

# Gait analysis as a complementary tool in the levodopa dose decision in Vascular Parkinson's Disease

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Meeting: [21st International Congress](#)

Abstract Number: 656

Keywords: [Gait disorders: Pathophysiology](#), [Gait disorders: Treatment](#), [Parkinsonism](#)

## Session Information

Date: [Tuesday, June 6, 2017](#)

Session Time: 1:45pm-3:15pm

Session Title: [Technology](#)

Location: Exhibit Hall C

**Objective:** Evaluate the role of objective gait analysis in the decision of levodopa dose in VPD, in particular when compared to clinical assessment.

**Background:** Vascular Parkinsonism (VPD) is typically characterized by lower body Parkinsonism, marked gait difficulty, relatively symmetrical symptomatic distribution, and poor response to levodopa treatment. There is no consensus on clinical criteria for VPD, even less on how much levodopa should be given and if higher doses of levodopa should be aimed.

**Methods:** Thirteen patients with VPD, average age of 80 years-old [76, 90] were included for gait analysis. Exclusion criteria were: presence of resting tremor, moderate-severe dementia ( $CDR \geq 2$ ), musculoskeletal disease and overt clinical progression since diagnosis, Hoehn-Yahr > 3. Demographic and clinical data – LEDD (mg), time after stroke to VPD diagnosis, MRI brain imaging – were collected. Patients were evaluated in “Off” phase (24h without L-dopa) and in “On” phase (after a suprathreshold L-dopa challenge, 150% of L-dopa morning dose), with MDS-UPDRS (part III) and gait analysis (GaitUp®) (60-meter continuous course in a self-selected walking speed). Clinical scores and spatial, temporal and clearance gait variables were statistically compared (“Off” vs. “On” phase) (Wilcoxon signed ranks test, significant P-values <0.05).

**Results:** In the “Off” phase, patients had a median MDS-UPDRS-III score of 44 [22, 70], (left/right) foot gait velocity of 0.63/0.66 (m/s), stride length of 0.68/0.67 (m), 10.53% of variability and 1.05 of symmetry. After L-dopa challenge, patients had statistically significant median change (%) (left/right foot) in: gait velocity (+9.9%/+8.3%); stride length (+8.3%/+5.9%); foot-flat (-1.5%/-1.3%) and pushing (+6.3%/+3.8%) %of stance time; and peak angle velocity (+5.1%/+4.1%). Overall, kinematic gait variables did not correlate with the magnitude of change (%) observed in the MDS-UPDRS-III (+12% [4, 23]) and rigidity (+25% [0, 42]) scores.

**Conclusions:** Increased doses of levodopa (50% extra), albeit with apparent clinical benefit in bradykinesia and rigidity, do not necessarily translate into an equivalent benefit in gait profile. Non-dopaminergic networks may be differently affected by vascular pathology, and therefore be less responsive to dopamine. Gait can be a complementary tool in the individualized decision of levodopa dose in VPD.

**References:** Vizcarra JA, Lang AE, Sethi KD, Espay AJ: Vascular parkinsonism: Deconstructing a syndrome. *Movement disorders : official journal of the Movement Disorder Society* 2015;30:886-894.

Takakusaki K: Neurophysiology of gait: From the spinal cord to the frontal lobe. *Movement disorders : official journal of the Movement Disorder Society* 2013;28:1483-1491.

To cite this abstract in AMA style:

M. Gago, F. Ferreira, N. Mollaei, M. Rodrigues, N. Sousa, E. Bicho, P. Rodrigues. Gait analysis as a complementary tool in the levodopa dose decision in Vascular Parkinson's Disease [abstract]. *Mov Disord.* 2017; 32 (suppl 2). <http://www.mdsabstracts.org/abstract/gait-analysis-as-a-complementary-tool-in-the-levodopa-dose-decision-in-vascular-parkinsons-disease/>. Accessed December 27, 2017.

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