ACTA NEUROBIOL. EXP. 1972, 32: 673-687

# FRONTAL LOBE AND MOTIVATION OF LEARNED BEHAVIOR

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*Abstract.* Dorsal prefrontal lesions in dogs elicit hyperreactivity in instrumental discrimination and perseveration in trained serial action pattern. Lateral premotor injury causes hyporeactivity in instrumental discrimination and prolongation of activity in the trained serial action pattern. When the dorsal prefrontal area was injured together with the premotor region the result was again hyporeactivity in instrumental discrimination and prolongation of activity in the trained serial action pattern. Instrumental discrimination and behavior in the serial action pattern changed in the same direction after a dorsal prefrontal lesion and in fasting, or after lateral premotor injury and satiation, respectively.

Of the disorders manifested in frontal lobe lesions, one group (hyperreactivity, distractability, deterioration of delayed reactions and discrimination, responses elicited by novelty, etc.) tends to direct attention to an impairment of the signalling role of external stimuli, and serves as a basis of relevant hypotheses (change in the reflexogenic strength of conditioned stimuli, Konorski and Ławicka 1964; sensory defect, Symmes 1967, etc.). Other disorders (such as the errors of alternation, poor performance on seriatim problems, overreaction in tasks of fixed interval and differential reinforcement of low rates of responding, augmented preference and perseveration, etc.) suggest that lesions affect such behavioral mechanisms that are less subjected to exogenous driving conditions. These underlie another group of theories (kinesthetic gnosis of spatial relations, Konorski 1967, Stamm 1970; impairment of programmed actions, Luria 1969, 1971, etc.). Finally, in the deficits on which the third group of explanations is founded (e.g., disinhibition, Kalischer 1911, Stanley and Jaynes 1949; hypermotivation, Fulton 1951, Brutkowski 1964, 1965; reduction of reinforcement effects, Pribram 1960; a greater persistence of the initial set, Brush, Mishkin and Rosvold 1961, Mishkin 1964; disturbance of the "corollary discharge", Teuber 1964; and so on), both kinds of functions are involved.

In forming one's opinion about these partly conflicting ideas, it seemed promising to measure the effect of frontal injuries by the changes revealed in both the behavioral patterns that were mainly directed by external stimuli, and others which were less subject to external stimuli, and more to the internal factors of behavior. The first series here reported concentrated accordingly to such reactivity that is directed primarily by external factors, namely, sensory discrimination. The second part of our work analyzed the time course of certain stereotype action patterns whose training procedure implied no change in external signals. Learned behavior was changed in both series of experiments. Consequently, our third series was focused at that class of mechanisms whose effect was detectable in both the reactivity to external stimuli and the trained stereotypes.

*Investigation of instrumental discriminative reactivity*

Frontal lesions may be associated with one of two syndromes having an opposite nature: either with agitation, euphoria and hyperexcitability or else with hypomotility, apathy and depression (Feuchtwanger 1923, Rylander 1939, Freeman and Watts 1942, Hécaen 1964). Some of the authors explain this contradiction by personality traits and claim that pycnic patients are liable to become euphoric whereas leptosome ones succumb to depression, lively persons grow restless while sluggish ones become torpid (Rylander 1939, Freeman and Watts 1942). Others suggest the possibility that different frontal structures may work dissimilarly (Fulton 1951, Luria 1969). In animal experiments, where lesions conform to boundaries, only the syndrome of hyperactivity and hyperreactivity could be reproduced by dorsal prefrontal (DPF) lesions (Kalischer 1911, Allen 1939, 1941, 1943, 1949*ab*, Brutkowski et al. 1956, Brush et al. 1961, Konorski 1961, Konorski and Ławicka 1964, Brutkowski 1964, 1965, Mishkin 1964, McEnaney and Butter 1969, etc.). However, more extensive lesions, particularly if they involve the lateral premotor area (LPM) as well, will impair learned reactivity (Jacobsen 1934, Kennard 1939, Pribram et al. 1955/56, Gerbner 1959, 1962, 1965, Stępień et al. 1960, Yamaguchi et al. 1963, Gerbner and Pásztor 1964, 1965, Pásztor and Gerbner 1964, 1965). This area appears to correspond to the postero-inferior portion of the human convexity in the lesions of which Luria (1969) observed a syndrome of deficient spontaneity and initiative. Thus, the lesions of the prefrontal, medial and lateral premotor areas are likely to provide information on the question whether conflicting syndromes were due indeed to injuries involving different structures.

