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The Neostriatal Inhibition of Catalepsy, but not of Muscle Rigidity, Evoked From the Substantia Nigra Pars Reticulata

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KOLASIEWICZ, W., A. COOLS, K. OSSOWSKA AND S. WOLFARTH. *The neostriatal inhibition of catalepsy, but not of muscle rigidity, evoked from the substantia nigra pars reticulata.* PHARMACOL BIOCHEM BEHAV **28**(4) 453–457, 1987.—The effects of a bilateral blockade of neo- and palleostriatal GABAergic mechanisms on catalepsy and muscle rigidity resulting from picrotoxin injection into the substantia nigra pars reticulata (SNR) were studied. The catalepsy and rigidity were induced by a unilateral injection of 100 ng/0.5 μl of picrotoxin. Bilateral injections of 250 ng/1 μl of picrotoxin into the intermediate-ventral parts of the caudato-putamen (CP) abolished the catalepsy but had no effect on the muscle rigidity induced by an intranigral injection of the drug. Bilateral injections of 250 ng/1 μl of picrotoxin into the globus pallidus (GP) did not influence the catalepsy and rigidity induced by the intranigral injection of the drug. The results indicate that the impulses, connected with the catalepsy evoked from the SNR seem to be transmitted back to the CP and blocked therein by inhibition of GABAergic synapses in its intermediate-ventral part. The impulses, connected with the muscle rigidity evoked from the SNR, presumably do not return to the striatum.

Catalepsy Muscle rigidity GABA mechanisms Neostriatum Substantia nigra pars reticulata

γ-AMINOBUTYRIC acid (GABA) is known to act as a transmitter in all main efferent systems of the neostriatum that project to the globus pallidus, entopeduncular nucleus, and substantia nigra pars reticulata (SNR) [1, 13, 27, 28, 31]. According to DiChiara *et al.* [13], Scheel-Krüger *et al.* [27,28], Turski *et al.* [33] and Ellenbroek *et al.* [15], the strionigral GABAergic pathway is the major neuronal tract transmitting catalepsy, rigidity, turning behaviour and other symptoms related with stimulation or inhibition of striatal dopamine receptors.

Many authors tried to determine which nigral efferent systems might be engaged in transmitting the above-mentioned behavioural effects farther on—beyond the region of the SNR [13, 15, 23, 32, 34]. A number of data indicate that the strionigral pathway forms monosynaptic inputs to nigrothalamic and nigrotectal GABAergic pathways [4, 6, 7, 9, 12, 13, 17, 30, 35] and mono- and/or polysynaptic inputs back to the nigrostriatal dopamine pathway [6–8, 26, 29]. However, Cools *et al.* [11] demonstrated that behavioural effects of picrotoxin microinjections into the SNR most probably were not transmitted back to the striatum via the nigrostriatal pathway, as they were only poorly modified by a direct intrastriatal administration of haloperidol and apomorphine. On the other hand, Kemmel *et al.* [22] showed that stimulation of nigral GABA receptors results in changes in the release of [^{3}H]GABA in the striatum. Therefore it may be supposed that the effects evoked by the stimulation of nigral GABA receptors are transmitted to the striatal GABA system with the avoidance of the nigrostriatal pathway.

To answer this question we decided to find out whether behavioural effects of inhibition of the GABA transmission in the SNR may return to the striatum via pathways other than the nigrostriatal one. To this end we administered picrotoxin into different parts of the SNR and studied the influence of simultaneous intrastriatal injections of that drug on the behavioural effects of picrotoxin administration to the SNR.

The seemingly most important connection between the SNR and the striatum passes through some thalamic nuclei [6, 7, 12, 20, 22, 32, 34, 35]. As the axons leaving the SNR in the direction of thalamus partly cross the midline and reach the bilateral thalamic nuclei [6, 17, 22], picrotoxin was injected intrastriatally onto both sides.

