndrome is larger than originally suggested, a fact which may partially account for restoration of inhibitory CRs during postoperative training.

Second, the lesions in subcortical structures related to inhibition of hunger drive–lateral amygdala (Fonberg, 1969) and ventromedial hypothalamus (Rozkowska and Fonberg, 1971)–also produce the disinhibitory syndrome closely connected with increase of hunger drive. Interestingly enough, after ventromedial hypothalamic lesions, the intertrial responses were noticed, but the no-response to the CS– was not affected.

Finally, Dabrowska (unpublished experiments) performed an extensive study on R–no R Pavlovian differentiation after hippocampal lesions. She found that these lesions produce in many dogs (but not in all of them) a severe, disinhibitory syndrome. This finding would support Kimble's (1968) thesis claiming an important role of the hippocampus in "internal inhibition." It remains to be elucidated whether hippocampal lesions affect the same or different aspects of the inhibitory mechanism than that affected by prefrontal lesions.

Investigations on R–no R Pavlovian differentiation in monkeys are less numerous. In experiments carried out in the Wisconsin General Test Apparatus, Brutkowski, Mishkin, and Rosvold (1963) have shown that after orbitofrontal lesions R–no R differentiation to visual stimuli is strongly impaired. Dorsal ablations involving principal sulcus and surrounding area, including the anterior bank of the arcuate sulcus, fail to produce these effects. In a modified Wisconsin General Test Apparatus (no screen between the animal's cage and the food well, with manipulandum permanently available to animals) Lawicka, Mishkin, and Rosvold (1966, 1972) performed experiments with auditory stimuli analogous to those carried out in dogs. It has been found that orbital ablations in monkeys produce almost exactly the same impairment of R–no R Pavlovian differentiation as that obtained in dogs after medial ablations (or complete prefrontal lobectomies). The intertrial responses became very abundant and the animals vigorously responded to the CS–. After a lapse of time the correct CR performance was restored, but the number of errors on the negative trials in postoperative retraining was much higher than it was preoperatively. On the contrary, dorsolateral lesions failed to produce this effect.

To sum up, we see that after removal of a definite part of the prefrontal region there occurs a dramatic impairment of alimentary (but not defensive, see Soltysik and Jaworska, 1967) inhibitory CRs. The problem arises as to what is

the mechanism of this phenomenon. In my recent book (Konorski, 1970, Chapters 7 and 10) it was postulated that besides the hunger drive system responsible for alimentary instrumental CRs, there is a higher order "antihunger center" whose role is to suppress the hunger drive in those situations in which food is not available. This "antihunger center" may be considered an extension of the limbic system, serving for the most delicate adaptation of alimentary behavior to the environment.

When at the start of the Pavlovian differentiation training a stimulus similar to the CS+ was presented without reinforcement, its center was already connected with the hunger drive center, owing to generalization. This is why the new stimulus elicits the instrumental response. During differentiation training, however, apart from these connections, which remain intact, new connections are formed between the new CS center and the antidrive center; these connections are responsible for the suppression of the instrumental response to this CS [Fig. 6(A)].

The mechanism of conditioned inhibition is a little more complicated. The CI, being dissimilar to any of the CSs used and never being reinforced by food, forms no connections with the hunger drive center (cf. the notion of the "primary inhibitory stimulus", Konorski and Szwejkowska, 1952), but it forms connections exclusively with the antihunger center [Fig. 6(A)]. The fact that the instrumental response to the CS+ which shortly followed the CI is suppressed, is primarily due to the inhibitory aftereffect which the CI exerted upon this response (the slight contamination of the CS itself by inhibitory properties is here neglected).

Now, the fact that lesions sustained in the mediofrontal area in dogs, or in the orbitofrontal area in monkeys, produce disinhibition of inhibitory CRs indicates that the postulated higher order antihunger drive center is localized precisely in this part of the prefrontal cortex. Accordingly, removal of this area [Fig. 6(B)]

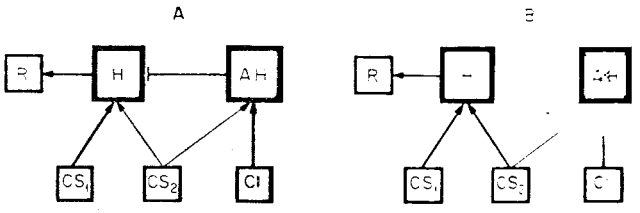

Fig. 6. Block model of the mechanism of inhibitory instrumental CRs (A), and their impairment after medial prefrontal (in dogs) or orbitofrontal (in monkeys) lesions (B). CS_1, positive CS center; CS_2, negative CS center; CI, conditioned inhibitor center; H, hunger system; AH, antihunger center situated in the prefrontal extension of the limbic system; R, instrumental response center. Arrows, excitatory connections; stopped lines, inhibitory connections. Thin lines denote weak connections. For simplicity, the direct connections between CSs and R are not drawn.

will not affect the excitatory CRs, but will impair those inhibitory CRs which have a mixed excitatory-inhibitory character. Thus the animal will perform the intertrial responses, because the experimental environment *was* originally a CS+ (see preceding section), and will also perform the responses to negatively differentiated CSs. As far as the CI is concerned it will not produce the instrumental response, because it has never been linked with the hunger center. However, since the antidrive center with which the CI center was connected has been destroyed, CI will no longer exert any inhibitory influence upon the CS+ following it; in effect, the inhibitory CR to the CI-CS compound will be dramatically disinhibited.

Since the prefrontal antihunger center is considered the rostral extension of the limbic system, it is clear that it is connected functionally with both the lateral amygdalar nucleus and the ventromedial hypothalamic nucleus. Accordingly, the disinhibitory syndrome is produced after lesions in these structures, although its symptomatology is not quite the same.

