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THALAMOGENIC MOVEMENTS IN CATS: THEIR CHARACTERISTICS AND DEPENDENCE ON THE CEREBRAL CORTEX

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Numerous studies concerning the effects of electrical stimulation of the cerebral cortex, performed either in waking animals by means of implanted electrodes, or in waking patients during neurosurgical operations, have shown that not only the motor area, but also a number of other areas are electrically "excitable"; that is, their stimulation gives rise to various motor activities (Foerster 1936, Penfield and Boldrey 1937, Dusser de Barenne et al. 1941, and others). In particular, stimulation of the sensory area in waking cats was shown to produce isolated movements of the contralateral limb (hindlimb or forelimb), similar to, but not identical with, those arising from stimulation of the motor area (Tarnecki 1962a, Tarnecki and Konorski 1963). The chief differences are: longer latencies, gradual and not abrupt initiation and termination, higher thresholds, and a more natural character. The fact that, with respect to the hindleg, only those movements produced by stimulation of the sensory area, but not of the motor area, are instrumentally conditionable (Tarnecki 1962a, Tarnecki and Konorski 1963) proves that the former movements have a "reflex" origin (cf. also Konorski 1967).

To our knowledge there were no systematic studies on the effects of stimulation of the ventrolateral (VL) and ventral posterolateral (VPL) thalamic nuclei, that is, those nuclei which have direct connections with the motor and sensory cortex respectively. Therefore, the purpose of the present study was to fill this gap.

MATERIAL AND METHODS

The experiments were performed on twelve cats in an experimental box of the size 85×50×75 cm. In the front side of the box there was a moving glass wall through which the experimenter observed the animal.

In each cat in two operations separated by an interval of a few days bipolar stainless steel electrodes of a diameter of 0.2 mm and 0.5 mm tip separation were implanted into the VL and VPL nuclei respectively using a stereotaxic apparatus. The VPL implantation was performed on one side under 35 mg/kg nembutal anesthesia, the VL implantation was performed on the other side under 50 mg/kg chloralose anesthesia. The localization of the electrodes was determined by stimulation with the needle electrodes of the contralateral forepaw and hindpaw and recording evoked potentials from the corresponding nuclei of the thalamus. The thalamic electrodes were fixed in the position of the maximal amplitude of evoked potentials. It should be noted at once that it was much easier to find points responding to stimulation of the forepaw than of the hindpaw.

Two weeks after the final operation the animals were brought into the experimental box (to which they had been habituated beforehand) and the electrodes were connected with a socket connected with the Grass stimulator through a loose soft wire hanging from the ceiling of the box. Thus free locomotion of the animal inside the box was insured.

In a few animals stimulation of various parameters was used in order to choose those which were most adequate for eliciting the isolated movements of either the forelimb or the hindlimb. The current was measured from the voltage drop on ten ohms resistance connected in series with the electrodes. When this was achieved eight animals were trained in instrumental conditioning by reinforcing the thalamogenic movement with presentation of food. This part of the experiment will be described in a subsequent paper (Tarnecki and Konorski 1969).

When the CR training was completed, or, in those animals which were not trained, the stimulation testing was terminated, ten animals out of twelve were subjected to a cortical operation under nembutal anesthesia. In this operation the unilateral sensorimotor area of the cortex, and in some animals in addition the premotor cortex, was removed. The side of the lesion was contralateral to that limb which was used in instrumental conditioning.

Two weeks after this operation the animals were tested in respect to their instrumental CRs (to be described in the next paper) as well as the effects of thalamic stimulation. Testing of stimulation was performed in a part of the box remote from the food well in order to be certain that no instrumental responding was admixed to the evoked movement. In some test experiments the animals were satiated before the session, a measure completely abolishing instrumental responding on that day.

After stimulation experiments and conditioning experiments were terminated, neural tissue surrounding the points of the electrodes was coagulated, the animals were sacrificed and their brains perfused with 10% formalin. The reconstruction of cortical lesions was made and the location of the electrodes was verified using the Klüver staining technique.

RESULTS

Parameters of stimulation. In a pilot study the optimal frequencies of stimulation were established. Beneath we present an example of this exploration. The pulse duration was 1 msec.

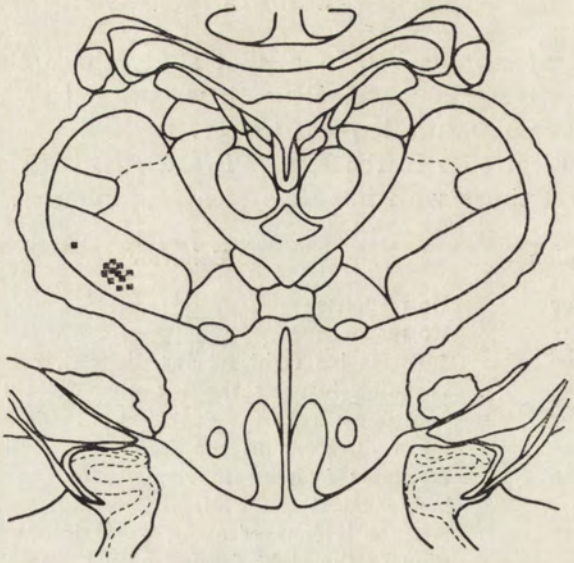
Cat no. 1. Electrodes in the left VPL nucleus. Each stimulation was repeated several times with intervals of a few minutes.

Stimulation	Behavior
1.0 v 50 c/sec	No response
3.0 v 50 c/sec	No response
5.0 v 50 c/sec	Inspects his right foreleg, licks it, remains quiet.
8.0 v 50 c/sec	Disquiet, inspects the leg, then tries to escape.
3.0 v 150 c/sec	Lies down, rubs the contralateral side of the body against the wall, looks at the forepaw and licks it.
5.0 v 150 c/sec	Irregular isolated movements of the foreleg, rubs his body against the wall, after stimulation licks the leg.
3.0 v 300 c/sec	After a latency of about 4 sec isolated and prompt flexion of the right foreleg with a slight turn of the head to the right; sometimes licks his paw. The responses are rapid and regular.

As seen from this protocol, the optimal frequency of stimulation was in this cat 300 c/sec. Since the similar picture was seen in other cats, this frequency was adopted in this series of experiments.

Stimulation of the VPL nucleus. The characteristics of the responses of the animals are given below. The frequency of stimulation was always 300 c/sec, the pulse duration 1 msec. The optimal current of stimulation was 0.2—0,4 ma. The locations of electrodes are presented in Fig. 1a.

Cat no. 1.	3 v.	Isolated and regular lifting of the contralateral foreleg, slight turn of the head towards the leg, licking the paw.
Cat no. 2.	4.5 v.	Rapid high lifting of the contralateral foreleg with the turn of the head in its direction. When during the stimulation food is presented the movement is discontinued and the animal runs normally towards the food well.
Cat no. 3.	1.5 v.	The cat gets up, inspects his body, sniffs.
	3.0 v.	Isolated and regular lifting of the contralateral foreleg.
Cat no. 4.	3.0 v.	Sniffs, lies down.
	5.0 v.	Lifting of the contralateral foreleg, sometimes with turning of the head. Irregular latencies.
Cat no. 5.	4.5 v.	Isolated lifting of the contralateral foreleg, sometimes looks at it. Low amplitudes of the movements.
	6.0 v.	The cat becomes immobile, then tries to escape.
Cat no. 6.	3.0 v.	Rapid and regular lifting of the contralateral foreleg. Continuation of stimulation produces movements of head and neck.



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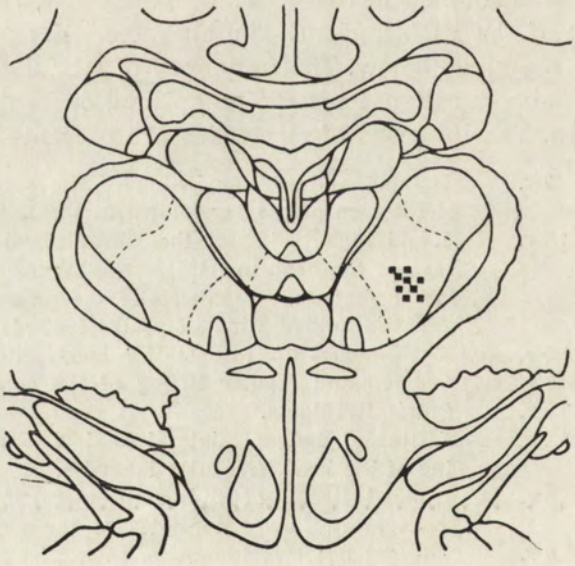
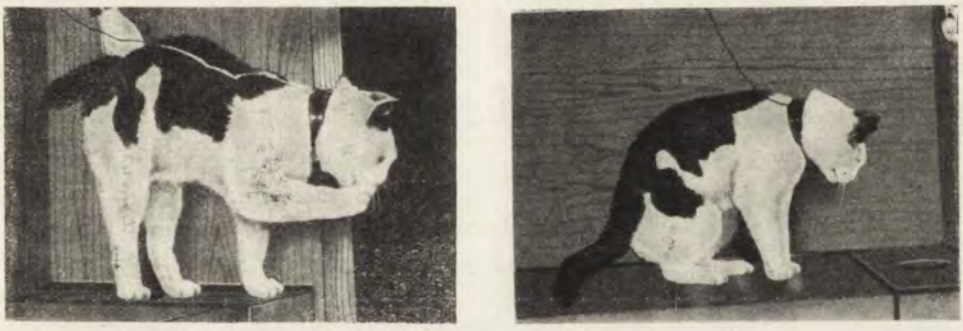


b

Fig. 1. The placement of the electrodes in the VPL nucleus (a) and in the VL nucleus (b)

Cat no. 7.	5.0 v.	Isolated flexion of the contralateral hindleg obtained when the cat is standing. After stimulation he inspects the leg.
Cat no. 8.	3.5 v.	Small movements of the contralateral foreleg, inspects the leg and sniffs it. Sniffs the floor round the leg.
	4.8 v.	Isolated and rapid movements of the leg. After stimulation inspects it for a long time.
Cat no. 9.	1.3 v.	Lies down and licks various parts of the body.
	3.5 v.	Isolated and rapid movement of the contralateral foreleg. Licks the leg or shakes it as if it were wet (Fig. 2a). If during stimulation the food is presented the animal puts his leg on the floor and runs to the food well.
Cat no. 10.	3.5 v.	Isolated lifting of the contralateral foreleg, sometimes turning the head towards it. After stimulation inspects the floor and sniffs it.
Cat no. 11.	3.5 v.	The cat lies down and rubs his body against the floor, licks his contralateral foreleg or shoulder.
	7.2 v.	High, rapid and repeated lifting of the foreleg, after stimulation long and intense licking of the paw.
Cat no. 12.	7.0 v.	High lifting of the leg with shaking movement (wet leg symptom). After stimulation holds the leg high as if he were afraid to put it down. Sniffs the leg and the floor. After several stimulations tries to escape.



a b

Fig. 2. The effect of VPL stimulation in cat no. 9, before (a) and after (b) removal of the sensorimotor cortex. Strength of stimulation 3.5 v

We have presented above the most salient fragments of the protocols which throw light on the character of the movements elicited by electric stimulation of the VPL nucleus with 300 c/sec.

The most frequently observed response consisted in lifting the contralateral foreleg with occasional turning of the head towards it. Only in one animal was the movement of the hindleg elicited. The movements were rapid with veriable amplitudes and latencies amounting to a few seconds. The size and character of the movement often depended on the posture

taken by the animal before stimulation. The responses were regular, if the intervals between stimulations were long (several minutes).

When stimulation was subthreshold with respect to the elicited movement, indefinite, sometimes bizarre, types of behavior were seen: the animal lay down on his back, rubbed his body against the floor or the wall, sniffed around and so on. The effective stimulation was also usually accompanied by additional responses like inspecting the leg involved in the movement, licking or sniffing it, shaking movements, sniffing the place on which the cat stayed. These responses appeared both during stimulation and particularly after its termination.

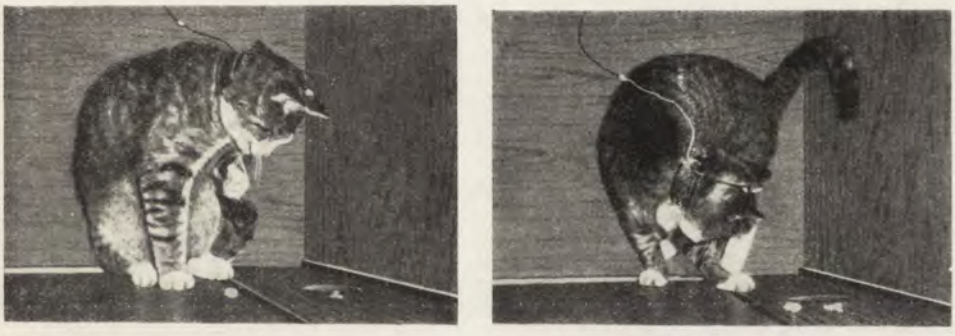
In most cases stimulation of the VPL nucleus did not elicit any aversive symptoms. The animals were not afraid of the exsperimental cage and willingly ate food presented after and even during stimulation. However, if the voltage of stimulation was high (about 7 v) the animals clearly manifested a defensive attitude which consisted in trying to get out of the cage and/or refusing to take food. No epileptic seizures were seen. Finally, it should be mentioned that the motor response elicited by VPL stimulation could be easily inhibited by other activities of the cat. In connection with the instrumental training of the elicited movements, we had many occasions to notice that when the animal heard the click of the bowl placed in the aperture of the food well, he ran towards it on four legs and could use the leg involved for taking food out of the bowl.

Stimulation of the VL nucleus. The optimal current of stimulation was 0.4—0.8 ma. The locations of electrodes are presented in Fig. 1b.

Here are the fragments of the protocols recorded for particular cats:

Cat no. 3.	6.0 v.	Isolated movement of the contralateral foreleg. Long and irregular latency. If stimulation is protracted the movement of the hindleg also appears.
Cat no. 5.	5.0 v.	Isolated and very regular lifting of the contralateral leg with supination of the paw. Supination means that the foreleg turns along its long axis so that the palm is directed inwards. Long latency, the amplitude of the movement gradually rises. The movement is not inhibited by the act of eating.
Cat no. 6.?		Lifting of contralateral forelimb and hindlimb. Isolated movements cannot be obtained.
Cat no. 7.	8.0 v.	Lifting of the contralateral foreleg and stiffening of the neck.
	10.0 v.	Lifting of the foreleg with strong supination. Turning of the head producing a loss of balance, so that the cat falls down on his side and cannot rise till the end of stimulation. No signs of aversion towards stimulation.

Cat no. 8.	6.0 v.	Gradually increasing isolated movement of the contralateral foreleg. Protracted stimulation leads to a strong supination (Fig. 3a). Latency 7—10 sec.
Cat no. 9.	6.5 v.	Isolated movement of the contralateral foreleg with long latency and gradually increasing amplitude; slight supination of the paw. When food is presented the cat tries to approach the food well with the foreleg lifted.
Cat no. 10.	4.0 v.	Clear muscle contractions of the distal part of the contralateral foreleg with clawing. Latency of about 10 sec.
	7.0 v.	Isolated lifting of the foreleg with supination. Latency about 10 sec and the performance of the movement extremely slow. If the animal received food during stimulation he ate it with the foreleg lifted. If stimulation was given at the moment when the animal took food with that foreleg he stopped doing so, or lifted the leg holding a piece of meat, but not putting it into the mouth.
Cat no. 11.	8.0 v.	Isolated lifting of the contralateral foreleg, occasional co-movements of the shoulder and ear. Protraction of stimulation elictis in addition small movements of the hindleg.
Cat no. 12.	8.0 v.	Isolated lifting of the contralateral foreleg with slow forward extension and supination. Latency till 15 sec.



a b

Fig. 3. The effect of VL stimulation in cat no. 8 before (a) and after (b) removal of the sensorimotor and premotor cortex. Strength of stimulation in a 6 v, in b 10 v

As seen from these protocols the character of the elicited movements is quite different from those producted by VPL stimulation. The latency is longer, the increase of the amplitude much slower, the effective strength of current nearly twice as large as in VPL stimulation. The movement has a stereotyped character, the lifting of the leg being accompanied by supination and sometimes extending the leg forward. If the intervals between stimulations are long (3—5 min), the responses are very regular. If stimulation is protracted, or its strength increased, the

movements become generalized: there appears the lifting of the hindleg, movements of the mouth, ear and shoulder, as well as turning the head. The isolated movement of the hindleg was never obtained in these experiments. There were no accompanying symptoms of "paying attention" to the limb, so characteristic for VPL stimulation. Rather there was impression that the animal did not "notice" the movement performed.

The movement was not inhibited by the antagonistic activities (e.g. locomotion or food intake) as was the case with VPL stimulation. Stimulation never had an aversive character and no epileptic seizures were observed. The subthreshold stimulation with regard to the movement of the foreleg did not produce any observable effects.

The effects of cortical lesions on the VPL-produced movements. In five animals (no. 2, 4, 6, 7, 9) the sensorimotor area of the cortex was removed on the same side on which the VPL electrode was implanted. The reconstructions of lesions are presented in Fig. 4. After the ablation, stimulation of the VPL nucleus was retested.

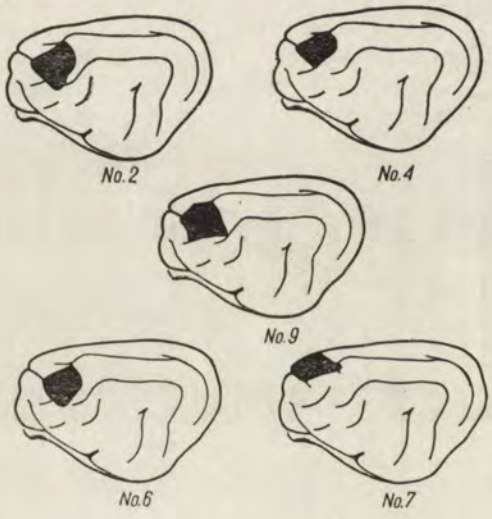


Fig. 4. Cortical lesions ipsilateral to the electrodes implanted in the VPL nucleus. Note that in cats no. 2, 4, 6 and 9 the sensorimotor cortex for the forelimb was removed, whereas in cat no. 7 the sensorimotor area for the hindlimb was removed

The results of these experiments were quite uniform. In all animals the movements observed before surgery were completely abolished (Fig. 2b). When the voltage was increased till 10 or 12 v clear and unmistakable defensive responses were observed accompanied by strong fear. Beneath we give the protocols of some sessions.

Cat no. 4. Removal of the left sensorimotor cortex limited to the foreleg area.

Before operation a regular isolated movement of the right foreleg was elicited by VPL stimulation with 5.0 v, 300 c/sec. The same sti-

mulation after operation produces no response. When stimulation is protracted till 30 sec the animal becomes disquiet, walks around the cage, sniffs and looks round.

8.5 v. After 2—4 sec the cat looks at his foreleg, licks it, then flattens his ears, moves backward and becomes disquiet. After several stimulations he tries to flee from the cage, and becomes aggressive. When touched or stroked, he mews, tries to escape and after more trials attempts to bite the hand of the experimenter. Refuses to take food.

10.5 v. Runs round the cage, sometimes stops abruptly, sniffs, mews, flattens the ears, tries to grasp by teeth the skin on the shoulder. After stimulation sits down in the corner of the cage, licks his claws, paw and the whole right side of the body. Avoids being touched. After prolonged stimulation jumps out of the cage.

12.0 v. Moves hindleg clumsily, then freezes, crouches, pants, looks fearfully around. The movements of the experimenter produce flight reaction.

Exactly the same picture was observed in other cats.

The effects of cortical lesions upon the VL-produced movements. In cats no. 5, 8, 10, 11 and 12 cortical lesions were sustained on the side of the VL electrodes (Fig. 5) and the results of those lesions are as follows.

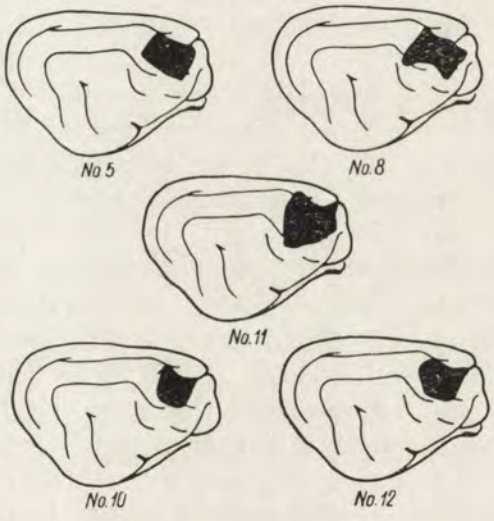


Fig. 5. Cortical lesions ipsilateral to the electrodes implanted in the VL nucleus. Note that in cats no. 10 and 12 the sensorimotor area was removed, whereas in cats no. 5, 8 and 11 the lesion involves also the premotor cortex

Cat no. 10. Ablation of the sensorimotor cortex for the foreleg. Stimulation of the VL nucleus produces the same motor response as before operation. The latency is somewhat prolonged.

Cat no. 12. Ablation of the sensorimotor cortex for the foreleg. The character of the movement produced by VL stimulation is similar to

that before operation, but its amplitude is reduced, and the supination clearly manifested before operation is absent. The response occurs less regularly and sometimes is absent. Increase of stimulation from 8.0 v to 9.2 v makes the occurrence of the movements more regular, but then the turn of the head and the tension of the muscles of the neck is observed. The amplitude of the movement remains low. If during stimulation food is presented the cat takes it willingly.

Cat no. 5. Ablation of the sensorimotor and premotor cortex. The lesion is shallow (in the cruciate sulcus) but extensive.

5.0 v (as before operation). No visible response.

7.0 v. No visible response.

10.0 v. The motor response is similar to that before operation, but its amplitude is lower and supination is absent. Its occurrence is irregular. If stimulation is prolonged till 30 sec, the tension of the neck and clenching of the jaws is seen which continues for 1—3 min after stimulation. After prolonged stimulation the cat is apathetic, is not interested in food, but remains calm. Rhythmic jerking of the head is also observed.

Cat no 8. Ablation of the sensorimotor and premotor cortex.

6.0 v (as before operation). No visible response.

8.0 v. No isolated movement of the leg, strong cramps of the neck, clenching the jaws, difficulties in respiration. Rhythmic jerking of the head. Afterwards the cat is apathetic and does not take food.

10.0 v. Tonic rigidity of the neck, clenching of the jaws, unnatural and tense turning of the head (Fig. 3b), followed by slow lifting of the hindleg and its stretching forward with spreading out the claws. Frequently this movement is stopped by increased turning of the head and body which leads to a loss of balance and the cat's falling down with tense muscles. After the stimulation is withdrawn it lies motionless and after 2—3 min strong and repeated jerking of the head is observed ending with jerking of the whole body. The seizures are followed by general relaxation of the body and immobility.

Cat no. 11. Ablation of the sensorimotor and premotor cortex.

Complete abolition of the isolated movements. Stimulation with 15 v produces general seizures similar to those described in cat no. 8.

Summarizing the effects of the VL stimulation after cortical lesions we can conclude that while ablation of the motor cortex produces only a slight impairment of the motor response, the additional removal of the premotor cortex leads to a complete abolition of that movement. The increased strength of stimulation elicits muscular contractions of a primitive character, terminating with more or less pronounced epileptic

seizures. Even a very high voltage of stimulation does not produce either pain or fear. Movements of the hindleg produced by strong stimulation may be due to the fact that hindleg area of the cortex was spared.

DISCUSSION

As seen from our results electrical stimulation of both the VL and VPL nucleus gives rise to isolated movements of the contralateral leg, provided that the parameters of stimulation are properly chosen. Why is it that only high frequencies (200—300 c/sec) of thalamic stimulation produce isolated movements of limbs is not clear, particularly if we take into account that the optimal frequencies of cortical stimulations are much lower. Thus, in the study of Tarnecki (1962a), in which the movements of the foreleg or the hindleg were elicited in waking cats by stimulation of the sensorimotor cortex, the frequency of 50 c/sec gave fluent and isolated responses, similar to those obtained in the present paper.

There are other striking differences between the effects of stimulation of the sensory cortex and the motor cortex on the one hand and the effects of stimulation of the VL and VPL nuclei on the other.

With regard to stimulation of the sensory cortex, to our best knowledge it is always emotionally indifferent, that is, it never has any nociceptive aspects. On the other hand, it was found by us that if the stimulation of the VPL nucleus is strong, the animal clearly shows the aversive response manifested both by typical local defensive flexion reflex and by emotional fear response. Moreover, the somesthetic sensations produced by VPL stimulation seem to be more durable than those produced by cortical stimulation, as judged from the long-lasting after-effect in the form of licking the leg, sniffing it, shaking movements, etc. It may be supposed that stimulation of the VPL nucleus throws into action some reverberating circuits which outlive the actual stimulation. Probably these reverberating loops are not activated by stimulation of the sensory cortex.

No less interesting is the comparison between the effects of stimulation of the motor cortex and stimulation of the VL nucleus. As is well known, stimulation of the motor cortex gives an abrupt lifting of the leg with very short latency and low threshold. On the other hand flexion of the leg produced by VL stimulation exhibits an exceedingly long latency, high threshold and very slow recruiting. Whereas stimulation of the motor cortex may be given with intervals of one minute or less without affecting the character of the response, VL stimulation

should be given once in 3—5 min, otherwise the response will deteriorate.

The problem of a long latency and very slow augmenting of VL produced movements is very difficult to understand. There is ample electrophysiological evidence to show that stimulation of the VL nucleus evokes a response in the pyramids after a latency of a few msec (Branch and Martin 1958, Amasian and Weiner 1966). Therefore one may ask why this stimulation does not exactly reproduce the cortical stimulation.

One possibility may be that repeated stimulation of the VL nucleus produces a potent recurrent inhibition of thalamic neurons, which blocks them for a length of time and then for some reasons becomes gradually removed (cf. Eccles 1966). If so, the question arises as to why the neurons of the VPL nucleus behave in a different way. It seems that electrophysiological experiments should answer that question.

To end the comparative analysis of the effects of cortical versus thalamic stimulations, it should be noted that whereas by stimulating the cortex (both motor and sensory) we can easily find points from which the movements of the hindleg are obtained, we were strikingly unsuccessful in this respect when stimulating the thalamus. The isolated hindleg movement was obtained only once by stimulating the VPL nucleus. The movements of the hindleg elicited by stimulation of the VL nucleus were never isolated, and we could not separate them from those of the fore leg.

Let us return now to the comparison of VL-induced movements with VPL-induced movements. Below we summarize once again their main differences:

The threshold of the VPL responses is lower than that of the VL responses, the latency shorter and the recruitment faster. The VPL responses have a clear somesthetic aspect, which is completely absent in VL responses. The aversive responses are more likely to occur with VPL than with VL stimulation. On the other hand, VPL responses are easily inhibited by other activities of the animal, such as locomotion or food intake, while VL responses are not.

Now we should discuss the effects of cortical lesions upon thalamogenic movements.

As was indicated above, stimulation of the VPL nucleus after the sensorimotor lesion fails to produce the isolated movement; however, an increase of the strength of stimulation leads to a general excitement of the animal of a clearly nociceptive character. This would suggest that the VPL nucleus contains not only relay neurons transmitting messages to the sensory cortex, but also those neurons which send messages to other cerebral structures. Whereas the former neurons are

responsible for isolated motor acts induced by thalamic stimulation, the latter are responsible for the general excitement produced by nociceptive stimulation. This conclusion is in good agreement with Tarnecki's findings (Tarnecki 1962b, 1963), showing that lesions in the VPL nucleus or medial leminiscus completely abolish the effects of nociceptive stimulation. In fact either strong squeezing of the distal parts of the body or strong pinching fail to produce any response.

It seems reasonable to assume that the structures responsible for general pain and fear reactions produced by VPL stimulation should be situated in the limbic system and/or hypothalamus. Thus the problem arises as to which are the pathways connecting the VPL nucleus with these structures.

One possibility is that separate neurons from those firing to SI area fire to SII area (Guillery et al. 1966) and they are preserved after removal of the former area. Since SII area has connections with amygdala (Albe-Fessard, personal communication), the thalamic messages may reach this structure by this way. On the other hand, the impulses firing from the thalamus may reach the limbic structures through other pathways, possibly through intralaminar thalamic nuclei, connected with the VPL nucleus. This problem must remain open for further investigation.

As far as the VL nucleus is concerned the situation is different. Removal of the sensorimotor cortex is not sufficient for the abolition of the isolated motor responses originating from its stimulation. These responses disappear only after additional ablation of the premotor cortex. The increase of the strength of stimulation does not produce any general aversive responses, as is the case with VPL stimulation, but instead gives rise to muscular tonic contractions terminating in Jacksonian seizures.

The failure to obtain isolated movements of limbs after sensorimotor-premotor ablations is in good agreement with the fact that the thalamo-cortical fibers originating in the VL nucleus have their end-station in both the motor and premotor area (Walker 1936). The question arises as to what is the pathway responsible for the primitive motor effects and epileptic seizures produced by VL stimulation in the absence of these cortical areas. The question is difficult to answer. It may be supposed that some fibers of the VL nucleus terminate in the basal ganglia through the thalamic fasciculus (cf. Crosby et al.) and that these structures intervene in the occurrence of these phenomena.

Irrespective of which are the pathways intervening in the general responses produced by VPL and VL stimulation in the absence of the cerebral cortex, one point seems to be clear. This is that the isolated

movements of the contralateral limbs elicited by stimulation of these structures are mediated by the cortex, since these movements are abolished by lesions sustained in the appropriate areas. This result seems to be puzzling, if confronted with the fact, described in the next paper (Tarnecki and Konorski 1969), that these lesions fail to abolish the instrumental responses originating from the thalamogenic movements.

SUMMARY

1. Stimulation of the VPL or VL thalamic nuclei by implanted electrodes in waking cats with a rate of 300 c/sec produced isolated movements of the contralateral limbs. Usually only the movements of the foreleg were obtained, whereas the isolated movement of the hindleg was seen only in one cat.

2. VPL-produced movements were accompanied by various orienting responses directed towards the limb, like licking, sniffing or gazing at it. With strong stimulation clear aversive responses were observed. The movements could be inhibited by locomotion or food intake.

3. VL-produced movements had long latency and gradual recruitment. Increased strength of stimulation produced involvement of muscles of the neck and mouth and/or movements of the hindleg.

