Monoaminergic modulation of motor axon recurrent collateral pathways in the neonatal rat spinal cord.

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Recurrent circuitry modulating the functional motor output of the *in vitro* neonatal rat spinal cord was examined using a conditioning/test paradigm. Prior to pharmaceutical manipulation, the effects of ventral root conditioning stimulation on test monosynaptic reflex amplitude could produce either inhibitory (n=14/35) or facilitatory actions (n=10/35).

Inhibitory effects are consistent with activation of Renshaw cells via the axon collaterals of motoneurons resulting in recurrent inhibition of motoneurons. Recurrent inhibition was unmasked by the 5-HT receptor antagonist methiothepin (n=5/7) supporting modulatory depression of recurrent inhibition via descending serotonergic pathways.

The excitatory effects of conditioning were likely induced by activation of motor axon collaterals since they were partly blocked by addition of the cholinergic antagonist mecamylamine (3/4). Recurrent excitation could be unmasked with noradrenalin (30-50 μM; n=9/12) supporting a modulatory facilitation via descending noradrenergic pathways. Interestingly, recurrent excitation was occasionally (4/35) so strong that ventral root stimulation on its own recruited motoneurons as far as 3 lumbar segments away. These “ventral root-evoked reflexes” were blocked in 0 external Ca2+ as well as by glutamatergic antagonists showing an action via excitatory chemical synaptic transmission.

In the disinhibited synchronous bursting cord (bic/strych) ventral root stimulation could entrain bursting (n=2/4) demonstrating that recurrent excitatory pathways also access interneuronal systems.

The monoaminergic modulation of recurrent motor axon pathways and the suggestion of the existence of a recurrent excitatory interneuron suggests a greater range of motor output control by recurrent circuitry than previously considered.

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