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Dopamine mediated circling behaviour is modulated by lesions of the ventromedial nucleus of the thalamus

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The destination of basal ganglia efferent pathways responsible for apomorphine-induced circling in rats with striatal dopamine receptor imbalance are unknown. Output pathways from the zona reticulata of the substantia nigra are involved, but not the major outflow to the superior colliculus (Jenner, Leigh, Marsden & Reavill, 1979). Other structures receiving fibres from the substantia nigra include the ventromedial (VM) and parafascicular (PF) nuclei of the thalamus (Herkenham, 1979). We now report the effect of lesions of these bodies on circling behaviour in male Wistar rats.

Unilateral electrolytic lesions of VM (A 4.8; L 1.2; V − 1.3) or PF (A 3.4; L 1.0; V − 0.8) (De Groot, 1959) 15 days previously did not cause spontaneous turning. Apomorphine hydrochloride (0.5 mg/kg s.c. 15 min previously) or amphetamine sulphate (3 mg/kg i.p. 30 min previously) caused slow wide ipsiversive turning (<2 turns/min) in both VM and PF lesioned animals.

Animals receiving unilateral 6-hydroxydopamine (6-OHDA; 8 μg/3 μl 0.9% saline) lesions of the medial forebrain bundle (MFB) at the level of the left lateral hypothalamus (A 4.6; L 1.9; V − 3.0) 9 days previously showed tight contraversive circling to apomorphine (18.8 ± 3.9 turns/min) and ipsiversive circling to amphetamine (10.5 ± 0.7 turns/min). Subsequent electrolytic lesioning of VM on the side of the 6-OHDA lesion caused a 60% reduction in apomorphine (control animals 18.8 ± 3.9 turns per min; VM lesion 7.5 ± 1.8 turns/min; $P < 0.05$). Amphetamine-induced circling was unaltered (control animals 10.5 ± 0.7 turns/min; VM lesion 8.5 ± 0.4 turns/min; $P > 0.05$). Further, electrolytic lesioning of VM on the opposite side to the initial 6-OHDA lesion caused a 86% reduction in amphetamine-induced circling (control animals 7.1 ± 2.4 turns/min; VM lesion 1.0 ± 0.5 turns/min; $P < 0.5$) while apomorphine-induced circling was unaltered (control animals 11.4 ± 2.7 turns/min; VM lesion 11.0 ± 1.6 turns/min; $P > 0.05$).

Circling to apomorphine or amphetamine in animals with an unilateral 6-OHDA MFB lesion was unaltered by a subsequent unilateral PF lesion irrespective of side.

This evidence would suggest an important role of VM in the efferent pathway controlling circling behaviour. However, subsequent bilateral lesioning of VM or PF in animals with a unilateral MFB lesion failed to inhibit circling in response to apomorphine or amphetamine. This may indicate that VM modulates striatal induced circling rather than acting as a critical basal ganglia outflow station mediating the turning behaviour.

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