4. Ablation of the sensorimotor cortex ipsilateral to the VPL electrodes led to total abolition of the VPL-produced isolated movements. Increased strength of stimulation elicited a general restlessness of the animals of an aversive character.

5. Ablation of the sensorimotor cortex ipsilateral to the VL electrodes led to a slight impairment of the VL-produced movements. If the premotor cortex was additionally removed, the isolated movements disappeared. Increased strength of stimulation elicited tonic contractions in the neck and jaws and turning of the head. These effects outlasted the period of stimulation and often passed into Jacksonian seizures.

6. The mechanisms involved in the above described effects are discussed.

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INSTRUMENTAL CONDITIONING OF THALAMOGENIC MOVEMENTS AND ITS DEPENDENCE ON THE CEREBRAL CORTEX

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In the previous paper (Tarnecki and Konorski 1969) it was found that high-frequency stimulation of the nucleus ventralis posterolateralis (VPL) and nucleus ventralis lateralis (VL) thalami produces in waking cats isolated movements of the contralateral limbs. In most instances lifting of the foreleg was observed.

In our earlier studies (Tarnecki 1962a, Tarnecki and Konorski 1963) we were concerned with the problem of instrumental alimentary conditioning of cortically induced movements. It has been established that movements of the hindleg are transformed into instrumental responses only in those cases in which they are elicited by stimulation of the sensory cortex, but not by stimulation of the motor cortex. It was concluded from this experiment that instrumental responses can be formed only from those movements which are initiated by stimuli impinging upon the afferent side of the nervous system.

The aim of the present experiments was twofold. First, it had to be established whether the movements produced by thalamic stimulation could be instrumentally conditioned. If so, the next problem arose concerning the role in instrumental conditioning of the cortical areas indispensable for the occurrence of the thalamogenic movements.

MATERIAL AND EXPERIMENTAL PROCEDURE

Experiments were performed on 10 cats used in a preceding study (Tarnecki and Konorski 1969). The experimental box is represented in Fig. 1. On the right side of the box there was a feeder in which successive bowls were placed by an automatic device.

After the electrodes were implanted into the VPL and VL nuclei (see Tarnecki and Konorski 1969), and the optimal strength of electrical stimulation was selected for elicitation of an isolated movement of the limb, the daily experimental sessions ran as follows.

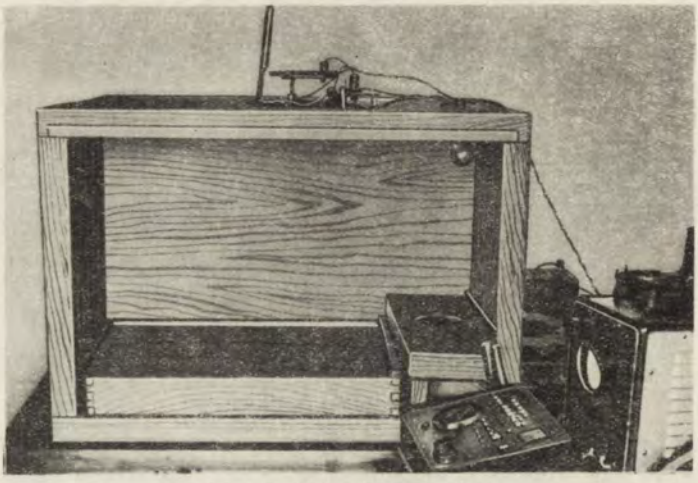


Fig. 1. Experimental box

The animal was placed in the box and electric stimulation was applied twenty times per session. When the cat performed the required movement in response to the stimulation, a bowl containing a piece of raw meat was put into position. The cat hearing the click of the moving bowl immediately approached the food and ate it. The intertrial intervals lasted 3—5 min.

When the cat started to perform active movements similar to those elicited by stimulation, they were immediately reinforced by food. Gradually their number increased so that stimulation was applied less frequently and eventually was completely withheld. Each session continued to consist of 20 reinforced trials irrespective whether the movement was elicited by stimulation, or was active.

When the training reached the stage in which the animal immediately after entering the box started to perform the instrumental movements and did so with regular frequency, a cortical operation was made in which the sensorimotor area, or sensorimotor and premotor area contralateral to the trained movement was removed. Two weeks after surgery the CR experiments were resumed and the instrumental responses were tested. After several weeks of retesting the cats were sacrificed and the anatomical examination of the brains was carried out (cf. Tarnecki and Konorski 1969).

RESULTS

Instrumental conditioning of VPL-evoked movements. The CR experiments were performed in five cats (no. 1, 2, 3, 7, 9). We present below the descriptions of training in each cat.

Cat no. 1. At first the cat was habituated to the experimental box

and feeding procedure, and the optimal current of stimulation of the left VPL nucleus was chosen. It was found that stimulation with 300 c/sec, pulse rate 1 msec, and strength 3.2 v (0.2 ma) produced an isolated regular flexion of the right foreleg. After stimulation the animal often licked his forepaw.

Thereafter, the regular instrumental training began consisting in reinforcing with food every movement elicited by VPL stimulation. Already at the end of the first session the animal turned to the feeder immediately after stimulation was turned on even before performing the movement. At the end of the second session four active movements of the foreleg appeared. They were quite similar to those produced by stimulation.

In the following sessions the animal performed active movements only when a few stimulations of the VPL nucleus were given beforehand. We present below the protocol of the fourth session.

After coming to the box cat is sitting at the feeder for 5 min, looks at his right leg, puts his leg into the empty bowl. The further course of the session runs as follows:

No. of trial	Time	Stimulation	Movement	Food	Comments
1	5 min	300 c/sec 3.2 v	VPL-evoked	+	From trial 7 till the end
2	8 min	300 c/sec 3.2 v	VPL-evoked	+	of the session regular ac-
3	8 min 20 sec	— —	active	+	tive movements of high
4	9 min	— —	active	+	amplitude are performed.
5	9 min 30 sec	— —	active	+	Sometimes before per-
6	13 min	300 c/sec 3.2 v	VPL-evoked	+	forming the movement
7	13 min 15 sec	— —	active	+	the cat licks his foreleg
8	14 min	— —	active	+	
	.	.	.	.	
	.	.	.	.	
	.	.	.	.	
20	21 min 30 sec	— —	active	+	

A similar picture is seen in the fifth and sixth session in which the performance of the active movements also had to be facilitated occasionally by VPL stimulation, particularly at the beginning of the session. It was observed, however, that the effective strength of current could be reduced to 2.0 v (0.12 ma). Beginning from the seventh session VPL-evoked movements were no longer needed, since the cat started to perform active movements soon after entering the box. Since the movements became regular and were performed immediately after the cat ate the preceding portion of food, the duration of the session (20 trials) was reduced to about 4 min, that is the instrumental responses were separated by about 12 sec.

It is worthwhile to note that when the instrumental reflex was firmly established the character of the motor response underwent some change. Whereas the response to stimulation consisted of a flexion of the leg in wrist, elbow and shoulder, the active movement in the later period of training consisted only in the flexion of the elbow. Interestingly, VPL stimulation produced in that period also the same simplified movement.

Cat no. 2. VPL stimulation, 300 c/sec, 4.5 v, evoked a quick and high lifting of the foreleg in the standing position with forward extension. After 12 trials stimulation was followed by approaching the feeder and expecting food. In the third session, after 7 stimulation trials (that is 67 trials in total), the active movements appeared which were identical to those produced by stimulation (Fig. 2a). In the fourth session the active movements appeared after two stimulation trials. From the fifth session stimulation was no longer needed, and the whole set of 20 movements was performed during 4—7 min.

From the twelfth session on, the character of the active movements was changed, because the cat began to perform them from the sitting position without extending the leg forward (Fig. 2b).

Cat no. 3. Stimulation of the VPL nucleus, 300 c/sec 3.0 v, produced a typical flexion of the foreleg performed in the sitting position. In the second session, when the current was turned on the animal became

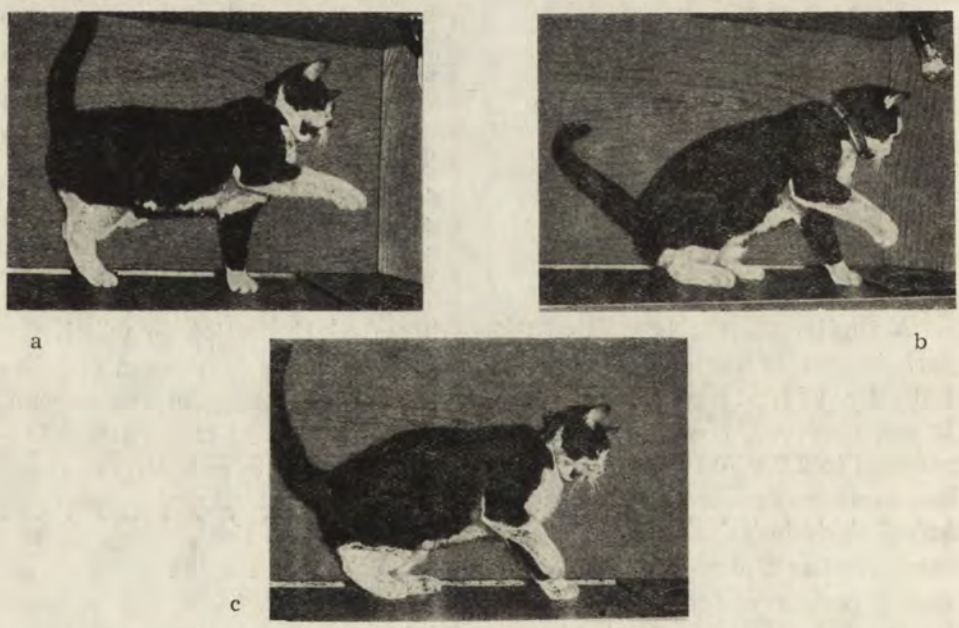


Fig. 2. Instrumental conditioned responses in cat no. 2 in the early period (a), in a later period (b) and after removal of the sensorimotor cortex (c)

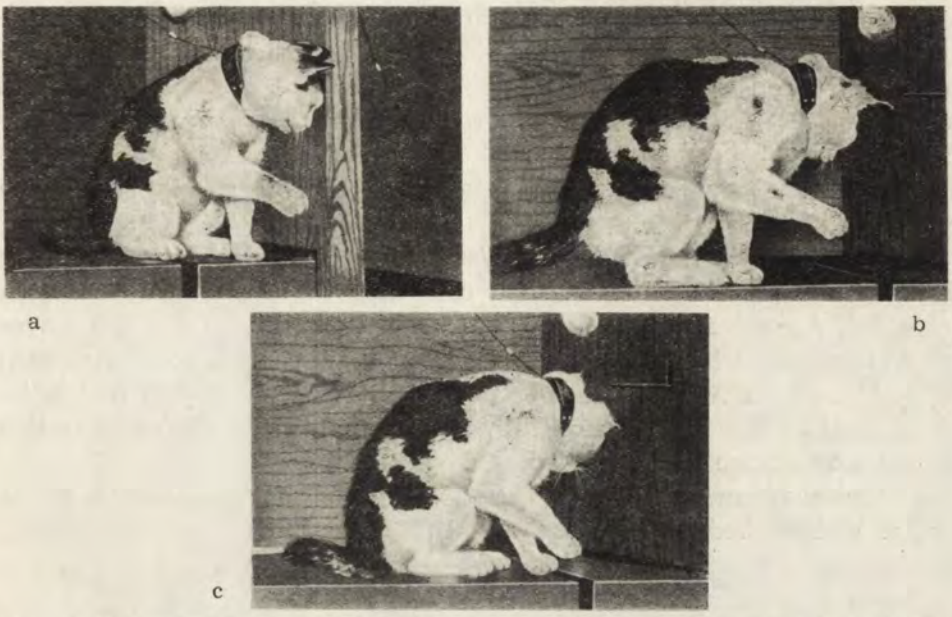


Fig. 3. VPL-induced movement (a), instrumental conditioned response (b) and instrumental response after removal of the sensorimotor cortex (c) in cat no. 9

disquiet, put his head into the empty bowl, sniffed the feeder or licked it. After six stimulation trials the cat, after having eaten his portion of food, began to lick his foreleg, lay down on the floor of the box, rolled for some time, then after a while returned to the sitting position. This bizarre behavior was repeated again and again during the second and third session. In the fourth session, after the forth trial (that is, after 64 stimulation trials in total) the active movements appeared which were quite similar to those produced by stimulation. As in cats no. 1 and 2 in subsequent two sessions the active movements followed a few stimulation trials. The effective strength of the current was reduced to 1.2 v.

From the seventh session active movements appeared as soon as the cat entered the box and stimulation was no longer needed. The animal performed his quota of 20 movements with reinforcement in about 4 min.

Cat no. 7. VPL stimulation, 300 c/sec, 5.0 v, evoked the movement of the hindleg performed in standing position.

After 14 trials the cat began to sniff the feeder and licked intensively his right hindleg or the right side of his body. When stimulation was turned on he became quiet, looked at the feeder, then got up and performed the movement. Sometimes when the movement was very small or absent, food was not presented and stimulation was turned off. Then the cat became disquiet and intensively licked his hindleg and body.

The first active movements appeared after 16 stimulation trials in the seventh session (136 trials in total). During six succeeding sessions active movements appeared always after a few stimulation trials, the threshold of stimulation being dramatically reduced: the current of only 1.0 v produced regular movements with short latency.

After 14 sessions the active movements became regular and were performed with maximal speed.

Cat no. 9. Stimulation by 300 c/sec, 3.5 v current produced a quick isolated movement of the fore leg consisting in its forward extension (Fig. 3a). After 7 stimulation trials in the third session the active movement appeared, which was an exact copy of the VPL produced movement (Fig. 3b). In the two following sessions the active movements had to be facilitated by movements elicited by stimulation. In the sixth session this facilitation was no longer needed.

Below we sum up the main characteristics of the instrumental training of VPL-evoked movements (Table I).

Table I

Main characteristies of the instrumental training of thalamogenic movements

No. of cat	Stimulation	Movement of the	Voltage applied	First session in which instrumental movement appears	Number of stimulation trials till the first instrumental movement appears	First session in which instrumental movement became stable
1	VPL	foreleg	3.2	second	36	seventh
2	VPL	foreleg	4.5	third	47	fifth
3	VPL	foreleg	3.0	fourth	64	seventh
7	VPL	hindleg	5.0	seventh	136	fourteenth
9	VPL	foreleg	3.5	third	47	sixth
5	VL	foreleg	5.0	fifth	93	sixth
8	VL	foreleg	6.0	fifth	93	sixth
10	VL	foreleg	7.0	seventh	130	eighth

1. In the first stage of conditioning the stimulation of the VPL nucleus — or, to put it more precisely, the somatic sensation produced by this stimulation — becomes a classical food CS manifested by the animal approaching the feeder, sniffing and licking the empty bowl,etc.

2. The instrumental movements of the foreleg appear after about 50 stimulation trials and those of the hindleg after 136 trials, but they do not occur regularly and must be preceded by thalamic stimulation. Only

in the further training is their rate increased and they appear immediately after the animal enters the box.

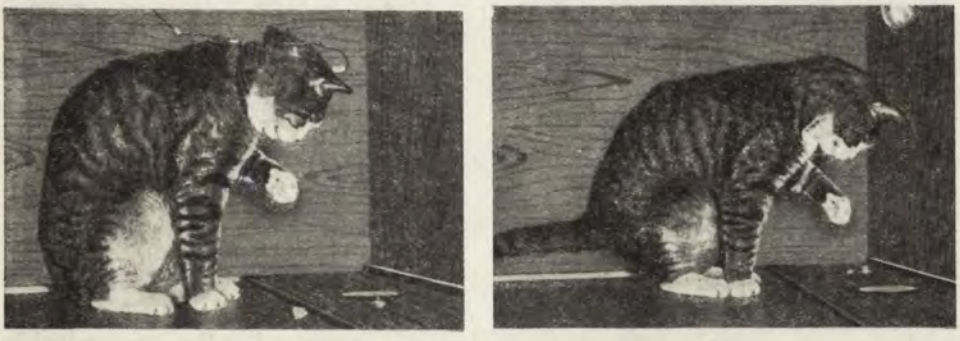
3. In the transitional period the threshold of thalamic stimulation evoking the movement is dramatically decreased.

4. With prolonged training the instrumental movement which initially completely mimics the VPL-evoked movement may become somewhat simplified.

5. In the early stage of instrumental conditioning the animal exhibits clear symptoms of sensory paresthesias of the contralateral leg and side of the body.

Instrumental conditioning of the VL-evoked movements. The experiments of this type were performed on three cats (no. 5, 8 and 10). Since the course of instrumental training was nearly identical in each cat, we present below its joint description (Table I).

Stimulation of the VL nucleus (300 c/sec, 5.0—7.0 v) elicited in all three cats a slow and gradual raising of the contralateral foreleg in extended position combined with supination (Fig. 4a). The latency was about 10 sec. After a number of stimulation trials (93, 93, 130 respecti-



a b

Fig. 4. VL-induced movement (a) and instrumental conditioned response (b) in cat no. 8

vely) the animals began to perform the active movements. In contradistinction to the VPL group, once the first active movements appeared, they were performed regularly and no facilitation by applying thalamic stimulation was needed. The shape of the movements was exactly the same as that in stimulation trials (Fig. 4b), except that their performance was swift and fluent and they did not possess a tonic and coerced character observed in VL-evoked movements. The movements did not undergo any changes till the end of training (about 30 sessions). The intervals between instrumental responses, which in the previous group were as short as about 15 sec, remained very long till the end of training

and amounted to one or two minutes. It should be reminded that VL-evoked stimuli also required very long intervals, otherwise they became very poor or even failed to occur (Tarnecki and Konorski 1969).

The effects of cortical lesions on instrumental responses of the thalamic origin. After the CR training was completed, six animals (three of the VPL group and three of the VL group) were subjected to cortical operations. In three cats (no. 2, 7, 9) of the VPL group and one cat (no. 10) of the VL group, only the sensorimotor cortex was removed; in two other cats of the VL group (no. 5, 8) the sensorimotor cortex and the premotor cortex was ablated (see Tarnecki and Konorski 1969, Fig. 4 and 5).

Below we present short descriptions of the animals' CR performance after these operations.

Cat no. 2, VPL group. Movement of the foreleg. Sensorimotor lesion. The general motor efficiency was only slightly impaired and soon returned to normal. On the other hand, the instrumental responding was very strongly impaired. The amplitude of the movements was very small (Fig. 2c), and the intervals between responses were much prolonged. The fatiguability of the instrumental responses was so great that it was not possible to enforce a full quota of 20 movements per session from the animal.

After several weeks the instrumental responses became more regular and the cat was able to execute all the 20 movements in 20—24 min (before operation 4 min). The general character of the movements did not improve.

Cat no. 9, VPL group. Movement of the foreleg. Sensorimotor lesion. In spite of a very slight impairment of the general motor efficiency, the instrumental responding was very poor. The movements were small (Fig. 3c), irregular and prone to fatigue. After about 10—12 responses, the movements became so small that they were hardly recognizable as instrumental responses.

Cat no. 7, VPL group. Movement of the hindleg. Sensorimotor lesion. The CR performance was strongly deteriorated. The trained movements became so small that they were difficult to be identified. Strong fatiguability and very long intervals between responses.

Cat no. 10, VL group. Movement of the foreleg. Sensorimotor lesion. A few weeks after operation the animal's motor efficiency hardly differed from that before operation. His CR performance was, however, impaired. Although the animal was able to perform from time to time a movement of normal amplitude and shape, this occurred rarely, because of the conspicuous fatiguability.

Cat no. 5. VL group. Movement of the foreleg. Sensorimotor and premotor lesion. The general motor skill after operation was much worse

than in the preceding cats. The animal crouched instead of walking and his motor coordination was very poor. He was not able to perform any manipulative movements. For four weeks after operation the instrumental CR was absent.

Thereafter the motor efficiency of the cat gradually improved, although it was still very poor. In that time the instrumental movements were restored but they were very low and irregular. This condition failed to change for several months.

Cat no. 8. VL group. Movement of the foreleg. Sensorimotor and premotor lesion. The general motor impairment, although prominent, was less severe than in cat no. 5. The instrumental CR was strongly impaired and was limited to a slight jerking up of the leg from the floor. Since the affected foreleg was generally hyperactive, it was sometimes difficult to identify a movement as a true instrumental response.

In sum the CR performance after the cortical lesions may be characterized as follows. Although in all our cats the instrumental CRs were preserved, their amplitude was very small and they were readily fatiguable. The general motor efficiency was much more strongly affected after sensorimotor and premotor lesions than after pure sensorimotor lesions. Similarly, the instrumental CRs were still poorer after the larger lesions than after the smaller ones.

DISCUSSION

Two findings reported in this paper should be discussed. One is the instrumentalization of the thalamogenic movements, the other, the survival of the instrumental CR after destruction of the cortical area intervening in the occurrence of those movements.

The ready instrumentalization of the VPL-evoked movements is easy to understand. In the preceding paper (Tarnecki and Konorski 1969) it was pointed out that stimulation of the VPL nucleus mimics the sensory peripheral stimulation of the noxious or subnoxious character of the limb concerned, stimulation producing the flexion reflex. According to the general rules of instrumental conditioning discussed elsewhere (Konorski 1967) this flexion reflex under proper reinforcement is easily transformed into an instrumental CR.

Further features of the instrumental CR derived from VPL-evoked movements fully confirm this thesis. It was found that in early stages of instrumental training active movements are facilitated by VPL-evoked movements. Conversely, the latter movements are now elicited by a much lower strength of current than before training. These facts are easily explained by the assumption that some center intervening in the

performance of the VPL-evoked movement plays an active role in conditioning. For, after the CR is established, this center is activated both by the "unconditioned" stimulus applied to the VPL nucleus, and by the conditioned stimulus represented by the experimental situation. In consequence, there is a mutual facilitation between the responses deriving from those two sources. The fact that the instrumental conditioned response exactly imitates the VPL-evoked movement, at least in the first stage of conditioning, also speaks in favor of that assumption.

Another curious phenomenon observed in the early stages of the instrumental training should be commented upon. As follows from our protocols, in the first experimental sessions the cats show signs of paresthesia of the side of the body contralateral to the VPL electrodes. This paresthesia observed in the intertrial intervals is manifested by the cat licking his paw, rubbing the body against the wall, etc. These phenomena strongly support a hypothesis claiming the formation of connections between the center of the given drive (here hunger drive) and the center of the stimulus provoking the trained movement (Jankowska and Sołtysik 1960, cf. also Konorski 1967, Chapter XI). It should be added that all the features characteristic of instrumental conditioning of the VPL-evoked movements are also observed in instrumental conditioning of "natural" responses such as scratch reflex.

Instrumental conditioning of VL-evoked movements follows a somewhat different course than that of VPL-evoked movements. It is true that the shape of the instrumental movement is an exact copy of the VL-evoked movement, but whereas the latter is performed very slowly, the instrumental movements are prompt and fluent, as if the recruitment were not needed for their occurrence. On the other hand, the trained movements do not follow each other with maximal frequency (as is the case with all other instrumental responses known to us), but are separated by intervals of more than one minute. No paresthesias of the body are seen in the intertrial intervals and the once established instrumental response does not need any facilitation. Certainly all these features are connected with the fact that the mechanism of the VL-provoked movement is different from that of the VPL-provoked movement, but we are far from a clear understanding of this mechanism.

Let us turn now to the second major finding described in this paper, namely that of the partial preservation of the instrumental response after the ablation of the sensorimotor cortex and even after the ablation of the sensorimotor and premotor cortex. This result is even more puzzling if we take into account that these lesions completely abolish the thalamogenic response from which the instrumental response takes its origin.

In previous works of this laboratory it was established that when a relatively simple motor response is instrumentalized, it is strikingly resistant to the damage sustained to the nervous system. We know that even extensive lesions sustained to the cerebral cortex (Jankowska and Górska 1960, Stępień et al. 1961), afferent pathways (Jankowska 1959, Górska and Jankowska 1961, Tarnecki 1962b), thalamic nuclei (Tarnecki 1962c, 1963) and efferent pathways (Górska 1967, Górska et al. 1966a, b) do not abolish the instrumental response although they impair it to a greater or lesser extent.

The same is true of instrumental movements derived from thalamic stimulation. Although after cortical lesions these movements become simplified or even abortive, there is no doubt that they are the remnants of instrumental responses. What the structures controlling these responses are is not clear without further experimentation.

SUMMARY

1. Movements of the limbs evoked by electrical stimulation of the VPL nucleus or VL nucleus with the frequency of 300 c/sec were used in instrumental alimentary conditioning in cats.

2. The VPL-evoked movements were instrumentalized after about 50 trials for the foreleg and after 136 trials (one cat) for the hindleg. In the first stage of conditioning there was mutual facilitation between the instrumental movements and the VPL-evoked movements, consisting in triggering of the instrumental movements by VPL-evoked movements on the one hand, and in lowering the threshold of the VPL-evoked movements by instrumental conditioning on the other. After the instrumental CR was firmly established the animals performed the trained movement immediately after eating food presented after the preceding movement.

3. The VL-evoked movements were instrumentalized after about 100 trials (for the forelimb). When instrumental conditioning was completed, the intervals between the performances of the trained movement remained long, amounting to one minute or more.

4. The instrumental responses transformed from the thalamogenic responses have exactly the same shape as the former ones.

5. Lesions sustained in the sensorimotor and premotor cortex do not abolish the instrumental responses derived from thalamic stimulation, but make them low, irregular and fatiguable.

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THE EFFECT OF "PARTIAL REINFORCEMENT" ON CLASSICAL AND INSTRUMENTAL CONDITIONED REFLEXES ACQUIRED UNDER CONTINUOUS REINFORCEMENT

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Our knowledge of the effect of partial reinforcement (PR) on the classical conditioned reflexes is disproportionately small as compared with the vast literature on the intermittent reward in instrumental learning. There are but few reports on this subject and most of them are inconclusive. Thus, all experiments on the effect of partial reinforcement in motor aversive conditioning, as for example, finger withdrawal (Platonov 1912), leg flexion (Brogden 1939), withdrawal response in earthworm (Wyers et al. 1964), or eye blink (Humphreys 1939a, Grant et al. 1950), to mention those in which PR did not produce a decrement in performance observed under continuous reinforcement, may be questioned simply because the omission of the noxious US constitutes a reward for the motor response [2]. In Pavlov's laboratories several of his co-workers (Virjikovskii, Fedorov, and Podkopaev) applied a sort of PR schedule to salivary conditioning with food reinforcement. They reinforced every second, or, in some instances, every third presentation of the CS and found that the first conditioned responses appeared as soon as with the continuous reinforcement. Later Usievich (1938) confirmed

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[2] Classical defensive motor CRs are easily transformed into instrumental avoidance CRs (cf. Petropavlovskii 1927, Bregadze 1953) although they are initially real classical CRs reinforced and maintained by the aversive US (Sołtysik and Jaworska 1962).

this finding using even a lower ratio of PR, namely, he reinforced every fourth application of the CS. In all these experiments the behavior of animals reflected the patterned presentation of reinforcements. They learned to react differentially according to the alternation of non-reinforced and reinforced trials. For example, in the later experiment (Usievich 1938) the dog [3] reacted with barking and withdrawal from the feeder during the first three applications of the CS, whereas to the fourth (reinforced) presentation of this stimulus he responded positively approaching the feeder and salivating copiously. Apparently, the employing of a fixed and non-random schedule of reinforcement, where the animals could learn the order of reinforced and non-reinforced trials, represents an entirely different procedure from the random PR training. Pavlov and his collaborators considered it rather as a "difficult discrimination" task whereas the problem of intermittent reinforcement has not yet been raised in their work.

Similar results were obtained by Longnecker et al. (1952) who compared the performance of conditioned GSR in human subjects trained under 50% PR; in half of the subjects the US was applied randomly, while the other half received the US in every second trial. Such a patterned PR resulted in a patterned responding. In effect, the average performance was slightly better in the randomly reinforced group.

More recent experiments devoted to the problem of PR in classical salivary conditioning yielded the following results. In salivary food conditioning (Wagner et al. 1964) the performance of the 100% reinforcement dogs was significantly superior to that of the 50% PR dogs. Fitzgerald (1964) obtained corresponding results by using the acetic acid injection into the mouth as an US in dogs. However, it is not clear from these reports whether the smaller conditioned salivation in PR dogs was an overall effect occurring in all trials, or whether it was caused by a diminished salivation in the trials that followed the non-reiforced presentation of CS. The phenomenon of reduced secretion of saliva after longer intertrial intervals was observed in dogs by Bruner and Kozak (1954) and Czarnecka and Sołtysik (1962), and since the PR experiments usually contain longer intervals between USs (i.e., between successive full activations of the glandular tissue) than the sessions with continuous reinforcement, the poorer performance in the PR groups may be related to this glandular phenomenon (cf. Babkin 1950, Chapter 28 and 29) and not necessarily to central factors.

The most similar design to ours was used by Brogden (1939) who first trained dogs in salivary food CR using continuous reinforcement

[3] The only subject of this study.

and afterwards tested the percentage of occurrence and intensity of these CRs under PR with fixed ratio of 80%, 60%, 40% and 20%. He noticed that the higher percentage of non-reinforced trials the lower was the frequency of salivary responses and the lower was also the intensity of these CRs. But the very fact that salivation did not occur on each trial makes the reader suspicious that the average drop in intensity was largely due to the above mentioned glandular factors.

Other studies on classical conditioning, e.g., fear conditioning (tested by the CER method) in rats (Willis and Lundin 1966), or the strength of secondary reinforcement (Armus et al. 1964), also showed the superiority of the continuous over partial reinforcement.

Thus, the general impression from all these experiments is that PR in classical conditioning produces inferior performance as compared with the continuous reinforcement. On the contrary, the PR, or more strictly (cf. English and English 1958), a partial *reward,* applied in instrumental learning often results in a higher performance level [4] than the 100% reward (Weinstock 1958, Goodrich 1959, Haggard 1959, Wagner 1961, Amsel et al. 1964, Amsel 1967). Of course, only the data from discrete trials experiments may be compared with the experiments on the classical conditioning, so we are not concerned here with the abundant body of evidence obtained in a Skinnerian free responding situation.

The aim of the present study was to analyse *simultaneously* the effect of a random intermittent omission of reinforcement upon the classical salivary CR and instrumental bar press response, both occurring in the same trial. The application of the Ellison-Konorski technique (Ellison and Konorski 1964, 1965), by which the salivary and instrumental responses are separately elicited in each trial, enabled us to observe the changes in either type of CRs in the course of the partial reinforcement training. Subjected to analysis were both the overall effect of the PR upon the average performance level and the immediate effect of non-reinforcement on the next CR.

