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EFFECTS OF ELECTRICAL STIMULATION IN THE STRIATUM ON BLADDER ACTIVITY IN CATS

Hypothesis / aims of study

In Parkinson's disease (PD) in which basal ganglia is affected, micturition disturbance is common[1)]. Because PD is caused by degeneration of nigrostriatal dopaminergic projections, dysfunction of these systems might be related to the pathogenesis of bladder hyperactivity. Recent studies reported that neural circuitry of basal ganglia has an inhibitory effect on micturitional reflex[2),3)]. However, little has been known about the relationship between the striatum and bladder function. We therefore examined the effects of electrical stimulation of the striatum on the bladder contraction, and recorded bladder activity related neuronal firing in the striatum in cats.

Study design, materials and methods

Experiments were done on 12 adult male cats under anesthesia with ketamine. The continuous periodic bladder contraction/relaxation cycles were induced by infusing saline volume into the bladder slightly exceeding a threshold volume (20-50ml). After the continuous periodic bladder contraction/relaxation cycles were generated, we stereotaxically inserted microelectrode into the striatum, and performed electrical stimulation and extracellular single unit recording in the striatum. We have examined striatal neuronal activities with relation to bladder contraction/relaxation cycles.

Results

Electrical stimulation applied in the posterior ventral caudate nucleus and the adjacent putamen elicited inhibition of the bladder contraction. None of the responses were facilitatory. The effective amplitude of the electrical stimulation for evoking inhibitory responses was 80-100 □A and higher. Forty-six neurons were recorded in the striatum that were related to bladder contraction/relaxation cycles. Thirty-five neurons were found to be tonically active throughout bladder relaxation phase, and a remaining 11 neurons were firing during bladder contraction phase, with almost constant firing activities. These neurons were located within the area where the electrical stimulation inhibited the bladder contraction.

Interpretation of results

These results indicated that the net effect of electrical stimulation on bladder contraction was inhibitory. Therefore, degeneration of striatal neuron might prevent the inhibition of bladder contraction, which ultimately leads to detrusor hyperactivity. On the contrary, neuronal activities in both the bladder relaxation phase (n=35) and the bladder contraction phase (n=11) were recorded in the posterior ventral striatum in single unit recording. Although we could not explain these discrepancies, several speculations are possible. If we assume that relaxation phase related neurons might actively participate in inhibiting bladder contraction, since the number of the relaxation phase related neurons was larger, the net bladder responses by the electrical stimulation can be inhibitory. Another possibility is that bladder contraction phase related neurons reflect afferent input from the bladder, whereas they might not actively participate in bladder contractions.

Concluding message

In the present study, we have shown that electrical stimulation inhibits the bladder activity and that there is urinary cycle related neuronal firing in the posterior ventral striatum.

References

1) J Neural Transm (Suppl) 1995;45: 11-19
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3) Br J Pharmacol. 2003;139:1425-1432.

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