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Multi-muscle synergies: A sensitive tool for Parkinson's disease

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Postural instability is one of the cardinal signs of Parkinson's disease (PD). Quantifying postural stability is commonly used to measure PD severity. We hypothesized that postural synergy indices in the space of activation of muscle groups (M-modes) may be used to measure changes in motor coordination due to PD and dopamine-replacement therapy. Synergy indices stabilizing the center of pressure (COP) were compared between 11 patients without clinical symptoms of postural instability (Hoehn-Yahr stage-II) and 11 age-matched controls, and between 10 patients (stage II and III) tested off-drug and on-drug. Electromyographic signals from 13 leg and trunk muscles, recorded during cyclic body sway and releasing a load from extended arms, were used to quantify synergy indices by comparing the variance that had no effect on the COP coordinate and the variance that changed COP coordinate. Since this analysis needs multiple trials to identify the variance structure, we also quantified components of motion in the space of M-modes that had (non-motor equivalent) and did not have effect (motor equivalent) on COP coordinate using individual sway cycles. PD patients showed significantly lower synergy indices, and reduced ability to adjust these indices in preparation to an action. Motor equivalence analysis confirmed these differences. Impaired ability to adjust synergy indices in preparation to an action may contribute to rigidity and episodes of freezing. We conclude that analysis of motor equivalence in muscle activation space, using a few trials, can be used as a clinical measure for early diagnosis of PD and tracking disease progression.

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Identifying Park-weight phenotype in Parkinson's disease: Implications on disease management

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A number of phenotypes are being identified in the neurodegenerative Parkinson's disease, mostly in reference to the non-motor symptoms. One purpose of identifying phenotypes is to manage disease process more effectively. PD patients have variable impairment of olfaction; high proportions develop body-weight change (gain or loss) as the disease advances. PD patients have a lower body weight as compared to non-PD controls. Weight loss in PD is not a benign phenomenon. Lower initial body weight and weight loss during the course of the disease predispose to the risk of dyskinesia; there being a relationship between body weight and levodopa dose per kilogram for dyskinesia. Additionally weight loss increases the risk of under-nutrition, frailty, poor quality of life and mortality. Patients at the risk of weight loss may be identified by their severe olfactory loss (anosmia) at an early stage, since anosmia, as compared to hyposmia, seems to represent more severe neurodegenerative process predisposing to weight loss and dyskinesia describing the "olfaction-weight-dyskinesia" phenotype in Parkinson's disease. Weight loss is not due to higher energy expenditure or lower energy intake. The basis of severe neurodegenerative process and weight loss might be a longer pre-clinical phase in this phenotype. PD patients should be monitored for weight loss and the dose of levodopa adjusted accordingly as the disease advances. Measures should be taken to prevent weight loss in such patients to prevent the low body-weight related non-motor and motor adverse effects. This may result into better quality of life.

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