MATERIAL AND METHOD

The subjects were six adult male dogs, previously used in another study (Miyata and Sołtysik 1968). It should be mentioned at once that the dogs had some experience with acute extinction but they had never been exposed to any form of chronic extinction or differentiation, and of cource, were absolutely naive in respect to the partial reinforcement procedures.

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[4] We are not concerned here with the persistence of CR, i.e., with the resistence to extinction, which was the main subject in most studies of the PR effect in operant behavior.

Five dogs had "shortened" salivary duct fistulae prepared surgically by the Sołtysik and Zbrożyna (1957) method and the sixth dog, Kyu-chian, had the classical parotid fistula prepared by the Glinski-Pavlov method (Podkopaev 1936). All the animals were trained in a combined instrumental-classical food CRs by the Ellison--Konorski (Ellison and Konorski 1964, 1965) procedure. Each trial consisted of presenting of two different CSs in succession and followed by the food-US. The first CS (a metronome) elicited a series of 14 bar presses. When this instrumental chained response was accomplished the CS_1 was terminated and replaced by the CS_2 (a buzzer) which was always followed by food. The buzzer, consequently, became a classical CS signalling food. Its duration was 8 sec, while the metronome had a variable duration depending on the rate of bar pressing (in a dogs: Kyu-chian, Kunio and Hideki). In the other three dogs (Susumu, Takeshi and Taro) we decided to keep the duration of CS_1 also constant, so that the number of bar presses could be used as an index of the instrumental CR, but this caused progressive deterioration of the response and masked the effect of the PR.

Similarly to the study of Ellison and Konorski (1965) also in our dogs there were no bar presses performed during the CS_2 and, contrariwise, the salivation was by far greater during this CS_2 than in response to the CS_1. Thus the instrumental and salivary CRs were relatively well separated from each other and the effect of PR could be observed independently on both types of CRs.

Experiments were carried out in the following way. First, those animals which had not been trained for several months were retrained in the same experimental situation (a sound-proof CR-chamber, Pavlovian stand with feeder, registration of salivary secretion and bar presses) using the same stimuli and procedure as those applied during the acquisition training. After the dogs had reached a stable level of responding to both CSs the experiment consisting of 60 daily sessions was started with the following program.

During the first 10 sessions and the last 10 sessions the dogs were reinforced on each trial. Each of these sessions (denoted henceforth as 100% pre-PR, and 100% post-PR) consisted of four trials, i.e., four presentations of CS_1 followed by CS_2 and US. During the middle 40 sessions a PR procedure was applied. That is to say, in sess ons 11th through 30th a 2 : 3 (or 67%) PR fixed ratiod, quasi-random schedule was applied; each of these 20 sessions consisted of six trials, two of which were not reinforced. The non-reinforced trials were distributed randomly with two restrictions: the first trial was always reinforced, and the non-reinforced trials were never presented in succession. In the next 20 sessions, i.e., in the sessions 31st through 50th, a 50% PR schedule was applied. The number of trials in the daily session remained 6 but only three of them were reinforced. The distribution of the non-reinforced trials was random with no restriction except that the first trial was always reinforced and considered a "warm up" trial. Table I gives a summary of the training procedure throughout all the 60 sessions of the present experiment. It is seen that in an attempt to keep the number of reinforcements constant we had to increase the number of trials from 4 to 6 in 67% PR phase. But during the 50% PR phase we compromised and reduced the number of reinforcement to 3 rather than further increase the number of trials to 8.

In comparing the performance in different phases of training only the trials 2, 3 and 4 were used. Trials 5 and 6 were present only during the phases with PR and they could not be legitimately compared with any trials during continuous reinforcement training.

Table I

Number of trials and pattern of reinforcement in consecutive phases of training

Phase of training	Number of sessions	Number of trials in a session	Number of nonreinforced trials in a session	Distribution of nonreinforced trials in each session
Continuous reinforcement 100% pre-PR	10	4	0	—
Partial reinforcement fixed ratio = 67%	20	6	2	Random, but the 1st trial always reinforced and the nonreinforced trials never presented in succession
Partial reinforcement fixed ratio = 50%	20	6	3	Random, except for the 1st trial which was always reinforced
Continuous reinforcement 100% post-PR	10	4	0	—

Several parameters of conditioned responses were selected and subjected to statistical analysis.

A. Classical salivary CR

Conditioned salivation started usually during the CS_1 but it reached its full rate, counted in drops per minute, during the CS_2 after the animal ceased to press a bar and stood motionlessly staring at the feeder and waiting for the delivery of food. Thus the only measure used was the average rate of salivation during the CS_2.

B. Instrumental CR

Three different indices of the motor CR were employed.

1. *Latency* of the bar pressing response, i.e., the time interval between the onset of the CS_1 and the first bar press.

2. *Total reaction time* of the instrumental CR; in three dogs the total time necessary to perform 14 bar presses was used as a measure of vigor or intensity of the motor response. It was equal to the duration of the CS_1.

3. *Number of bar presses* during 15 seconds of CS_1; in the other three dogs the duration of the CS_1 was kept constant and the number of bar presses was used as a measure of a vigor of CR. The duration of the CS_1 was set in such a way animals could easily, at their average rate of responding, reach the previously required number of 14 presses.

RESULTS

Conditioned salivation

Table II shows changes in an average rate of conditioned salivation in the course of this experiment. Only the responses on trials 2, 3 and 4 contributed to the calculation. Two dogs, Kunio and Taro had very small responses and did not show any changes. The remaining dogs salivated profusely and all of them showed the deteriorating effect of the PR. This effect could be noted already in the second phase, i.e., under PR = 67%, and reached its maximum during the third phase, when reinforcement was presented only in half of trials. During the fourth phase (100% post-PR) when the continuous reinforcement was reinstated, the salivary CRs remained lower, except for the dog Susumu, who was able to recover the rate of salivation equal to that from the first (pre-PR) phase.

The data from the third phase (FR [5] = 50%) were subjected to a more detailed analysis in order to find out what was the immediate effect

Table II

Rate of conditioned salivation (number of drops per minute) during the classical conditioned stimulus (buzzer)

Dog	10 sessions of continuous reinforcement 100% pre-PR	20 sessions of partial reinforcement FR = 67%	20 sessions of partial reinforcement FR = 50%	10 sessions of continuous reinforcement 100% post-PR
Kyu-chian	146.7	101.2***	103.2***	85.7***
Kunio	9.0	9.1°	12.5°	8.8°
Hideki	61.0	58.4°	26.0***	30.3***
Takeschi	82.8	68.4**	52.1***	57.3***
Susumu	112.0	88.8°*	71.4***	109.0°
Taro	13.4	8.5°	9.6°	14.2°

° = risk level above 0.05
°* = risk level close to 0.05
* = risk level below 0.05
** = risk level below 0.01
*** = risk level below 0.001

FR = fixed ratio
Data in collumns FR = 67%, FR = 50% and 100% post-PR are compared with the initial scores presented in the collumn of 100% pre-PR.

[5] Fixed ratio or the proportion of the reinforced trials to the total number of trials expressed as percentage. In operant conditioning the FR refers to the proportion of rewards to the CRs, however, in classical conditioning (and in discrete trials instrumental conditioning) the variables concerned are CSs (or trials) and reinforcements.

of the omission of reinforcement upon the next CR. Similarly as in the Table II only the 2nd, 3rd and 4th trials were analysed, and the changes in the rate of salivation following the reinforced or non-reinforced trial are shown in Table III. The results were not uniform but yielded an interesting picture. In three dogs the salivary CRs were, as one might have expected, smaller after non-reinforced trials. Two dogs did not show significant changes, but in one, Kyu-chian, the opposite effect was obtained. This dog showed a decrease of CR after reinforcement (i.e., the usual pattern in salivary CR session with large portions of food cf. Sołtysik and Czarnecka 1960, Miyata and Sołtysik 1966) but an *increase* in the rate of conditioned salivation after a non-reinforced trial. As it was mentioned earlier, Kyu-chian was the only dog with the Glinski-Pavlov type of fistula which is known (Sołtysik and Zbrożyna 1957, Sołtysik 1957) to be a source of artifacts such as false increase of salivation due to increased motility; the collected in the Stensen's duct saliva may be squeezed out by cheek movements or lowering the head. We could not, however, tell whether this might have caused the paradoxical effect of increased salivation after non-reinforced trials.

Table III

Immediate effect of non-reinforcement on the salivary CR

Dog	Change in salivary CR after reinforced trial	Change in salivary CR after nonreinforced trial	Difference	Statistical significance by two-tailed test	
				t - test	Wilcoxon -White
Kyu-chian	− 14.7	+ 8.3	+ 23.0	$p < 0.05$	$p < 0.04$
Kunio	+ 5.7	− 4.1	− 9.8	$p < 0.05$	$p < 0.13$
Hideki	0.0	− 6.3	− 6.3	$p < 0.01$	$p < 0.05$
Takeshi	− 5.0	− 6.3	− 1.3	n.s.	n.s.
Susumu	− 9.1	− 1.9	+ 7.2	n.s.	n.s.
Taro	+ 4.0	− 5.3	− 9.3	$p < 0.05$	$p < 0.16$

Vigor of the instrumental CR

Vigor of the instrumental CR was measured either as a total reaction time (in three dogs) or as a total number of bar presses during the fixed length of the CS_1 (in the other three dogs), so this should be kept in mind when reading Tables IV and V. The results presented in Table IV show that the introduction of a PR schedule caused a decrease in vigor

Table IV

Vigor of the motor instrumental conditioned response [a]

Dog		Consecutive phases of training			
		10 sessions of continuous reinforcement 100% pre-PR	20 sessions of partial reinforcement FR = 67%	20 sessions of partial reinforcement FR = 50%	10 sessions of continuous reinforcement 100% post-PR
Kyu-chian	Reaction time in seconds	12.95	14.15°	17.03***	12.05°
Kunio		6.77	5.75°	7.93°	6.30°
Hideki		11.41	15.53°	12.61°	11.70°
Takeshi	Number of bar presses	11.27	9.52°	6.10***	7.53**
Susumu		27.27	21.28***	12.60***	13.70***
Taro		11.53	13.23°*	8.87°*	6.87***

a Two indices of vigor were used. In the first three dogs a total reaction time necessary for executing fourteen bar presses was measured. In the other three dogs the duration of the instrumental CS was constant and the mean number of bar presses performed during the CS is presented in the table. Decrease in the total reaction time or increase of the number of bar presses was considered as an increase of a vigor of instrumental response. Statistical significance labelled as in Table II.

Table V

Immediate effect of non-reinforcement upon the vigor of instrumental conditioned response

Dog		Change in vigor of CR after reinforced trial	Change in vigor of CR after non-reinforced trial	Difference	Statistical significance
Kyu-chian	Reaction time	— 0.54	— 0.89	— 0.35	n.s.
Kunio		— 0.20	— 0.90	— 0.70	n.s.
Hideki		— 0.65	— 1.41	— 0.76	n.s.
Takeshi	Bar presses	— 0.53	— 0.44	+ 0.09	n.s.
Susumu		— 0.64	— 0.29	+ 0.35	n.s.
Taro		+ 1.00	0.00	— 1.00	n.s.

of motor CR [6] and this effect is seen most clearly in the third (FR = 50%) phase of the experiment. The only exception is the dog Taro, who in the second phase (FR = 67%) showed a slight increase in the number of bar presses and this change in behavior approaches a statistical signifi-

[6] Decrease in vigor is assumed when there is an *increase* of reaction time, or *decrease* in number of bar presses delivered by the animal in a time unit.

cance. There are, moreover, some other points noticeable in this table. First, the changes in behavior are less marked in the upper three dogs than in the lower ones. This, obviously, was the effect of the change in procedure to which Takeshi, Susumu and Taro were subjected. Until the first phase of this experiment had started, these dogs also had to perform 14 bar presses to secure the application of a buzzer and food. When the duration of a CS_1 (a metronome) had become fixed and the rate of presses no longer influenced its duration, a process of gradual diminution of the motor response began, and, in fact, was detectable already in the first (100% pre-PR) phase of the experiment. Thus, at least part of the deterioration of the motor CR in these three dogs must be attributed to that factor. The dogs in the upper half of Table IV show very little effect of the PR (except for Kyu-chian in the 3rd phase) and a full recovery of the initial values in the 4th phase.

Let us see how the non-reinforcement influenced the vigor on the next following trial (Table V). Although the changes were small and statistically for each dog insignificant, the direction of a change was in all the dogs but one (Taro) towards an increase of vigor after a non-reinforced trial. Thus, all the dogs in the upper half of the Table V show a decease of a total reaction time after any preceding trial (reinforced or non-reinforced) but this decrease is stronger after non-reinforcement. In the dogs in which a number of presses was measured in a constant time of a CS_1 the number of presses tended to drop trial to trial, but after non-reinforcement this drop was smaller than after the reinforced trial. It should be reminded that both groups of dogs differed from the very beginning of the experiment in that the dogs which were shifted from the 14 bar presses to a fixed duration of CS_1 (Takeshi, Susumu, Taro) showed a gradual decrement in vigor within the session, while the dogs which remained on the previous procedure (i.e., 14 bar presses secured the CS_2) tended to increase their vigor from trial to trial. Nevertheless, on the background of these trends, the effect of PR, namely, the increase of vigor after each omission of reinforcement, could be noticed.

Latency of the instrumental CR

Changes in the latency of motor CR, presented in Table VI, were not uniform in our dogs. Hideki did not changed at all. Kunio, Takeshi and Susumu increased their latencies. But Kyu-chian and Taro showed a decrease in *average latencies,* that is, a positive PR effect consisting in an improvement of the instrumental CR. Taro shows this change after an introduction of PR in the phase of PR = 67%. Kyu-chian also

Table VI

Latency of motor instrumental conditioned response

Dog	Consecutive phases of training			
	10 sessions of continuous reinforcement 100%	20 sessions of partial reinforcement FR = 67%	20 sessions of partial reinforcement FR = 50%	10 sessions of continuous reinforcement 100%
Kyu-chian	4.52	4.04°	6.63***	3.17***
Kunio	1.02	1.14*	2.06***	1.17°
Hideki	1.48	1.41°	1.66°	1.74°
Takeshi	5.40	6.60*	6.42°	7.99**
Susumu	4.16	5.07*	6.65***	7.23***
Taro	2.87	2.00*	3.49°	2.54°

Table VII

Immediate effect of non-reinforcement upon the latency of instrumental conditioned response

Dog	Change in latency after reinforced trial	Change in latency after nonreinforced trial	Difference	Statistical significance by two-tailed test	
				t - test	Wilcoxon -White
Kyu-chian	+ 0.11	— 0.77	— 0.88	n.s.	n.s.
Kunio	+ 0.29	— 0.13	— 0.42	$p < 0.05$	$p < 0.01$
Hideki	+ 0.22	— 1.38	— 1.60	$p < 0.01$	$p < 0.25$
Takeshi	+ 1.20	— 0.14	— 1.34	n.s.	n.s.
Susumu	+ 0.42	— 0.40	— 0.82	n.s.	n.s.
Taro	+ 1.47	— 0.35	— 1.82	$p < 0.02$	$p < 0.015$

shows the same drop in latency during the second phase (though not significant at the 0.05 level), then during the third phase his latencies considerably lengthened, but in the final phase they shorten below the initial level. Both changes were significant at the 0.001 risk level.

Even more spectacular was the immediate effect of non-reinforcement upon the latency observed during the third phase when the food US was presented only in half of the trials. As seen in Table VII all the dogs showed an increase of the latency after the reinforced trial and, contrariwise, a shortening of the latency after the omission of reinforcement. This is a significant result by any appropriate non-parametric test

(a change in the same direction in all cases when N = 6), and moreover, in three dogs this change was significant also by intra subject comparison.

DISCUSSION

The problem of PR has attracted many authors and there is an abundant literature devoted to its effects on behavior. Needless to say, an overwhelming majority of publications concerns the instrumental emitted responses, i.e., a behavior not separated into trials. For this kind of experiments a classification of schedules of reinforcement was elaborated (Ferster and Skinner 1957) and in a free responding situation the most striking effects of PR was the increased vigor of CR and increased restistance to extinction. This line of research was initiated by Skinner (1938) and much of our present knowledge on PR we owe to his and his followers' investigations.

Three other types of behavior, where the PR was also studied, are instrumental conditioning in a discrete trials situation, classical conditioning, and verbal expectancy. All these were first investigated by Humphreys (1939a b, 1940), who demonstrated an increased resistance to extinction (a greater "reflex reserve" as it was called after Skinner at this period) and explained it in terms of a "more difficult formation of the expectation of nonreinforcement".

Several explanations of the PR effect were proposed by a number of authors. Formulated in various scientific languages they are difficult for concise reviewing. Generally, however, their main task was to explain the increased resistance to extinction. Since this is not the subject of our study, we shall concentrate only on the hypotheses that postulated a greater (at least temporarily) vigor of CRs resulting from intermittent non-reinforcement as compared with the performance in the 100% reinforcement situation.

An excellent example of the PR effect upon the vigor of the instrumental CR was provided in the studies of Goodrich (1959) and Amsel et al. (1964). Goodrich compared the speed of a running response in two groups of rats: one was rewarded continuously, whereas the other received a reward in only 50% of trials. The PR group showed faster running in the two first segments of a runway. However, in the third (i.e., the last) segment the continuously reinforced group was consistently faster. In the Amsel et al. (1964) study, the comparison of a speed of running response was made within the same subjects, i.e., each rat was partially rewarded in a black alley and continuously rewarded in a white one. Here again, the rats were faster in the black alley, or more exactly, in

the first two segments of this PR alley; in the last segment they were slower in the black alley than in the continuously rewarded white one.

How were these results, explained by the authors? In his several publications Amsel (1951, 1958, 1967) presented an outline of a theory, which combines a neo-Hullian approach (i.e., an analysis of behavior based on Hullian system supplemented by a classical conditioning model) with a concept of frustration as an implicit reaction to a non-reward in a situation where a reward is expected. Frustration, in this theory has two aspects. Firstly, it adds to a general drive, and secondly, it produces specific internal stimuli. There are a few consequences of these assumptions. One is that frustration, being a sort of unconditioned reaction can be classically conditioned. Secondly, and the most important one, is that the uncoditioned or conditioned frustration exerts a definite influence upon the behavior because it increases the "general" drive. Thus, it results in an intensification of any ongoing activity. Finally, being an increase of drive, frustration acts as a punishment so that any precedent response tends to be stamped out, i.e., inhibited or replaced by other responses. Amsel's theory further parallels the conditioned frustrative responses with Hullian antedating fractional goal responses (with accompanying internal stimuli) in order to explain the greater resistance to extinction of a CR acquired under PR, but this extension of his theory is not directly related to our data and need not be discussed here.

Another theory of frustration which also may be pertinent in this context is the Brown and Farber (1951) formulation. It has also its roots in the Hullian system but differs from Amsel's theory in some points. Brown and Farber regarded the frustration as a hypothetical state of an organism (for Amsel it was a response) resulting either from an inhibition (e.g., due to non-reinforcement, blocking or excessive amount of work) or from a conflict with some competetive tendency. The consequences of frustration in this formulation are much the same as in Amsel's theory, i.e., an increase of general drive and the provision of specific stimuli.

What is important in both these theories is that one can make some predictions for the situation where continuous reinforcement is replaced by partial reinforcement. If frustration appears in response to the omission of the expected reward and acts both as an energizer of the ongoing activity (increment of drive) and as a puishment of the precedent responses [7], two effects should be predicted. First, the increased drive pro-

[7] In the Hullian theory a reward is defined as a reduction of drive; henceforth any increase of drive is aversive and should exert a punitive effect upon the preceding behavior.

duced by frustration should result in an increase of the vigor of the CRs, particularly on trials that follow the non-reward. Secondly, there should be a progressive decrease in the vigor of CRs because they are punished on each omission of reward by the frustrative increase of drive.

Before, however, any attempt of applying the aforementioned theories to our results, one has to point out, that these theories deal with the PR effects (using the concept of frustration as an intervening variable) only within the instrumental conditioning paradigm. Could they also cope with the classical conditioning? Amsel (1967) expressed his doubts recently, suggesting that "different explanatory schemes may be required to understand PR effects in those two" (i.e., classical and instrumental) "kinds of experiment".

Let us examine now what explanatory possibilities are offered by the integrated instrumental-classical conditioned reflex model developed in the Nencki Institute (Sołtysik 1960, Sołtysik and Konorski 1966, Konorski 1967). As a departure point for our explanation of the PR effect, a schematic diagram of the hypothetical structure of the neural mechanism involved in an instrumental food CR is presented in Fig. 1. CS_1 and

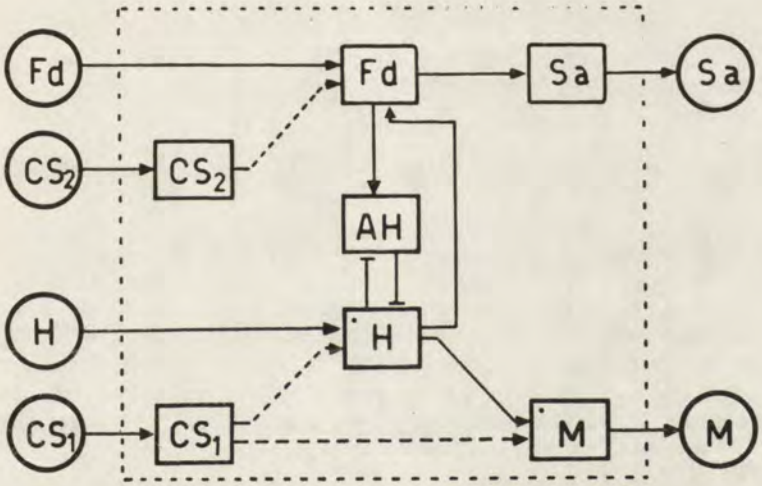

Fig. 1. A block model of the instrumental-classical CR arc. Circles represent observable or manipulable variables: Fd, food, or more exactly the taste of food. CS, conditioned stimulus; H, hunger; M, motor instrumental response; Sa, salivation. Rectangles represent centers: CS, center or CS; Fd, center of the consummatory feeding reflex; Sa, salivary nucleus; H, hunger center; AH, antihunger center; M, center of motor response. $\rightarrow$, facilitatory or activating connection. $\dashv$, inhibitory connection. Solid line, unconditioned connection; broken line, conditioned connection. Note that the hunger center is activated jointly by H and CS_1 and inhibited through AH by the Fd center. But H facilitates the Fd center establishing thus a stabilizing negative feedback controlling the output of the hunger drive center.

hunger H are factors eliciting and determining the strength of the instrumental response M. CS_2 and, to some extend, a feedback from the M elicits consummatory food reflex (Fd) exemplified on the effector side by salivary response (Sa). The interconnections between the hunger and consummatory centers are shown in the central part of the diagram. They are assymetrical. The consummatory reflex is facilitated by hunger drive but the latter is inhibited by the former via an anti-hunger center. The details of this model need not be discussed here as they were treated in much detail, in Konorski's monograph (1967).

Let us assume now, after Brown and Amsel, that frustration produced by an omission of reinforcement acts as an agent increasing motivation. This could be effected in one of several ways or by a combination of some of them. Fig. 2 illustrates these possibilities. Thus, frustration

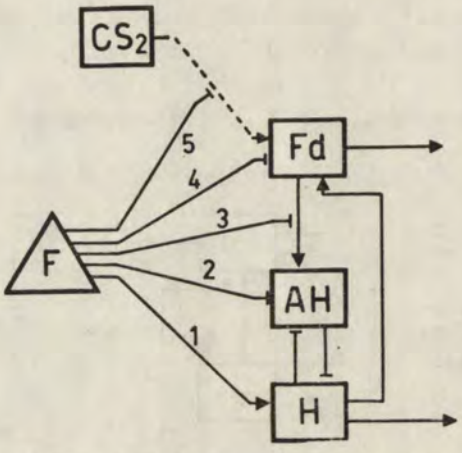

Fig. 2. A block model of the hypothetical action of frustration on the instrumental and classical food CRs. Only the central part of the diagram from Fig. 1 is presented for simplicity. Explanations the same as in Fig. 1. Note that the frustrative increase of drive may be obtained by five different ways, but only the "connections" 4 and 5 produce a simultaneous increase of drive and depression of the consummatory behavior

(F in a triangle — to avoid for a moment a question on the nature of this variable) may either activate a hunger-drive center (1), or inhibit an anti-hunger center (2), or inhibit a feeding center (4), or block the connections between the two latter centers (3), or even inhibit the conditioned connection of the consummatory CR (5).

How this can be applied to our data? In order to recall our results presented above in the detailed but not easily readable form of tables, Fig. 3 is added. It shows graphically what happened with the salivary and

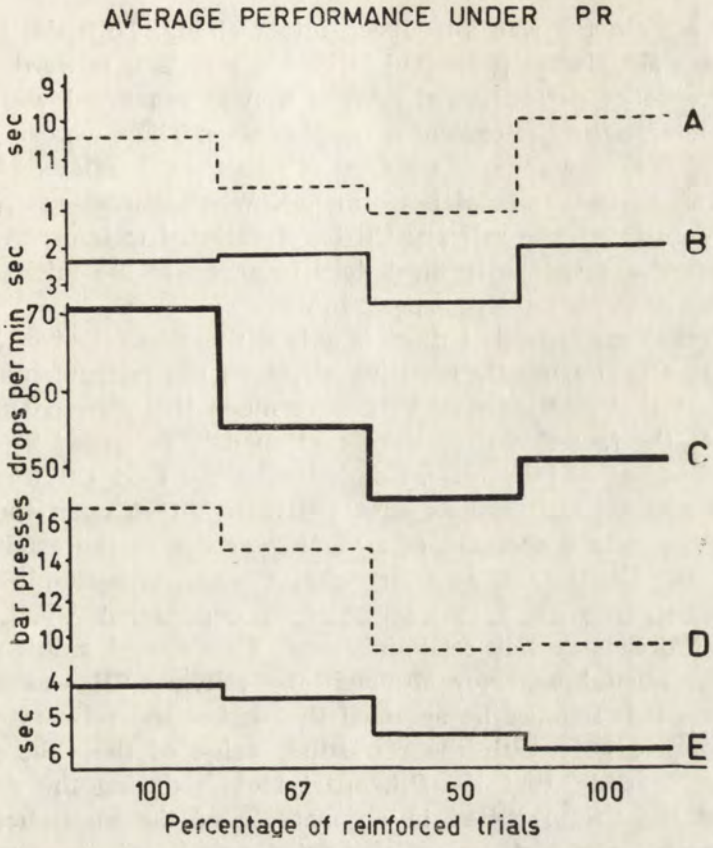

Fig. 3. A, Total reaction time in three dogs (Kyu-chian, Kunio and Hideki). Vigor of this response (14 bar presses) decreases after introduction of PR but fully recovers in the fourth phase when the continuous reinforcement is reinstated. B, Latency in the same three dogs. Only during the third phase of training (under 50% PR) is there an increase of latency but it also fully returns to initial level in the fourth phase. C, Conditioned salivation in all six dogs. The deteriorating effect of PR is perfectly evident. There is only slight improvement of this CR in the fourth phase, suggesting that the extinction of this response was much deeper than of the instrumental CR. D, and E, Vigor (number of bar presses) and latency of the instrumental CR in three dogs (Takeshi, Susumu and Taro). In those dogs the duration of CS_1 was kept constant and the number of bar presses indicated the vigor of CR. This procedure caused progressive deterioration of the CR so the effect of PR is largely masked. In fact, when the number of bar presses remained during the third and fourth phase on the same level, it should be interpreted that there was some increase of vigor in the fourth phase, otherwise a further drop in number of bar presses would have occurred. Ordinates for A, B and E are seconds; for C are drops per minute; and for D are bar presses during CS_1. Thus, deflection downwards always means decrease in response and deflection upwards corresponds to an increase of response

motor CR after a PR was introduced into training. To make it clearer the ordinates are always presented in such a way that upward direction means increase of performance, even if that is connected with a drop of a parameter, as in the case of a reaction time. Thus, we can visualize easily that PR caused generally a drop of salivary CR followed by a less marked drop in instrumental performance. When continuous reinforcement was reinstated, the salivary CR hardly started to improve whereas the instrumental response in most dogs returned to the initial level of performance or even slightly surpassed it.

This picture suggests that there is only a negative effect of PR upon the salivary CR and mostly negative effect on the instrumental behavior. The fact that instrumental CRs were much less affected may have been due to the fact that they were continuously rewarded by the CS_2, so that the effect of intermittent omission of the food US was greatly attenuated and we can assume that the main effect upon the instrumental CR, namely a decrease of a vigor, was due to the gradually diminishing value of the CS_2 as a secondary reward. This simple explanation is insufficient in the fourth phase of the experiment, when the continuous reinforcement was readministered. This caused a full recovery of the instrumental behavior although the salivary CR was still evidently depressed. It must be assumed that either the salivary CR does not change parallelly with the rewarding value of the CS_2, or, what seems more feasible, that the PR, apart from reducing the rewarding function of the CS_2, resulted in an increase of the instrumental CR. This enhancement of instrumental behavior, was largely masked by the reduction of the secondary reward provided by the CS_2, but could be revealed when continuous reinforcement in the fourth phase reversed the progressive extinction of the consummatory CR elicited by the CS_2.

The latter conclusion is supported by our data concerning the immediate effect of non-reinforcement. In most cases the instrumental CR was facilitated whereas the salivary CR was diminished. If we wanted now to point at a possible way in which this "frustrative" effect could have occurred, the simplest explanation is offerred by the effects 4 and 5 on our hypothetical diagram in Fig. 2, that is, by postulating the inhibition of food consummatory center or by supressing the conditioned consummatory CR. In other words, the primary effect of PR would consist in the reduction of the consummatory CR. This by itself explains also the changes in the instrumental behavior, for such an extinctive diminution of the consummatory CR releases from partial suppression (through the anti-hunger center) the hunger-drive center which in turn leads to an enhanced instrumental behavior. It should be obvious from this formulation that the hypothetical variable F (frustration) is super-

fluous and can be removed from our diagram, because the extinction of consummatory CR being the primary effect of PR (or more correctly, partial non-reinforcement) does not need it. "Frustrative competetive responses" hypotheses of extinction may be justified only for instrumental reward training and even here a batter explanation is provided by the analysis of changes in the underlying classical conditioned drive and consummatory CRs. The weakening of the consummatory CR occurs when, due to intermittent non-reinforcement, the CS_2 becomes an ambiguous signal activating both the Fd (food) and N-Fd (non-food) centers.