Dogs were trained in instrumental discriminitaion (for details of technique *see* Gerbner 1971). Sessions consisted of presenting nine trials of four kinds of stimuli in random sequence, six of them being instrumentally conditioned and three discriminative ones. Movements of the leg that were elicited by the conditioned stimuli (either a 300 cycle/sec generator tune or the sound of a buzzer) were rewarded by food. The simple discriminative stimulus was a 700 cycle/sec generator tune while the complex discriminative stimulus was a buzzer sound presented 5 sec after the ringing of a bell. The instrumental responses occurring during discriminative stimuli were not reinforced. Surgery took place when the animals committed less errors than 5%, or after the 200th session, at latest. Lesions were given by subpial suction under aseptic conditions. To locate the lesion at the end of the experiment Nissl staining was used.

In the DPF lesion group discriminative errors became more frequent than pre-operatively. The number of intersignal errors increased as well. As reflected by the errors, the previously adequately learned reactivity

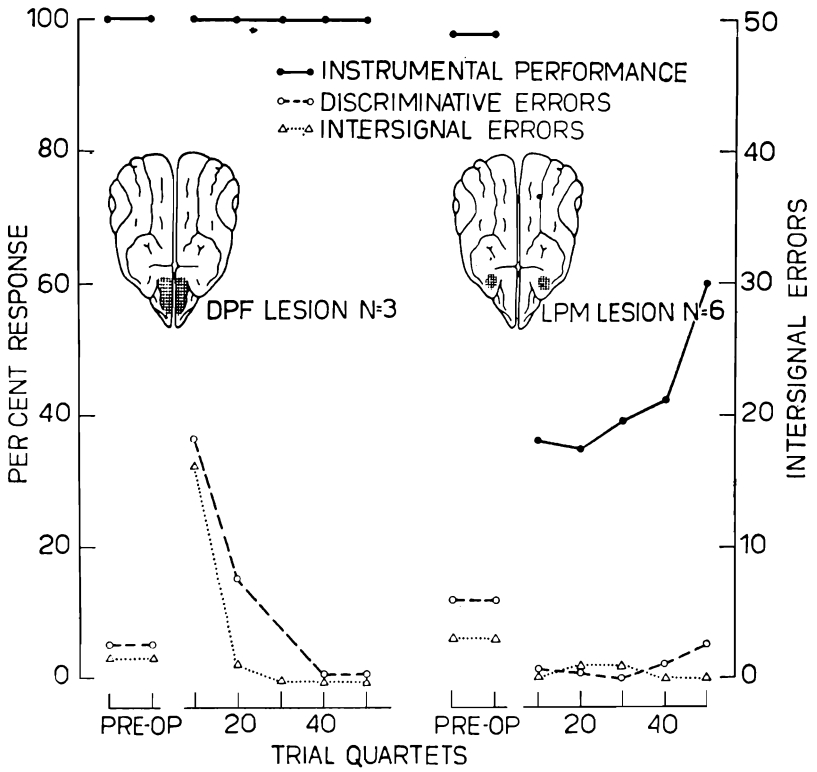


Fig. 1. Mean changes of discriminative reactivity following dorsal prefrontal and lateral premotor lesions.

became enhanced. On the other hand after LPM lesions instrumental performance became depressed: the animals responded less often both to conditioned and discriminative stimuli. Reactivity previously learned to criterion fell below the adequate level. In case of medial premotor (MPM) lesions the changes were not significant. Thus, reactivity was affected by DPF and LPM lesions in opposite direction (Fig. 1).