METHOD

The experiments were carried out on male Wistar rats, weighing 200–250 g at the time of surgery. The rats were stereotaxically implanted under pentobarbitone (Vetbutal, Biowet, Poland, 30 mg/kg IP) anaesthesia with stainless steel guide cannulae (0.4 mm o.d.) unilaterally in the central or caudal part of the substantia nigra pars reticulata (SNR) (A=1.9–1.7, L=2.0, H=−2.3 for the central group and A=1.6–1.2, L=2.2–2.4, H=−2.0–2.4 for the caudal group)

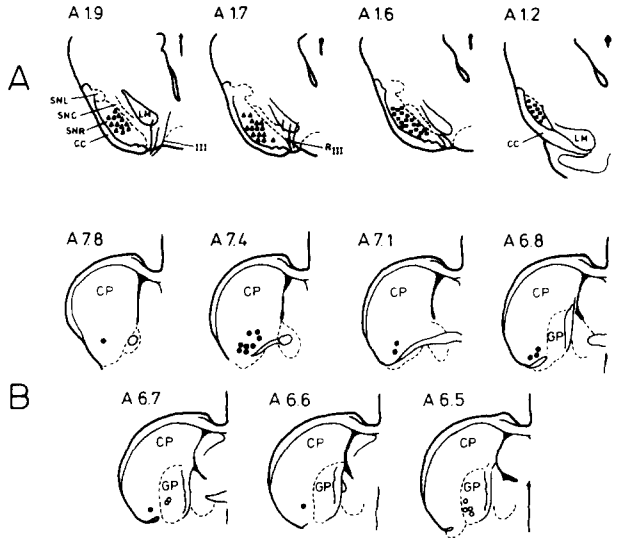

FIG. 1. A diagrammatic presentation of the localization of cannula tips on frontal sections of the rat substantia nigra (A) and caudate-putamen (B), according to König and Klippel [24]. A: anterior plane; CP: caudate-putamen; GP: globus pallidus; LM: lemniscus medialis; R III: radix nervi oculomotori; SNC: substantia nigra pars compacta; SNL: substantia nigra pars lateralis; SNR: substantia nigra pars reticulata; filled squares: cannula tips in SNR (wherefrom picrotoxin induces motor disturbances and catalepsy); filled triangles: cannula tips in SNR (wherefrom picrotoxin induces motor disturbances only); circles: cannula tips in CP or GP, where injections of picrotoxin were effective (filled) or ineffective (open) against the intranigral picrotoxin-induced catalepsy; two bilateral cannula tips in each rat were presented on one (left) side of the frontal section.

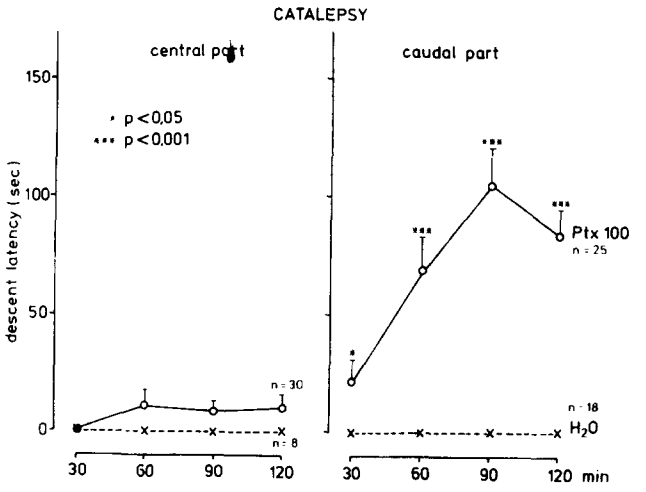

FIG. 2. Catalepsy (9-cm cork test) evoked by 100 ng of picrotoxin (Ptx) injected unilaterally into either central or caudal parts of the substantia nigra pars reticulata (SNR).

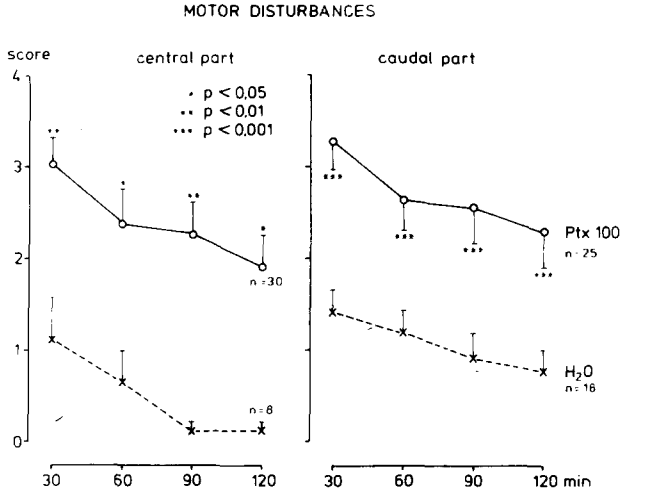

FIG. 3. Motor disturbances (pull-up test) evoked by 100 ng of picrotoxin (Ptx) injected unilaterally into either central or caudal parts of the substantia nigra pars reticulata (SNR).