There is one procedural aspect of our experiment on which we would like to comment. By eliciting a fair rate of salivation on each trial the CS_2 prevented the artifact caused by longer intervals between consecutive activations of the salivary gland. As known from acute experiments on the nerve-gland preparations (Babkin 1950) the salivary response to the same stimulus becomes smaller and smaller when the interval or resting period increases and this effect is most pronounced for intervals of 1 to 10 min. In normal unanaesthetized dogs this phenomenon was described for both unconditioned and conditioned CRs (cf. Bruner and Kozak 1954, Czarnecka and Sołtysik 1962). The fact that our dogs salivated on each trial enables us to ascribe the change in the salivation rate after non-reinforcement primarily to some central and not glandular factors. Thus, in contradistinction to the earlier studies on the PR effect in salivary conditioning (Brogden 1939, Wagner et al. 1964, Fitzgerald 1964), the conclusion that PR resulted in our dogs in partial extinction of the consummatory CR (the salivary CR being the index of it) cannot be criticized on the ground that some longer intervals between consecutive activations of glandular tissue were present in the sessions with PR.

An additional evidence for the latter statement is provided by the dog Kyu-chian. The salivary CRs in this dog were significantly greater after non-reinforced trials than after the reinforced ones. However, this is an embarassing evidence as it seems to disprove our hypothesis that the "frustrative increase of drive" is caused by the partial extinction of the consummatory CR. It should be mentioned here that Kyu-chian was the only dog with a sizable salivary CR to CS_1. It was therefore possible in this dog to analyse the changes in conditioned salivation accompanying the instrumental response. The results were as follows. The average rate of salivation during the first phase of training (i.e., under the continuous reinforcement) was 146.7 drops per minute (d.p.m.) during the CS_2 and 58.9 d.p.m. during the CS_1. In the third phase (under the 50% PR) the salivary CR to CS_2 dropped to 103.2 d.p.m., which equals to 70% of the previous value, but the CS_1 elicited only 34.6 d.p.m.

what amounts to 59% of the initial pre-PR value. Thus the consummatory CR to CS_1 suffered more than the same CR to CS_2. But even more important was the immediate effect of nonreinforcement upon the following salivary CR to both CSs. In contrast to the increase of the salivary CR to CS_2 after nonreinforced trials, the salivation to the CS_1 in the same trials, was diminished, and the average drop of rate was about 3 d.p.m. After reinforced trials the change was in the opposite direction and the average response to CS_1 was greater of about 1 d.p.m. when compared with the preceding one.

Thus, even Kyu-chian does not contradict our general line of reasoning. The consummatory CR was in fact diminished to the CS_1 and the vigor of the instrumental CR was slightly greater after non-reinforcement. On the other hand, the remarkable increase of the salivary CR to CS_2 after non-reinforcement cannot be at present satisfactorily explained. It could be an artifact from the Stensen's duct which often contains a certain amount of saliva (Sołtysik 1957, Sołtysik and Zbrożyna 1957) and this residual saliva is likely to be squeezed out if any increased motor activity occurs. Kyu-chian was the only dog with the classical Gliński—Pavlov fistula, whereas the remaining dogs had the Stensen's duct surgically shortened.

SUMMARY

The effect of 67% and 50% fixed ratio quasi-random partial reinforcement upon classical salivary and instrumental motor CRs was studied in six dogs. The dogs were first well overtrained under continuous reinforcement and then the 67% and 50% schedules were applied. The instrumental CR was a chained response of 14 bar presses to a metronome (an instrumental CS) followed by a buzzer (a classical salivary CS) and food, so that the instrumental and salivary CRs were elicited by two different CSs and could be studied simultaneously though separately (the Ellison—Konorski procedure). The effect of partial reinforcement was observed on the rate of conditioned salivation, latency of the first bar press and on the vigor of the instrumental CR.

The partial reinforcement caused a deterioration of salivary CRs. The instrumental CRs were also diminished but some evidence was provided that there was an increase of a drive CR, masked by the decrease of incentive motivation. The immediate effect of non-reinforcement consisted in a diminution of salivary CRs, considered as a partial extinction of the consummatory CR, and a simultaneous increase of instrumental CRs, explained by the release of the drive centre from inhibitory influence of the consummatory center.

Thus the "frustration effect" was explained in terms of changing proportions of consummatory and drive CRs, due to the partial extinction of the former and a release of the latter CR in conditions of an intermittent reinforcement.

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THE EFFECT OF CARDIAC SYMPATHECTOMY ON INHIBITORY AND EXCITATORY CARDIAC CONDITIONED REFLEXES IN CATS

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Several authors have reported that the action of a classical defensive conditioned stimulus produces a decrease of heart rate in the cat (Jaworska 1959, Santibañez-H. 1962, Santibañez-H. et. al. 1963). The physiological mechanism underlying this phenomenon is as yet poorly understood. Santibañez-H. et. al. (1965) have demonstrated that extirpation of the sygmoid cortex prevents the appearance of conditioned bradycardia, while excision of "gyrus genualis", in the middle of the hemisphere, facilitates it. The aim of the present paper is to investigate the role of the sympathetic nerve in cardiac CRs.

METHOD

Subjects. Ten cats divided into two groups were used: 4 cats formed the normal group (NG) and 6 cats were sympathectomized (SG).

Each cat was kept in an individual cage and fed on meat and water "ad libitum". They were fed 3 hr before training, receiving during this period milk and vitamins in addition to their ordinary diet. Operatees were kept at a constant temperature in a thermoregulated room.

Apparatus and Recording. Training was carried out in a cage (90×90×120 cm) placed inside a sound-proof chamber bearing a one-way vision window. Animals were placed in a hammock and cardiac recording and stimulating electrodes were adjusted to the skin of the limbs. The EKG was recorded by a Gilson poligraph. The rewarded stimulus (CS) a tone of 500 c/sec, and a non-rewarded differential

stimulus (DS), a tone of 1000 c/sec, were both of 5 sec duration, while a combination of 1000 c/sec for 3 sec and 500 c/sec for 2 sec provided an inhibitory compound stimulus (ICS). These tones were generated by two Philips audioscillators. US was an electric shock (300 c/sec frequency, 1 msec pulse duration and 30—80 volts) given to the right fore-paw with a Grass stimulator and capable of producing retraction of all four legs.

Operations. Following a modification of the technique described by Hodes (1939), sympathectomy was performed under deep Pentobarbital anesthesia in three stages: a) neck, bilateral removal of cervical sympathetic trunk and medial and superior cervical ganglia; b) right thorax, removal of stellate ganglion and of lateral chain up to the 5th ganglion; c) left thorax, same procedure. Operations were performed in one week intervals, while animals received antibiotics and special diet in post-operative periods. 20 days after the last operation, training started.

Training. Before initiation of training, animals were adapted to the experimental situation through seven sessions. Spontaneous cardiac rate and modifications induced by the acoustic stimuli to be used in the conditioning process were recorded during the 7th session. The effect of electric shock to be used as US was also tested.

All the animals were trained through 70 sessions of 12 trials each (6 times 500 c/sec reinforced, 3 times 1000 c/sec not reinforced and 3 times 1000 + 500 c/sec not reinforced). CS (500 c/sec) was reinforced after 5 sec with an electric shock. EKG was recorded throughout the session but, for statistical reasons was analyzed in 3 periods: a) 10 sec before the onset of acoustic stimulus ("before"); b) 5 sec from the onset of acoustic stimulus ("during") that is until the shock in the case of CS and c) 10 sec from the shock onwards, or the temporal equivalent period for DS and ICS ("after").

Electrophysiological control. At the end of the training period animals were anesthesized with chloralose, the thorax opened and the sympathetic trunk stimulated electrically in order to observe if sympathectomy was complete.

RESULTS

Normal group

Before training acoustic stimuli produced either no effect or heart rate increase. Electrical shock produced a general motor excitement with a tendency to become free of the hammock, midriasis, piloerection and increase of muscular tone. Heart rate was increased and micturition and defecation was sometimes observed. As training continued, the defensive motor response to shock progressively diminished, requiring an increase in voltage output from the stimulator. It was also observed that while reactivity to shock at the beginning of a session was moderate, it increased towards the end of the session.

Positive bradycardic conditioned response was very soon established, as has been indicated in other studies (Santibañez-H. et al. 1963). In the initial ten sessions differences between "before" and "after" were statistically significant. These difference became more obvious in the last 20 sessions, being significant at a level of $p < 0.02$ (Fig. 1, Table I).

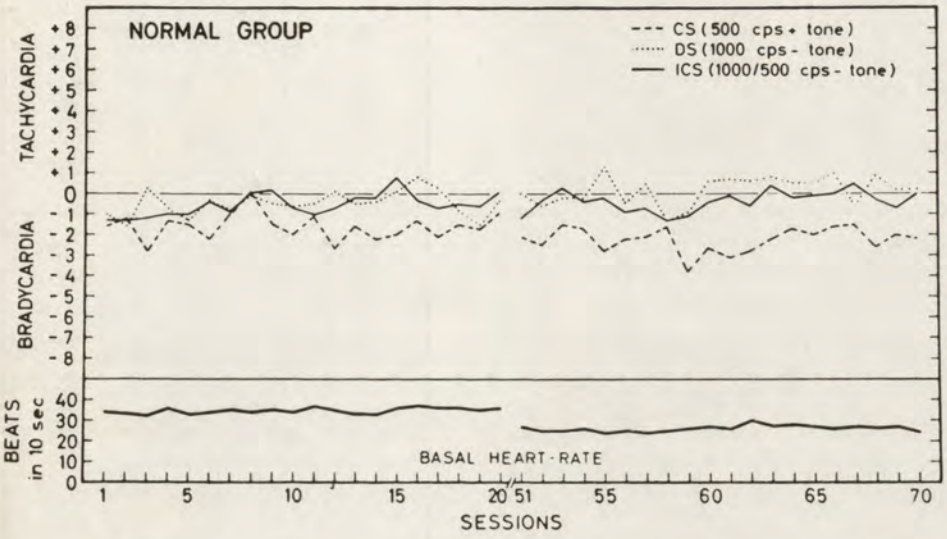

Fig. 1. Evolution of cardiac responses resulting from the comparison between 10 sec "before" and "during" CS, DS and ICS application in the normal group (NG) through 70 sessions. Note that positive response is bradycardia and that inhibitory responses are not different from 0. Evolution of basal heart rate in 70 sessions

Inhibitory conditioning progressed differently. In the first 10 sessions there was a period of generalization. The inhibitory process started to develop slowly and was clearly and steadily established in the last 20 sessions. Establishment of conditioned inhibition was much more difficult than that of differential inhibition (Fig. 1, Table I).

Another fact to be stressed is the way in which spontaneous heart rate was altered through training, as measured in the experimental chamber (Fig. 1). During the 20 initial sesions heart rate frequency was maintained at around 35—38 beats per 10 sec, but in the last 20 sessions this was slowed down to around 25 beats per 10 sec. This phenomenon is statistically significant at the 0.05 level.

Table I

Normal group. Comparison of the mean heart rate of the last 20 sessions before, during and after the action of CS, DS and ICS

Cat	Stimulus	Before	During	After
1	CS (positive stimulus, 500c/sec)	27.96	25.85	28.61
2		26.57	23.53	27.21
3		22.41	19.78	24.88
4		26.46	25.37	27.37
		$\bar{x} = 25.85$	23.63	27.01
		t =	5.2**	2.8
1	DS (differential stimulus, 1000c/sec)	28.10	28.60	26.70
2		23.90	22.50	21.50
3		23.10	23.30	20.70
4		24.60	24.20	23.30
		$\bar{x} = 24.90$	24.60	23.10
		t =	0.7	5.8***
1	ICS (inhibitory conditioned stimulus 1000 + 500 c/sec)	28.20	28.20	25.60
2		23.00	21.80	21.10
3		23.50	23.00	18.30
4		27.00	27.20	25.00
		$\bar{x} = 25.40$	25.00	22.50
		t =	1.2	3.8*

* $p < 0.05$.
** $p < 0.02$.
*** $p < 0.01$.

The relation "before" — "after" is interesting to analyze (Fig. 2). As CS was always reinforced by a shock, obviously the heart rate response was increased after the operation of the CS. This increase was observed during the whole training period. However, the relation was quite different with regard to the DS and ICS, because here, bradycardia appeared in the "after" period.

Table II shows the analysis of variance of heart frequency in the "during" period for CS, DS and ICS. The test shows that there are no differences between the inhibitory stimuli but both of these are statistically significant compared to CS at a level of $p < 0.001$.

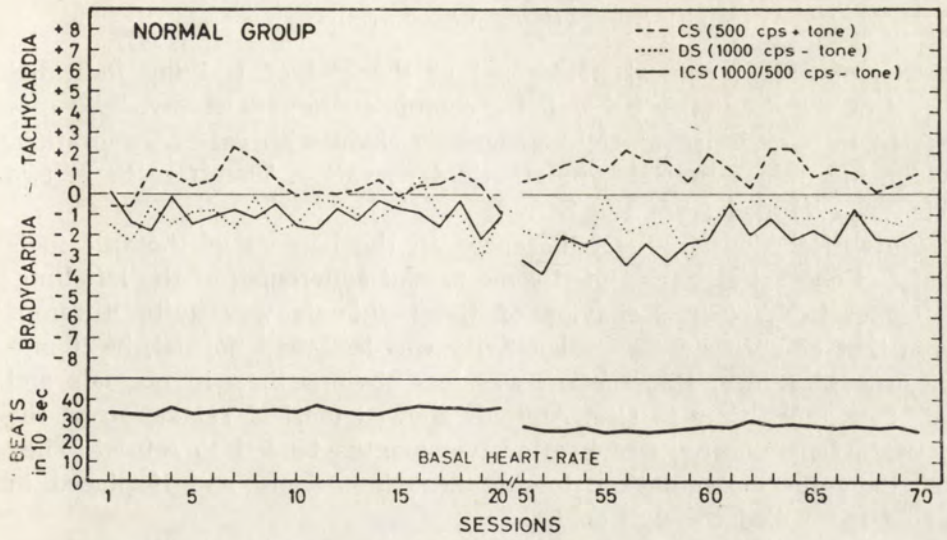

Fig. 2. Evolution of cardiac responses resulting from the comparison of 10 sec "before" and "after" CS, DS and ICS applications in the normal group (NG) through 70 sessions. Evolution of basal heart rate in 70 sessions

Table II

Normal group. Analysis of variance of mean heart frequency for CS, DS and ICS in the last 20 sessions

Source of variation	Sum of squares	D.F.	Variance estimate	F (varience rate)	P
Between sets	158.00	2	79.00	6.27	0.01
Within sets	718.35	57	12.60		
Total	876.35	59			

Groups	Difference	SE of difference	Difference (SE)	P
CS and DS	2.63	0.355	7.4	0.001
CS and ICS	2.90	0.355	8.2	0.001
DS and ICS	0.27	0.355	0.8	

Sympathectomized group

The operation control performed at the end of training indicated that 4 of the 6 animals presented a complete sympathectomy. Two cats reacted to stimulation of right ganglionic chain with cardioacceleration, evidencing only a partial sympathetic denervation. Therefore these two cats are excluded from the SG.

Autopsies showed lung adherences on the borders of thoraxic incision and one cat also presented some partial adherences of the left lung.

General behavior. Behavior of these animals was quite different from that of normals. General activity was less than normal, be it motor or exploratory. Piloerection was less obvious than in normals and hair took more time to rise. Animals showed marked relaxation of the nictitating membrane, which after two months tended to retract. They lost their thermoregulation, loosing heat intensively and if placed in a cold room died through cold.

Pretraining period. Isolated acoustic and electric stimuli produced a heart rate increase. Cardiovascular responses to electric shock were doubtless stronger than those observed in normals, and they were accompanied by intense liberatory reflexes.

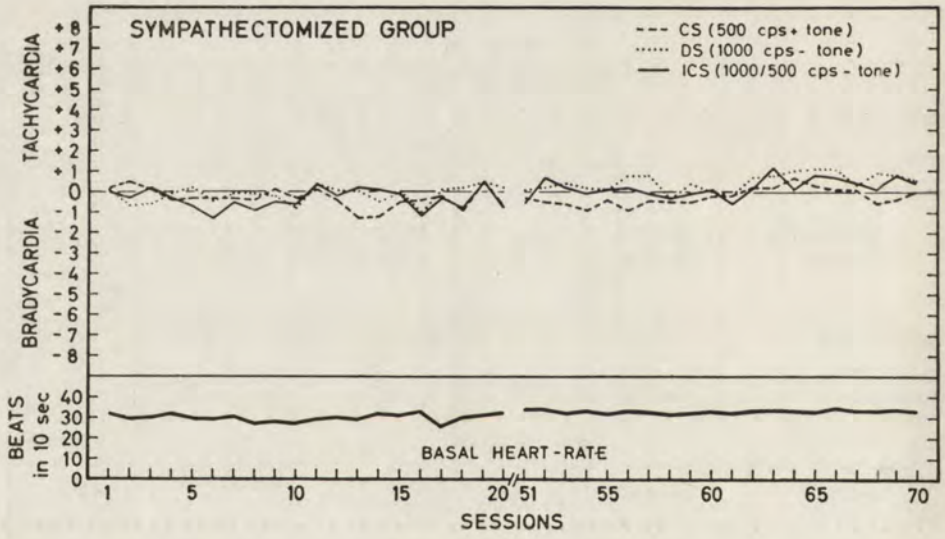

Fig. 3. Evolution of cardiac response resulting from the comparison of 10 sec "before" and „during" CS, DS and ICS application in the sympathectomized group (SG) through 70 sessions. Positive conditioned response is bradycardia, but it is not present in this group. Evolution of basal heart rate through 70 sessions

Training period. Fig. 3 and Table III show that SG not only failed to establish a bradycardic CR to the CS, but, on the contrary, developed a clear tachycardic CR (significant at the 0.05 level). Basal heart rate did not undergo any alterations through the whole training period and spontaneous frequency was maintained at about 30 beats per 10 sec.

Table III

Sympathectomized group. Comparison of the mean heart rate of the last 20 sessions before, during and after the action of CS, DS and ICS

Cat	Stimulus	Before	During	After
1	CS	36.97	37.84	39.73
2		32.38	33.31	34.20
3		26.25	28.51	30.61
4		37.63	38.97	42.67
		$\bar{x}$ = 33.31	34.66	36.83
		t =	4.21*	4.76**
1	DS	38.09	39.25	36.34
2		32.51	31.85	29.87
3		27.73	28.53	26.25
4		38.52	39.27	37.61
		$\bar{x}$ = 34.21	34.73	32.52
		t =	1.23	4.69**
1	ICS	37.82	38.50	36.25
2		33.23	33.30	28.88
3		27.79	28.15	26.22
4		38.89	38.86	37.74
		$\bar{x}$ = 34.43	34.70	32.27
		t =	1.79	2.93

* $p < 0.05$.
** $p < 0.02$.

While examining Fig. 4 we see that in the "after" period the SG presented marked bradycardia to DS and ICS, and tachycardia to US. Table III shows that differences "before" — "after" is statistically at a level of $p < 0.02$ for DS and US.

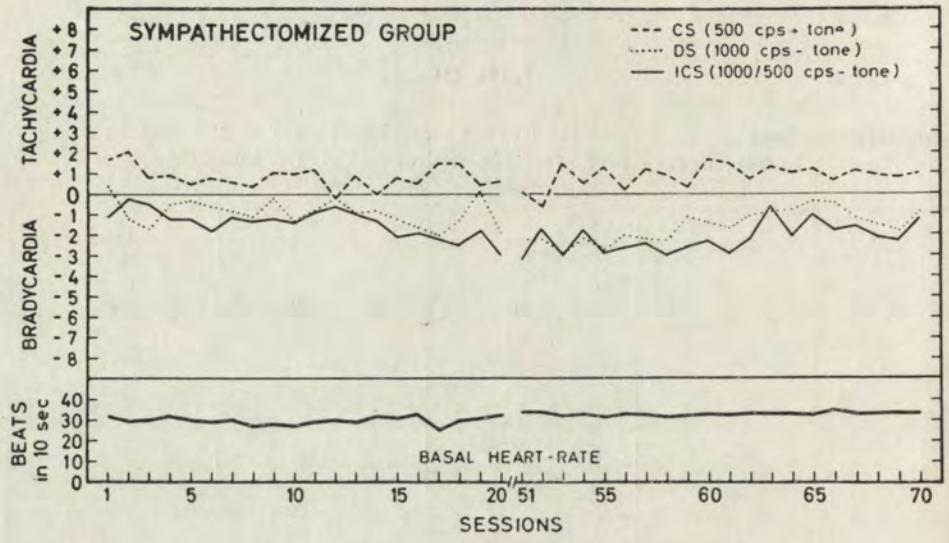

Fig. 4. Evolution of cardiac responses resulting from the comparison of 10 sec "before" and „after" CS, DS and ICS application in the sympathectomized group (SG) through 70 sessions. Evolution of basal heart rate through 70 sessions

Comparison of the two groups

Basal cardiac rate is different for both groups. Table IV shows that this difference is significant at the 0.01 level. Fig. 5 shows that basal heart rate in NG was around 35 beats per every 10 sec, in the first 30 sessions, coming down to 25 beats per sec, in the last 20 sessions. Heart rate in the SG, on the contrary, oscillated around 30 beats per every 10 sec in the first 20 sessions and thereafter rose to the level of 33 beats.

SG differed clearly from NG in that it developed tachycardic CR instead of bradycardic CR to CS. On the other hand the reaction to the DS and the ICS in the "before" — "after" comparison was in both groups exactly the same.

SG presented more orienting reflexes and more motor conditioned responses than NG in the experimental situation, in spite of the fact that SG was spontaneously less dynamic and exploratory.

Table IV

Analysis of variance comparing basal heart-rate between NG and SG

Source of variation	Sum of squares	D.F.	Variance estimate	F (variance rate)	P
Between sets	394.40	1	394.40	7.6	0.01
Within sets	1973.60	38	52.04		
Total	2368.00	39			

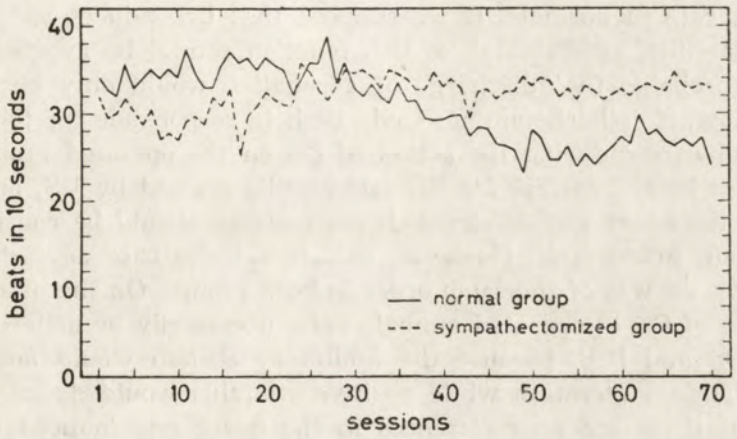

Fig. 5. Comparison of the evolution of basal heart rate in the normal and sympathectomized group through 70 sessions. Note that NG presents decrease of heart rate frequency from the 30th session onward, while SG does not show any evident alteration

Santibañez-H. et al. (1963) observed previously that in normal cats motor defensive conditioned responses tended to disappear, at the same time as bradycardic responses became more stable. This principle was confirmed and extended to the sympathectomized preparation during periods of motor excitement.

DISCUSSION

As the present experiments show, sympathectomy produces complete abolition of defensive bradycardic CR to a positive CS and its substitution by the tachycardic CR. On the other hand bradycardia which follows the application of inhibitory CSs (DS and ICS) remains unimpaired. What is the explanation of this seemingly paradoxical result? We shall

attempt various hypothetical explanations. If we accept that the operation produced sensitization of the heart by denervation, the response of the experimental group to CS and US would be induced by circulating cathecholamines. During CS, the vagus nerve would not be able to produce a decrease of the heart rate. DS and ICS would not produce additional cathecholamines so cardiac rate would not change. This explanation sounds quite reasonable, logical and based on facts solidly established, but not all the facts observed in our results fit in it.

If the principal mechanism involved in the experimental situation were mediated by adrenaline, the action of CS would hardly slow down the heart rate in normals. If we suppose that US induces secretion of cathecholamines, conditioning of this reaction cannot be avoided and it seems improbable that discharge of adrenaline would only be induced by US. Now, if cathecholamines could be held responsible for the absence of bradycardia during the action of CS on the operated group, they would also be responsible for the tachycardia elicited by US, and as in this case the heart was denervated, the reaction should be considerably higher than in normals. However, this is not the case as tachycardia induced by US was of the same order in both groups. On the other hand, the action of CS in operated animals must necessarily be different than that of DS and ICS, because the inhibitory stimuli would not induce cathecholamines secretion while positive stimulus would do so. Results show that there are no differences in the heart rate induced by both kinds of stimuli. Consequently this interpretation does not satisfy the factual requirements of our results.

If we accept that bradycardia may be either due to the increase of the tonus of the vagus nerve or to the inhibition of the tonus of the sympathetic nerve, it follows that bradycardic defensive CR observed in normal cats was produced exclusively by the latter and not by the former mechanism, since it was affected by sympathectomy. Bradycardia following the inhibitory CS, on the contrary, must depend, not on the decrease of the sympathetic tonus but on the increase of the vagal tonus which is not affected by our surgery.

This hypothesis suggests that the mechanism of the two bradycardic reflexes is quite different. The defensive bradycardic reflex would belong to the class of passive fear reflexes (Konorski 1967) involving freezing. The animal was immobile because in the given circumstances this is biologically the optimal defense and not because he was calm. On the contrary, bradycardia following the operation of an inhibitory stimulus was due to the state of security and relief.

The general conclusion which may be drawn from this interpretation is that all fear drive activities, wether an active fear producing

motor excitment and flight or a passive fear producing freezing, are controlled by the sympathetic system, whereas fear antidrive producing calmness and relief is controlled by the parasympathetic system. Further experiments should be performed in order to verify this hypothesis.

On the basis of this concept we should assume that another bradycardic effect, namely deceleration of the heart rate observed in the intertrial intervals in the late periods of training, also depends on the inhibition of the sympathetic system since it was abolished after sympathectomy. This means that during this interval the animals did not relax but on the contrary were frozen in expectation of the imminent CS and the shock.

The last question which arises is why in sympathectomized cats a positive CS elicits tachycardic conditioned response, and why acceleration of heart rate occurs as a response to shock. This question is very difficult to answer without additional experimentation. It seems that perhaps the most probable hypothesis is that suggested in our earlier discussion, namely that the heart is sensitized to cathecholamines due to the severance of the sympathetic nerve. This would also explain the high level of the heart rate throughout the experimental sessions.

SUMMARY

Four normal cats and six cats with surgical denervation of the sympathetic cardiac nerve were submitted to training in both positive and inhibitory classical defensive CRs. Normal animals exhibited a bradycardic conditioned response to the positive CS, while operated animals failed to establish this response. However, both groups of animals presented a decrease in heart frequency in the 10 sec following inhibitory CSs. Basal heart rate recorded in the intervals, decreased after 30 sessions of training in the normal group, but not in the operated group. The autonomic mechanism involved in the regulation of the cardiac defensive conditioned reflex is discussed.

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EFFECTS OF ALIMENTARY REFLEXES ON MOTOR GASTRIC ACTIVITY

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In his analysis of the central mechanism of the alimentary reflex Sołtysik (1960) separated its two phases denoted as the drive reflex and consummatory reflex. He further suggested that the cosummatory food reflex inhibits the hunger drive reflex, a hypothesis allowing one to understand the reinforcing character of food intake in instrumental conditioning according to the drive reduction theory. This concept found further support both in old and new experimental data (cf. Konorski 1967). Among other things it was shown by Ellison and Konorski (1965) that it is possible to separate completely the instrumental food response depending on the hunger drive, and the conditioned salivary response; the latter being an element of the food consummatory reflex.

So far the only clear and unambiguous indicator of the alimentary hunger reflex was provided by the occurrence of the instrumental response trained in alimentary conditioning. Therefore it was desirable to look for an autonomic indicator of that reflex which would be, however, more specific than the heart rate or respiratory responses. It was thought that the well known phenomenon referred to as gastric hunger contractions might serve this purpose.

The phenomenon of hunger contractions of the empty stomach was discovered at the turn of this century by several authors both in animals (dogs) and man (for references see Carlson 1916, Cannon 1929,

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Galperin 1960), and thereafter those were the subject of many investigations. Among other things a striking fact was soon revealed showing that these contractions are immediately inhibited when the subject takes tasty food into the mouth, a phenomenon referred to as "receptive relaxation" of the stomach. This fact strikingly confirms Sołtysik's idea on the inhibition of the hunger reflex by the food consummatory reflex.

Unfortunately there are only few studies in which hunger contractions were examined under the rigorous schedule of CR experiments. In an extensive experimental study on dogs performed by Galperin and Rubinov (1968) the following interesting facts were found: the CSs signalling presentation of the liquid food produce a conditioned response being a full replica of the coresponding unconditioned response, that is, a complete relaxation of the stomach. On the other hand, both eating of bones and the appropriate CS produce increase of the tonus of gastric musculature and/or waves of stomach contractions. This seems to show that whereas the intake of liquid food has a purely consummatory character, the intake of bones does not inhibit the hunger reflex and it can even facilitate it.

The purpose of the present study was to yield further information on the behavior of the stomach muscles during various phases of alimentary activity in the routine CR experiments.

MATERIAL AND METHODS

The experiments were performed on three male mongrel dogs 12—14 kg of weight, called Fafik, Morus and Dudek. In aseptic conditions and under Nembutal anaesthesia a gastric fistula was made, the walls of which were formed from the fragment of colon traversum according to the Glavcheva method (1964). A few weeks after surgery the daily experiments in the standard Pavlovian radiophonized CR chamber were initiated.

First, the animals were fully accustomed to stay quietly on the Pavlovian stock in a standing position, being on a leash and in the harness. They learned to take food from the bowls put into position by remote control in a feeder 16 cm high situated in front of the animal. Each portion of food consisted of 50 g of dry biscuits, in cubes of the size of about 8 cm^3, mixed with minced boiled meat. They were also accustomed to the procedure of recording stomach activity. For this purpose the animal's stomach was carefully rinsed by water at room temperature before he was brought to the experimental chamber and all the remnants of food left after the previous feeding were removed. After the animal was placed on the stock, a rubber balloon was introduced into the stomach filled with air and connected with the recording system located in the pre-chamber on the experimenter's desk. The experimenter observed the dog through a one way window. After each session which lasted usually from 30 to 60 min, the dog was taken to the animal house and after 2 to 4 hr fed with his routine meal (cereal with broth and meat). Accordingly each session took place after 18—22 hr of fasting.