In certain actions of some frontal patients as well as in the learned responses of animals after prefrontal lesions perseveration and hyperactivity were repeatedly observed (Harlow 1848, 1868, Fulton 1951, 1952, Kirschbaum 1951, Jarvie 1954, 1960, Lauber 1958, Toczek 1960, Milner 1964, 1966, animal experiments: Kalischer 1911, Allen 1939, 1941, 1943, 1949*ab*, Brutkowski et al. 1956, Brush et al. 1961, Konorski 1961, Konorski and Ławicka 1964, Brutkowski 1964, 1965, Mishkin 1964, McEnaney and Butter 1969). Our experimental result suggests the possibility that this inadequate hyperreactivity may be related to the activating effect of the uninjured LPM area. The mechanism located here might be the very

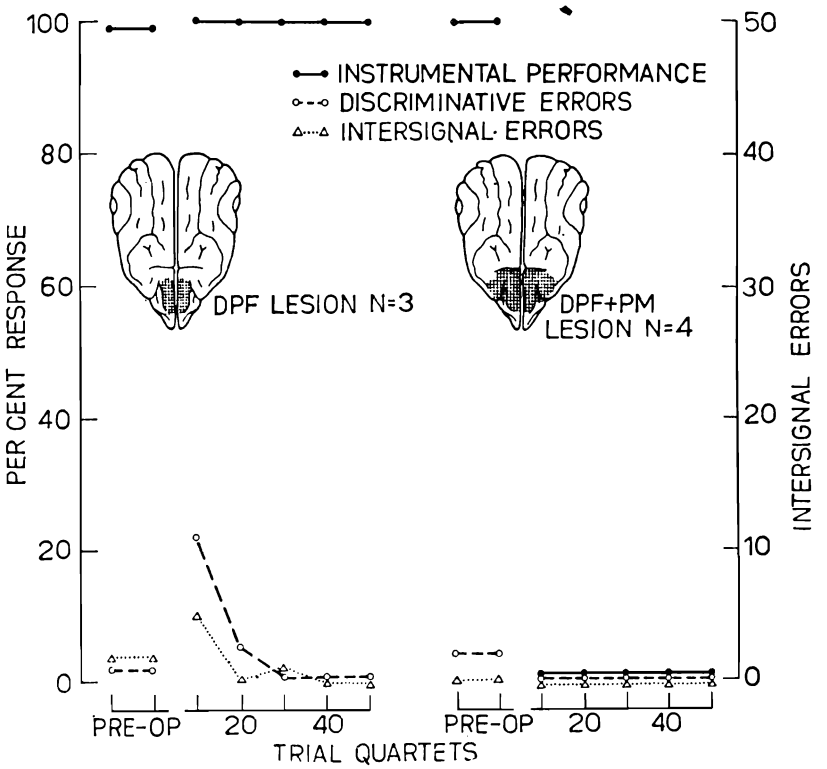


Fig. 2. Mean changes in discriminative reactivity following solitary dorsal prefrontal and combined prefrontal-premotor lesions.

"spring" that makes "Jack jump out of the box". Assuming this to be true we may postulate that in combined prefrontal and premotor lesions hyperreactivity associated with prefrontal lesions would fail to develop.

In pure DPF lesions a similar hyperreactivity was observed as in the former experiment. On the other hand, when the premotor area was injured along with the prefrontal cortex, reactions were completely lost. The hyperreactivity ensuing prefrontal injuries thus seems to be related to the activating influence of the LPM cortex (Fig. 2).

