Lectures

CO59-001-e
Gait and postural disorders in Parkinson’s disease
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Gait disorders and axial symptoms are the main therapeutic challenges in advanced Parkinson’s disease (PD). Gait disorders in PD are characterized by spatial and temporal dysfunction. Gait hypokinesia appears first and is responsible for the decrease in velocity. High responsiveness to levodopa is well established. It is demonstrated that the ability to modulate walking cadence remains intact in PD, and could correspond to a compensatory mechanism. More advanced stages are characterized by abnormal temporal parameters, which are unresponsive to levodopa. After a few years of evolution, paradoxical episodic phenomena may occur: festination and Freezing of Gait (FOG). Both are incapacitating for PD patients, because of their resultant loss of independence and their poor response to levodopa therapy. Kinematic studies of FOG reveal a decrease in velocity, stride length and an exponential increase in cadence, prior to a FOG episode. New approaches (functional MRI, wavelets, ...) should offer new prospects. Posture is also affected in Parkinson’s disease. Generally, postural dysfunction induces clinical impairment at the latest stages of the disease. Posture may be affected in its orientation component (stooped posture, camptocormia, Pisa syndrome) or in its balance component (loss of postural reflexes).

http://dx.doi.org/10.1016/j.rehab.2014.03.1205

CO66-001-e
Neurorehabilitation in Parkinson’s disease: Towards patient deparkinsonization?
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Keywords: Parkinson’s disease; Aerobic training; Motor strengthening; Cueing

Patients with parkinsonism tend to self-limit their physical activity. This may prove twice detrimental to their function: beyond general deconditioning, this self-imposed sensorimotor restriction could accelerate cell death and disease progression, based on recent animal model data.

To increase striatal dopamine production, research has involved exogenous strategies bringing heterologous trophic factors into the striatum to foster cell regeneration and dopamine production (initially surrenal and mesencephalic cell transplants, more recently gene and stem cell therapies). Endogenous strategies are being tested today in a number of neurorehabilitation programs aiming at natural trophic factor and dopamine production from the surviving cells, after prolonged and intense aerobic training in particular.

Animal model research has impressively demonstrated both symptomatic and neuroprotective effects of physical exercise, requiring a double threshold of exercise duration and intensity to occur (3 to 4 months of continuous exercise in rodent models). In PD patients, controlled trials have mostly shown symptomatic effects to date: while endogenous dopamine is produced immediately after an aerobic exercise, a few weeks of high-intensity aerobic training improves walking, upper limb and executive functions. Motor strengthening programs, Tai Chi and attentional strategies (cueing) have also produced symptomatic effects in controlled protocols. Early personal results will be shown.

http://dx.doi.org/10.1016/j.rehab.2014.03.1207