A few weeks after the operation, the CR experiments began and lasted for

about two months. The training sessions consisted of four trials separated by about seven minutes intervals. In each trial the beating of a metronome, 80 beats per min lasted for one minute. After 50 sec of its operation the food was presented, the act of eating continuing for 30—60 sec.

When the animals were well trained two kinds of trials were additionally given. Occasionally, the CS—US interval was protracted to two or three minutes and/or the CS was presented without food reinforcement.

RESULTS

Prefeeding activity of the stomach. In the first experimental sessions, when the dog was placed on the stand in the CR chamber, his gastric motor activity was almost nil. Only very rarely could stomach contractions be observed (Fig. 1A). However, after several sessions, the picture changed radically, since the gastric activity appeared almost immediately after the dog was brought to the experimental situation. This activity

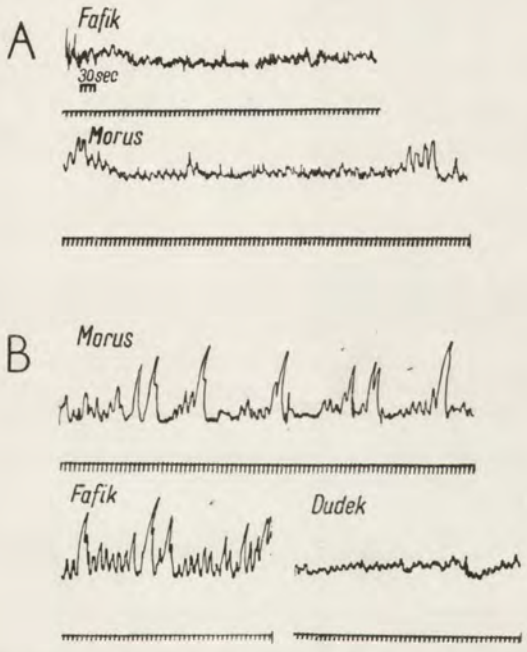

Fig. 1. A, Typical records of motor activity of the empty stomach in a "neutral" situation for individual dogs. The small oscillations of the frequency of about 15—20 per minute are due to respiratory artefacts. In both records only a few true stomach contractions are observed. B, Typical records of rhythmic contractions of the empty stomach in a regular alimentary CR session before the first trial. Note that in Morus and Fafik slow frequency oscillations of large amplitude are seen: in Dudek fast frequency oscillations of small amplitude are seen, but the tonus of the stomach is increased as may be concluded from Fig. 2E. Time marked in 10 sec intervals

had a twofold character. First, it consisted of phasic contractions of the stomach muscles with varying amplitude and a frequency of about two to four per minute; the lower the frequency the larger the amplitude of

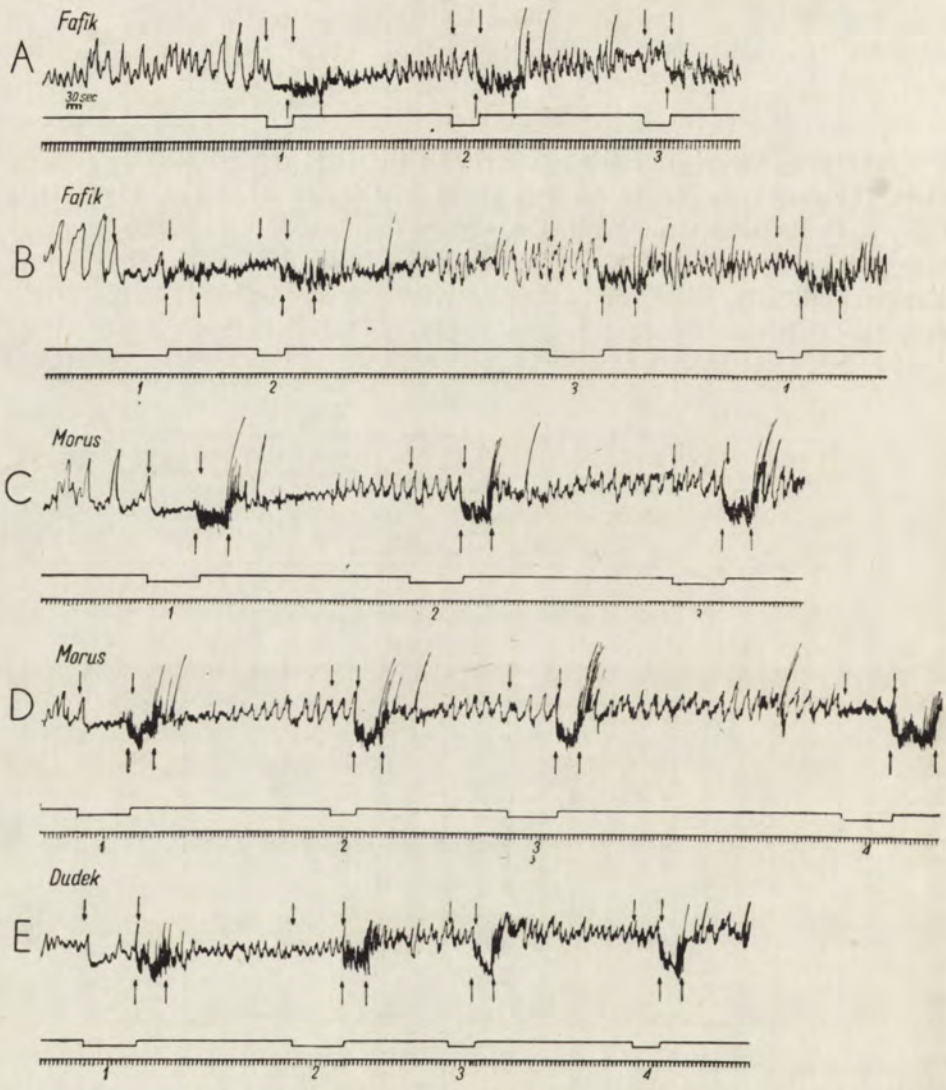

Fig. 2. Records of motor activity of the stomach in particular fragments of CR sessions. From top to bottom: kymographic records of stomach activity; the operation of the metronome; time marked in 10 sec intervals; numbers below time recording indicate the number of successive trials in the session. The arrows above the record indicate the note, the onset, and the termination of the sound of the metronome; the arrows below the record indicate the note, the onset and termination of the act of eating

oscillations. Secondly, the stomach activity consisted in an increased tonus of the musculature; against its background the small oscillations of the frequency of four to five per minute were observed (Fig. 1B). Whereas in Fafik and Morus the former type of activity prevailed, in Dudek almost exclusively the latter type was observed.

The stomach activity during the food intake. Without exception the whole period of food intake was manifested by relaxation of the gastric tonus with some fast artefacts arising from breathing or chewing movements. This relaxation is manifest in all our figures. In most instances the level of the record during the act of eating was either flat (as in Fig. 2A, trial 1, Fig. 2C trial 1) or concave (as in Fig. 2D, trials 2 and 3, Fig. 2E, trials 3 and 4). In one dog (Fafik) stomach contractions of the usual frequency were often observed against the background of a decreased tonus (as in Fig. 2A, trials 2 and 3, Fig. 2B, trials 3 and 4).

Stomach activity in the intertrial intervals. Immediately after the act of eating was terminated the tonus of the stomach increased either abruptly or gradually. Then after various periods of time the waves of the stomach contractions reappeard. Their amplitude was at first small, but, if the next trial was delayed, it grew larger, (see Fig. 2A, after trial 1, Fig. 2C after trial 1, Fig. 3B after trial 2). In most instances both the gastric tonus and the rate and form of waves did not differ from those observed before the first trial (i.e. in the empty stomach), except that the very high amplitude low frequency oscillations were rarely observed. As seen in Fig. 2E the intertrial oscillations in Dudek (in whom they were very small) were exactly the same as they were before the first trial.

Stomach activity in response to the CS. It should be noted that all three dogs manifested a very clear motor conditioned response to the metronome consisting of pricking the ears and turning the head to the feeder. As far as the stomach activity was concerned it was different in the first trial of a given session, that is before eating, and in the following trials when food was already in the stomach.

In the first trial the clear relaxation of the stomach was observed and the record was quite flat (Fig. 2A, C, D and 3C). The difference between the CR record and the UR record consisted only in the lack in the former record of fast artefacts connected with the act of eating. Quite often against the background of relaxation very small rhythmic contractions could be observed (Fig. 2B, E, 3A, B, D). When the CS was protracted, the "escape phenomenon" was present — the larger oscillations and/or the increase of tonus being manifested (Fig. 2B, E, 3B, E, trials 1 and 2).

The behavior of the stomach was quite different in the following trials, given after some food was consumed. In the majority of cases the picture depended on the character of the pretrial period. If the oscillations in this period were large they diminished slightly with onset of the

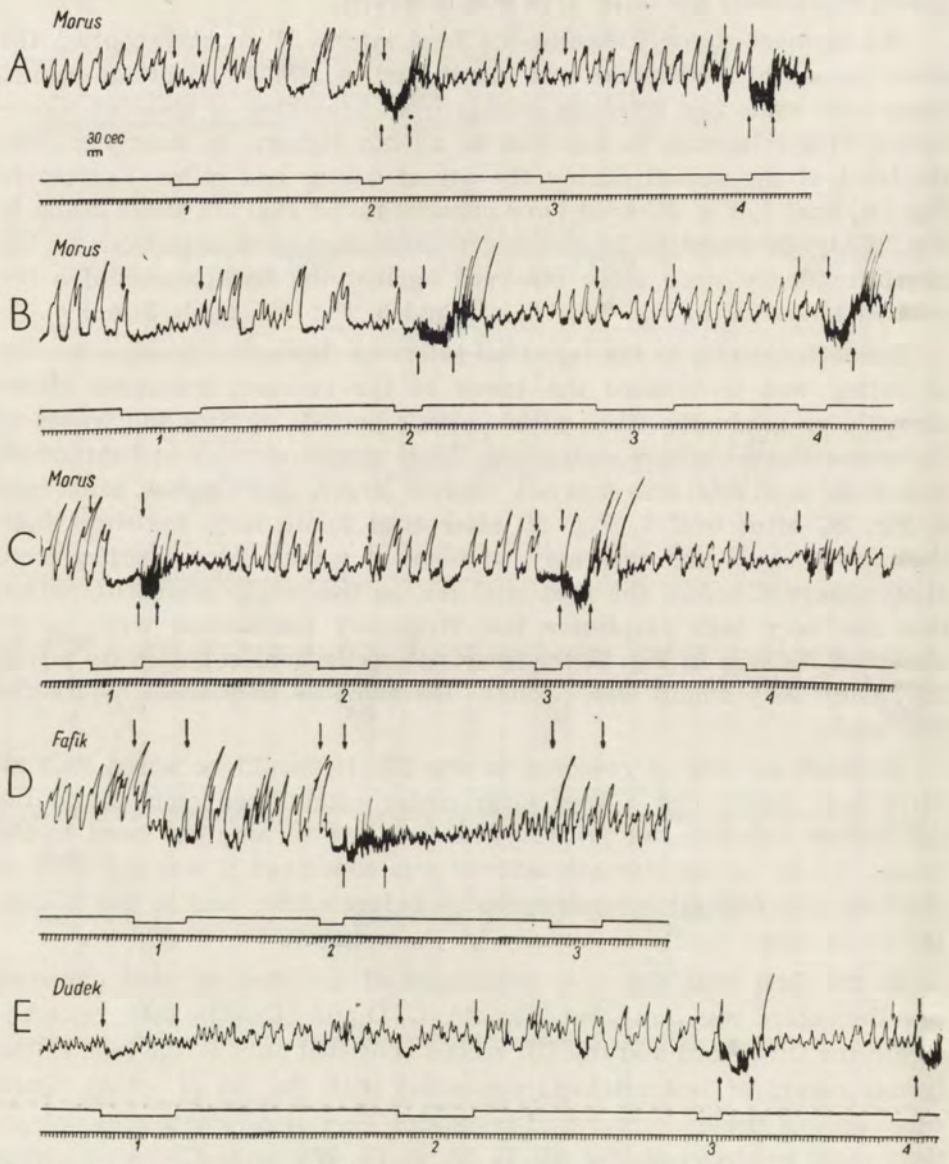

Fig. 3. Records of motor activity of the stomach in the sessions in which the reinforcement was occasionally omitted. This is seen by the lack of arrows below the record of gastric activity. In the unreinforced trials the operation of the metronome is often prolonged to 2 or 3 min. Other explanations as in the Fig. 2

CS (Fig. 2D, trial 4, Fig. 3A, trial 3, Fig. 3B, trials 3 and 4, Fig. 3C, trials 2 and 3) with or without "escape phenomenon"; when they were moderate their increase was noticed (Fig. 2A, trial 3, Fig. 3A, trial 4, Fig. 3D, trial 3). In many cases it was not possible to detect any change of the oscillations in comparison with the pretrial period.

Finally, it should be noted that when a trial was given a short time after a previous feeding, when the stomach was still in a period of inactivity, the CS did not produce any visible effect (Fig. 2B, trial 2).

The effect of non-reinforcement of the CS. As mentioned earlier, when the food CR to the metronome was firmly established, we occasionally presented this stimulus without reinforcement either at the beginning of the session or in later trials (Fig. 3A—E). When the non-reinforced trial was given at the beginning of the session (as in Fig. 3A, B and D) the large oscillations of the stomach were immediately restored and usually they were even bigger than before the CS (rebound phenomenon). The same is seen in Fig. 3C, after the second trial. In the first trial the dog received a very small portion of food. When the oscillations were restored, the metronome presented without reinforcement produced relaxation of the stomach followed by strong contractions after the stimulus was terminated. On the other hand, when the non-reinforced stimulus is presented in a later trial and fails to produce relaxation of the stomach, then there is no increase of oscillations after the stimulus is terminated (Fig. 3B after trial 3).

Finally, we shall describe an interesting result obtained in Dudek, a dog which never displayed large oscillations in stomach activity. As seen in Fig. 3E the session began with double presentation of the metronome for three minutes without reinforcement. Both times the relaxation effect, with later "escape phenomenon" was observed. However, as an after-effect a gradual increase of the amplitude of oscillations took place, ending with large slow waves — a phenomenon never before observed in this dog.

DISCUSSION

As was hypothesized by Sołtysik (1960) and Konorski (1967), the alimentary behavior of the animal, both unconditioned and conditioned, can be regarded as a continuous interplay between hunger drive and consummatory reflexes. We can ascertain on the basis of our present experiments that the stomach motor activity reflects this interplay very precisely.

If the animal is brought to a given situation to which it is habituated and where he does not receive any food, his stomach is almost comple-

tely still, although he was not fed for a number of hours (Fig. 1A). But after the animal was fed several times in this situation, slow contractions of the stomach appear immediately after he is placed in it (Fig. 1B). These contractions are the manifestation of the hunger CR established to the situation.

Whereas the situation in which the dog receives food becomes a hunger CS, the stimulus immediately preceding the presentation of food becomes a consummatory CS (Konorski 1967, Chapter VI). This is documented by the fact that this stimulus in our experiments, as well as in those of Galperin and Rubinov (1968), produced a relaxation of the stomach similar, to, or even identical with, the relaxation produced by food intake.

When after the presentation of the CS, food is not given to the animal, the large waves of contractions immediately reappear, being sometimes even higher than before its presentation. Here the experiment with Dudek (Fig. 3E) is particularly illustrative. This return or augmentation of contractions is in good agreement with the fact that non-reinforcement of a CS increases the hunger CR (Konorski 1967, Chapter IX). Sometimes, if the operation of the CS is prolonged, the "escape phenomenon" occurs consisting in gradual increase of the contractions; thus the consummatory CR is gradually replaced by the hunger CR.

We should now turn to a discussion of another fact, regularly obtained in our experiments, which does not seem to be so obvious as those findings which we discussed above. This is the complete change of the conditioned response of the stomach to the CS, when it is presented not at the beginning of the experimental session, before food intake, but after one or a few reinforced trials. In these cases the conditioned relaxation of the stomach to the CS is rarely seen, because usually we observe either no change in rhythmic contractions of the stomach, or even some increase of their amplitude.

How are these facts to be explained?

The food presented to the animals in our experiments had a mixed character: the minced meat provided a direct taste stimulus to the oral cavity, whereas the dry biscuits provided at first rather tactile stimuli, which only after thorough chewing, accompanied by salivation, were converted into taste stimuli. In consequence, we have reasons to believe that even during the very act of eating the hunger drive was not fully inhibited (as is the case when eating liquid or pulpy food), but it remained, facilitating strong mastication. This suggestion is confirmed by the fact that in some of our records clear rhythmic contractions of the stomach were seen during the act of eating against the background of relaxation of the stomach (cf. Fig. 2A, B). If it is so, we may assume that

the CS signalling the consumption of such food can also produce a double effect, namely both consummatory and/or hunger response. The fact that, after one or a few portions of food were already eaten, there was an increase of the hunger CR and weakening of the consummatory CR may be due to the postconsummatory increase of hunger postulated by Konorski (1967, Chapter I). It should be also noted that long CS—US intervals (amounting at least to 60 sec) used in our experiments favored the hunger CR and disfavored the consummatory CR (cf. Konorski 1967, Chapter VI).

An alternative hypothesis could be that when food is in the stomach, inhibition of the gastric contractions is more difficult than in the case when the stomach is empty.

The next problem to be discussed, a much more controversial one, is this. It is generally accepted that the so called hunger contractions appear only when the stomach is empty. On the other hand, when food is in the stomach, peristaltic movements come into being whose character, although somewhat distinct from the hunger contractions, has some features in common. In most studies on animals and man concerning "hunger contractions" only the empty stomach was used because either of oesophagotomy or of the block of oesophagus due to disease. Such a procedure is not quite reasonable if we take into account the fact that a subject, after consuming a small portion of food, is still hungry.

We could establish in our experiments, in which each portion of food freely passed to the stomach, that exactly the same waves of contractions or the same tonic contractions which appeared before the first feeding, when the stomach was empty, were observed after the second or the following feedings. The only difference was that whereas in the empty stomach there appeared waves of various amplitudes up to the very high ones, the latter were hardly seen after feeding. But even that was not the general rule, since, as we see in Fig. 3C, when the portion of food offered to the animal was small, the high amplitude waves appeared as a rebound after an unreinforced trial.

There are two possible explanations of these findings. For one thing the postfeeding contractions may be regarded as "hunger contractions", that is, they are dependent on the hunger drive in the same way as are the contractions of the empty stomach. After all, the animal after eating the first or the second portion of food is not less, but even more hungry (in the sense of hunger drive) than before eating, thus it would be completely incomprehensible why the hunger contractions should disappear, taking into account that these contractions are the result of hunger drive and n o t of the stomach being empty.

For another thing it should be realized that the phenomenon of hunger contractions of the empty stomach is somewhat mysterious and its physiological function is far from being clear. Therefore we can assume that these contractions are nothing else but ordinary *peristaltic* contractions which appear in the empty stomach in *anticipation* of taking food on the same basis as salivation appears before the consummatory feeding act. Therefore, it may be claimed that there is no principal difference between the "hunger contractions" and peristaltic contractions, as there is no significant difference between the conditioned and unconditioned salivation.

It should be noted that both these views are roughly equally represented in the literature. Of course, both Carlson (1916) and Cannon (1929) regarded "hunger contractions" as a phenomenon sui generis quite separate from the peristaltic contractions. However, as early as in 1915 Rogers and Hardt carefully analysing the contractions of the empty stomach and those of the filled stomach did not regard them as separate events, considering "hunger contractions [to be] powerful peristaltic contractions, which, arising at or near the cardiac sphincter, swept downward over the entire stomach".

The same discrepancy of views may be seen in the contemporary literature. For instance Chu, Jen-Pao et al. (1961) stress the difference between rhythmic contractions in the empty stomach and those in the filled stomach, considering them separate processes. On the other hand, Bogatch (1965) and Starcev (1961), after a thorough analysis of both types of contractions come to the conclusion that they cannot be regarded as separate phenomena.

The advantage of the second hypothesis over the first one is that it explains reasonably the origin of the hunger contractions. However, its drawback is that it cannot explain the relaxation of the stomach during the consummatory reflex (both conditioned and unconditioned), a fact which is fully understandable in view of the first hypothesis.

It seems that it is possible to prove which of these two hypotheses is correct by a more detailed analysis of the type of contractions occurring when the stomach is empty and when it contains small portions of food producing no satiety.

Irrespective of which of these two hypotheses is correct, the large amplitude of contractions in the empty stomach should be simply attributed to the fact that they appear against the background of its full relaxation. In fact, it may be seen that the valleys of these contractions are exactly at the same level as that observed during the act eating. The higher the tonic contraction of the stomach the lower is the ampli-

tude of the phasic contractions; this thesis is true with regard to both the empty stomach and the stomach partially filled with food.

Our concept of the mechanism of alimentary activity (cf. Konorski 1967, Chapter I) implies the assymetrical relation between the taste center and the hunger center. Whereas the activation of the taste center (due to the taste stimulus or its conditioned signal) inhibits the hunger center, the activation of the latter, on the contrary facilitates or even excites the taste center. This being so, it is easy to prove that the rhythmic activity of the hunger drive center, reflected in hunger contractions, is intimately connected with the structure of the system, because the higher the activation of the hunger center, the stronger its positive influence upon the taste center, which leads to its inhibition. Speaking psychologically, when a subject is very hungry, he may easily imagine a tasty food, begin to salivate and in this way temporarily suppress his hunger. This, however, leads to the cessation of the activation of the taste center and to the re-activation of the hunger center.

SUMMARY

1. In three dogs subjected to routine alimentary CR experiments the motor activity of the stomach was observed throughout each session. For reinforcement there served portions of food, 50 gr in weight, composed of small cubes of dry biscuits mixed with minced boiled meat.

2. After a number of trials a fasting dog brought to the CR chamber manifested either rhythmic motor activity of the stomach of large amplitude and low frequency (so called hunger contractions) or increased tonus with small rapid waves.

3. The food intake procedure produced the relaxation of the stomach and the disappearance of the rhythmic activity; however, it happened occasionally that the rhythmic activity was present against the background of a low tonus.

4. After the act of eating the rhythmic activity was absent for some time and then gradually emerged, resembling the activity in the prefeeding period. The only difference was that the oscillations of very high amplitude and low frequency failed to occur. The tonus of the stomach rose after the act of eating, either abruptly or gradually.

5. During the operation of the CS in the first trial there occurred the relaxation of the stomach and the disappearance of high oscillations. The low oscillations were either absent, the record being flat, or they were present. Occasionally, with the protraction of the operation of the CS the "escape phenomenon" was observed — the oscillations gradually

growing larger. When the CS was not reinforced, the high oscillations returned immediately, usually even larger than before the presentation of the CS.

6. In the following trials (that is after the dog ate some food) the stomach motor activity was variable: if in the pretrial period the contractions were high, they usually diminished, but when they were low, they rather increased; in some cases there was no change in the motor activity of the stomach.

7. All the results obtained are discussed with reference to the interplay between the hunger drive reflexes and consummatory food reflexes.

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LATERALISATION OF EFFECTS OF ACUTE EXTINCTION OF AN ALIMENTARY INSTRUMENTAL RESPONSE

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The effects of training of various behavioral responses have been shown to be capable of being lateralised in one hemisphere in some cases and in others not. That is to say, if an animal has one cortical hemisphere (more particularly, neocortex) non-functional during conditioning with the other intact, restoration of function may show no evidence of such conditioning when testing is confined to that hemisphere. For example, an avoidance reaction to shock may be so lateralised (Bureš and Burešova 1960, 1968, Myers 1956) as may a food-reinforced lever press (Russell and Ochs 1961), whereas a classical conditioned reflex may have a neural substrate elaborated in both hemispheres (Bureš and Burešova 1960, Ross and Russell 1967). All such reports, however, have been concerned with the elaboration of a reflex, usually testing for retention; in this study a particular case of reversal learning where experimental extinction procedures are instituted, was examined.

A further problem which arose out of the findings in those cases where the "engram" or neural trace could be lateralised was concerned with the possibility of interhemispheric transfer. In the case where one hemisphere has been exposed to a given training situation whereas the other has not, at least one trial with both hemispheres functional may be required before the "naive" (untrained) hemisphere can benefit from

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the other side's training. Thus "passive transfer" rarely occurs, and the phenomenon of "one-trial learning" is commonly reported. It was desired to examine this possibility in the extinction situation.

Most studies employing hemilateral preparations have used aversive stimulation. The relatively greater ease of extinguishing a positively-reinforced response, coupled with the lack of literature on this type of situation determined the choice of food as the drive stimulus.

Cortical spreading depression of Leao (SD) has been employed in various behavioral situations as some approximation to a functional reversible decortication. It is questionable whether the effects of SD (evoked by topical application of 25% KCl, for example) are analogous to those of surgical ablation (Bureš and Burešova 1960, Bureš et al. 1967), the invasion of subcortical structures in the case of SD being highly probable to a limited extent (Bureš and Burešova 1968). However, behavioral and electrophysiological evidence indicates that SD certainly produces a functional cortical deficit which is reversible in the treated hemisphere of a lissencephalic brain. This technique is therefore an interesting tool in its own right for the elucidation of the role of the cortex in learning situations, and of the conditions under which transfer of the effects of training from one hemisphere to another may occur.

METHODS

30 male albino rats, 90—120 days old at the time of the experiment, and food-deprived to 80% of their ad lib. feeding weight, were used.

Subjects were hand-tamed and pre-trained to run in a straight alley for pellet reinforcement. Non-retraceable doors were employed, and in order to facilitate their running through these, on the first pretraining trial the doors were open. On subsequent trials, the doors were successively more and more closed, until the rats were passing through closed doors. After criterion was achieved (nine successive runs in ten trials in less than thirty seconds each) surgical procedures were instituted. Two days of extinction training followed, in which no food-reinforcement was available to the animals in the apparatus. The non-retraceable doors operated relays activating counter-timers, thus giving a measure of latency of exit from the start-box and running time in the alley. These measures were noted form the moment when the doors were sufficiently closed to operate the relays.

Apparatus. A straight runway 1.5 m long and 10 cm wide by 20 cm high connected two boxes 40×25×20 cm. The sides of the runway and start and goal boxes were opaque bakelite, the floor being 1 cm diameter. Dinsmoor bars at 2 cm centers, over which was a wire mesh floor of 8 cm^2 lattice. The start box had a hinged lid the closing of which activated a relay and triggered two panels of a counter-timer. Between the start-box and the runway was a non-retracing door which, when opened, opened a second relay, switching the pulse activating one panel of the counter-timer to a third panel — thus a measure of the latency

of exit from the start-box was obtained. At the junction of the runway and goalbox another non-retracing device operated a relay so that the remaining two panels were switched off (registering alley running time and total, latency plus alley, running time). The pulse now operated a timer to allow ten seconds for eating. Thus measures of latency, limb running time, and total running time, were registered. The entry door to the goal-box also when operated activated a pellet dispenser which discharged two pellets into a food-cup at the far end of the compartment.

Surgery. Surgery was performed under light ether anaesthesia. Animals' scalps were clipped, and a midline incision performed. The skin and muscles were retracted, the periosteum removed, and trephine holes (5 mm diameter) made between the Lambda and Bregma sutures and 1 mm lateral to the midline, care being taken to ensure that the dura mater remained intact. The wound was then sutured and the animals allowed to recover for twentyfour hours, when, under very light ether anaesthesia, the incision was reopened, the wound revised, and 3 mm diameter pledgelets soaked in 25% KCI placed directly on the dural surface over the desired hemisphere. After testing, the pledgelets were removed, the dura thoroughly washed with Ringer's, the sides of the incision loosely sutured together in order to facilitate revision before the second day's testing, and antibiotics administered. All surgical procedures were carried out under aseptic conditions. After experiments animals were routinely examined for neurological damage.

Procedure. Animals were weighed daily throughout the experiment; their food intake being adjusted to keep their weight at the desired 80% ad lib figure. Water was freely available in their home cages. The paradigm of the experiment is illustrated in Fig. 1.

Thus, group E1 had bilateral function during acquisition, and during the two test days had the same hemisphere depressed. Groups E2 and E3 had the con-

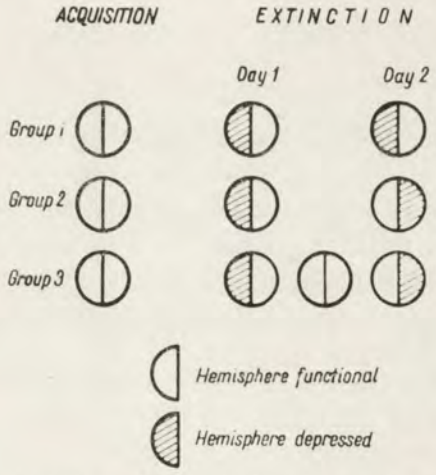

Fig. 1. Experimental paradigm. Each circle represents a cerebral hemisphere. Hatched circles indicate the presence of spreading depression. Open circles represent functional hemispheres. In group 3 one trial with bilateral function was given before the second day's testing. Controls are not diagrammed

tralateral hemisphere depressed on the second test day, and Group E3 and one extinction trial before the second day's testing with both hemispheres functional. Sessions were held daily throughout training-testing, and there were ten trials per session. At the beginning of a trial the animals were placed in the start-box, care being taken to ensure that this was carried out in as constant a fashion as possible. During acquisition and extinction, the animals were allowed ten seconds after entry of the goal-box to consume the pellets, two of which were supplied on each trial, during pretraining this was extended upwards to thirty seconds. After each trial the animal was returned to the transfer cage, and after each session returned to his home cage. The inter-trial interval was two minutes; the whole procedure for a group of ten rats taking some thirty to forty minutes. Animals were fed some hours after training. After each trial latency of exit from the start-box and running time in the alley were noted.

Controls. Since the comparison of interest in this study was between two groups where treatment was identical except for the fact that on day one of extinction one group (E1) had the same hemisphere depressed as on day two, whereas the other groups (E2 and E3) had the opposite hemisphere depressed on the second day, and since it was found in an earlier study (Rentoul 1968) that the surgical interference produced in the open preparation had no measurable effect on the received indices of performance either during acquisition once the habit was established or during extinction training (vis a vis unoperated controls) surgical controls are not reported on here.