(Fig. 1). Other groups of rats were additionally implanted with stainless steel guide cannulae (0.4 mm OD) bilaterally in the intermediate-ventral part of the caudate putamen (CP) (A=7.8–6.5, L=2.6–3.2, H=−0.6–1.4) or in the globus pallidus (GP) (A=6.5, L=2.7, H=−1.4) (Fig. 1)—according to the atlas of König and Klippel [24].

Intracerebral injections were made in conscious rats with a stainless steel injection cannula (0.3 mm o.d.), protruding from the guide cannula by 0.3 mm, at a speed of 0.2 μl/min. After the termination of injection the injection cannula was left in place for another 1 min. After at least 1-week recovery period, picrotoxin (Ptx) (Sigma) was administered intranigrally—unilaterally in a dose of 100 ng/0.5 μl, and/or intrastriatally or into the GP—bilaterally in a dose of 250 ng/1 μl. Intrastriatal injections were started at 1 min after the termination of intranigral administration. Bidistilled water was injected as a control at a volume of 0.5 μl into the SNR, and at a volume of 1 μl into the CP. Behavioural tests were carried out 30, 60, 90 and 120 min after the intranigral injection. Catalepsy was assessed using a 9-cm cork test. The time of keeping both fore paws on the cork up to 180 sec was measured. In the pull-up test (used as an efficiency test for hind limbs) the animals were hung with both fore paws on the edge of a wooden bar (2.5×2.5×30 cm) placed 60 cm above the floor. The test was scored using a 5-point scale, where 0 was scored by a rat which pulled up both its hind limbs and instantly climbed the bar, and 5 points were scored by a rat which was able neither to climb the bar (within 15 sec) nor to pull up its hind limbs and grasp the bar with either of them; 4 points were given to a rat which was unable to climb the bar within 15 sec and was able to pull up one limb only. Details of the scoring system were described previously [35].

After the completion of experiments localization of all the cannula tips within the rat brain was checked histologically (Fig. 1). The statistical evaluation of the results was performed using the Student *t*-test.

RESULTS

The effects of unilateral injections of picrotoxin into the SNR were dependent on the position of the tip of the injection cannula on the rostro-caudal plane (Fig. 1). Accordingly, the SNR was divided into regions located rostrally or caudally to A=1.7 (last trace of the *radix nervi oculomotori*), i.e., placed between A=1.7–1.9 and A=1.6–1.2 respectively, and further on called central and caudal parts of the SNR.

Already after 3 min the unilateral injections of picrotoxin into both parts of the SNR evoked a marked enhancement of

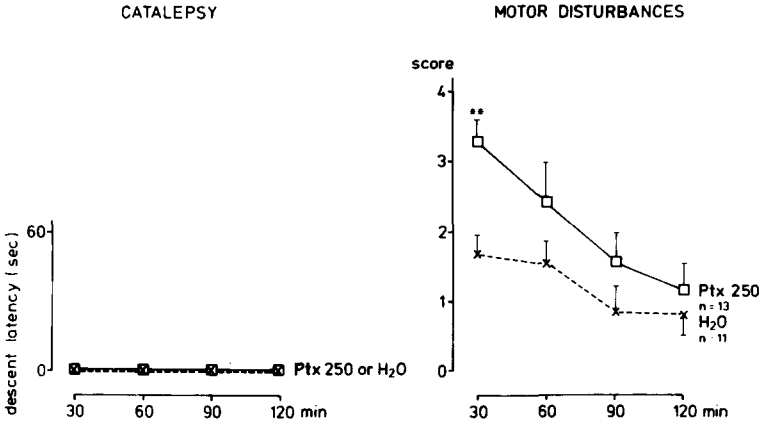

FIG. 4. Catalepsy (9-cm cork test) and motor disturbances (pull-up test) evoked by 250 ng of picrotoxin (Ptx) injected bilaterally into the intermediate-ventral parts of the caudate-putamen (CP).