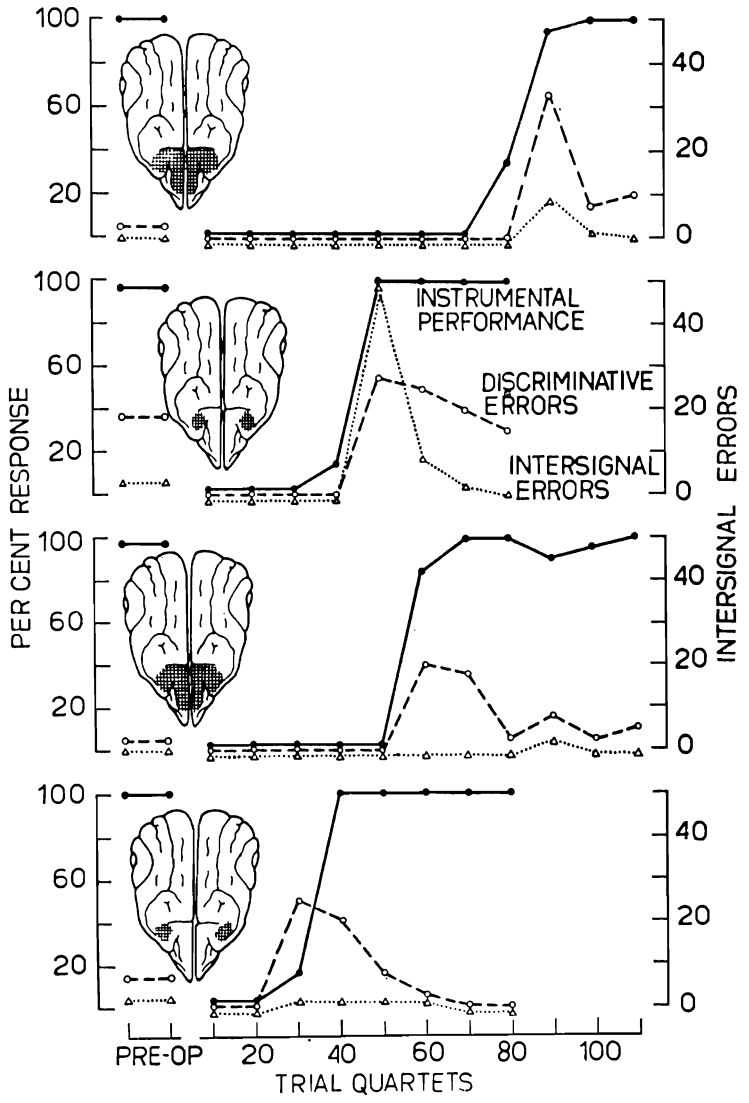


Fig. 3. Spontaneous restoration of injured discriminative reactivity (single dogs).

We believe that during instrumental discriminative learning, i.e., in activating rewarded responses and deactivating (inhibiting) unreinforced reactions, two submechanisms are cooperating. The assumption is supported from the fact that during spontaneous recovery of instrumental reactivity observed in some animals subjected to LPM injury, the frequency of erroneous discriminative and intersignal reactions increased. This overshoot of the recovered reactivity before adjusting itself again to the adequate level is rather similar to the "behavior" of regulating systems (Fig. 3).

*Activation of trained stereotype seriatim patterns*

With prefrontal injuries it is a common observation, both clinically and experimentally, that actions in process tend to perseverate, serial patterns become simplified, and programmed activity deteriorates (Jacobsen 1931, 1936, Luria 1969, 1971). One group of our experimental animals was trained in a simple stereotype pattern of behavior. From beyond a grating, the dogs had to pull through 10 pieces of meat in each session. The succession of foot movements was very rapid already by the seventh of eighth session, thus the total time of the reaction series changed very little later. Surgery was performed after the tenth session. Acquisition of food by the foot movements continued to be very swift after DPF lesions, and the speed of the reaction pattern did not change. As it is, the trained rate could not increase any more. Following LPM lesions, however, the intervals between the forelimb movements became longer making a growth in the total time of the pattern. A slighter, but still significant elongation was found with MPM lesions. When the premotor area was injured, together with the prefrontal cortex the series of trained actions showed similar changes like a pure LPM injury: the behavioral pattern took a longer time (Fig. 4).

Another group of animals was trained in an action pattern consisting of alternating elements. In this case the grating was divided into two parts by a plate. When the first piece of meat had been pulled through, the subsequent piece was placed beyond the other half of the divided area, thus the dog had to run over to the other side of the partition in order to get the meat. The reaction then continued in this alternate fashion: acquisition of the meat alternated with running to the other side.

DPF lesion performed after the tenth session caused a perseveration in the food acquiring movements. In another version of this experiment the visual cue was excluded by placing meatlike but unpalatable polyurethane pieces beyond the same side of the grating as soon as the dog had pulled through its piece of meat. Here DPF lesions forced the ani-

