

## MAIN THEME: COGNITIVE DISORDERS IN NEUROLOGICAL DISEASES

### COGNITIVE AND BEHAVIOURAL ASPECTS OF GAIT

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The gait of patients with Parkinson's disease (PD) is typically marked by reduced speed, shortened stride length, start hesitation and longer double support phase. In addition, gait dynamics are characterized by exaggerated stride-to-stride variability, that reflects a disturbance in gait rhythmicity and an inconsistency of the locomotor pattern, and is a marker for increased fall risk. Walking and standing are not purely automatic tasks, regulated by subcortical control mechanisms and requiring little if any conscious attention. Instead, gait is now increasingly seen as a complex 'higher-order' form of motor behavior, with prominent and varied influences of mental processes. This becomes evident under complex circumstances, when patients with PD are unable to deal with multiple tasks simultaneously, either because the central processing abilities have become too limited (a limited attentional resource that interfere with their ability to execute more than one task at the same time), or be-

cause patients fail to properly prioritize their balance control over other, less important secondary tasks (difficult switching of this resource between tasks). Poor responsiveness to levodopa replacement therapy suggests that gait deficits may, at least partially, result from additional non-dopaminergic lesions.

Freezing is probably the most debilitating symptom of PD, usually observed in the advanced stage of the disease and characterized by a sudden inability to initiate or continue walking, as well as when approaching a destination (freezing of gait; FOG), especially while turning, in stressful time-constrained situations (i.e., crossing busy street), and upon entrance into and through narrow spaces such as doorways (patient feel as if foot is glued to the ground). We will present preliminary result of our MRI voxel-based morphometry study, suggesting significance of frontal and posterior parietal cortex deficiencies in etiopathogenesis of FOG in PD.