


BULBAR AND PONTINE SOURCES OF CATECHOLAMINE INNERVATION OF THE RAT SPINAL CORD INVESTIGATED BY MONOAMINE FLUORESCENCE AND RETROGRADE LABELING TECHNIQUES

The organization of sources providing catecholaminergic innervation of the upper thoracic spinal cord was investigated in rats using a sensitive technique specific to tracing connections between catecholaminergic neurons. Large numbers of pontine catecholaminergic neurons belonging to groups A5, A6, and A7 and some single cells from medullary group A1 send out direct projections to the spinal cord. It is postulated that pontine catecholaminergic neurons contributing to innervation of the spinal cord and the medulla are noradrenaline-containing and adrenaline-containing respectively.

INTRODUCTION

The spinal cord is generously innervated by catecholaminergic fibers. Especially large numbers of noradrenergic terminals have been discovered in the sympathetic intermediolateral trunk, the ventral horn, and the substantia gelatinosa of the dorsal horn of the spinal cord gray matter [9, 16]. Catecholaminergic terminals in the spinal cord are known to be of exclusively supraspinal origin. The main sources of catecholaminergic innervation of the spinal cord were previously thought to be bulbar neuronal groups A1 and A2 according to the findings of Dahlstrom and Fuxe [8], on the evidence of findings obtained during experiments determining the degree of fluorescence in brainstem catecholamine-containing cells following section at different points on the spinal cord [9, 16, 25] and the results of studies on the location of horseradish peroxidase (HP) labeled neurons within the brain following microinjection of this enzyme into different segments of the spinal cord [15]. The development and use of techniques for examining connections between chemically identified neurons radically changed previously held views on supraspinal catecholaminergic innervation of the spinal cord. A major role in catecholaminergic innervation of the spinal cord is now attributed to pontine neurons, although findings on this subject have proved highly contradictory [19, 23, 27].

Descending influences on the spinal cord implemented by monoaminergic pathways are known to modulate or control the activity of spinal neurons belonging to the autonomic, somatomotor, and sensory systems, thereby largely determining the course of spinal reflexes. By shedding light on the detailed structure responsible for integrating catecholaminergic neuronal systems

Fig. 1. Distribution of catecholamine-containing and retrogradely primulin (Pr)-labeled neurons in the rat bulbar caudal enlargement following injection of fluorochrome into the upper thoracic segments of the spinal cord; diagrams 1-3) frontal planes of brain slices in a rostro-caudal direction. Dots denote Pr-labeled; circles: catecholaminergic cells; triangles: Pr-labeled catecholaminergic neurons; AP: anterior field; C: cuneate nucleus; DP: decussation of pyramids; G: nucleus gracilis; IO: inferior olive; LRN: lateral reticular nucleus; MV: medial vestibular nucleus; NTS: nucleus tractus solitarius; P: pyramids; RF: reticular formation; SNV: nucleus of the spino-cerebral pathway of the trigeminal nerve; X: dorsal motor nucleus of the vagus nerve; XII: nucleus of the hypoglossal nerve.

Fig. 2. Location of retrogradely Pr-labeled catecholaminergic neurons in the medulla following injection of Pr into the upper thoracic segments of the spinal cord. Notations as for Fig. 1.

with spinal cord cells, a number of mechanisms underlying descending supraspinal control of brain function can thus be clarified.

This article analyses the contribution of catecholaminergic (i.e., noradrenaline- and adrenaline-containing) neuronal groups of the hindbrain to innervation of the upper thoracic segments of the rat spinal cord. The "Faglu" highly specific and sensitive catecholamine fluorescence method was combined with HP retrograde axonal transport techniques and primulin (Pr) labeling during this study.

METHODS

Experiments were conducted on 15 white rats of both sexes weighing 150-250 g. The animals were injected on one side of segments Th₁-Th₃ of the spinal cord with 4-6 µl of 10% Pr prepared in a 2% aqueous dimethylsulfoxide solution using a microsyringe and a glass micropipet under Nembutal-induced anesthesia (35 mg/kg). The animals were perfused transcardially under deep anesthesia 3-6 days later. A warm hypertonic solution of sodium chloride was briefly passed through and then perfusion proceeded for 1.5-2.0 h with a cold fixing agent at a temperature of 2-4°C consisting of 3.5% paraformaldehyde, 0.25% glutaraldehyde, and 5% sucrose in solution in phosphate buffer (0.1 M; pH 7.2); volume of fixing agent: 1-1.5 liters. To end perfusion a cold 10% sucrose solutions was passed through for 0.5-1 h. The perfusing a cold 10% sucrose solution was passed through for 0.5-1 h. The perfusing system