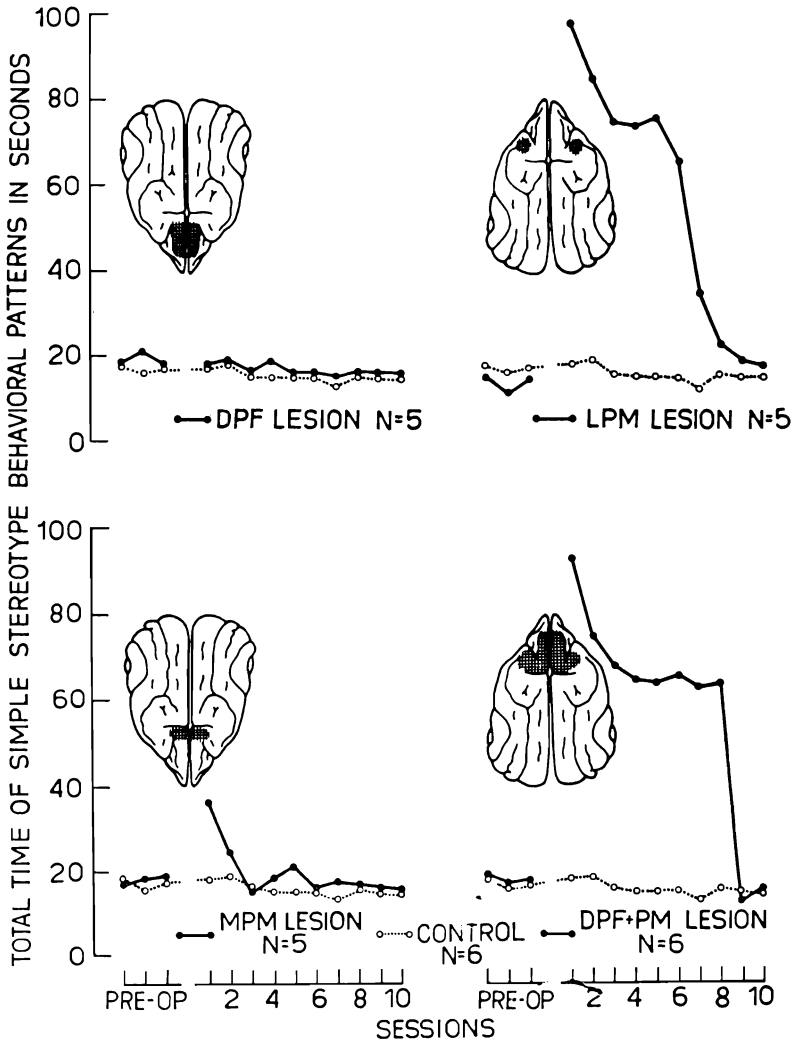


Fig. 4. Mean changes of the duration of simple trained stereotype action patterns following dorsal prefrontal, lateral and medial premotor, and combined prefrontal and premotor lesions.

mals to pull through a greater number of plastic pieces than pre-operatively, moreover, the dogs sometimes even ate them. Running over to the opposite side of the partition became thus delayed and the total time of the behavioral pattern grew longer. With LPM lesions the forelimb movements were rarely repeated, yet the total time of the action pattern was greater, this time owing to the elongation of the interelement interval of the action (Fig. 5).