As noted before, lesions in the dorsolateral prefrontal area in dogs produced disinhibition in R–no R differentiation when the intertrial intervals were very short (15 sec instead of 1 min) (Brutkowski and Dabrowska, 1963, 1966). Although this disinhibition is quite significant, it differs from that produced by medial lesions in that during the intertrial intervals the dogs are quiet and fail to perform instrumental responses. Since these dogs fail also to display any increase of the hunger drive manifested by searching and sniffing movements, disinhibition of their instrumental responses to the CS– cannot be attributed to impairment of drive inhibition. It may be supposed that their defect is due to an inability to switch rapidly from excitatory to inhibitory responses, a capacity required in rapid succession of positive and negative CSs.

IV. R–no R Both Reinforced Differentiation

The effects of prefrontal lesions upon the performance of this test were recently studied in dogs (Dabrowska, 1971) in the following way. First, the dog was trained to lift his right foreleg to a 1000 cps tone and to place it on the feeder situated before him. Each such trial was reinforced. Thereafter, a 700 cps tone was presented in random order with the 1000 cps tone. At first, the animal performed the trained movement in response to the 700 cps tone, but since no food reinforcement followed, after a number of sessions he stopped doing so. If during 5 sec of the 700 cps tone no trained movement occurred, food was offered. This led to restoration of response to the 700 cps tone, until after several successions differentiation was achieved: the dog performed the trained movement to the 1000 cps tone, while during the operation of the 700 cps tone he clearly refrained from doing so.

After the animals reached criterion they were operated upon: in one group ablations included the medial part of the prefrontal area, in the other group the

dorsolateral part was removed. The results of these lesions were exactly opposite those obtained by Brutkowski and Dabrowska (1966) with Pavlovian differentiation: medial lesions which were detrimental for Pavlovian differentiation produced only a slight effect, while the dorsolateral lesions produced total and irreversible disorder of symmetrical differentiation: the animals either performed the trained movement to both CSs, or refrained from performing that movement to either one. A comparison of data obtained in experiments on R–no R Pavlovian differentiation and in studies on R–no R both reinforced differentiation is presented in Fig. 7.

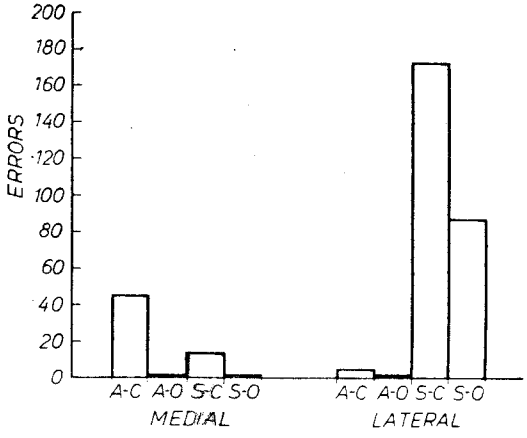

Fig. 7. Commission and omission errors in dogs in Pavlovian and both-reinforced R–no R differentiation after medial and lateral lesions. A–C, commission errors in Pavlovian differentiation; A–O, omission errors in Pavlovian differentiation; S–C, commission errors in both-reinforced differentiation; S–O, omission errors in both-reinforced differentiation. (After Brutkowski and Dabrowska, 1963, 1966; Dabrowska, 1971.)

Further experiments by Dabrowska (1971) have shown that whereas selective dorsal ablations, involving the proreal gyrus only, are not harmful for the performance of the R–no R both reinforced task, lateral ablations involving the orbital gyrus produced the same effect as dorsolateral lesions. Thus, the lateral and not the dorsal prefrontal area has appeared to be responsible for the integrity of an R–no R both reinforced differentiation.

Analogous experiments were performed much earlier on monkeys by Weiskrantz and Mishkin (1958), Gross and Weiskrantz (1962) and Gross (1963). In these experiments, too, auditory CSs were used to demonstrate that removal of the dorsolateral prefrontal cortex produced severe impairment of an R–no R both reinforced differentiation. It should be noted that in Gross and Weiskrantz's experiments (not supported by those of Gross, 1963) only ablations of cortex surrounding the principal sulcus but not its depth were responsible for this impairment.

How should we explain the fact that in spite of a seemingly small difference between two procedures of R–no R differentiation, the areas responsible for these two programs are different. This fact implies that the mechanisms underlying these programs should also be distinct.

In view of our concept of Pavlovian differentiation, the difference between the two programs is quite understandable. In the Pavlovian R–no R program, hunger drive is the essential link for occurrence of differentiation. The animal stops performing the instrumental response to the CS– because this stimulus becomes a signal of antihunger, or to speak freely, the animal does not expect food in its presence. On the contrary, in learning that no–R to CS_2 is reinforced, the animal must decide whether the R response or the no–R response should be performed to a given stimulus.

V. R_1–R_2 Differentiation

The program of this task consists in the formation of two instrumental responses: CS_1–R_1 and CS_2–R_2. In our own experimental practice we used two methods. In Lawicka's experiments the left feeder and the right feeder in the setup presented in Fig. 2 were used, and two auditory stimuli served as cues which evoked locomotor responses to each of them respectively. In Dobrzecka's experiments the animal was required to place either the right or the left foreleg on the feeder (Fig. 1) in response to one or the other auditory CS, respectively. Before discussing R_1–R_2 differentiation, it should be noted that we shall deal here exclusively with the situation where both CSs are noncontiguous to the feeders. When, in locomotor R_1–R_2 differentiation, the cues are contiguous with the goals (e.g., buzzers are placed on the respective feeders), then practically no training is necessary and the animals make no errors even if a triple choice is presented; the CS simply "pulls" the animal to the corresponding feeder (Konorski and Lawicka, 1959). The situation is, however, quite different when the CSs are not contiguous to the feeders. In that case the task requires a more or less prolonged discriminatory training before the animal learns which CS signals which feeder.