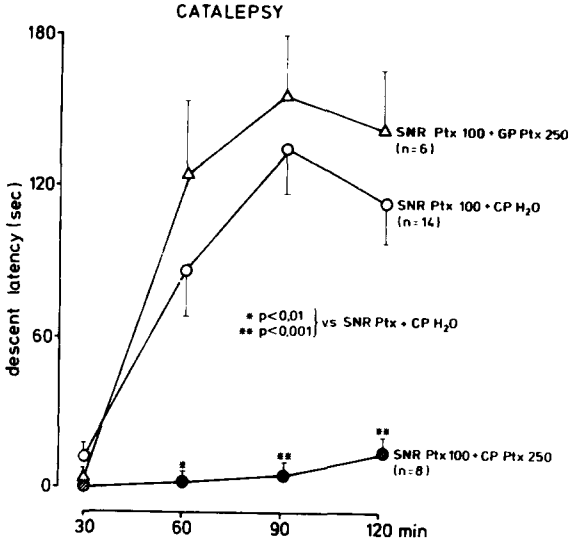

FIG. 5. The effects of bilateral picrotoxin (Ptx, 250 ng) injections into either intermediate-ventral parts of the caudate-putamen (CP) or the globus pallidus (GP) on the catalepsy evoked by a unilateral injection of 100 ng of picrotoxin into a caudal part of the substantia nigra pars reticulata (SNR).

the locomotor activity, episodes of recognizing the environment, sniffing, and a particular kind of "oral dyskinesia" consisting of unnatural movements of the tongue, chewing movements and catching with teeth (not really biting) of anything accessible in the environment including parts of their own body. Simultaneously also asymmetric movements were seen: large, slow, mainly ipsilateral turns after injections into the central part of SNR, and quick, mainly head-to-tail, contralateral turns after injections into the caudal SNR. Those effects lasted for a shorter time after an injection into the caudal SNR and were followed, approx. 30 min after the injection, by an almost total long-lasting immobility.

A unilateral injection of picrotoxin (100 ng/SNR) into the caudal part of the SNR evoked, beginning at 30 min thereafter, a strong and long-lasting catalepsy, whereas similar injections into the central parts were unable to evoke it (Fig. 2). Injections of picrotoxin into both those parts of the SNR evoked similar motor disturbances, as measured by the pull-up test (Fig. 3).

A bilateral injection of 250 ng of picrotoxin into the CP (intermediate-ventral part of the caudatus-putamen, Fig. 1) evoked only slight motor disturbances (pull-up test) within the first 30 min after the injection, and no catalepsy (Fig. 4). Within the first 20 min after the injection also a moderate enhancement of the locomotor activity and sniffing and chewing episodes could be seen. Bilateral injections of 250 ng of picrotoxin into the CP, started 1 min after intranigral administration, abolished the catalepsy evoked by a unilateral injection of 100 ng of the drug into the SNR (caudal part) (Fig. 5). Injections into the CP were without effect on motor disturbances evoked by an intranigral injection of picrotoxin. Bilateral injections of 250 ng of picrotoxin into the GP had no effect on both the catalepsy and motor disturbances (pull-up test) evoked by an intranigral injection of the drug (Fig. 5).

DISCUSSION

The present results point to the separability of GABA mechanisms responsible for catalepsy and muscle rigidity within the SNR and CP. This assumption is supported by the observation that inhibition of the GABA transmission in the caudal part of the SNR by picrotoxin evokes catalepsy and motor disturbances, whereas inhibition of the GABA transmission in the central parts of the SNR induces motor disturbances only. Havemann *et al.* [19] reported the occurrence of muscle rigidity, which was measured electromyographically after injection of bicuculline—another antagonist of the GABA transmission, into that region of the SNR. It seems, therefore that motor disturbances observed after injection of picrotoxin into the SNR are due to muscle rigidity. This concept is supported by our own unpublished results (Kolasiewicz *et al.*), according to which enhanced resistance of flexor and extensor muscles of the hind paw to a forced extension and flexion of the paw occurred after intranigral injections of picrotoxin. Therefore the motor disturbances assessed by the pull-up test are further considered to be due to muscle rigidity.