To control for the possible effect of differential hemisphere preference, in all experimental groups one half were depressed on the left side on day one and one half on the right. Groups E2 and E3 were of course depressed on the opposite hemisphere on day two of extinction, that is, those animals depressed on the left hemisphere on day one were depressed on the right on day two and vice versa.

Monitoring of SD during testing was done by means of taking placing reflexes.

RESULTS

When a hemilateral animal was placed in the maze after bilateral training, he characteristically displayed some hesitancy expressed in the measures taken as a slightly longer running time. This would disappear by the third trial, when shorter than acquisition running times and latencies might be registered. Extinction procedures would begin to "bite" by the sixth or seventh trial on the first test day in all groups, the same being true for groups two and three on test day two.

A criterion of extinction was imposed during testing. If after 30 sec an animal had not left the start-box, he was removed. On test day two many of the group one animals had extinguished to this criterion, which therefore had the effect of reducing their total running-times on that day (Fig. 2).

The first question of interest is whether the animals' behavior on test day one is comparable to that on the last day of acquisition. The

mean running times at the end of acquisition were of the order of 2 sec, whereas for all groups this figure was approximately four times as large on the first day of extinction (Fig. 2).

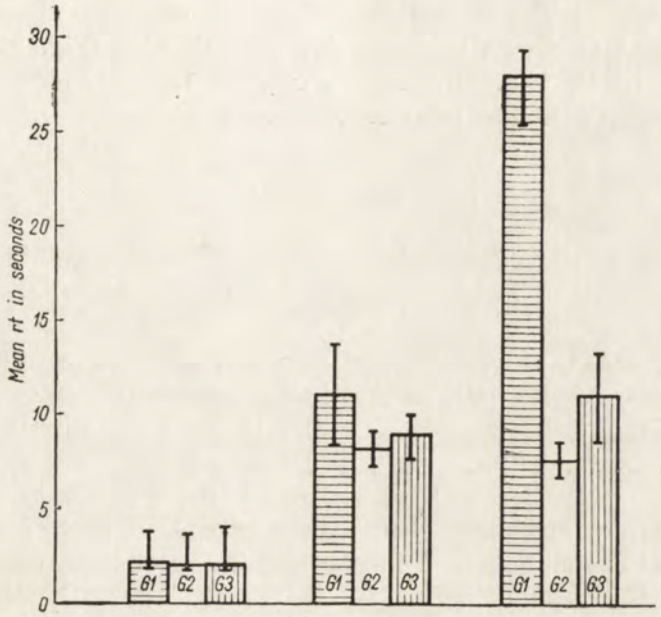

Fig. 2. Extinction data. Mean running time for each group is presented for the last day's traning and for the two days of extinction testing. The range is indicated by the vertical lines. G1, ipsilateral hemisphere depressed on both test days. G2 and G3, opposite hemisphere depressed. G3, one interpolated trial with bilateral function before test day two

The experimental design, however, does not permit one to attribute this to SD alone or the initiation of extinction procedures, or their interaction, since this was not the point at issue.

Comparing the three groups on test day one indicates that the data is still homogeneous; there being no statistical significance in the received differences. However, on day two this is no longer the case. Group one, which had the same hemisphere depressed on both test days shows quite clearly on the second day the effects of the first, their mean running times for the last ten trials being of the order of 25—30 sec, whereas in the two groups where a naive hemisphere was exposed on test day two, the received indices are entirely comparable to the previous test day. This can be evaluated by a comparison of the results of groups one and two on the second day of extinction, the difference in results being significant at the 0.001 level (Student's T test).

The effect of interpolating one trial with bilateral function was studied in group three; the trial preceding the second test day, A slightly higher mean running time than in group two was recorded; this was, however, without statistical significance. Indeed, the results of this group on the second day of extinction were almost identical with the first group on the first day of extinction (Fig. 2).

The results for latency and alley running time examined separately, were comparable to the total running-time data.

DISCUSSION

As described above, lateralisation of the effects of training in various behavioral situations has been reported in the literature, although there are no studies of the effects of unilaterally evoked SD during extinction procedures. This is of some theoretical importance for learning theorists, and the above results indicate that in some respects extinction procedures represent for the organism another learning situation analogous to others, at least in the property that the neural "trace" of such learning, if acquired by one hemisphere, will not transfer passively across commissural fibres. That is, a hemisphere placed in such a situation is "naive", and cannot benefit from the training received by the other hemisphere. It has been demonstrated that such a naive hemisphere may perform at the level of the "trained" hemisphere if only one trial with bilateral function is given, and this has been termed "one trial learning". It is undeniable that it is one-trial learning of a sort; however, since the initial training of the other hemisphere took many more trials than one, and since it is reasonable to assume that, without access to this trained hemisphere, the naive hemisphere would require about as many (indeed, where the possibility of one such bilaterally functioning trial is excluded, the data show precisely this); it might be more precise to term this effect "one-trial commissural retrieval" or "one-trial access".

Ross and Russell (1964) have shown that in some situations this one-trial transfer may not be complete, but that it does not occur at all unless the trial in which both hemispheres are functional is reinforced. It is arguable whether one may say that in extinction reinforcement is absent; certainly the reinforcement that motivated the original learning is being withheld, but the alteration in behavior consequent upon instituting extinction procedures must have antecedent stimulus "reinforcing" conditions, and these stimuli are of course those associated with the absence of food. The results of group E3 indicate no such transfer with one trial and both hemispheres functional, and one must conclude that the reinforcement for extinction training — the stimuli associated with

the withholding of pellets — cannot be equated with the positive reinforcement supplied in a situation such as food-reinforced bar-pressing, since the effects of such reinforcement on measureable behavior differ.

Viewed as a particular case of learning, extinction might be termed as a special type of redundancy learning, where the animal learns that a response in its repertoire that was previously significant (reinforced) is so no longer, and is in this respect analogous to habituation, where a stimulus initially eliciting a response comes not to do so. The physiological substrate of extinction will no doubt depend on the analysers involved in the specific task employed, and on the reinforcement history of the subject, as well as on more basic variables such as arousal. That is, habituation is a more strictly physiologically passive process with a different time-course, whereas extinction is in this respect a form of "non-contingent" learning analogous to an operant response. In the normal operant situation the subject learns that reinforcement is contingent upon a given response; in extinction the subject is required to learn that such reinforcement is no longer so contingent. Thus, extinction remains a distinct psychological category requiring independent theoretical treatment.

SUMMARY

Thirty rats were run in a straight alley for food reinforcement, and after reaching criterion were subjected to two days extinction training. All rats had one hemisphere depressed during extinction sessions. Group 1 had the same hemisphere depressed on both extinction sessions, whereas Groups 2 and 3 had the opposite hemisphere depressed on the second day. Group 3 had one extinction trial between both test days with bilateral function. The results indicate that exposure of one hemisphere to extinction sessions has no effect on performance mediated by the other, when subsequently exposed to the same experimental situation, and that one unreinforced (extinction) trial with bilateral function is not sufficient to precipitate passive transfer.

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DIFFERRING EFFECTIVENESS OF AUDITORY QUALITY AND LOCATION CUES IN TWO FORMS OF DIFFERENTIATION LEARNING

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In the initial series of experiments concerned with the go left-go right task it was found (Ławicka, unpublished experiments) that normal animals had difficulty with the choice of the correct response if these were signalled by two different auditory stimuli (a buzzer and a metronome) presented from the same locus. The failure of the animals to perform this task could be avoided if the stimuli were spatially separated. If however, under these experimental conditions the location of the stimuli was interchanged, then it appeared that the animal's response was determined by the stimulus location and not by its auditory quality. This striking fact deserved additional analysis since it is well known that Pavlovian go-no go differentiation may be based on very fine differences in the quality of auditory stimuli. The present experiment proceeds with further analysis of this finding.

MATERIAL AND METHOD

Experiment I

Go left-go right task. The Ss were 8 naive mongrel dogs trained in a rectangular room with two food dispensers situated on the side walls of the room. The starting platform was in the middle of the room at 2 meter distance from each food dispenser. Every food dispenser contained bowls with food, which could be moved

into position by remote control by the experimenter, who was sitting at a small table behind the starting platform. In this experiment two auditory test stimuli were used, each of them signalling food in one of the two available foodwells. Whenever the test stimulus was on, the animal was required to approach the correct foodwell.

In the first stage of preliminary training the test stimuli were not used and the animals learnt simply to approach the foodwell in which the food was delivered. Every delivery of food produced an audible click coming from the baited foodwell and the animals learned very quickly to associate it with the presence of food, approaching the foodwell from which the click was heard. In every preliminary session, food was thus delivered 18 times, 9 in the left and 9 in the right foodwell, presented in random order. If an animal made 18 correct runs within the session, on the following, day a pair of test stimuli was introduced.

These were two auditory stimuli, one of them signalling food in the left, and another in the right foodwell. The stimuli were presented in 1 min intervals and were followed by delivery of food in the appropriate foodwell; on the first 3 days food delivery was delayed for 3, 4, and 5 sec respectively, whereas on the next successive 3 days for 10, 15 and 20 sec respectively. Usually, the effect of this procedure was that the animals started approaching the foodwells as soon as the test stimuli were on, without waiting for the click coming from the baited foodwell. Whenever the animal approached the correct foodwell, food was immediately presented and the stimulus turned off. If the animal made an error, the stimulus was discontinued, no food was presented and the stimulus presented again after the usual 1 min interval (correction run). In the case the animal did not correct his error in the three successive extra-runs, the stimulus was repeated once again, and in 2 sec from its onset the food presented in the correct foodwell. Thus, in the case of three successive extra-errors, running to the sound of a click ended the trial.

Every experimental session consisted of 18 rewarded trials, food in each foodwell being presented in random order in 9 trials. The trial was scored as correct whenever the animal chose the correct foodwell at the first presentation of a stimulus. If the animal chose the wrong foodwell, the response was scored as an error. The number of erroneous extra-runs was scored as number of extra-errors.

If it happened that at the first stimulus presentation of a given trial the animal did not approach a foodwell but remained on the starting platform, 20 sec after the stimulus onset the food was delivered in the correct foodwell and the trial scored as "passive". Only those trials are included in the number of daily trials in which the animals made an active choice response approaching one of the foodwells.

Two gorups of dogs were trained according to this procedure. In group I two different tone frequencies, 300 c/sec and 1500 c/sec, were used as the test stimuli. The tones were presented from a single loudspeaker situated at 2 m from the starting platform. This group will be further referred to as the frequency group. In group II only a single tone frequency, the 300 c/sec, was used, presented from one of two (up- or down-) loudspeakers, differring in location. One loudspeaker was located on the floor of the starting platform and another 1.2 m above it. This group will be further referred to as the location group.

Five dogs in the frequency group (no. 1—5) and three dogs in the location

group (no. 6—8) were trained until they reached a criterion of 90% correct responses in 90 trials (5 successive experimental sessions). If an animal failed to reach criterion within the set limit of 50 sessions, training was terminated.

Experiment II

Go-no go task. The Ss were 10 naive mongrel dogs trained in the same experimental room as the two previous groups from Experiment I, but in this task only a single food dispenser, situated on the left side wall of the room, was used. Two auditory test stimuli, one positive and one negative, were used. The animals were required to approach the foodwell to the positive stimulus and to refrain from this response if the negative stimulus was presented. Approaching the foodwell to the positive stimulus was immediately rewarded by food, whereas the same response made to the negative stimulus was never followed by food delivery.

At the beginning of preliminary training the test stimuli were not used and the animals learned to approach the foodwell to the click associated with delivery of food which was presented 9 times during the session. After an animal made 9 responses to the successive clicks in a single session, on the following day the positive stimulus was introduced. The positive stimulus was presented 9 times during the session and the food delivered after its onset: on the first day after 3 sec, on the second and third — after 4 and 5 sec respectively. Whenever the animal approached the foodwell in response to onset of the positive stimulus, the food was immediately presented and the stimulus turned off. If the animal made 9 successive responses to the tone alone with a latency not exceeding 5 sec, on the following day the negative stimulus was introduced. Starting with this day 9 positive and 9 negative stimuli were applied in random order. The stimuli were applied with 1 min interval. The duration of the negative stimulus was always 5 sec. The duration of the positive stimulus depended within 5 sec on the response latency. If the animal did not approach the foodwell within this limit, food was presented to him 5 sec after the stimulus onset. Thus, in this case the response to the click ended the trial. Due to this procedure every presentation of the positive stimulus was followed by a food reward.

The positive trial was scored correct whenever the animal approached the foodwell to the positive stimulus alone; if the animal did not approach the foodwell until the click was heard, the trial was scored as an error in the positive trial. The negative trial was scored as correct, if the animal refrained from responding within the 5 sec duration of the negative stimulus. If the animal approached the foodwell during the negative stimulus, the response was scored as an error in the negative trial.

Two groups of dogs were trained according to this procedure using the same two pairs of auditory stimuli as in Experiment I. Group I was the frequency group trained with the tones 300 c/sec and 1500 c/sec presented from a single loudspeaker, and group II was the location group with the single tone frequency 300 c/sec presented from two differently located loudspeakers.

Five dogs in the frequency group (no. 9—13) and five dogs in the location group (no. 14—18) were trained until they reached a criterion of 90% correct responses in 90 consecutive trials (5 experimental sessions). If an animal did not reach this criterion within the set limit of 20 experimental sessions, training was terminated.

RESULTS

Experiment I

The animals performance on the go left-go right task is summarized in Table I. As may be seen, there is a significant difference among the groups in number of trials to criterion ($p < 0.036$, Mann-Whitney U test, two-tailed, Siegel 1956), the location group being much superior to the frequency group. Three Ss in the frequency group failed to reach criterion within the set limit of 50 experimental sessions. One of them (no. 5) developed a very strong alternating tendency remaining at the chance level until the end of testing.

Table I

Number of errors and trials to criterion of individual Ss on the acquisition of the go left-go right and go-no go task

	Experiment I go left-go right task			Experiment II go-no go task		
	Ss	Errors[a]	Trials	Ss	Errors[b]	Trials
Frequency group	1	401	848* (77%)	9	6	18
	2	297	873* (85%)	10	19	54
	3	331	643	11	9	36
	4	196	524	12	8	18
	5	505	839* (48%)	13	5	54
Location group	6	160	354	14	181	360* (47%)
	7	127	230	15	78	270
	8	103	269	16	168	360* (54,4%)
				17	175	360* (50%)
				18	157	360* (54%)

[a] Including extra-errors.
[b] Including errors in positive and negative trials.
* Indicates failure to attain criterion in the stated number of trials (the corresponding percent score is percent of correct in the last 90 trials).

Experiment II

The animals performance on the go-no go task is summarized in Table I. The location group in this task showed significantly lower acquisition score than the frequency group ($p < 0.008$, Mann-Whitney U test, two tailed, Siegel 1956). Four Ss failed to reach criterion in 360 trials.

The single animal (no. 15) who was able to learn took 270 trials to reach criterion.

Five animals in the frequency group reached criterion in 18—54 trials after the maximal number of 19 errors. As may be seen in Table II all Ss in this group made a greater proportion of errors in the negative than in the positive trials. In the location group 4Ss made errors both in the negative and positive trials.

Table II

Number of errors in positive and negative trials for individual Ss on acquisition of the go-no go task in Experiment II [a]

	Ss	CS^+	E_{pos}	E_{neg}
Frequency group	9	1500 (c/sec)	1	5
	10	1500	1	18
	11	300	0	9
	12	300	0	8
	13	300	0	5
Location group	14	Down	164*	17*
	15	Up	8	70
	16	Up	55*	113*
	17	Up	152*	23*
	18	Down	0*	157*

[a] Abbreviations: CS^+, positive stimulus for individual dogs; E_{pos}, errors in positive trials; E_{neg}, errors in negative trials.

* Indicates errors (without attaining criterion) in 360 trials.

DISCUSSION

The results of Experiment I show that the auditory location group needed much less trials to learn the go left-go right task than were required by the auditory frequency group. The much lower learning scores of the animals in the frequency group cannot be explained by their poor discriminatory capacity related to the pair of presented stimuli, since a group of animals from Experiment II trained with the same tone frequencies was able to learn the go-no go task in very few trials.

The data from the go-no go task in the Experiment II show, on the contrary, the superior acquisition of the frequency group to the location group demonstrating that all animals in the frequency group were able to reach criterion at the time, when the animals in the location group were still showing no progress in learning.

Comparison of the results obtained in Experiment I and II suggest therefore that the learning scores depend not only on the animal's ability to discriminate the stimuli used in training, but also on the task in which they are presented. Thus, as shown by the present study, the auditory location cues, as compared to the tone frequency cues, appear to be much more effective in an experimental situation which requires learning of two instrumental responses both rewarded by food. The tone frequency cues, however, are superior to the location cues in a Pavlovian differentiation task, in which only one form of instrumental response is involved and the animal is required to respond to the positive stimulus reinforced by food, and to refrain from responding to the negative stimulus, never followed by a food reward.

How may one account for this difference? Why is it that these stimuli, otherwise well discriminable by the animals, depending on the task, differ so much in their ability to signal the correct response? Let us consider first the role of the conditioned stimuli in these two different tasks.

As has been shown by Konorski (1939) the animal's behavior in an experimental situation may be considered as a function of both evoking and determinig stimuli, the first being responsible for the instrumental response *to appear*, the second — for *determining* the form of an evoked instrumental response. A lot of experimental evidence suggests that in the go-no go task, the sporadic conditioned stimulus possesses evoking properties only, whereas some other, more constant components of the experimental background are playing the determining role (Konorski 1939, Wyrwicka 1956, 1958, Wyrwicka et al. 1960).

Since in the go left-go right task two forms of instrumental responses are involved, the continuous stimuli of the experimental situation are related to both instrumental responses and cannot play any selective, determining role. In this case each of the two sporadic conditioned stimuli must acquire not only evoking but also determining properties responsible for the selection the correct one from two different responses.

Thus, according to this analysis, the role of the sporadic positive conditioned stimuli in a Pavlovian differentiation task and in the go left-go right task is not the same: although in both cases they possess an evoking role with regard to the trained responses, in the go left-go right task they acquire additionally determining properties, enabling the animal to respond with the *correct* movement.

Considering the physiological mechanism of instrumental conditioning Wyrwicka (1952, 1960) in her theoretical model assumed the existence of two types of functional connections between the central repre-

sentation of conditioned stimulus and instrumental response: the direct CS-R connections, as well as indirect CS-D-R connections, the latter mediated through the drive center (Sołtysik 1960). Since, as has been shown (Konorski 1939, Wyrwicka 1955) the same sporadic stimulus presented in two different experimental situation can, depending on the experimental background, evoke one or another of two different instrumental responses both based on the same drive, it may be assumed that in the go-no go differentiation a positive sporadic stimulus evokes the response only on the basis of CS-D-R connections. The determining role, or the function of the direct CS-R connections in that case, is taken over by experimental background. We may assume further that the negative stimulus in this task evokes no response due to its intercentral connections with the corresponding antidrive (Konorski 1967).

However, in the go left-go right task with two different positive responses based on the same drive, the indirect CS_1-D-R_1 and CS_2-D-R_2 connections cannot ensure the selection of the correct response. Neither can the continuous stimuli from experimental background, since they are related to both instrumental responses. Therefore, learning of the go left-go right task requires the formation of additional direct CS_1-R_1 and CS_2-R_2 links between the sporadic conditioned stimuli and "their" instrumental acts, thus enabling the correct instrumental response to the presentation of CS to appear.

According to what has been discussed above, the positive sporadic stimulus in a go-no go task forms only evoking, or indirect CS-D-R connections, whereas a positive stimulus in a go left-go right task must form both indirect, as well as direct, or determining CS-R connections with respective movement. As demonstrated by the present data, there is a great difficulty in forming the direct CS-R connections if the tone frequencies are used as cues. If, on the other hand, auditory location cues are presented, they acquire the determining properties relatively easy.

In interpreting different rates of learning, Konorski (1948, 1967), in his theory of plastic changes in the central nervous system, assumed that they depend on different strength of potential connections linking corresponding nerve centers. According to this theory, the potential connections are, in the course of learning, transformed into the actual connections.

The data presented in this paper would suggest therefore that the auditory localization cues, i.e. presumably the afferent feedback from turning of the head and eyes towards the sound-sources, possess more potential connections with respect to the motor acts than do the audi-

tory quality stimuli. On the other hand, auditory quality cues possess more potential connections with respective drives, a last condition being crucial for solving the go-no go task.

The difficulty of the auditory location group in acquisition of the go-no go task deserves some additional analysis. In this group the positive and negative stimulus consisted of the same auditory component and two different, one positive and one negative, loci. If we assume that the auditory component forms strong connections with drive, then in the effect of being reinforced only in positive trials (i.e. 50% of presentations) becomes associated both with alimentary drive and its antidrive (Konorski 1967). This might explain why in this group errors both in positive and negative trials were observed.

Since the animals could not solve this task by relaying upon the established associations with the tone, they might still learn it using different components of orienting response towards the source of the positive and negative stimulus. However, since in the negative trials the stimulus is not reinforced by food, it seems probable that with its repeated presentation the orienting response towards its locus becomes extinguished, loosing thus the possibility to mediate the acquisition of the "no go" response.

That the localization of a stimulus does not, indeed, acquire the property of a negative alimentary cue is recently shown by experiments of Szwejkowska (1967). In her study the dogs were confronted with the positive and negative cues differring both in quality and location and could base the performance in the go-no go task on either of them. In the test trials the loci of the positive and negative stimuli were interchanged to find out whether the animals will react according to the stimulus quality or its locus. In the effect of the test trials it has been shown that the "no go" response was associated not with the locus of the auditory stimulus but nearly exclusively based on its auditory quality [1].

Also the "go" response in that experiment appeared to be associated in most cases (although not so exclusively as the negative one) with the auditory quality of the positive cue and not with its location.

Since the animals in our location group in the go-go go task could not use the auditory quality cue because the same tone was presented in positive and negative trials, nor could they use two orienting re-

[1] The only seeming exception — a single dog out of a group of 10 animals showing a consistent positive response when the negative stimulus was transferred into the locus of the positive one — might be interpreted as evidence that in this dog the "go" response was associated mainly with the locus of the positive cue.

sponses because one of them habituated in the course of training, they might still solve the task basing the positive response exclusively on the locus of the positive stimulus neglecting completely its auditory quality. It seems possible that this was the case with the single animal (no. 15) who was able to reach criterion within the limit of testing. Four others failed in this task because, as is also supported by experiments of Szwejkowska (1967), in the go-no go task the association with the auditory quality of presented stimuli is playing the dominant role.

It should be mentioned that since the time of first preliminary report (Ławicka 1964) and discussion of its results (Konorski 1962, 1964) demonstrating selective effectiveness of the auditory quality and localization cues, there have appeared a number of studies (Dobrzecka et al. 1966, Dobrzecka and Konorski 1967) showing, that the superiority of auditory localization cues found for acquisition of two locomotory responses to two differently situated foodwells, may be also extended for a task with two manipulatory movements (left limb vs. right limb) both reinforced by food delivery from a single foodwell.

SUMMARY

Two groups of naive dogs, one with tone frequency cues (frequency group) and the other with auditory location cues (location group) were trained on a task with two instrumental responses (go left-go right) both reinforced by food. It was found that the acquisition of the location group was much superior to the frequency group.

Two other groups of dogs with the same two pairs of frequency and location cues were tested on the acquisition of a Pavlovian differentiation task (go-no go) in which only one stimulus was reinforced by food. Contrasting to the results of the previous task here the acquisition rate of the frequency group was significantly higher than that of the location group.

The results are interpreted in terms of differential effectiveness of cues, indicating that auditory localization cues may become easily associated with motor acts, whereas tone frequency cues are easily associated with respective drives.

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MYELOARCHITECTONICS OF THE WHITE MATTER OF THE SENSORIMOTOR CORTEX IN DOGS

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The present paper is the continuation of my two previous studies on the myeloarchitectonics of the prefrontal white matter in the dog brain. In the dog the sensorimotor cortex is situated between the presylvian fissure and the coronal and ansate fissures. It corresponds to the motor and sensory areas of the primates (Woolsey 1933, Woolsey and Settlage 1950; Hamuy et al. 1956, and others) and includes the precruciate, anterior composite, precentral, postcentral, and coronal gyri.

Twelve complete series of microscopic sections cut in three main planes and stained by the Weigert-Wolters, Woelcke, Klüver-Barrera, Gros-Schultze, and Nissl methods were used for this study. Special attention was given to the arrangement of projectional, commissural, and associational fibres in relation to each other in the semioval center and gyral white matter, the relative number of fibres reaching the gyri, their direction, thickness, density, and the degree of staining, accumulation in bundles or dispersion. The course of medium and short association bundles and their description are also presented.

I. SEMIOVAL CENTER

The semioval center is the region of white matter of the cerebral hemisphere into which commissural, projectional and associational fibres penetrate and within which they cross each other, mingle or run parallel to leave it and enter the gyral white matter. The semioval

center, therefore, constitutes a passage between the internal capsule and corpus callosum on one side and the gyral white matter on the other side.

As regards topography, the semioval center extends along the whole brain, latero-dorsally to the lateral ventricle, above the corpus striatum. Its anterior and posterior portions slope downwards and thus it has an arcuate shape in longitudinal sections. The boundaries between the semioval center and the adjacent formations cannot be defined exactly. The ventral boundary has been presented alternatively as a conventional plane of decussation of commissural and projectional fibres, separating the semioval center from the internal capsule (Crosby et al. 1962), or lower, as a slanting line, also conventional, leading from the upper border of the caudate nucleus to the upper edge of the putamen (Dejerine 1895—1901). In this last case the part of projectional fibres between the internal capsule and their decussation with commissural fibres would be called the base of the corona radiata (pied de la couronne rayonnant, Dejerine 1895—1901). Since myeloarchitectonically the part which forms the base of the corona radiata in the dog corresponds in structure rather with the corona radiata than with the internal capsule, the second alternative has been adopted in the present study. The dorsal, medial and lateral boundaries are also arbitrary, and it has been assumed that they are marked out by the points where the nerve fibres leave the semioval center for particular regions of the gyral white matter.

In the longitudinal aspect the semioval center of the dog brain may be divided into three parts, an anterior, a middle and a posterior, and next each of these parts subdivided into smaller portions. The principle on which this subdivision is based is the distribution of nerve fibres before they enter particular cerebral regions. Thus, in the anterior part of the semioval center one can distinguish 2 subdivisions, an anterior and a posterior, in the middle part three subdivisions, an anterior, a middle and a posterior, whereas the posterior part has two subdivisions, an upper and a lower. The present paper is concerned with the middle part of the semioval center.

So far as myeloarchitectonic structure is concerned, the whole semioval center has it in common that the projectional fibres approach it from below and from the lateral side, and the commissural fibres from the medial side. Besides, the superior fronto-occipital bundle occurs at the base of the semioval center, under the corpus callosum. The particular parts and subdivisions, in addition to the distribution of their fibres before they leave the semioval center, often differ from

each other in the arrangement of their nerve fibres, mainly projectional and commissural, in relation to each other.

In the dog the middle part of the semioval center serves the cerebral cortex bounded by the cruciate fissure in the front and the suprasylvian and median ectosylvian fissures at the back. It is the most compact part of the semioval center, extending above the corpus striatum. Three layers can be seen in its vertical sections.

The lower layer, which forms the base of the corona radiata, is differentiated in respect of its myeloarchitectonic structure into an anterior portion (lying along the dorsal margin of the head of the caudate nucleus) and a posterior. In the anterior portion the area occupied by the base of the corona radiata is shorter in the vertical aspect and wider in the horizontal aspect than in the analogous area of the posterior portion. Its myeloarchitectonic structure is fairly uniform. Medially and dorsally small numbers of fibres leave the head of the caudate nucleus and contribute to the superior fronto-occipital bundle. Laterally to them a large number of projectional fibres are grouped into bundles. These are all well stained and run medio-oro-dorsally. On the medial side they are more closely packed and contain a larger number of fibres, being looser and composed of paler fibres in the middle of the layer. On the lateral side the bundles are again compact, and fibres, single or in thin bundles, run among them. The fibres of the superior longitudinal bundle, limiting the base of the corona radiata from the outside, are fairly thin, pale and few in number.

In the posterior portion of the base of the corona radiata two layers can be distinguished, a dorsal and a ventral, the latter constituting a kind of passage from the internal capsule to the dorsal layer. The medial portion contains well-myelinated bundles of fibres coming from the thalamus and, laterally to them, compact bundles of a small number of well-myelinated fibres from the internal capsule running medio-dorsad. There are also some thin fibres which extend transversely. The lateral portion, too, has fibres arising in the internal capsule but running in the vertical plane and deflecting somewhat mediad. They form thin and very compact bundles which are limited on the outer side by a certain number of fibres running in the horizontal plane.

The middle layer presents the following arrangement of nerve fibres: fibres of commissural radiation, well-stained and of medium thickness, have a medial position; some of them extend from the medial side upwards, others laterad, without being divided into bundles. The middle of the layer is occupied by fibres of the superior fronto-occipital bundle. They are paler and thinner than the commissural fibres and have a lon-

gitudinal course. The projectional fibres of the base of the corona radiata are situated laterally and form compact bundles with a latero-oro-dorsal direction. Externally to them there are some commissural fibres, which form a thick bundle. Those situated more superficially run dorsolaterally and the deeper ones mediodorsally.

The dorsal layer, thicker than the previous one, has a far more confused appearance. Fibres of all types are mixed up, but not in a completely disorderly manner. The rule is that the fibres go the nearest way to their appropriate cortical fields. As a result, fibres which tend to the medial cortical areas can be seen on the medial side and those for the areas situated laterally on the lateral. In the middle of the layer there occur fibres going to the superior areas and those traversing the region from one side to the other. The commissural fibres are present throughout this layer, on the medial side they extend upwards in the vertical plane, in the lateral portion run slantingly oro-laterally, and in the middle have a dorsal or lateral direction. The projectional fibres go obliquely right through the layer in a broad bundle directed medio-dorsally. At the dorsal margin of this layer this compact bundle of fibres become somewhat looser and some of them bend sidewards and forward. Before entering the gyral white matter, these fibres deflect towards the vertical plane. As for the association fibres, twa types are observed: short fibres, which run superficially, and long ones, piercing singly through the others. They are fibres coming from the cingulate bundle and from the superior longitudinal bundle and transverse fibres connecting the medial and lateral portions of the sensorimotor cortex.

In the longitudinal aspect this part of the semioval center is divided into portions which give off fibres to different gyri of the cortex. Thus, the anterior portion of the middle part sends fibres to the pre- and postcentral gyri and the middle portion of the middle part to the coronal gyrus. The anterior composite gyrus receives fibres from different regions of the semioval center. The posterior portion of the middle part gives off no fibres to the sensorimotor cortex and for this reason it will not be discussed here (Fig. 1).