An important rule should be followed in this training, originally discovered by Lawicka (1964, 1969a) for locomotor CRs, and later confirmed by Dobrzecka and Konorski (1967, 1968) for manipulatory CRs. If the two CSs differ only in quality but operate from the same place (e.g., two tones emitted from the same loudspeaker), the task is very difficult and requires prolonged training. If, however, the CSs differ in location, e.g., the source of one sound is above the source of the other (Lawicka's experiments), or one source is in front and the other behind the animal (Dobrzecka and Konorski's experiments), then the task does not present serious difficulties to the subject. Incidentally, the same rule appears to be valid not only for dogs but also for monkeys (Lawicka *et*

al., 1966, 1972). Konorski (1970, Chapter 10) explains this phenomenon by assuming that the quality of a sound is an inadequate cue for establishing direct CS–R connections because potential connections linking auditory and kinesthetic gnostic units are poorly developed. Consequently, the CS–R connections are mediated by kinesthesis produced by orienting responses to the CSs; the greater the difference between the kinesthetic feedback of those responses (as is the case when the CSs operate from different places), the easier is the discriminatory training.

Let us now analyze in more detail the formation of the R_1–R_2 differentiation to two auditory CSs, with the help of the block model presented in Fig. 8. Since in the experimental situation the animal has learned to perform both instrumental responses, the situation itself becomes a subthreshold CS, producing readiness for response occurrence. Releasing stimuli are provided by both CSs, due to the fact that they increase excitation of the hunger drive center and thus elicit both responses.

Since "direct" connections between auditory stimuli centers and centers of particular movements are poor, the instrumental responses to both CSs are indiscriminate, unless the CSs differ also in eliciting two distinct orienting responses. Orienting responses can easily establish connections with corresponding instrumental movements (e.g., look up–go right, look down–go left), and these connections determine which movement is performed to which stimulus.

Returning now to the problem of prefrontal representation of a R_1–R_2 differentiation, it should be noticed that when the CSs are contiguous with the appropriate feeders, even complete frontal lobectomies fail to impair the locomotor R_1–R_2 differentiation (Lawicka and Konorski, 1959).

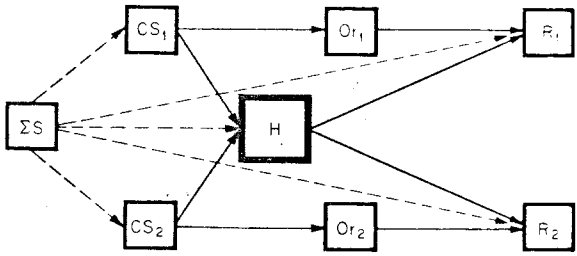

Fig. 8. Block model of R_1–R_2 differentiation to CSs noncontiguous with the place of feeding. CS_1, CS_2, centers of CSs; Σ S, experimental situation center. H, hunger system; Or_1, Or_2, centers of orienting reactions to CS_1 and CS_2; R_1, R_2, centers of instrumental responses. Arrows, excitatory connections; interrupted lines, facilitatory connections. $Or_1 \rightarrow R_1$ and $O_2 \rightarrow R_2$ are supposed to be situated in the prefrontal extension of the kinesthetic analyser.

The situation is, however, different when the CSs are not contiguous to the feeding places. As shown in some preliminary experiments, complete prefrontal lobectomies do impair locomotor (Lawicka, unpublished) and manipulatory (Dobrzecka *et al.*, unpublished) R_1-R_2 differentiation; however, the effects of partial prefrontal lesions have not yet been fully investigated. Recently Lawicka (1969b) reported that ablations of the proreal gyrus failed to impair this task (see Fig. 10). Let us recall that the same negative effect was obtained after this lesion with regard to R–no R where both were symmetrically reinforced (Dabrowska, 1971).

The study of this problem is more advanced in monkeys. In recent experiments by Lawicka *et al.* (1966, 1972) dorsolateral prefrontal lesions, including the rostral bank of arcuate sulcus, impair considerably the R_1-R_2 differentiation to CSs, differing from each other either in direction (up versus down) or in quality (frequency of tones). Remember (Section III) that R–no R Pavlovian differentiation in monkeys was not impaired after dorsolateral lesions. On the other hand, orbitofrontal lesions, which strongly impaired the R–no R Pavlovian differentiation, failed to affect seriously R_1-R_2 differentiation, at least to directional cues (up–down). The R_1-R_2 differentiation established to frequency cues, being an exceedingly difficult task for normal monkeys, *is* impaired also after ventral lesions–a fact which is elaborated upon elsewhere (cf. Lawicka *et al.*, 1972).

In this connection a recent study by Goldman and Rosvold (1970) should be cited, because they clearly demarcate the extent of lesions producing impairment of R_1-R_2 differentiation to directional auditory CSs (up–down). They have shown that the optimal lesions are those affecting the arcuate sulcus. Dorsolateral lesions around the principal sulcus produce a less severe effect, while those involving the principal sulcus alone are virtually without effect.

Similar results were obtained by Stepien and Stamm (1970a). These authors trained monkeys in the R_1-R_2 test with cues spatially opposed to the feeders–the cue near the left feeder signaled "go right," and that near the right feeder signaled "go left." They found that dorsolateral prefrontal lesions, surrounding the principal sulcus and involving the arcuate sulcus, severely impaired this test. On the other side, lesions inside the principal gyrus were almost ineffective. Lesions sustained in the premotor and the orbitofrontal areas produced a very insignificant deficit.