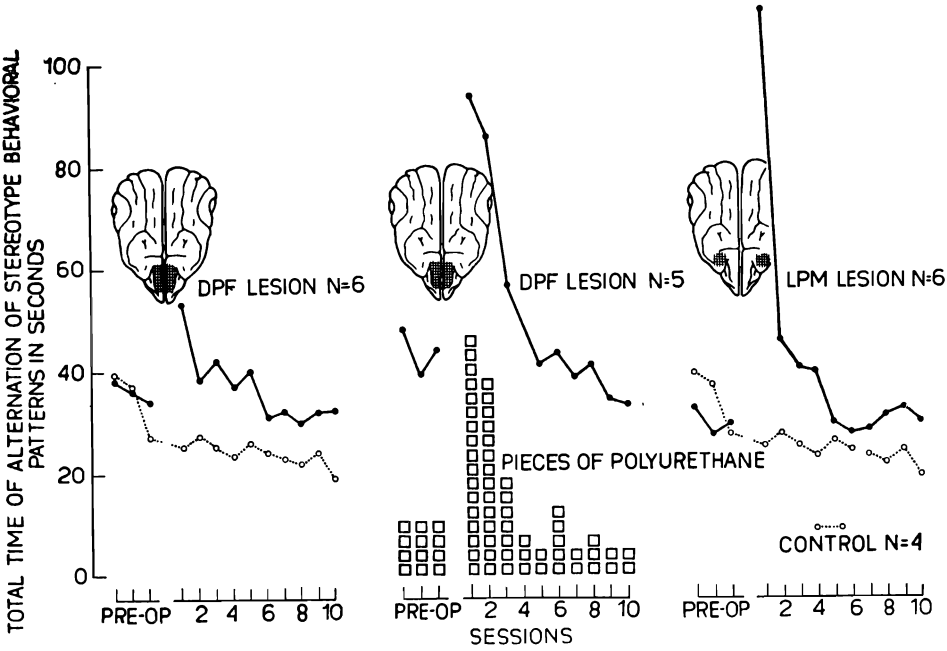


Fig. 5. Mean changes of the duration of trained alternating stereotype action patterns in dorsal prefrontal injury, after dorsal prefrontal lesions investigated by means of plastic meat substitutes, and in lateral premotor lesions.

DPF lesions were thus manifested by an inadequate, perseverative activation of the trained movements, whereas after LPM injuries the activation proper of the elements in the pattern suffered reduction. In the case of combined prefrontal and premotor lesions, the trained stereotypic pattern was impaired in the same way.

### *Frontal lobe and motivation of learned behavior*

Similar changes in both externally induced reactivity and trained stereotype performance gave an impetus to search for common causative factors. One such common source might be the impairment of the mechanism which had been most often related hypothetically to disinhibition (Kalischer 1911, Stanley and Jaynes 1949), motivation (Fulton 1951, Brutkowski 1964, 1965), reinforcement (Pribram 1960), the initial set (Brush et al. 1961, Mishkin 1964), and to "corollary discharge" (Teuber 1964). These conceptions appeared to us to rely on some internal relationship. Stronger motivation may bring about disinhibition and a defect of discrimination, it may increase reactivity, and under certain circumstances (as for example, in reversal) it may give rise to a greater persistence or inertia

of initial sets. Under the effect of reinforcement, stimuli induce definite actions. In this sense, reinforcement is a motivating agent. Central receptor structures may be preset by the "corollary discharge" of proprioceptive impulses associated with movements. In case of being supported by reinforcement this presetting may be the basis of discriminative reactivity and of the performance in trained stereotypes. The accentuation of motivation among these related conceptions may be justified by pointing out that it may be a common basis for all the other mentioned mechanisms and its structural relationships are relatively better elaborated.

The postulated relationship between motivation and frontal lobe functions derived from the connections of the thalamus with the frontal lobe, on the one hand, and on the other, from the already outdated suggestion that the organ of affectivity was the thalamus. It was believed that affective sensations were connected with the process of action by thalamofrontal pathways, backing it up by dynamic power and volitional motivation (Herrick 1926). Only medio-basal frontal lesions were able to elicit behavioral changes associable with affective-motivational functions (Fulton 1951, Brutkowski 1965). Consequently, merely the effects of the latter injuries used to be explained by the impairment of the affective-motivational functions. This belief was founded on the fact that medio-basal-limbic connections had been much sooner discovered than the limbic connections of dorsal frontal structures had become clarified (Nauta 1964). As a consequence, even the representatives of the motivation concept fostered the opinion that the function of dorsal frontal structures were of a different nature, and associated it with intellectual processes or with "act inhibition" (Fulton 1951, Brutkowski 1965).

In view of this argumentation the effect of DPF and LPM lesions was experimentally compared to those of satiation and fasting influencing motivation. Instrumental discrimination was studied as well as the fate of trained stereotype behavioral patterns. Each group of animals were subjected to the effect of two opposing but related agents.

Reactivity and stereotype changed in the same direction under the effect of DPF lesion and fasting, and of LPM lesion and satiation, respectively. Whereas the effects of prefrontal injures were most pronounced in the early post-operative period, fasting and satiation, on the other hand, caused maximal effects in the later sessions (Fig. 6 and 7).