II. GYRAL WHITE MATTER

In the dog brain the gyral white matter occurs as a number of discrete longitudinal parts which radiate from the semioval center. They are 6 in number. Starting from the medial side of the brain, part 1 serves the prorean, subprorean, precruciate and cingulate gyri, part 2 the anterior composite, pre- and postcentral gyri and upper areas of the parietal

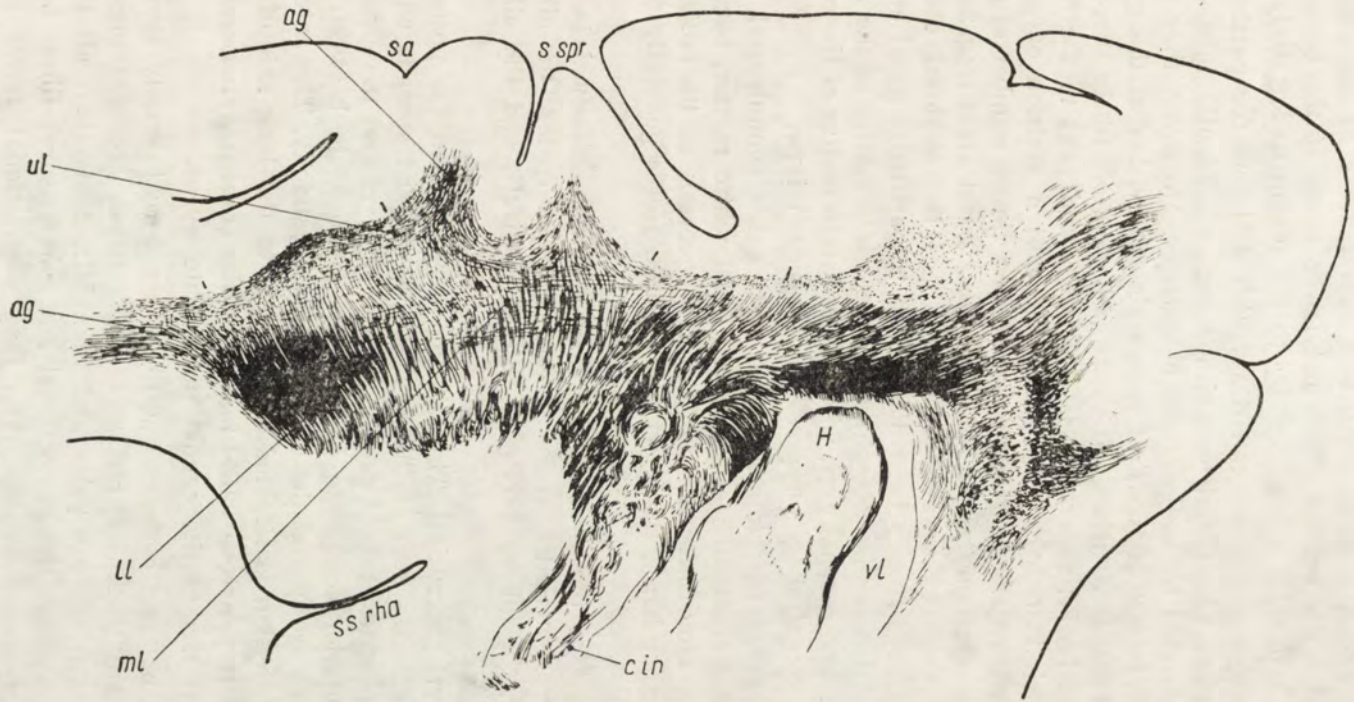

Fig. 1. A parasagittal section through the semioval center, showing the lower layer, which contains fibres belonging to the corona radiata, the middle layer with long associational fibres, the upper layer and the entries into particular portions of the gyral white matter. The division of the semioval centre into longitudinal parts is also shown. Weigert-Wolters, semi-diagrammatic

cortex situated posteriorly to them, part 3 the anterior composite gyrus (dorso-lateral portion), coronal gyrus and lateral areas of the parietal cortex, and part 4 the orbital gyrus and lower areas of the temporal cortex. Parts 5 and 6 lie caudo-laterally to the region under study.

In each of these parts one can distinguish divisions for particular gyri of the cortex. These divisions either merge gradually into each other or are separated by well-defined boundaries. Moreover, they present two typical forms: in the first case a single division of the gyral white matter is situated within one gyrus and gives off fibres successively, first to the lower, fissural or lateral, areas and next to the main gyral area, which is only one. In the other case a division of gyral white matter contains fibres which either go to a larger number of gyri (more rarely) or reach one gyrus with 2 or more gyral areas (Fig. 2a, b). This may be considered as a "second level" of the semioval center, before its fibres have finally been distributed to particular gyral areas. The first type of division is exemplified by the white matter of the prorean, subprorean and coronal gyri, whereas the white matter of the orbital, pre- and postcentral gyri represents the second type.

It is, as often as not, impossible to distinguish commissural and projectional fibres from each other in the gyral white matter, because they run in the same direction and enter the cortex as its radiation fibres. On the other hand, the short commissural fibres generally form tangential bundles.

Four divisions of the gyral white matter can be observed in the part of the dog brain that contains the sensorimotor cortex: the album gyri precruciati, the album gyri pre- and postcentralis and the album gyri coronalis.

a. Album gyri precruciati. This division departs from the posterior portion of the anterior part of the semioval center and spreads within the transverse precruciate and caudal precruciate gyri and the anterior areas of the anterior composite gyrus. It is squeezed in between the presylvian fissure orally and the cruciate fissure caudally. The anterior portion of this division is situated transversely to the long axis of the brain. Laterally it joins the white matter of the coronal gyrus and its posterior portion lies in the caudal precruciate gyrus.

The projectional and commissural fibres are here intensely stained, mostly of medium thickness, but some thick fibres are also present. The associational fibres are paler and thin. Mixed together, all these fibres run dorso-orally, the projectional and commissural fibres being grouped in fairly loose bundles. The long associational fibres come from the cingulate bundle, superior longitudinal bundle and superior

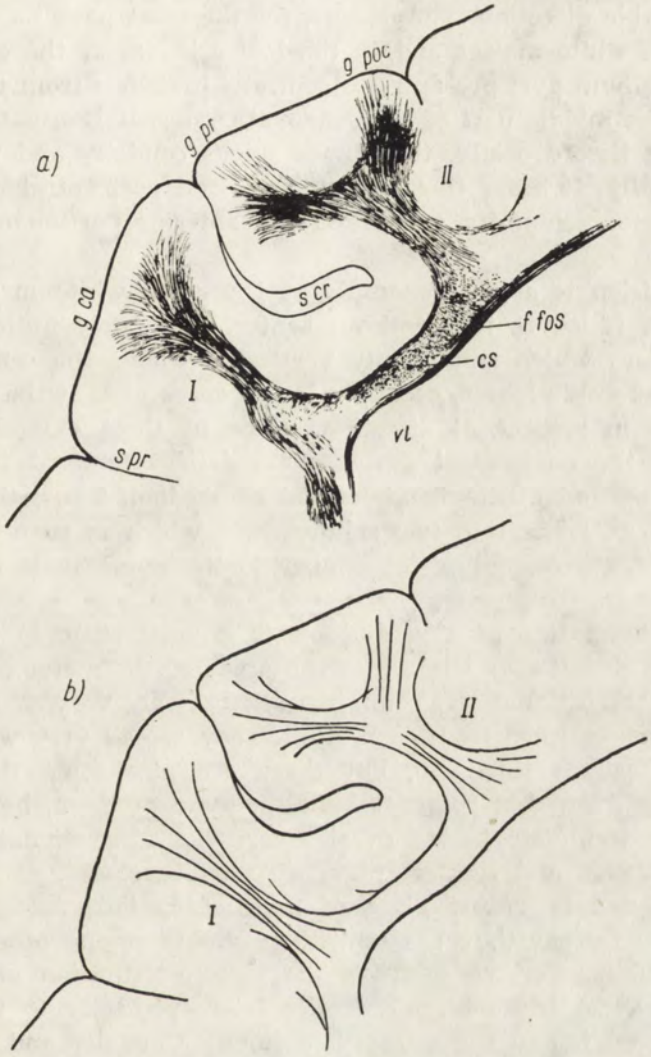

Fig. 2a, b. A parasagittal section through the semioval center and album gyri, showing two types of the gyral white matter semi-diagrammatically and diagrammatically. The description in the text. Weigert-Wolters

fronto-ocipital bundle, but they can be traced only to the place where they enter the gyral white matter. Higher, they are mingled with other fibres and it cannot be established in which areas they terminate. The medium and short associational fibres extend, as a rule, in three directions, i.e., vertically, horizontally and medio-laterally. Some of them

have a veriable direction. They occur, for the most part, on the surface of the gyral white matter and in the lower layers of the cortex.

b. The album gyri pre-and postcentralis branches from the anterior portion of the middle part of the semioval center. It is situated between the cruciate fissure orally, the ansate sulcus caudally and the coronal sulcus laterally. It bears fibres for the pre-and postcentral gyri, a part of the anterior composite gyrus and the anterior portion of the marginal gyrus.

This division is a good example of the gyral white matter of the second type. It leaves the semioval center as a fairly uniform assemblage of fibres which run mainly vertically. Under the central sulcus these fibres become looser, occupy a larger space and, at the same time, differentiate in respect of direction. Some of them extend vertically upwards to the postcentral gyrus, some deflect forward to the precentral gyrus, and others bend laterad or mediad. Thus, the division, so far uniform, splits into two subdivisions, which in turn divide into smaller units, corresponding in number to the gyral areas in the gyri (Fig. 1).

The white matter of this division is a very strongly myelinated region. It receives fibres tending to the areas of the motor and sensory cortex. The projectional and commissural fibres are very numerous and cannot be distinguished from each other. They are all very well stained, mostly of medium thickness, but there are also some thick fibres. They usually form thin compact bundles; some of them, however, run singly. They terminate in the gyral areas. The associational fibres are generally thinner and lighter; they are numerous and, as previously, can be traced only to the place of their entry into the gyral white matter, since farther they are undistinguishable among other fibres.

c. The album gyri coronalis is the foremost division of the third part of the gyral white matter. It lies latero-ventrally to the coronal sulcus, between it and the ectosylvian sulcus. Caudally and dorsally it is joined directly with the white matter of the ectolateral gyrus. It serves areas KA and KP in the coronal gyrus, the lower portion of area CA and areas CE I and CE II in the anterior composite gyrus.

Nerve fibres for this division of the gyral white matter come from the middle portion of the middle part of the semioval center, projectional fibres from the lateral side of the anterior portion of the middle segment of the corona radiata and the commissural fibres from the commissural trunk (Świecimska 1968).

As compared with the pre- and postcentral gyri, the number of fibres for the coronal gyrsu and areas CE I and CE II of the anterior composite gyrus is smaller. At first all these fibres run obliquely dorso-

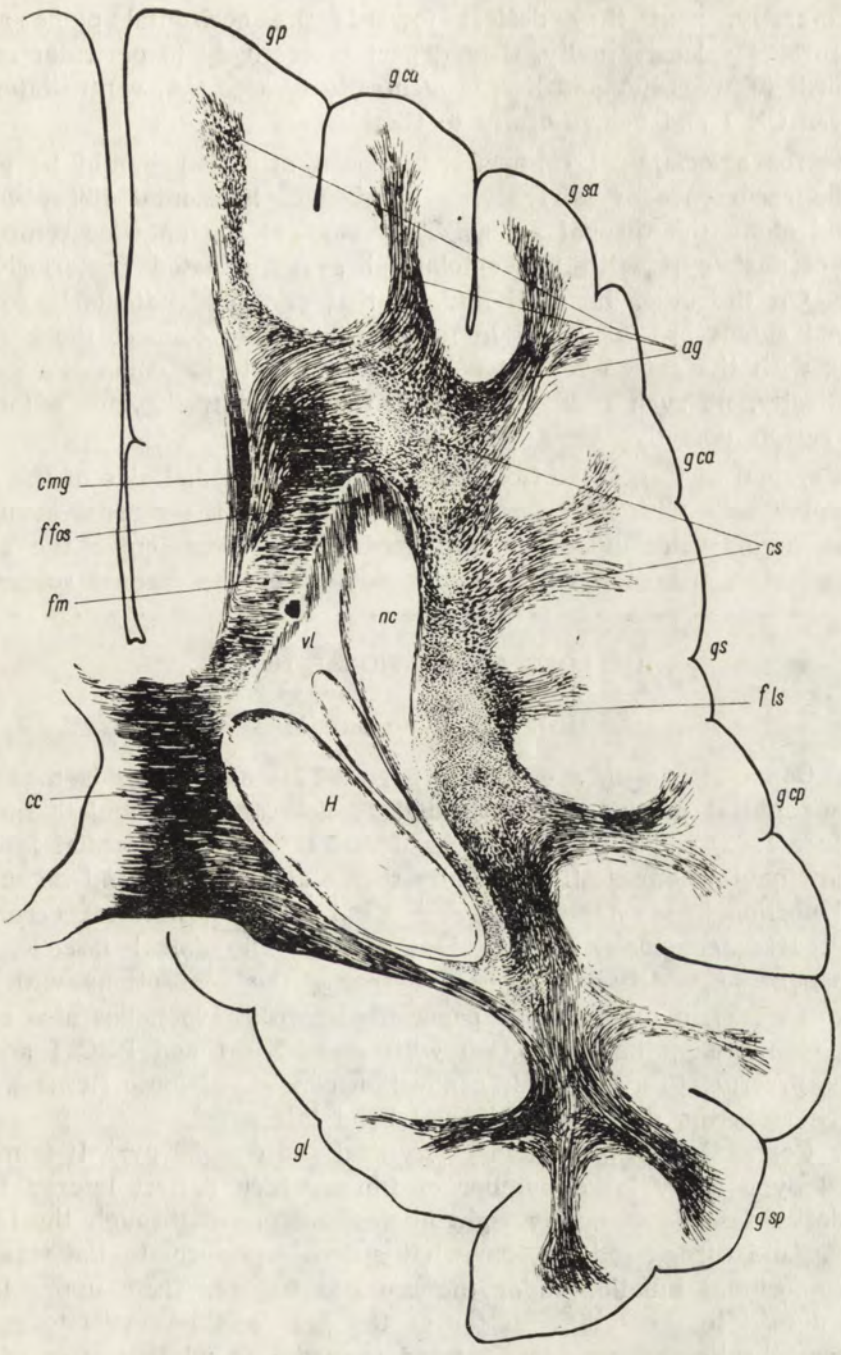

Fig. 3. A horizontal section through a dog brain. The description in the text. Weigert-Wolters, semi-diagrammatic

latero-orally, next they deflect towards the horizontal plane along the coronal sulcus. Finally, they depart successively to paricular areas, upwards to areas KA and KP, oroventrally to area CA, ventro-laterally to area CE I and ventro-orally to CE II.

As for associational connections, special attention should be given to the occurrence of fairly large number of horizontal fibres which extend along the coronal sulcus. They connect the anterior composite and coronal gyri with the ectolateral gyrus situated posteriorly to them. On the other hand, the fibres that run perpendicularly to the coronal sulcus are very few in number and they connect the coronal gyrus with the antero-ventral portion of the anterior composite gyrus. Practically, no such connections with the postcentral gyrus, adjoining this region dorsally, have been demonstrated.

A system of transverse fibres, connecting the medial side of the sensorimotor zone with its lateral side, is visible in the coronal gyrus as a bundle of well-stained medium-sized fibres which enter the gyral white matter from the dorso-medial side under the coronal sulcus.

III. LONG ASSOCIATIONAL TRACTS

A. Cingulate bundle

a. Connection with precruciate gyrus. This connection is made by the superficial portion of the medial fascicle of the cingulate bundle (Kreiner 1962) and a portion of the dorsal fascicle. The medial fascicle of the cingulate bundle lies medially to the axial fascicle and its superficial portion bends dorsally to area XP I of the posterior precruciate gyrus. It is composed of thin loose fibres. The dorsal fascicle, one component of which was discussed among the connections with the prorean gyrus in my previous paper (Świecimska 1968), has also other components, e.g., the connection with areas XP I and PRC I and II in the precruciate gyrus. This connection consists of loose fibres which branch from the dorsal fascicle and bend laterad.

b. Connection with pre-and postcentral and coronal gyri. It is maintained by a fairly large number of fibres which deflect laterad from the dorsal fascicle at nearly right angles and pierce through the lesser forceps and corona radiata somewhat antero-superiorly to the superior fronto-occipital bundle, under the cruciate fissure. Their upper layer runs dorsad to the white matter of the pre- and postcruciate gyrus, whereas the lower layer goes laterad towards the white matter of the coronal gyrus. In the gyral white matter they intermingle with other fibres and it cannot be established which areas they reach. They are

either moderately thick or thin, generally well stained, though not so well as the adjacent commissural fibres. They do not form bundles.

B. Superior fronto-occipital bundle

A small number of well-stained fibres from the dorsal fascicle of the superior fronto-occipital bundle radiate to the sensorimotor cortex. This fascicle is situated on the dorso-lateral side of the superior fronto-occipital bundle. Its fibres bend dorsolaterally and enter the semioval center laterally to the knee of the corpus callosum and medially to the corona radiata. They are not very numerous but very well stained. Some of them end in the anterior portion af the orbital gyrus, some in the precruciate gyrus, and those situated farthest in the rear in the gyri beyond the cruciate sulcus.

IV. SHORT AND MEDIUM ASSOCIATIONAL TRACTS

Particular authors differ in opinion about the classification of associational fibres. These fibres are divided into groups on the basis of their length and position in the white matter and cortex. Most authors apply a division into two basic groups: 1) long fibres and 2) short fibres, these last being sometimes divided into two subgroups: a. intracortical fibres and b. subcortical Fibres (Bechterew 1889, Ariëns Kappers et al. 1936, Clara 1959, Ranson and Clark 1959, House and Pansky 1960, Crosby et. al. 1962, Bochenek and Reicher 1963, Miller et al. 1964, Marciniak 1965). Dejerine (1895—1901) distinguished 3 groups, namely, 1) short fibres, 2) long fibres and 3) own fibres of the occipital and frontal lobes, whereas Truex and Carpenter (1964) introduce still another division of associational fibres, that is to say, they assume two main groups, 1) intracortical fibres and 2) subcortical fibres, and subdivide these last into a. short fibres and b. long fibres.

Apart from these divisions, all the authors agree with the statement that the short fibres, running in the cerebral cortex or on its border with the white matter, connect regions neighbouring directly upon each other and the long fibres, situated deeper in the white matter, connect regions which are remoter from each other.

The division of fibres adopted in the present paper differs somewhat from those introduced by other authors. The cerebral cortex of the dog has been divided by Kreiner (1961, 1964a, b, c 1966) into myeloarchitectonic areas, which are connected with each other by associational fibres. The following types of connections have been distinguished: *a*, connections between particular parts of an area; *b*, connections between neighbouring areas; *c*, connections between areas separated by a sulcus

or another; area or several areas; *d*, intergyral connections; *e*, connections between remote cortical areas (e.g., interlobular connections).

As regards topography, connections *a* and *b* lie in the cerebral cortex, the first of them more superficially, connections *c* and most of *d* extend on the border of the cortex with the white matter, and connections *e* are sunk in the white matter of the pallium. All these connections occur one above another and they are often difficult to discern from each other. Fibres of one category may also change their position, e.g., when they run under a sulcus and can be seen on the border between the cortex and white matter. With respect to the above-mentioned classifications fibres *a* and *b* are treated in the present study as short, fibres *c* and *d* as medium, and fibres *e* as long associational fibres. Fibres *c* and some of *b* may be regarded as classical U-fibres.

In the dog most of the fibres *b*, *c* and *d* arise in the side-walls of a gyrus (in subareas *b* and *c*, Kreiner 1961) The same was found in man by Dejerine (1895—1901). Fibres *a* generally connect the tops of gyri (subareas *a*, Kreiner 1961). Short associational fibres usually form scattered bundles, whereas the medium ones are more closely packed. The long fibres form compact bundles only in their middle course (Dejerine 1895—1901, Ariëns Kappers 1947, and others), no differences being found in this respect in the dog.

In the present paper the description of particular bundles of short and medium associational fibres will be arranged according to the gyri to which they belong. Since the number of these bundles is very large, suitable designations will be used in the description. Thus, short fibres, tangential with the cortex, will be designated with the letter *a*, fibres which run on the boundary of the cortex with the white matter with the letter *b*, and fibres sunk shallowly in the white matter with the letter *c*. The places of occurrence of given bundles will be marked with numerals, 1, 2, 3, . . ., starting from the front backwards, and so the bundles designated with the numeral 1 are the foremost ones. The letter *m* will be used to designate the medial side of the brain and the letter *l* for the lateral side. In addition, each bundle will be marked with the abbreviation of the name of the gyrus from which it comes.

A. Precruciate gyrus

The precruciate gyrus is situated on the medial side, frontally to the cruciate fissure, on the upper edge of the brain. It has been divided (Kreiner 1964a) into the following myeloarchitectonic areas: the central precruciate area (XC), which forms the transverse precruciate gyrus, posterior precruciate areas I and II (XP I and II) and lateral precruciate area (XL), occupying the caudal precruciate gyrus, medial precruciate

areas I and II (XM I and II), situated on the medial precruciate gyrus, and two fissural areas, the area of the splenial fissure (FS) and that of the pregenual fissure.

Regarding the associational connections, the precruciate gyrus is a transition from the prefrontal cortex to the sensorimotor zone, because it contains bundles composed of both large and small numbers of fibres, running long or short distances. In comparison with the prefrontal region there are more, very short, local connections here which occur within one area.

1. Connections with the prefrontal cortex

These connections have been described in a previous paper (Świecimska 1968). They include the bundles; Pr 3am, which connects subareas Pr a and b with subarea XM I a, Pr 4am, connecting the posterior portion of subarea Pr a with area XC, Pr 3bm, connecting subarea Pr a with area XM II (Fig. 4), PRL 4al, connecting area PRL with areas CX, XC and CA in the anterior composite gyrus, and ORB 4 cl, which connects area ORB II with areas XM I and II. This last bundle belongs to the transverse system, which will be described below. In addition to the above-mentioned elements, there is also a very poor connection of the posterior portion of area Pr with area XP I by means of bundle XP I 6bm, which consists of very thin fibres, few in number, extending parasagittally on the boundary of the cortex and white matter.

2. Connections with the limbic cortex

These are represented by the connection with the cingulate gyrus and bundle, described in the section on the long associational connections (p. 10), and by bundle XM II 5 am, which connects area XM II with area C I in the genual gyrus (Fig. 4). This bundle is built of several well-stained fibres which run in the cortex under the genual fissure. Bundle XP I 7bm, connecting areas XP I and II with areas S II and LAV in the cingulate gyrus, belongs under the same group of connections. It consists of several well-stained fibres of medium thickness which come out of areas XP I b and XP II b, arch under the caudodorsal end of the genual fissure and enter the base of the anterior portion of the cingulate gyrus.

3. Connections with the anterior composite gyrus

a. Area XM I and II

Bundle XM I 5am connects area XM I with area CX. The fibres of this bundle run transversely from area XM I dorso-laterad and penetrate through the upper portion of the white matter of the prorean

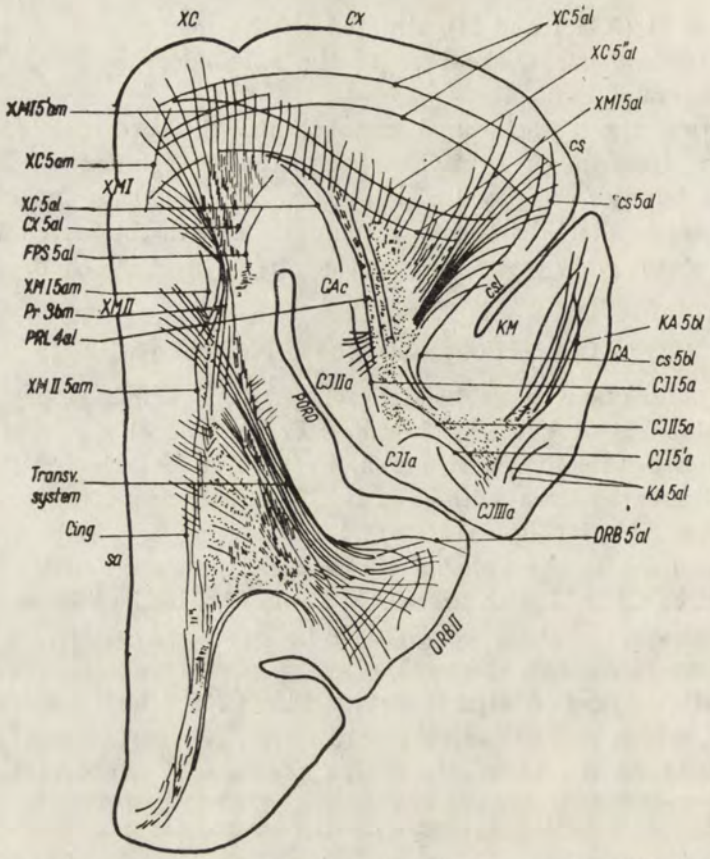

Fig. 4—7. A series of transverse sections showing the myeloarchitectonic organization of the white matter. The description in the text. Weigert-Wolters, diagrammatic

gyrus. They are several in number, moderately thick and well stained (Fig. 4).

b. Area XC

Bundle XC 5al connects area XC with area CJ II. It extends along the laterad brink of the presylvian fissure, next through the white matter of the precruciate gyrus, from area XC ventro-laterad to area CJ II. Its fibres are of medium thickness and well stained. They are few in number and run singly. Their more superficial portion terminates in area CA (Fig. 4, 8).

Bundle XC5'al connects area XC with areas CX and CS. It is a strong bundle running across the dorsal margin of the gyral white matter from the medial side laterad. It crosses the radiation fibres of the cortex over the whole length of area XC. Its fibres are numerous. Those situated

dorsally are the shortest and reach area CX. The underlying fibres are longer and go to area CS. They are all situated above the cructiate fissure (Fig. 4).

4. Connections with the precentral gyrus

a. Area XC

Bundle XC 5″al maintains connection between area XC and area PrC I. Its double dorsal portion runs together with bundle XC 5′al, being formed by its longest fibres, which on passing across areas CX and CS swing postero-ventrad while the ventral portion arises in area XC and descends nearly as far as the bottom of the presylvian fissure. Next it turns laterad and somewhat caudad to area PrC I. It contains fewer fibres than the previous one. All these fibres are moderately thick and pale (Fig. 4).

b. Area XM I

Bundle XM I 5al is the lowest portion of bundle XC 5″al. It runs from area XM I upwards and next laterad through the white matter (Fig. 4).

c. Areas XP I, XP II, and XL

Bundle XP 7bm connects the caudal precruciate gyrus with the precentral gyrus. It passes along the splenial fissure and is arranged so that the fibres which originate in area XL lie superficially and the fibres from area XP I farther to the front than those from area XP II. They all run from these areas in a ventrolateral direction and next bend dorso-latero-orally. The fibres from area XL turn dorso-medially. None of these fibres are numerous, they are all thin and pale.

5. Connections within the precruciate gyrus

a. Area XC

Bundle XC 5am, connecting area XC with area XM I, is loose and consists of very intensely stained thin fibres which extend as tangential fibres ventrally and slightly medio-orally.

Bundle XC 6am connects area XC with area XP I. This connection, observed in the deeper layers of the cortex as its tangential fibres, passes from area XC ventro-caudally over the whole length of area XP. The fibres are few in number, thin and scattered (Fig. 9).

Bundle XC 6′am, composed of several fine fibres, is a short local connection between areas XC and XL.

b. Areas XM I and II

Bundle XM I 5am, connecting areas XM I and XM II, is built of loosely arranged, thin and very well stained fibres, which run from

area XM I downwards with a slight caudal deviation. They are fairly numerous (Fig. 4).

Bundle XM II 7am connects area XM II with area XP I. It is a scattered bundle of thin and pale fine fibres, of which some extend in the lower layers of the cortex and either terminate in lateral subareas of both gyri or reach the gyral areas, whereas the others run somewhat deeper, on the boundary of the cortex with the gyral white matter and penetrate among the cortical radiation fibres of the gyral areas.

c. Areas XP I, XP II and XL

Bundle XP I 7am keeps connection between areas XP I and II. It consists of some dozen well-stained fine fibres, which run caudoventrally and connect the tops of both these areas.

Bundle XP I 6al consists of several single fine fibres which come out of the lateral subarea XP I c and are directed ventrolatero-caudally. They are thin and poorly stained.

B. Precentral gyrus

This gyrus is situated just behind the cruciate fissure, which borders it anteriorly and medially. Laterally it spreads to the coronal sulcus and posteriorly is limited by the central sulcus. It includes three gyral areas, precentral areas I, II and III (PrC I, II and Ill), two lateral areas, the lateral precentral area (PrCL) in the ansate fissure and the internal precentral area (PrCL) in the ansate fissure and the internal precentral area (PrCJ) in the coronal sulcus, and two fissural areas, the area of the central sulcus (CE I) and that of the cruciate fissure (FCr). The associational connections of this gyrus are typical of the sensori-motor cortex: the bundles contain very few fibres, which run for very short distances.

1. Connections with the precruciate gyrus

These connections have been described in the previous section. They include bundles XM 5al, XC 5″al and XP 7bm, as well as bundle PrC II 3a, connecting area PrC II with area CS. It passes in the horizontal plane across the dorso-lateral portion of the cruciate fissure and contains a small number of pale medium-sized fibres.

2. Connections with the parietal cortex

Bundle PrC III am extends in the horizontal plane in the deep layers of the cortex, as its tangential fibres, along area PrC III about halfway across area MA in the marginal gyrus. It is formed by a small number of thin fibres.

Bundle PrC III bm connects area PrC III with area MA in the marginal gyrus. It runs horizontally on the boundary of the cortex and white

matter. This bundle is composed of fairly numerous, moderately thick and well-stained fibres.

Bundle PrC II am, which connects area PrC II with area ND in the marginal gyrus, passes inferior and lateral to the previous ones, along the posterior wall of the cruciate sulcus. It consists of several medium-sized fibres which terminate in the slopes of the gyri.