To sum up, we see that, as far as dogs are concerned, total prefrontal lobectomy does impair the R_1-R_2 differentiation, but the area specifically concerned with this test has not so far been demarcated. In monkeys the R_1-R_2 differentiation is impaired after lesions located in the dorsolateral area excluding the principal sulcus. The crucial area for this test lies in the arcuate sulcus.

In order to explain these findings we should recall that the R_1-R_2 differentiation to cues noncontiguous to the place of feeding is mediated by the

orienting responses elicited by these cues (Fig. 8). Since the dorsolateral area of the prefrontal cortex including the arcuate sulcus may be regarded as a gnostic extension of the kinesthetic cortical region (Konorski, 1970), it may be supposed that precisely in this area associations are formed between kinesthetic patterns, representing particular behavioral acts, for instance, between patterns generated by orienting responses and those patterns generated by particular instrumental movements. When this area is removed, these associations are broken, and the animal is no longer capable of responding selectively to the corresponding cues. However, the more general connections, which link *both* CS centers with *both* instrumental movement centers via the hunger drive center, are fully preserved. Therefore the animal performs these movements indiscriminately, depending on the instantaneous higher excitability of a given motor center.

The fact that the R_1-R_2 test is *not* impaired when the animal is required to approach the feeder signaled by the contiguous CS is understandable; this response is based on a much more primitive mechanism previously discussed in Section II.

To end these considerations, we should ask the question whether the R_1-R_2 differentiation is fully equivalent to R–no R both reinforced differentiation. In experiments on monkeys the areas responsible for both tasks are, in fact, overlapping, but the crucial experiment, testing whether the arcuate sulcus, responsible for R_1-R_2 differentiation (Goldman and Rosvold, 1970), is also responsible for R–no R both reinforced differentiation has not yet been performed. In dogs the area responsible for R_1-R_2 differentiation is still unknown.

The difference between the two tasks is that one of them involves reciprocally related movements of one leg only (flexion–extension), while the other task involves two symmetrical movements (turn right–turn left, or lifting the right foreleg–lifting the left foreleg). As stated before, the R_1-R_2 task is acquired by the mediation of orienting responses; whether the same is true of an R–no R task, we do not know. Therefore, the problem of whether the two tasks are identical or different must still await its solution.[1]

VI. Delayed Responses

The program of delayed responses derives from the R_1-R_2 differentiation program, except that the animal is not allowed to display the instrumental

[1] Addendum in proof: Recent experiments of Dabrowska (*Acta Neurobiol. Exp.*, 1972, 32) and Stepien and Stepien (*ibid.*) have shown that R_1-R_2 differentiation and R–no R both reinforced differentiation depend on two different mechanisms since they are impaired after different prefrontal lesions. Probably the main difference between the two tests consists in that the first test involved only locomotor responses whereas the other one is based on two manipulatory responses, namely flexion of the fore leg versus its extension.

response in the presence of the corresponding CS but only to its trace. Since, however, in the majority of studies using this task the CSs were contiguous with food wells and the responses consisted of running toward them, or reaching for food by hand, the complexity of the R_1-R_2 programs with noncontiguous cues was omitted. Consequently, delayed response tests are typically concerned with the problem of mere delay, without being contaminated with the problem of making a learned choice. This is the advantage of using a procedure with cues contiguous to the feeders.

From the early 1930's, when Jacobsen (1936) performed his famous experiments showing impairment of delayed responses after prefrontal ablations in monkeys, studies on this problem were very numerous and the essential fact discovered by Jacobsen was fully confirmed. Most authors, however, did not agree with the original explanation, namely his attributing the delayed response deficit after prefrontal lesions to an impairment of short-term memory.

Since the experimental paradigm for the delayed response tasks with monkeys is well known to American readers, I shall describe here only the method used in our laboratory with dogs (Lawicka, 1959). Figure 2 illustrates the experimental setting. The buzzers are placed on each feeder. Before every trial the dog was attached by a leash to the starting platform, and in several seconds one of the buzzers operated for 3 sec; after various delay periods (usually 15 and 60 sec), the animal was released and allowed to approach the feeder. When he reached the correct feeder, a bowl of food was placed into position. An important factor introduced in these experiments was the application of trials with distractions during the delay period, the usual distractor consisting of a small portion of food being placed on the starting platform before the animal was released. This measure prevented the animal from preserving a directional posture toward the signaled feeder during the delay period. In this way pseudodelayed responses produced by the animal's maintaining his orientation posture to the buzzer were avoided.

Having established that delayed responses were dramatically impaired in dogs following prefrontal lobectomies (Lawicka and Konorski, 1959), the next aim was to isolate which part of this region was responsible for this deficit. In experiments by Lawicka *et al.* (1966) it was found that the dorsomedial part of the prefrontal area (proreal gyrus, Fig. 4) was crucial for this test, whereas lesions sustained in the lateral area (orbital gyrus and presylvian sulcus) did not produce any deficit (Fig. 9). On the other hand, pure proreal lesions which severely impaired the delayed response test failed to affect the R_1-R_2 differentiation (Konorski and Lawicka, 1964; Lawicka, 1969b) (Fig. 10).

As far as monkeys are concerned, many investigations have shown that the area necessary for successful performance on the delayed response task (including the spatial delayed alternation) lies in the depths of the principal sulcus (Blum, 1952; Mishkin, 1957; Gross and Weiskrantz, 1964; Goldman and Rosvold, 1970).