The changes of learned performance were not accompanied by general behavioral changes in case of lesions. On the other hand, fasting often produced hypermotility and enhanced affectivity while satiation elicited unresponsiveness. As mentioned above, the connection of dorsal frontal structures with motivation had been denied primarily because former investigators could not observe such behavioral changes in the course of

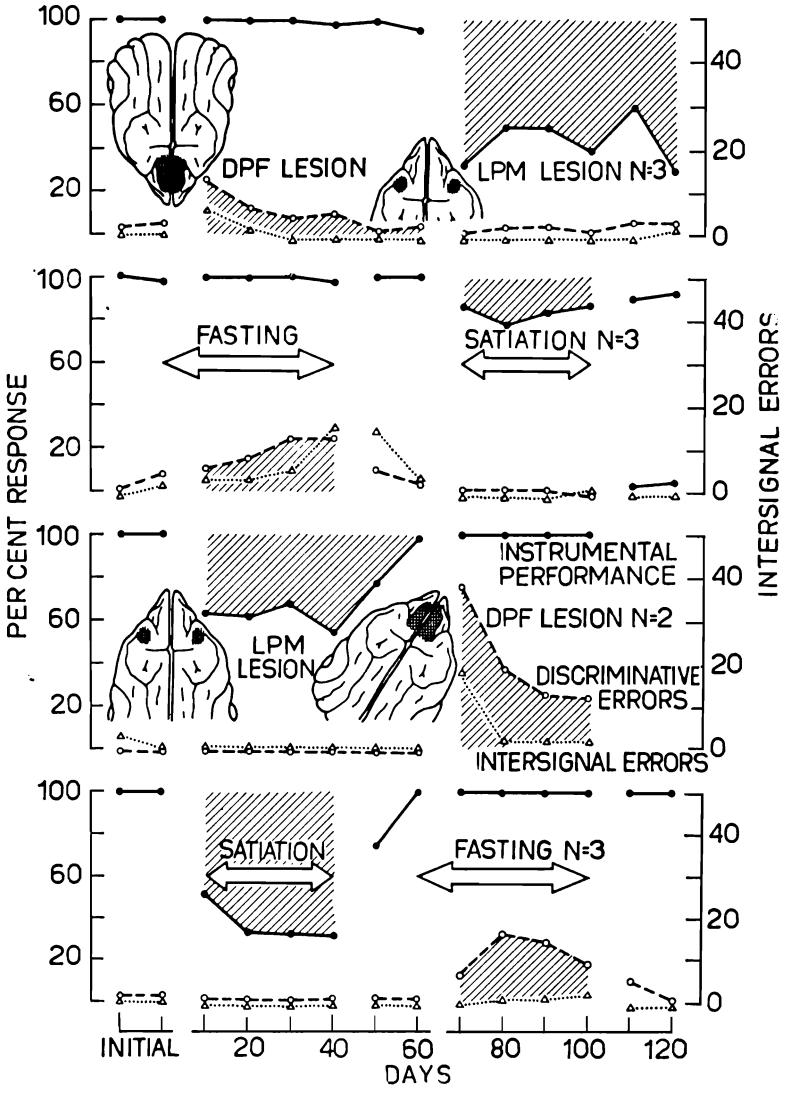


Fig. 6. Mean changes of discriminative reactivity after dorsal prefrontal and lateral premotor lesion, and after satiation and fasting, respectively. Upper row, DPF-LPM lesion; second row, fasting-satiation; third row, LPM-DPF lesion; bottom row, satiation-fasting. Full circles, instrumental performance; open circles, discriminative errors; triangles, intersignal errors. Hatched areas represent discriminative errors and deficit of instrumental responses.

dorsal lesions that were suggestive of motivation effects. It is possible, however, that merely the "cue" mechanism of motivation is affected by the dorsal frontal lesions, and the effect revealing itself in vigility, vigilance, affectivity, and motivational patterns of behavior remained rela-