3. Connections with the anterior composite and coronal gyri

These are maintained by the transverse system, which will be discussed below. Out of the short associational bundles only a weak connection by bundle PrCL 7al can be mentioned here. The fibres of this bundle go under the coronal sulcus as its U-fibres. Their number is very small and they are seen for a very short distance. They connect area PrCL with the medial coronal area (KM) in the coronal gyrus (Fig. 5).

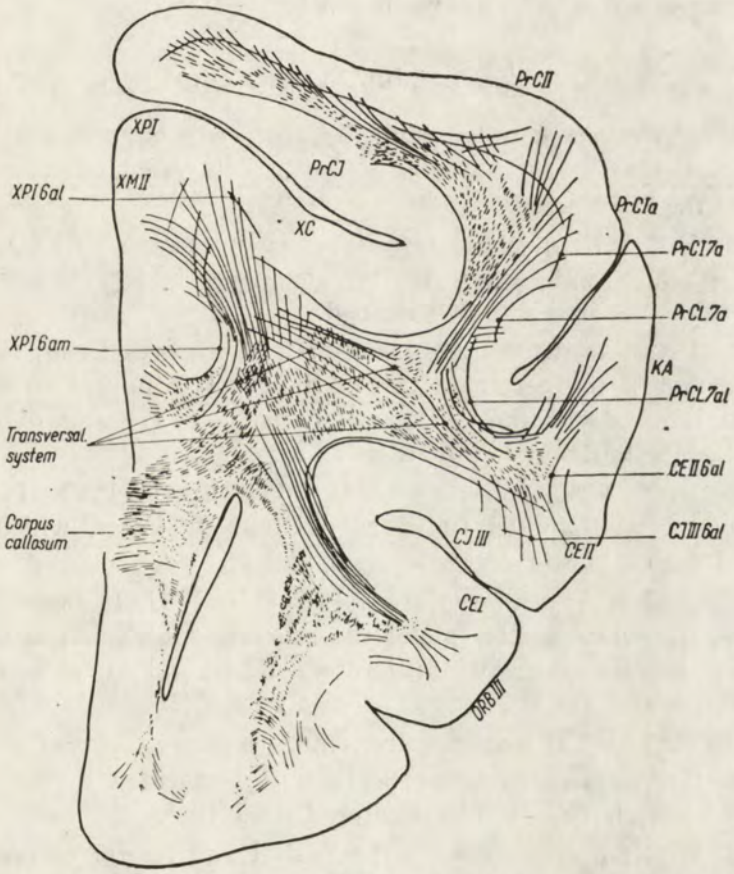

Fig. 5

4. Connections with the postcentral gyrus

These connections are few and spread in the parasagittal plane.

Bundle PrC 7bl, connecting areas PrC I and II with area PoC, runs for a very short distance in the parasagittal plane in the depth of the white matter, under the central sulcus, and comprises only several medium-sized fibres (Fig. 10).

Bundle PrC III 7b′m connects area PrC III with subarea PoC b. It is situated medially to the central sulcus and has a small number of short fibres of medium calibre.

Bundle PrC II 7bl, which connects area PrC II with subarea PoC a, is situated laterally to the central sulcus. It resembles the previous bundle only that it is richer in fibres.

Bundle PrC II 7′bl, connecting area PrC II with subarea PoC c, passes under the central sulcus. Its fibres, being very few, well-stained and medium-sized, are sunk in the white matter (Fig. 9).

5. Connections within the precentral gyrus

These connections can be divided into two groups. Some of them connect particular areas and subareas with each other, others particular regions of the same areas.

Bundle PrC I 7bm, whose few fibres connect area PrC I with area PrC III, extends in the white matter along and under the central sulcus. The fibres are thin and well-stained.

Bundle PrC I 7a, connecting, area PrC I with area PrCJ, lies in the lateral portion of the posterior wall of the cruciate fissure on the boundary of the cortex with the white matter. Its fibres are thin, delicate and scattered in the horizontal plane (Fig. 5).

Bundle PrCL I 7a connects area PrC I with area PrCL. It is made up of several well-stained fibres of medium thickness, which run as tangential fibres in the cortex (Fig. 5 and 9).

Bundle PrC II 7a connects areas PrC II and III. It extends on the surface of the white matter along the cruciate sulcus and is composed of a very small number of moderately thick and very well stained fibres (Fig. 8 and 9).

Bundle PrC II 7′al connects the anterior portion of the top of the area PrC II with the posterior portion. It is formed by several fine fibres which arch over the cortical radiation fibres.

Bundle PrC III 7am connects the medial and lateral cortex of area PrC III. This connection is also the most superficial.

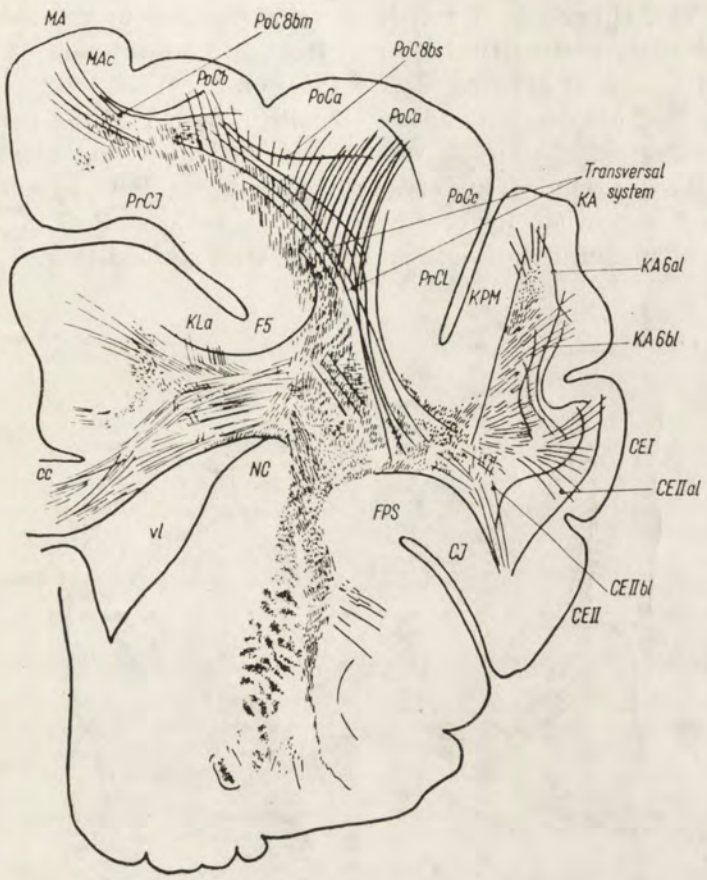

Fig. 6

C. Postcentral gyrus

This gyrus is situated posteriorly to the central sulcus, between it and the ansate sulcus and the side branch of the coronal sulcus. It contains a large area, PoC, divided into three gyral subareas (Kreiner 1964a) and a fissural area, CE II.

1. Connections with the parietal cortex

Bundle PoC8bm, connecting subareas PoC b and c with area MA in the marginal gyrus, is built of fairly thick or medium-sized U-fibres which run across the ansate sulcus on the boundary of the cortex and white matter. Some of them, occupying the highest position, cross the cortical radiation fibres at a high level and terminate in the top regions

of both areas. Other fibres run lower and pass on to the side wall of sub area PoC b, and still others run from the lateral side of area MA to the top region of subarea PoC c (Fig. 6).

Bundle PoC 8bl connects area PoC with area BA of the parietal cortex. It goes round the ansate sulcus, and its longer and more dorsally situated fibres extend from area BA to subarea PoC a, whereas the shorter and somewhat lower fibres end in subares PoC c. All these fibres are of medium thickness and fairly well stained (Fig. 7 and 9).

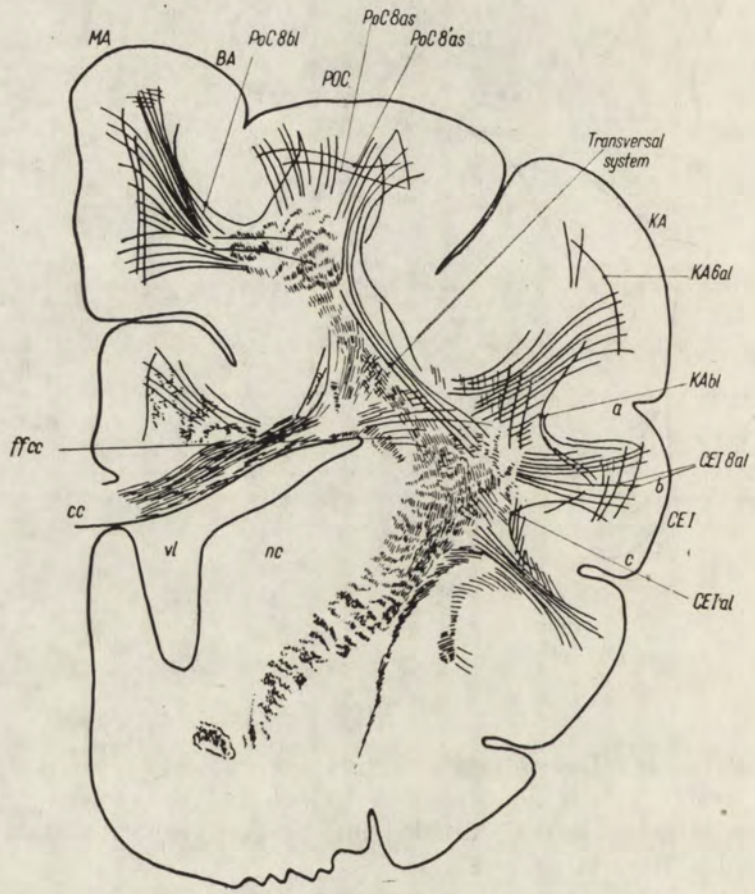

Fig. 7

Bundle PoC 9 is a connection between the floor of the ansate fissure and the coronal fissure. It runs horizontally over a very small area and connects subarea PoC c with area QA in the parietal cortex. It comprises some dozes of well-stained thin fibres (Fig. 10).

2. Connections with the precentral gyrus

These connections have been described for the precentral gyrus. They are: bundle PrC III 7b′m, connecting subarea PoC b with area PrC III, bundle PrC 7bl, connecting subarea PoC a with area PrC II, bundle PrC III 7′bl, which connects subarea PoC c with area PrC II and bundle PrC I 7bl, connecting subarea PoC a with area PrC I.

3. Connections within the postcentral gyrus

Bundle PoC8bs, connecting subarea PoC b with subarea PoC a, consists of several, intensely stained, fairly thick fibres which lie sunk in the white matter. They do not emerge on to the surface of the gyrus in the areas in which they terminate (Fig. 6 and 9).

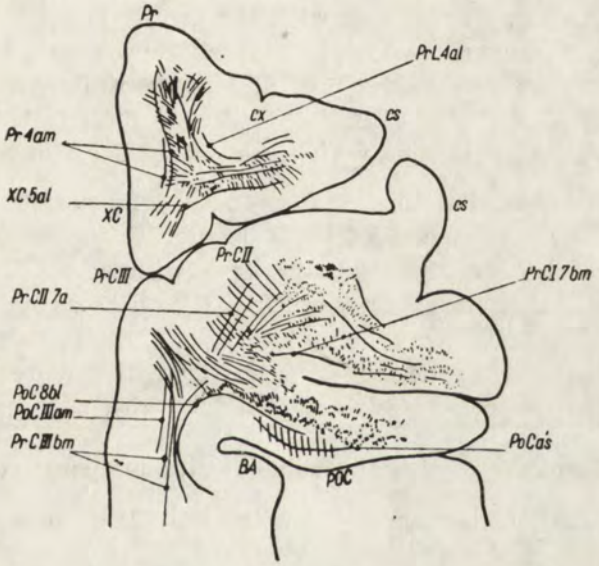

Fig. 8—10. A series of horizontal sections showing the myeloarchitectonic organization of the white matter. The description in the text. Weigert-Wolters, diagrammatic

Bundle PoC 8as connects subareas PoC a and b under the cryptosulcus which separates both these subareas. Its fibres cross the cortical radiation fibres, they are medium-sized and well-myelinated (Fig. 9).

Bundle PoC 8a′s, connecting the same subareas PoC a and c, runs superficially. It comprises very few, 8—10, fibres which are thin and scattered as tangential fibres of the cortex (Fig. 3).

D. Anterior composite and coronal gyri

This cerebral region stretches anteriorly, laterally, and ventrally to the precruciate, precentral, entolateral, ectosylvian and sylvian gyri. Its boundary with the above-mentioned gyri is blurred, whereas anteriorly the anterior composite gyrus is bordered by the presylvian sulcus. In the floor of the perfissura presylvia, Kreiner (1964) distinguished 13 myeloarchitectonic areas in this region. They include the following gyral areas: precruciate composite area (CX), sigmoid composite area (CS), anterior coronal area (KA) and ectosylvian composite areas I and II (CE I and II), the lateral areas: anterior composite area (CA), lateral sigmoid compasite area (CSL), internal composite areas I, II and III (CJ I, II and III) and medial coronal area (KM), and the fissural areas: the area of the presylvian fissure (FPS) and that of the coronal fissure (FK). The associational connections of the anterior composite gyrus both with the neighbouring areas and with the prefrontal, parietal and temporal cortex are numerous. They are often represented by bundles of longer fibres than those in the pre- and postcentral gyri.

1. Connections with the prefrontal cortex

These connections, listed below for general information, are described in a previous paper (Świecimska 1968) devoted to the prefrontal cortex.

Bundle Pr 4al, connecting subarea Pr a with area CX,

bundle PRL 4al, connecting area PRL II with areas XC, CX and CA (Fig. 4),

bundle PORD 4al, connecting area PORD with subarea CJ I a, and

bundle ORB 4bl, connecting area ORB II with subarea CA a.

2. Connections with the parietal and temporal cortex

Bundle KA 8al connects area KA with area KP. It runs on the boundary of the cortex with the white matter and in the deeper layers of the cortex. It contains a fairly large number of medium-sized and thin fibres which form a layer along the lateral margin of the coronal gyrus (Fig. 10).

Bundle KA 8bl, which connects area KA with area QA, lies in the white matter, medially to the previous bundle. It is composed of longer and thicker fibres, which are very intensely stained but less numerous than those in bundle KA 8al (Fig. 10).

Bundle CE I al, connecting area CE I with the sylvian gyrus, is a typical connection by means of U-fibres and it lines the ectosylvian sulcus. These fibres arise in the slopes of the gyri, are of medium thickness or thin and fairly numerous (Fig. 7).

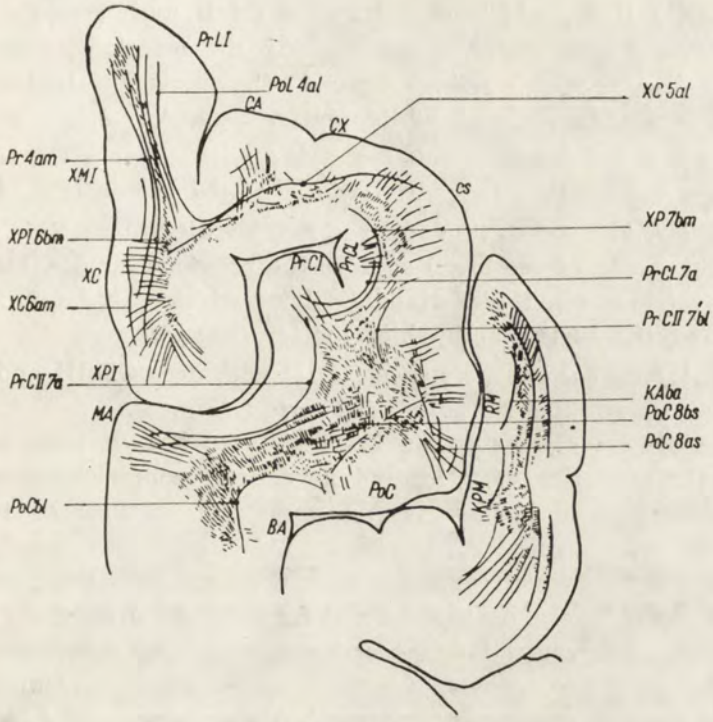

Fig. 9

3. Connections with the precruciate, precentral and postcentral gyri

These connections have been described above (p. 14) and are as follows:

bundle XM I 5′am, connecting area CX with area XM I,
bundle XC 5al, connecting area CJ II with area XC,
bundle XC 5′al, connecting areas CX and CS with area XC, and
bundle PrCL 7al, connecting area PrCL with area KM.

4. Connections within the anterior composite gyrus

Bundle CJ I 5a connects subarea CJ I a with subarea CJ II a. It is composed of several well-stained thin fibres which extend in the fairly deep cortical layers of the side-wall of the presylvian fissure (Fig. 4).

Bundle CJL5′a, connecting subarea CJ I a with subarea CJ III a, comprises more fibres than the previous bundle. The fibres are of medium thickness and lie on the boundary of the cortex and white matter along the presylvian fissure like the fibres of bundle CJ I 5a but inferior to them (Fig. 4).

Bundle CJ II 5a, which connects area CJ II with area CJ III, consists of several thin fibres. It goes along the inferior portion of the presylvian fissure which swings towards the parasagittal plane on the boundary of the cortex and white matter (Fig. 4).

Bundle KA 5al connects subarea KA c with subarea CJ III a. It is built of very short thin tangential fibres of the cortex (Fig. 4).

Bundle KA 5bl, connecting subarea KA a with subarea CJ III a, runs on the lateral side of the brain as does the previous bundle, but is composed of medium-sized well-stained fibres which can be seen on the boundary of the cortex and white matter (Fig. 4).

Bundle CJ III 6al connects area CJ III with area CE III and contains several pale tangential fibres of the cortex (Fig. 5).

Bundle CS 5bl connects area CS with area KA. It runs across the coronal sulcus over a very limited space and comprises several long fibres of medium thickness, visible under the surface of the white matter (Fig. 4).

Bundle CS 5al, which connects area CS with area CSL, extends in the fairly deep layers of the cortex, forming an arch from area CS laterad and next ventrad in accordance with the depression of the coronal sulcus. It is composed of several well-stained thin fibres (Fig. 4).

Bundle KA 6bl, connecting subarea KA a with area CE I, is a strong bundle of typical U-fibres extending across the suprasylvian fissure. The fibres enter fairly deep into the white matter, are very intensely stained and form a fairly thick layer (Fig. 6).

Bundle CE II 6a connects area KA with area CE II. It is made up of a small number of thin and poorly stained fibres which run as tangential fibres of the cortex.

Bundle KA 6al connects subarea KA a with subarea KA c. It consists of thin fibres scattered singly over a fairly large space. They cross the cortical radiation fibres (Fig. 6 and 7).

Bundle KA 6a, connecting area KA with area KM, is situated on the dorso-medial side of the coronal gyrus, in rather deep layers of the cortex. Its fibres are thin and fairly numerous (Fig. 9).

Bundle KA 6a′ connects area KA with area KPM. It resembles the previous bundle and lies ventrally to it. Both these bundles run in the horizontal plane caudally with a slight medial deviation (Fig. 10).

Bundle CE I 7al, which connects subarea CE I c with subarea CE I a, is a very short local connection of a small number of moderately thick and well stained fibres tangential with the cortex.

Bundle CE I 7a′l, connecting subarea CE I c with subarea CE I b, is analogous to the previous one (Fig. 7).

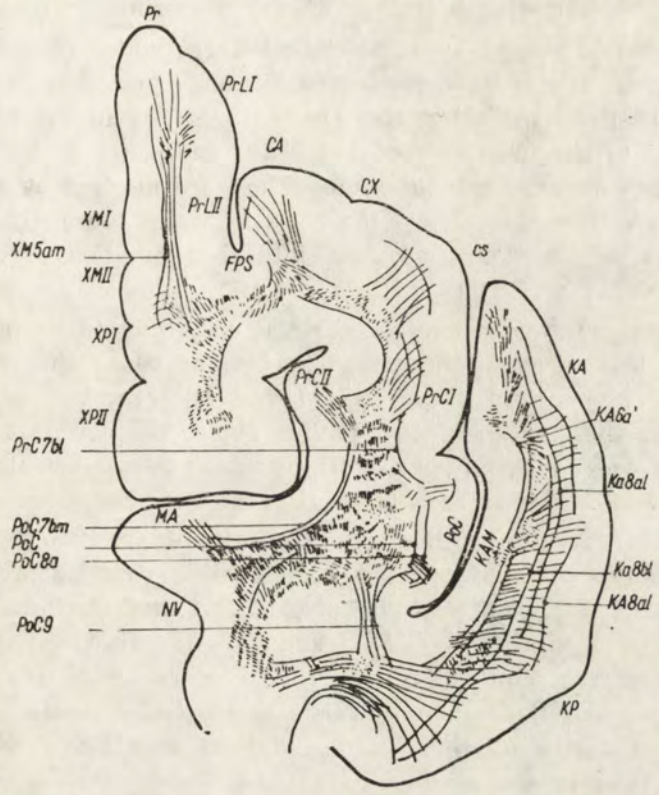

Fig. 10

Bundle CE II al connects area CE I with area CE II. It is a tangential connection of the cortex, superficial and composed of several thick fibres (Fig. 6).

Bundle CE II bl connects the same areas, CE I and II. It is a bundle of typical U-fibres, which form a thick layer across the ectosylvian sulcus. The fibres are very well stained and somewhat immerged in the white matter. The bundle resembles typical connections of the prefrontal cortex (Fig. 6).

Bundle CA 5bl has a horizontal course on the lateral side of the anterior composite gyrus. It connects together areas CA, KA and CE. It consists of a fairly large number of medium-sized fibres which can be seen on the boundary of the cortex and white matter. The bundle runs from area CA caudo-laterad and its shorter fibres end in area KA, whereas the longer ones disappear in the dorso-anterior portion of area CE I.

Transverse associational system of the sensorimotor region.

This system is formed by associational fibres which connect together particular areas of the sensorimotor cortex and pass through the semioval center. In the semioval center these fibres run in the plane which is transverse to the long axis of the brain and, on entering the white matter, deflect in different directions. They are marked by their being very well myelinated and by their medium or more than medium thickness. These fibres are grouped into thin bundles or run singly. They intermingle with the cortical radiation fibres.

The transverse system may be divided according to its position and connections into several bundles. The bundle connecting the medial areas of the precruciate gyrus (XM I and II) with the orbital gyrus is situated farthest to the front. It lies above the corona radiata and consists of a very large number of fibres which descend ventro-laterally from areas XM I and II area ORB II (Fig. 4).

Somewhat caudally to this connection there are two bundles, far poorer in fibres than the previous one. They connect areas XM II and XP with the precentral and coronal gyri and the lower areas of the anterior composite gyrus (CJ III and CE). The first of these bundles lies dorsally in the semioval center. Its fibres run ventro-laterad from areas XM II and XP to turn upwards under the cruciate sulcus. The lower-situated fibres of this bundle end in area CSL, whereas the upper ones tend to the areas PrC (Fig. 5).

The fibres of the second bundle, which has a ventral position in relation to the first one, extend straight ventro-laterally and reach the level of the coronal sulcus, where some of them bend towards the front to enter the coronal gyrus and the others deflect downwards to the anterior composite gyrus.

The next bundle connects the areas of the precentral gyrus with the lower areas of the anterior composite gyrus and the lower portion of the temporal cortex. The fibres of this bundle are not very numerous and they run ventro-laterad in the lateral portion of the semioval center.

The last in this group is the bundle which connects the anterior composite and sylvian gyri with area PoC in the postcentral gyrus and with the marginal gyrus. It is a broad bundle composed of about 10 fascicles which descend from the dorso-medial portion ventro-laterad. The upper portion of this bundle runs through the dorsal region of the semioval center, next under and across the coronal sulcus to penetrate into the postero-ventral part of the anterior composite gyrus. The lower portion passes through the middle of the semioval center and turns

ventrally and somewhat to the rear towards the sylvian gyrus (Fig. 5 and 6).

The occurrence of these connections may possibly be explained by the fact that throughout the sensorimotor cortex the layers of medium-sized fibres, which line the sulci and cryptosulci of the other regions in abundance, e.g., the prefrontal region, are very poorly developed. On the other hand, the short connections composed of tangential fibres of the cortex which connect particular cortical areas and subareas are numerous but not very rich in fibres. They can be found in greatest number in the anterior composite gyrus.

Longitudinal fibres of the sensorimotor cortex.

The short associational fibres running along the sulci have been included in the description of the associational bundles. There remains only the system of long fibres parallel to the coronal sulcus to be discussed. It consists of two components, of which one, having an inferior position, contains fibres belonging to the superior longitudinal bundle. These fibres run on the dorso-lateral edge of the corona radiata and mingle with the fibres of the corona radiata where, in the front, these last fibres bend towards the parasagittal plane. Together with them, the longitudinal associational fibres probably enter both the coronal gyrus and the ventrolateral region of the anterior composite gyrus.

The other component comprises fibres which go from the parietal cortex oro-ventrally along the coronal sulcus to the coronal and anterior composite gyri. They are situated dorsally to the first component and are slightly more numerous.

The fibres of the longitudinal system are all alike, for the most part of medium thickness, though there are also some thin ones, and generally well stained.

DISCUSSION

The sensorimotor cortex differs in respect of myeloarchitectonics from the prefrontal cortex in its larger number of projectional and commissural fibres and the smaller number of associational fibres. I think that this difference may be explained, on one hand, by the small area covered by this region in the dog and, on the other hand, by the huge number of fibres which conduct sensory stimuli from the thalamus and carry commands to muscles. The large number of commissural fibres can also be explained easily by the need of synchronization of the right and left sides of the body.

Strong myelination and lack of distinct differentiation of the projectional and commissural fibres of the sensorimotor cortex in so far

as their calibre is concerned are characteristic features of these fibres. This uniformity and the same basic direction of the fibres (both commissural and projectional fibres enter the gyral white matter from the ventral and medial sides) make it impossible to distinguish these two types of fibres from each other throughout their course. Neither is it possible to find whether the commissural fibres are collaterals of the projectional fibres, as some authors suppose (e.g., Krieg 1965), though the angle (nearly 90°) at which they cross one another in the semioval center makes this supposition probable.

The associational connections, mainly medium-sized and short ones, are decidedly less numerous in the sensorimotor cortex than in the prefrontal. This agrees with the opinion on the role of the prefrontal cortex, which is rather associatory. Only one major system of medium-sized associational bundles was found in the white matter of the sensorimotor region, i.e., the transverse system, described above and connecting the medial areas with the lateral ones and the medial and dorsal areas with the ectosylvian gyrus. These connections traverse the semioval center, which fact has hardly ever been observed in the prefrontal cortex; instead, here there are hardly any broad bundles of *b* and *c* type fibres, which so abundantly line the floors of the sulci of the prefrontal region. The superficial tangential bundles also comprise a smaller number of fibres; for instance, in the places analogous with those in which, in the prefrontal cortex, a bundle is composed of some dozen fibres, in the sensorimotor cortex it often consists of at most several and generally 2—5 fibres.

Bundles of associational fibres which run along sulci were also observed. Of these the bundle comprising most nerve fibres passes along the coronal sulcus and connects together the anterior composite, coronal and ectosylvian gyri. Two far weaker bundles can be traced along the ansate and central sulci.

The connection with the temporal cortex is maintained by the lowest portion of the sensorimotor transverse system (see p. 118). De Vito and Smith (1959) inferred the existence of this system from the data obtained by them during their degenerative studies on the macaque. They wrote about this system as composed of "intracortical fibres running through the semioval center". However, the anatomical observations made on the dog do not indicate that the fibres are intracortical, because this term is applied for short associational fibres which do not enter the white matter. I should rather regard them as medium-sized associational fibres which pass through the semioval center, enter the cortex as radiation fibres, and connect together the top areas of the

medial and lateral regions of the sensorimotor cortex and its medial areas with the cortex of the superior areas of the temporal lobe.

As for the connections with the cingulum, these have been found only for the precruciate gyrus, there being no connections of the cingulum with the sensorimotor areas situated posteriorly to the cruciate fissure. This would coincide with the results of De Vito and Smith (1959) who, on the basis of degeneration studies, described a connection in the macaque corresponding to the connection of the cingulum with the precruciate gyrus in the dog.

Myeloarchitectonically the white matter of the prefrontal region differs from that of the sensorimotor region. The chief differences may be found in the myelination and number of the projectional fibres and the organization and number of the associational fibres.

SUMMARY

The semioval center and gyral white matter have been divided into parts according to the direction of fibres in the upper layer of the semioval center and their reference to appropriate gyri of the cerebral cortex. The existence of two types of the gyral white matter has been demonstrated. The first of them contains fibres that go to only one gyral area and, eventually, several lateral ones, whereas the fibres of the white matter of the second type, which have ramified once within the semioval center, branch again, tending to two or more gyral areas. Stratified organization has been found in the portion of the semioval center related to the sensorimotor cortex. Layers, each having a somewhat different arrangement of fibres, have been observed. It is impossible to distinguish commissural and projectional fibres from each other in the gyral white matter.

The white matter of the sensorimotor cortex is marked by a larger number of fibres than in the prefrontal cortex and their heavier myelination. Almost no thick layers of associational fibres can be seen lining the floors of the fissures; instead, there is a transverse system which connects the medial side of the sensorimotor cortex with its lateral side as well as the temporal lobe. Associational fibres pass also along the fissures. Their largest bundle runs along the coronal sulcus and connects the anterior composite gyrus and the coronal gyrus with the ectosylvian.

In general, the sensorimotor cortex is connected by medium-sized and short associational fibres with the prefrontal, parietal and temporal cortex and by long fibres with the limbic lobe (cingulun) and parietal cortex (superior fronto-occipital and superior longitudinal bundles).

List of abbreviations

ag, album gyri
cc, corpus callosum
c in, capsula interna
cing, cingulum
cs, centrum semiovale
ffos, fasciculus fronto-occipitalis superior
fls, fasciculus longitudinalis superior
fm, forceps minor
g ca, gyrus compositus anterior
g ea, gyrus ectosylvius anterior
g ep, gyrus ectosylvius posterior
g l, gyrus lingualis
g p, gyrus proreus
g poc, gyrus postcentralis
g pr, gyrus precentralis
g s, gyrus sylvius
g sa, gyrus suprasylvius anterior
g sp, gyrus suprasylvius posterior
H, hipocampus
ll, lower layer
ml, middle layer
nc, nucleus caudatus
s a, sulcus ansatus
s cr, sulcus cruciatus
s pr, sulcus presylvius
s rha, sulcus rhinalis anterior
s spr, sulcus suprasylvius anterior
ul, upper layer

Symbols of the myeloarchitectonic areas and associational bundles are described in the text.

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