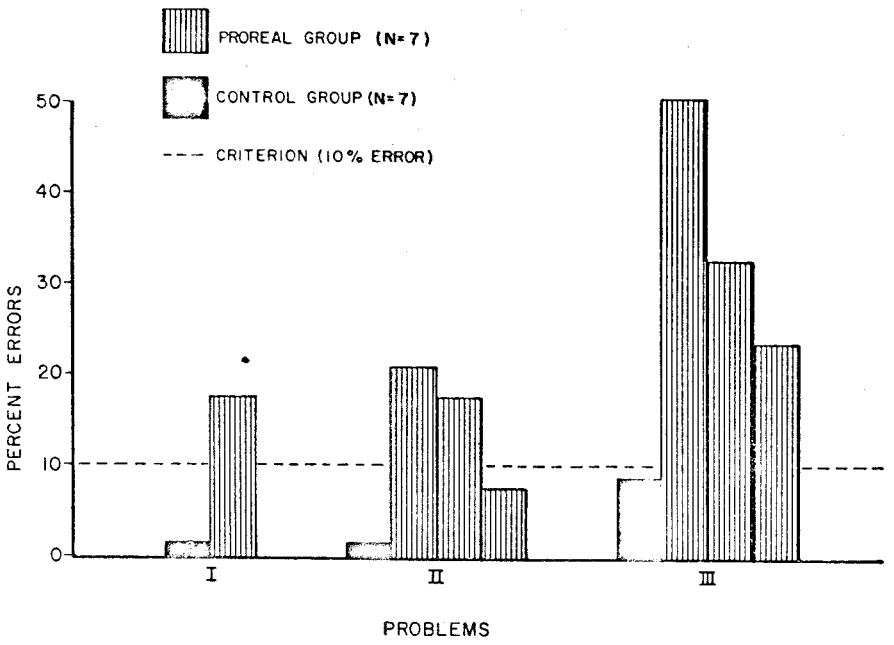

Fig. 9. Average performance of the proreal group (hatched columns) and of the combined orbital and presylvian group (black columns) for blocks of 60 trials on the three delayed response problems: I, 15-sec delay; II, 60-sec delay; III, 60-sec delay with intradelay feeding (Lawicka *et al.*, 1966).

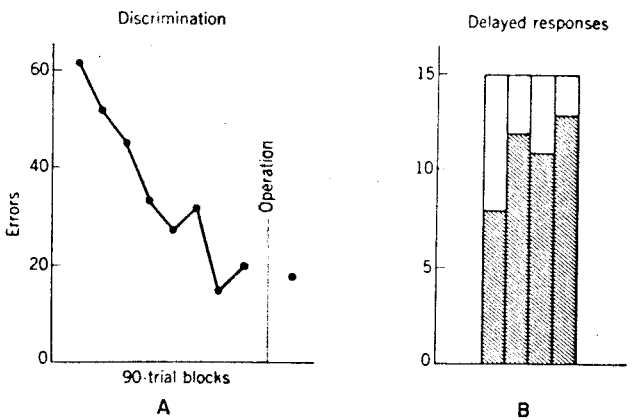

Fig. 10. Comparison of the effect of proreal lesions on R_1-R_2 differentiation and triple choice delayed responses. (A) Training of differentiation before operation, and its full preservation after operation. (B) Number of errors (hatched parts of the columns) in delayed responses with distractions; performance on the chance level (66.6% of errors). (From Konorski and Lawicka, 1964.) Reprinted by permission of McGraw-Hill, New York.

Now we proceed to the main problem of our discussion, namely the role of the prefrontal area in delayed response performance. Perhaps the best evidence to demonstrate that the deficit caused by a prefrontal lesion is not due to the abolition of short-term memory has been provided in experiments on cats in a three-choice situation (Lawicka and Konorski, 1961). These animals, although severely impaired after prefrontal ablation, behaved regularly in the following way; when released from the starting platform after the delay period, they approached the feeder from which they had received food on the preceding trial; not finding food in this feeder, they would turn immediately to the *correct* one. This means that they did remember the signaled feeder, but simply could not suppress the perseverative (or rather one-trial learning) tendency to perform that locomotor response which was just reinforced. The same results were obtained on dogs, although corrections were less frequent (Fig. 11).

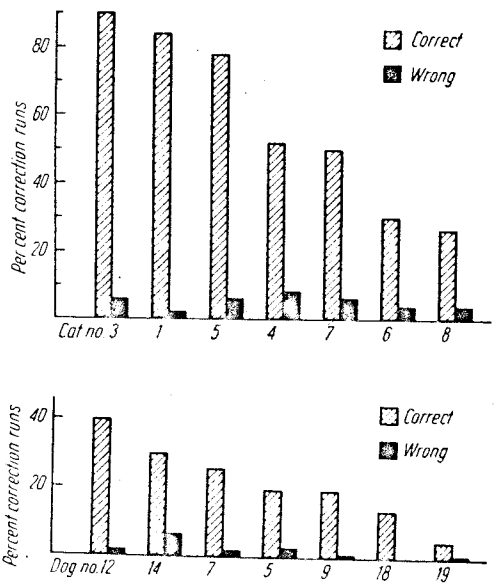

Fig. 11. The percentage of errors with corrections in prefrontal cats (upper graph) and dogs (lower graph). Each hatched column denotes the percentage of errors with "correct" corrections; each black column denotes the percentage of errors with wrong corrections (Lawicka, 1969b). Reprinted by permission of Polish Scientific Publishers, Warsaw.

Another important fact relevant for our discussion was recently obtained by Lawicka (1969b) on dogs. This author introduced a procedure described by her as the sham-trial method. It consisted of releasing the animal from the starting platform without a signaling stimulus. Normal animals, after being released in sham-trials remain, as a rule, on the platform or may slowly visit one of the feeders (Lawicka, 1969b). In contrast, the dorsofrontal animals, when released

without a signaling stimulus, rush quickly to one of the feeders, and repeat these responses over a long series of sham-trials. When, however, they finally stop doing so, and a true trial follows, the delayed response is correct. Moreover, with increasing delayed response impairment, the responses in sham-trials following each correct true trial become more numerous.