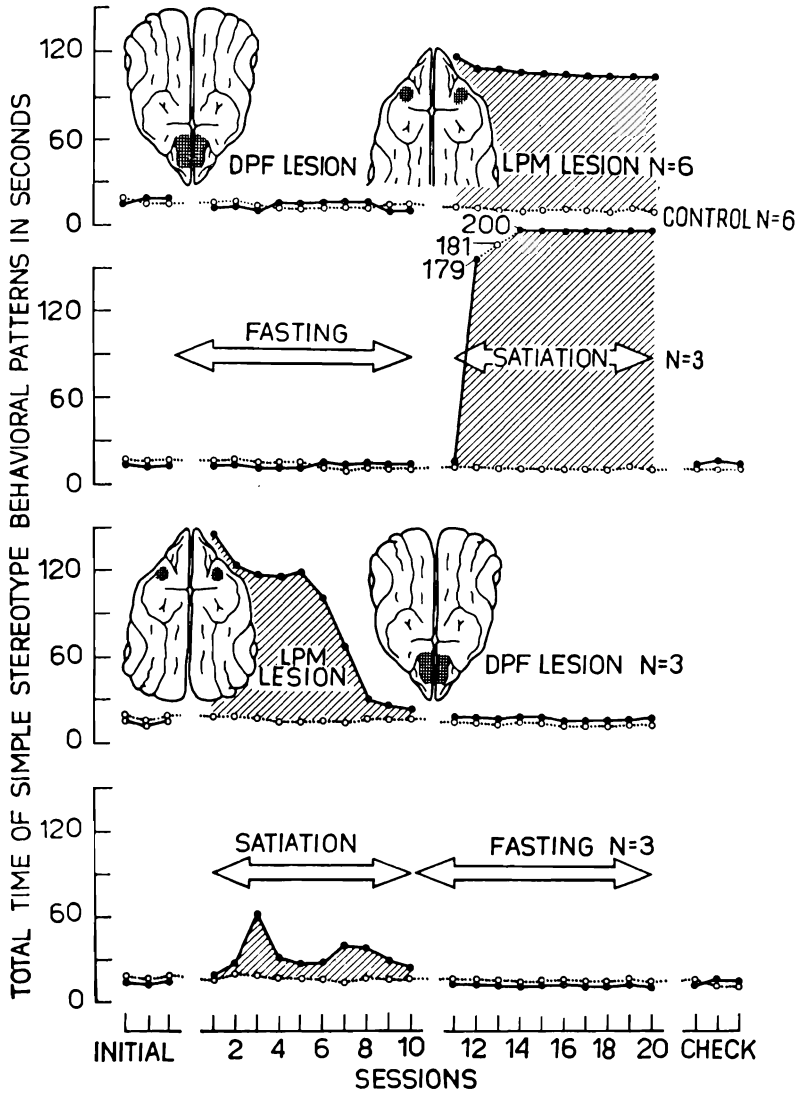


Fig. 7. Mean changes of the duration of a simple trained stereotype action pattern following dorsal prefrontal and lateral premotor lesion, satiation and fasting, respectively. Upper row, DPF-LPM lesion; second row, fasting-satiation; third row, LPM-DPF lesion; bottom row, satiation-fasting. Full circles, test animals; open circles, control animals. Hatched area denote prolongation of the total times of simple trained stereotype behavioral patterns.

tively unimpaired. The recently clarified connections between dorsal frontal structures and the hippocampal region, further the resemblance between hippocampal and prefrontal lesion effects (Kimble 1968, 1969), are suggestive of a hippocampal — dorsal frontal cooperation.

## *Conclusions*

Dorsal frontal lesions produce changes in discriminative reactivity and the time-course of stereotype action patterns. According to our assumption, the reason for these effects lies in the impairment of motivational "cue" mechanisms. These mechanisms are involved in the conceptions of disinhibition, reinforcement, initial set and "corollary discharge", and would underlie an alteration of the reflexogenic strength of conditioned stimuli and a deficiency of programmed actions.

On the basis of conflicting frontal syndromes indications of a regulation were found. Dorsal prefrontal lesions of the cortex caused defects in adequate discrimination and in the deactivation (inhibition) of alternating stereotype action patterns. Lateral premotor injury impaired instrumental reactivity and the activation of elements in serial action patterns. The effect of dorsal prefrontal lesions appeared to be related to the function of the lateral premotor mechanism. The overshoot of recovering reactivity, also was similar to the "behavior" of regulating systems. According to our hypothesis, adequate discriminative reactivity in learned behavior as well as the correct stereotype order of elements in trained serial action patterns would develop by the regulative interrelationship of these activating and deactivating (inhibitory) mechanisms.

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*Received 31 August 1971*

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