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Development and Plasticity of the Corticospinal Tract in Children with Neurological Disorders

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Introduction

Because the young human brain is highly plastic, brain lesions that occur during development interfere with the innate development of architecture, connectivity, and function mapping and cause changes in structure, wiring, and representations. The motor cortex and/or the corticospinal tract are common sites of brain damage in childhood, and the most common time for brain damage to occur is during the prenatal or immediately perinatal period. It is now widely recognised that the corticospinal system is capable of significant reorganisation following lesions, and that this reorganisation is likely to underpin the partial recovery of function [1-3].

For skilled movements, the corticospinal tract (CST) is the primary motor control pathway. It has a long postnatal development period, making it vulnerable to perinatal brain and spinal cord injury. According to research, the motor signs of cerebral palsy (CP) reflect the loss of CST connections as well as the development of abnormal motor system connections, particularly between the developing CST and spinal motor circuits. We discuss a feline model of CP that we developed in this paper. The animals develop an abnormal pattern of CST connections that is strikingly similar to that seen in hemiplegic CP and visuomotor impairments. We developed neural activity-based therapeutic approaches based on this model to repair the abnormal CST connections and restore normal skilled movement control [4]. According to our findings, more active CST connections can maintain strong synaptic connections with spinal motor circuits.

The corticospinal tract originates in several areas of the brain, including the motor areas as well as the primary somatosensory cortex and premotor areas. The majority of neurons originate in the primary motor cortex or the premotor frontal areas. About 30% of corticospinal neurons originate in the primary motor cortex, 30% in the premotor cortex and supplementary motor areas, and the remaining 40% in the somatosensory cortex, parietal lobe, and cingulate gyrus. These upper motor neurons begin in the neocortex's layer V pyramidal cells and travel through the forebrain's posterior limb of the internal capsule to enter the cerebral crus at the base of the midbrain. Both tracts then travel through the brain stem, from the pons to the medulla [5].

The corticospinal and corticobulbar tracts form two pyramids on either side of the brainstem's medulla, giving rise to the term pyramidal tracts. Corticospinal neurons synapse directly onto alpha motor neurons to control muscles directly.

Betz cells are very large, easily visible under a microscope cells that, despite accounting for only about 5% of cells projecting to the spinal cord, are widely regarded as the most important for motor signal communication.These cells are notable for their fast conduction rate, which exceeds 70m/sec, making them the fastest signal conduction from the brain to the spinal cord [6].

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