All these data indicate that the deficit in delayed response after proreal lesions in dogs does not consist in abolition of short-term memory of directional cues, but in the vulnerability of the response system controlled by this memory. Accordingly, dominance of the proper delayed response over various concomitant instrumental reflexes, such as reacting to the unleashing procedure itself, or repeating the last reinforced run, is now overthrown. To restore this dominance we must extinguish these concomitant reflexes in order to give way to the operation of the delayed response.

One can argue whether this overthrow of delayed response dominance results from an increase in conditionability of actual stimuli, which may be related to an increase of orientation reactions to exteroceptive agents (Lawicka, 1969b), or is it due to a weakening of the memory traces of locomotor kinesthetic cues. We have some evidence to believe that these cues are primarily affected. According to data collected by Mishkin, Vest, Waxler, and Rosvold (1969) after lesions in the dorsolateral area including principal sulcus, delayed spatial alternation (go left–go right) is much more severely impaired than object alternation. In fact, in the former test, in order to react correctly the animal must remember the last direction of run, whereas in the latter test he must remember the visual cue. This finding is in good agreement with the fact that the dorsolateral frontal cortex may be considered a rostral extension of the kinesthetic analyzer, being the gnostic representation of kinesthetic directional cues.

VII. General Discussion

The main set of findings described in this paper is that different "parallel" types of programs of animal motor behavior, each involving two or more motor tasks in response to various stimuli, are represented by different parts of the prefrontal cortex. These programs are: R–no R Pavlovian differentiation, R–no R both reinforced differentiation, R_1–R_2 differentiation, and R_1–R_2 delayed responses. It has been shown that these programs are, in fact, different as far as their operations are concerned and therefore it was only to be expected that their central representations should be different.

The important problem arises as to whether all other programs of complex motor behavior can be reduced to these four models. It should be realized that the tasks with which we have dealt in the present article are only a small part of all tasks of motor behavior used in experimental practice. Here belong: (1) drive reversal, (2) response reversal, (3) delayed spatial alternations, (4) delayed object

alternations, (5) delayed responses to cues not contiguous to the feeders, (6) delayed Pavlovian alternation, and many others. Furthermore, there are sequential types of programs such as locomotor maze habits on the one hand and chains of manipulatory movements on the other. The reason we are not concerned with these other programs in the present survey is simply because we do not have enough experimental documentation for both dogs and monkeys.

Let us try to analyze the enumerated programs in order to see whether they can or cannot be reduced to those described above.

Drive reversal consists in changing the signaling role of the CS with regard to *drive*. The simplest reversal of this kind is represented by extinction, in which the CS is no longer reinforced by food, and in consequence the instrumental response is inhibited. What is the role of the prefrontal cortex (or its parts) in this task? We have not studied this problem with dogs because we considered it to be identical with R–no R Pavlovian differentiation. Indeed, extinction is based on the formation of the connections between the CS center and the antihunger center (Fig. 6), exactly as is the case with a differentiated negative CS. Therefore the normal course of extinction should depend on the integrity of the same area as Pavlovian differentiation. In fact, as found by Butter, Mishkin, and Rosvold (1963) on monkeys, resistance to extinction after orbitofrontal lesions is much stronger than after dorsolateral lesions or in control animals.

Response reversal consists in changing the signaling role of the CS with regard to the instrumental response. The usual way of experimenting on this task is to confront the animal with two food wells and teach him, first, to approach one well in response to a given CS, and then to switch his response to the other one. This procedure is usually repeated many times in succession, and animals with various cortical lesions are compared.

In Lawicka's experiments (unpublished) performed on dogs, it has been established that proreal lesions fail to affect this test, at least when reversals begin after the animal has reached criterion in the preceding response.

An illustrative example of response reversal in monkeys has been provided by Mishkin (1964), who performed a number of successive reversals in two tests–spatial reversal and object reversal–after ablations sustained in the laterofrontal area, the orbitofrontal area, and inferotemporal area. His results are shown in a modified version (Fig. 12) presenting only the optimal case in which differences between the groups were most conspicuous; this took place when the task was neither too easy nor too difficult.

Notice that in spatial reversals orbital and lateral monkeys were equally impaired, while the temporal monkeys were not impaired. On the contrary, in object reversals orbital monkeys were as impaired as in spatial reversals, lateral monkeys were almost normal, and temporal monkeys were impaired.

These informative data have shown that: (1) the inferotemporal area plays the same role with respect to object reversal as does the laterofrontal area with

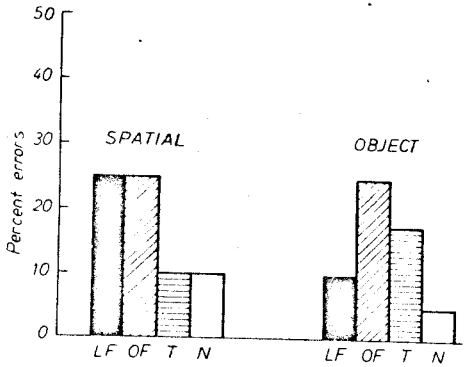

Fig. 12. Mean percentage of errors on object- and place-differentiation reversal in monkeys after lateral prefrontal lesions (LF), orbital prefrontal lesion (OF), inferotemporal lesion (T), and in normals (N). On the basis of Mishkin's data (1964), four reversals per day, each reversal includes eight trials.

regard to spatial reversals, and (2) both reversals are equally impaired after orbital lesions.

Thus the specific program concerning spatial reversal in monkeys depends on the integrity of the dorsolateral prefrontal area whereas the specific program concerning object reversal depends on the integrity of the inferotemporal area.

