Antidromic latency variations of nigral compacta neurones

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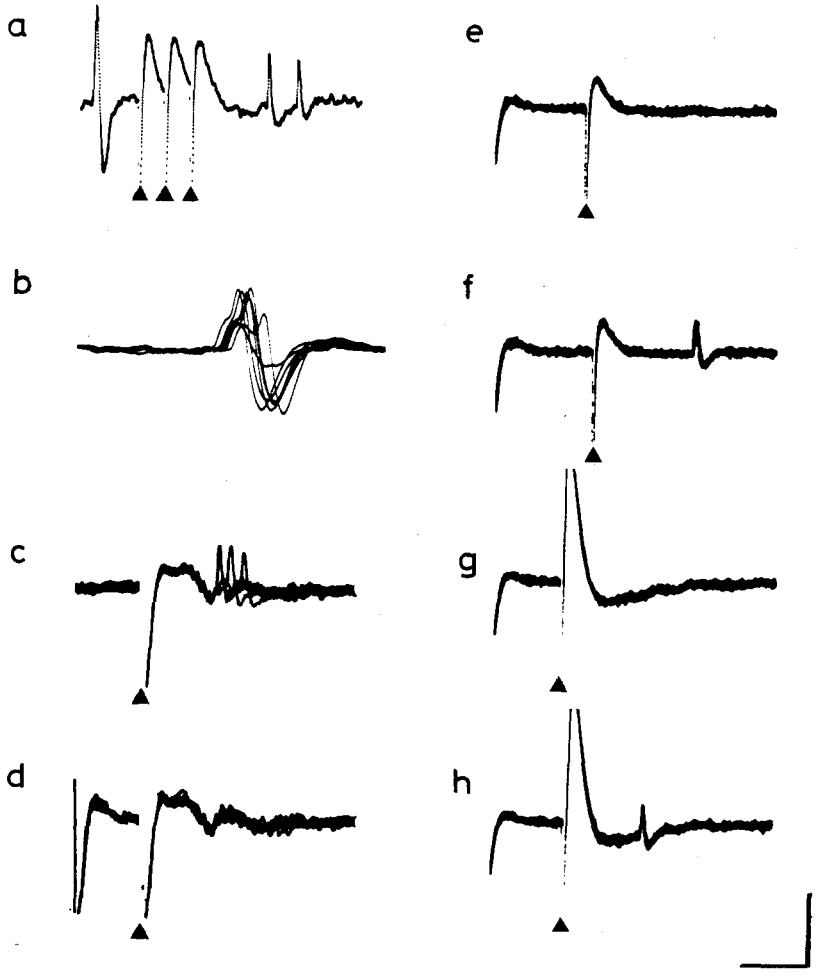
Summary. Variations in the antidromic latency of substantia nigra compacta neurones were commonly observed following striatal stimulation. These results provide electrophysiological evidence for a branched unmyelinated nigrostriatal pathway and demonstrate that the antidromic criterion of constant latency is not valid for this type of pathway.

The dopaminergic nigrostriatal pathway consists of highly branched, unmyelinated axons[1,2]. Recently, a pathway from the substantia nigra pars compacta to the striatum has been described using antidromic invasion techniques[3-5], and may correspond to the dopaminergic pathway as it is absent in 6-OHDA-treated rats[5]. In the present study, we report variations of the antidromic latency consistent with this pathway comprising of branched unmyelinated axons.

Experiments were performed on male and female albino rats weighing approximately 200 g anaesthetized with either urethane (1.2-1.4 g/kg i.p.) or halothane (0.5-1.0% in oxygen). The substantia nigra was approached dorsally with single barrelled glass electrodes filled with either 4 M NaCl or 0.5 M Na acetate containing 2% pontamine sky blue dye. Extracellular action potentials were amplified and displayed conventionally, and stimulation of the ipsilateral striatum (0.3 msec pulses, 0.05-3 mA delivered at 1-500 Hz) and histological verification of electrode placements were as described previously[6].

Following ipsilateral striatal stimulation 119 neurones were antidromically invaded with a mean ± SEM latency of 14.4 ± 0.4 msec giving a mean conduction velocity of approximately 0.5 m/sec. These cells had wide action potentials (4-6 msec) which often showed noticeable initial segment (IS) and large late positive components. Cell firing was usually regular and always below 8 Hz (mean firing rate 3.80 ± 0.20 Hz), and histogical examination showed that most cells were located in the zona compacta.

Antidromic invasion consistently produced an IS spike which in many cells sometimes lead to a full spike. In response to a stimulus of constant strength there were usually slight variations in the IS latency and larger variations in the somadendritic (SD) latency (figure 1,b). Additionally, in a few cells the antidromic spike appeared at 2 or 3 separate latencies (figure 1c-d). In a total of 20 cells a shorter latency could be produced by increasing the stimulus intensity. These latency jumps ranged from 1 to 10 msec and in some cells multiple latency jumps could be produced. For example, the cell illustrated in the figure, e-h, had 5 antidromic latencies (only 2 are illustrated). Some cells never displayed latency jumping despite exhaustive testing. The IS antidromic spike always followed stimulus