The occurrence of a peculiar kind of dyskinetic movements, found in the mouth region of rats receiving intranigral injections of picrotoxin, seems to be worth stressing. Similar diskinetic movements were previously described by Arnt and Scheel-Krüger [2] after injections of GABA antagonists into the same nigral region. Those observations may be of some importance in relation to the well-known late effects of the neuroleptic treatment (tardive dyskinesia).

The obtained results suggest that at least part of the impulses (increase or decrease in the impulse flow) evoked in the caudal part of the SNR and resulting in catalepsy return continously to the CP where they are blocked by inhibition of the GABA transmission by picrotoxin. Conversely, the impulses evoked by picrotoxin injection into the caudal part of the SNR and leading to a muscle rigidity do not seem to go back to the CP, as the latter effect is not influenced by picrotoxin injected into both the CPs. It might be supposed that the CP is involved in a neuronal loop responsible for maintaing catalepsy, i.e., the loop within which the impulses turn continously round. Conversely, as regards muscle rigidity, the CP seems to represent one of the stations in an open neuronal chain only, maybe the initial station.

Which pathways are taken by the impulses going back to the CP? In spite of many efforts made in the past three decades, the picture of connections which are responsible for catalepsy and muscle rigidity is still unclear. Therefore the explanation of the present results in terms of structures and pathways involved in mediation of these effects must be speculative. It was shown [22] that a unilateral injection of muscimol into the substantia nigra may affect the [^{3}H]GABA release in bilateral caudate nuclei. Moreover, the contralateral effects could be influenced by lesions of the thalamic nuclei [22], which seems to suggest involvement of polysynaptic uni- and bilateral projections, including nigro-thalamo(cortico)-striatal pathways, in the mediation of catalepsy [3–7, 12, 13, 17, 20–22, 25]. Therefore it might be supposed that in the present experiment picrotoxin injections into the SNR could influence not only the ipsilateral CP but also the contralateral one. In order to block also those crossing impulses which transmit the nigral influence to the contralateral CP, in the present experiment picrotoxin injections were made to both the bilateral CPs.

It was reported that bilateral injections of muscimol into the ventromedial thalamic nuclei [13, 23, 32, 35] evoke catalepsy. This finding is in line with our hypothesis that the impluses generated by picrotoxin in the caudal SNR and connected with catalepsy are transmitted back to the striatum via the nigro-thalamo(cortico)-striatal pathway. This hypothesis is further supported by the fact that, like intrastriatal injections, bilateral injections of picrotoxin into the ventromedial thalamic nuclei abolish the catalepsy evoked by injection of the drug into the SNR (Kolasiewicz, Ossowska, Wolfarth—unpublished data).

A number of results point to the striatum as an initial station not only for catalepsy but for rigidity as well. It was shown that both morphine [18] and neuroleptics [14–16] injected into the striatum are able to induce muscle rigidity. Moreover, muscimol injected into the same region of the striatum (CP) as in our experiment evoked both catalepsy and muscle rigidity (EMG study) [33]. The following station seems to be the SNR, as both picrotoxin (present study) and bicuculline [19] injected into it evoke rigidity. On the other hand, the SNR is an initial station of three GABA pathways: to the ventromedial thalamic nucleus, to the colliculus superior and to the reticular formation [4, 6, 7, 12, 13, 17, 20, 34]. Muscimol injected bilaterally into the ventromedial thalamic nuclei induces muscle rigidity and catalepsy [23], whereas its injection into the superior colliculus evokes muscle rigidity without catalepsy [15]. Neuronal connections conveying farther on the muscle rigidity are still unknown. However, our present results suggest, that the impulses connected with this symptom seem not to return to the striatum.

The neostriatum (CP) is the largest basal nucleus, and the non-homogeneity of its structure—both anatomical and functional—is well known [5, 10, 14, 16, 33]. Different synaptic mechanisms seem to mediate catalepsy and muscle rigidity within the CP [16]. Therefore it cannot be excluded that some other regions within the caudate nucleus would be more effective, maybe by means of different receptor inputs, in antagonizing the rigidity evoked from the SNR. Nevertheless, the results obtained so far speak for the involvement of the caudate nucleus in the neuronal loop responsible for catalepsy, whereas this structure seems to be only an initial station for the muscle rigidity.

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