An unspecific role in response reversals (both spatial and object) is played by the orbitofrontal area. This role is easy to conceive if one takes into account that response reversal necessarily involves extinction of the instrumental response leading to food, whether it is a spatially guided response (where to go) or a visually guided response (to which object to go).

Thus we see that response reversal is a complex task involving either spatial or visual gnosis secured by the laterofrontal or inferotemporal cortex respectively, *and* the normal ability to extinguish instrumental responses secured by the orbitofrontal cortex.[2]

Delayed spatial alternation again combines two mechanisms: one functions to remember what was the last response (left or right), the faculty depending in monkeys on the integrity of the principal sulcus (Mishkin, 1957; and others); the other mechanism functions to easily "extinguish" the preceding response in order to alternate, the faculty depending on the orbitofrontal area. Consequently lesions in both areas impair this test.

[2] Recent experiments by Butter (1969) seem to indicate that lesions in the lateral orbital area produce an impairment of reversal learning, while lesions in the posteromedial orbital area affect mainly extinction. This result suggests that there might be a difference between simple extinction and response reversal training. This problem requires more detailed investigation.

On the other hand, delayed object alternation is obviously impaired for the same reason after orbital lesion, but it is not (or at least less) impaired after lateral lesions (Mishkin *et al.*, 1969).

Tests on delayed responses to cues noncontiguous to the feeders were performed in monkeys by Stepien and Stamm (1970b). Cues were spatially opposed to the signaled feeders so this test combined two programs: delayed responses and R_1–R_2 differentiation. Therefore it should be impaired both after dorsolateral and principal sulcus lesions. As seen in Fig. 13, this is precisely what occurred. After dorsolateral nonprincipal lesions, R_1–R_2 differentiation with and without delay was severely impaired. Note that initially the no-delay task was even more "impaired" (errors much above chance), because of the magneto-reaction. The delay task begins with chance level responding and remains at this level throughout the experiment, even when the no-delay responses become normal—demonstrating that R_1–R_2 differentiation with delay is even more difficult than it is without delay. On the other hand, lesions in the principal sulcus destroyed irreversibly the delayed response, while the no-delay differentiation was only insignificantly impaired. After total dorsolateral lesions,

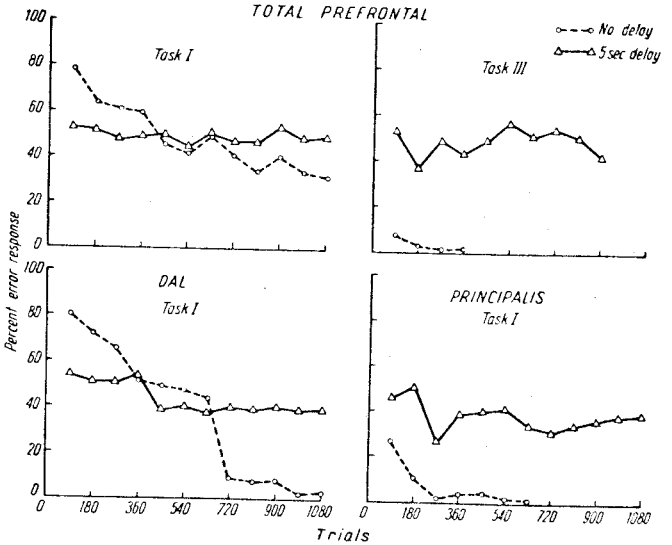

Fig. 13. Means of errors (two monkeys per group) for postoperative testing under no-delay and delayed response conditions. Task I, spatial opposition of CSs; Task III, spatial contiguity between CSs and food locations. Total prefrontal means dorsolateral cortex; "DAL" means cortex surrounding principal sulcus. In task I: total dorsolateral lesions produce severe impairment (chance level) on both no-delay test and 5-sec delay test, DAL lesions produce the same effect, principalis lesions produce impairment in only 5-sec delay test; on task III: total dorsolateral lesions produce impairment in 5-sec delay test, but not in no delay test (Stepien and Stamm, 1970b). Reprinted by permission of Polish Scientific Publishers, Warsaw.

R_1-R_2 differentiation was equally impaired with and without delay, but in the task with spatial contiguity between the cue and the feeder (task III in Fig. 13) only the delayed response was impaired.

Delayed Pavlovian alternation occurs when the same CS is reinforced every second time. These experiments were performed on dogs with 1-min intertrial intervals, which means presentation of food every 2 min (Szwejkowska, Lawicka, and Konorski, 1964). When the task was mastered, it turned out that the animal did not solve the problem according to the alternation program; instead, the act of eating on reinforced trials became a conditioned inhibitor with regard to the next CS, with the inhibitory aftereffect of about 1 min. This was proved by the following facts: (1) when food was given "gratis" without the CS, the dog never performed the trained movement on the next trial; (2) when CSs were presented twice or thrice during the inhibitory aftereffect (about 90 sec) the animal did not perform the trained movement, and (3) when the intertrial interval amounted to 2 min, that is, the negative trial was lacking, the animal did perform the trained movement, as if the negative CS were interspersed. Thus we have here a good example of a situation in which the program established by the experimenter and that adopted by the animals were quite different (cf. Section I).

In order to eliminate this pseudoalternation and teach the animal to perform true alternation, the intertrial intervals varied from half a minute to 2 min, and eventually, after long training the animals succeeded in solving the task (Szwejkowska, 1965a). Animals trained by a fixed or variable intertrial interval sustained prefrontal ablations either medial or dorsal (Szwejkowska, 1965b). The effects of these lesions are represented in Fig. 14. It may be seen that in the group with fixed intertrial intervals (which reacted according to the R–no R Pavlovian differentiation program) medial lesions produced a clear disinhibitory syndrome, whereas dorsal lesions failed to do so. However, in the group with variable intervals, the impairment was much more severe and concerned animals with both dorsal and medial lesions. This suggests that the true Pavlovian delayed alternation test is rather complex and involves both drive inhibition and some delay component. In fact the animal *must* now remember at each trial whether the preceding trial was negative or positive.