Response of compacta neurones to striatal stimulation. *a* Single sweep showing frequency following of IS spike at 333 Hz. No antidromic spike appeared after the first shock of the volley because of collision with the full spontaneous spike which can be seen at the extreme left of the trace. *b* Expanded time base of 7 sweeps showing variations in IS and SD latencies of the antidromic spike of another SNC neurone in response to a constant stimulus. *c* and *d* Each record is 5 superimposed sweeps from the same neurone illustrating the response to a constant stimulus. In (c) the IS antidromic action potential appeared at 1 of 3 separate latencies (10.5, 12.0 or 14.0 msec). In (d) no antidromic spike appeared due to collision with a spontaneous spike (partially illustrated on left of trace). *e-h* Each record is 10 superimposed sweeps from the same neurone, showing collision of the antidromic spike by stimulating 15 msec (e) but not 16 msec (f) after a spontaneous spike (stimulus intensity 0.28 mA, antidromic latency 14 msec). Similarly, collision 11 msec (g) but not 12 msec (h) after the spontaneous spike when stimulus increased to 2.6 mA (antidromic latency 11 msec). Calibration: horizontal a, c-h 10 msec, b 2 msec; vertical a-h 1 mV.

frequencies upto 250 Hz and sometimes upto 500 Hz (figure, a), and could always be collided with an appropriately timed spontaneous spike (figure, a, c–h).

In agreement with previous reports[3–5], a distinct population of cells could be recorded in the zona compacta of the substantia nigra which had wide action potentials, slow firing rates and were antidromically invaded following striatal stimulation. Also, in agreement with previous observations[4] slight variations in SD latency were seen which reflected the excitability of the somadendritic membrane and hence the rate of axon-soma invasion[7]. However, in the present study slight variations in the IS latency were also observed in many cells. These variations could be attributed to changes in axon excitability following conduction of the previous orthodromic action potential, a phenomenon described in other unmyelinated central axons[8–11].

More noticeable were the latency jumps of the antidromic spike in response to a constant or altered stimulus. 1, or occasionally 2, latency jumps have been reported in certain hypothalamic neurones[8,12–15] and have been attributed to activation along different parts of branched or twisted axons. Multiple latency jumps of the extent observed in some neurones in the present study have not been reported before. However, the dopaminergic nigrostriatal pathway is highly branched[2] and this extensive branching may account for the multiple latencies seen. Presumably, with increasing stimulus strength the current spreads to faster conducting branches or to activation points closer to the cell soma.

Finally, the variations in antidromic latency reported here may explain in part failure of earlier workers to antidromically identify compacta neurones[16].

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Diurnal variations in serum and liver zinc levels throughout the 4-day estrous cycle of the hamster

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Summary. Diurnal variations of serum zinc level and liver zinc level of the golden hamster were found throughout the 4-day estrous cycle, and on the different days of the cycle, the patterns of fluctuation differed.

It is well known that zinc metabolism in animals is regulated homeostatically[1]. Although the diurnal changes of zinc levels in serum have been noted in man and in other mammals[2–5], such changes in other organs or changes in relation to the reproductive cycle are scarcely known as yet. We therefore examined the serum zinc concentrations and liver zinc concentrations at selected times of the day throughout the 4-day estrous cycle in the golden hamsters *(Mesocricetus auratus)*.

Adult virgin female golden hamsters weighing 135±23 g (mean ±SD, n=133) were kept under controlled conditions of temperature (23±3 °C), relative humidity

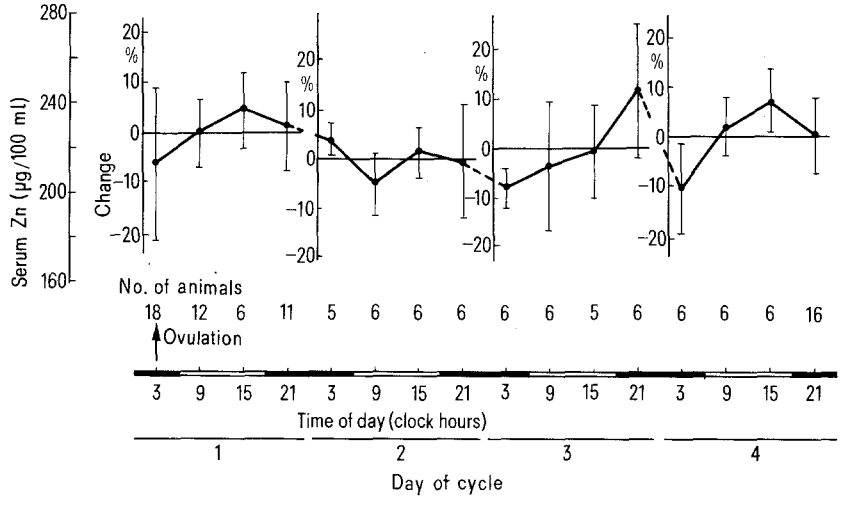

Fig. 1. Serum zinc concentrations of female hamsters throughout the 4-day estrous cycle. Points indicate the mean values ±SD in each group. The number of animals per group is shown in the figure. The analysis of variance revealed statistically significant diurnal changes on days 3 ($p < 0.05$) and 4 ($p < 0.01$) of the cycle. Also, significant changes with the day of the estrous cycle were observed at 09.00 h ($p < 0.05$) and 15.00 h ($p < 0.05$).