As far as sequential types of programs are concerned, we do know from experiments on rats (Dabrowska, 1964, locomotor sequences) and primates (Jacobsen, 1934, manipulatory sequences) that prefrontal lesions are detrimental for these tasks. We do not know, however, which particular anatomical areas are crucial.

To summarize all the programs discussed in this section, we may observe that in all probability they *can* be reduced to those dealt with in our previous sections. The only exception is object alternation and object reversal. They are essentially visual tasks controlled by inferotemporal cortex; however, the orbitofrontal cortex largely contributes to their proper performance.

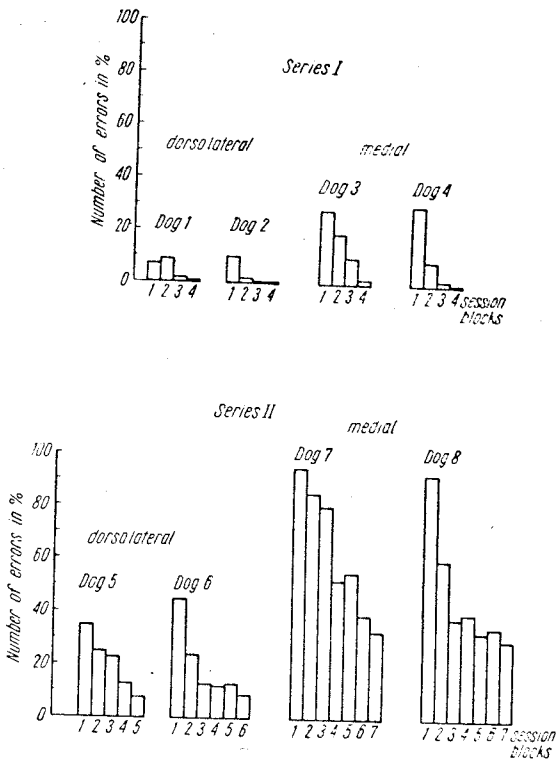

Fig. 14. The effects of prefrontal lesions on Pavlovian R–no R alternation. Series I: fixed intertrial intervals of the 1-min duration; Series II: varying intertrial intervals from ½ min to 2 min. Each column denotes 10-session block (80 inhibitory trials). (From Szewjkowska, 1965b.)

VIII. Summary

Acquired behavioral acts denoted as instrumental conditioned reflexes may be divided into two categories differing in their central mechanisms. To the first category belong simple instrumental responses to appropriate conditioned stimuli, according to the paradigm CS–R, where CS stands for the conditioned stimulus and R stands for the instrumental response. In the second category belong discriminative responses: a subject must select a particular instrumental response among two or more responses which are likely to occur in the given situation. In contradistinction to the first category, the second category demands that the animal make a decision what response should be performed in order to achieve the goal. The general paradigm for this category is: $CS_1–R_1$, $CS_2–R_2$, etc., and the corresponding training consists in learning which response

is correct and which is wrong in the presence of a given conditioned stimulus. This paper was concerned mainly with the second category of instrumental responding.

In experimental practice we may discern the following tasks in which decision making is necessary; for the sake of simplicity, only instrumental responses based on food reinforcement are taken into consideration.

1. The subject must learn that in the presence of a given stimulus he should perform a given movement in order to receive food, but in response to a similar stimulus he should not, because this stimulus is never followed by food. The appropriate paradigm is: $CS_1 - R_1 \rightarrow$ Food, $CS_2 -$ no $R \rightarrow$ no Food.

2. The subject learns a similar task, but whereas CS_1 is reinforced when R is executed, CS_2 is reinforced when R is *not* executed. The paradigm of this procedure is: $CS_1 - R \rightarrow$ Food, $CS_2 -$ no $R \rightarrow$ Food.

3. The subject learns to perform R_1 in response to CS_1, and R_2 in response to CS_2, both these responses, when performed to the proper stimulus, being reinforced by food. The corresponding paradigm is

$$CS_1 - R_1 \rightarrow \text{Food}, CS_2 - R_2 \rightarrow \text{Food}.$$

4. The subject first learns the task specified in the preceding paragraph, but thereafter performance of the response is allowed by the experimenter to occur only to the trace of the CS (the so-called delayed response). The corresponding paradigm is

$$\text{tr } CS_1 - R_1 \rightarrow \text{Food}, \text{tr } CS_2 - R_2 \rightarrow \text{Food}$$

where "tr" stands for "short-term memory trace of."

In experiments performed on dogs and monkeys it has been shown that correct performance of these tasks depends on the integrity of the prefrontal region of the cerebral cortex, but for a given species each task requires the integrity of a specific part of this region. Thus the first task is impaired after ablation of the medial part of the prefrontal region in dogs and the ventral part in monkeys; the second task is impaired after ablation of the lateral prefrontal region in dogs and dorsolateral part (excluding the principal sulcus) in monkeys; the third task is impaired in monkeys after ablation of the arcuate sulcus; the fourth task is impaired after ablation of the dorsal prefrontal area in dogs and the principal sulcus in monkeys.

This functional compartmentalization of the prefrontal cortex is understandable if we take into account that in these experiments we have to deal with at least three different physiological mechanisms: task one is based on drive inhibition, tasks two and three are based on discriminative connections between stimuli and responses, while task four is based on short-term memory of the decisions made in advance. The problem remains whether or not other tasks requiring the integrity of the prefrontal cortex are reducible to these three mechanisms.

Acknowledgments

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