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WEAK DETRUSOR CONTRACTILITY CORRELATES WITH MOTOR DISORDERS IN PARKINSON'S DISEASE

Hypothesis / aims of study

Limited attention has been paid to the relationship between urinary symptoms or urodynamic findings and motor disorders in Parkinson's disease (PD).

Overall urinary symptoms correlate well with the severity and duration of motor disorders [1]. However, among four routine urodynamic parameters (first sensation, bladder capacity [storage phase]; maximum voiding detrusor pressure, post-void residual [voiding phase]), Araki and colleagues reported that only the volume of post-void residual urine was related with motor disorder [2]. In order to further clarify this, we aimed to correlate pressure-flow urodynamic parameters with video-gait analysis parameters in PD.

Study design, materials and methods

We recruited 41 patients with PD: 25 men, 16 women; age 70.6 +- 8.5 years; Hoehn-Yahr motor grading 2 (1–3); disease duration 4 years (1–7 years); taking levodopa 300 mg/day (100-400 mg). All patients underwent pressure-flow urodynamics (parameters: first sensation, bladder capacity, detrusor overactivity [noted in 24 patients] and Watts factor) and video-gait analysis (parameters: time and the number of strides for 5-m gait (simple task) and time for timed up and go (complex task). Statistical analysis was made by Mann-Whitney's U-test for analyzing the relation between detrusor overactivity and gait, and Spearman's rank correlation coefficient test for analyzing the relation between the remaining parameters and gait.

Results

We found no relation between filling-phase urodynamics (detrusor overactivity, first sensation and bladder capacity) and video-gait analysis parameters. By contrast, we found a significant relation between voiding-phase urodynamics (Watts factor, reflecting detrusor power) and all three video-gait analysis parameters (reflecting lower-half bradykinesia and loss of postural reflex) in our PD patients ($p<0.01$) **(Figure 1)**.

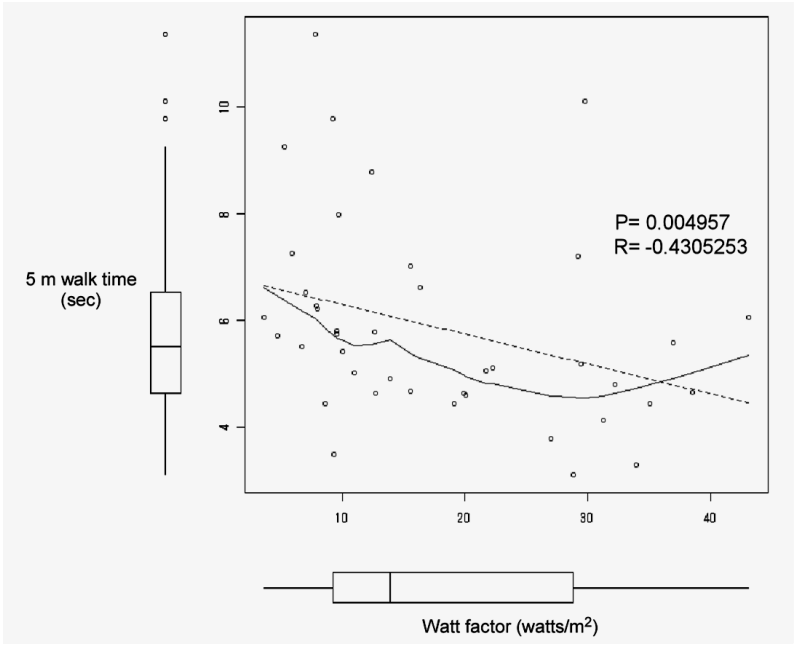
Interpretation of results

We did not know the exact reason for the lack of correlation between voiding-phase urodynamics and motor disorders. Not only basal ganglia dopaminergic pathways but also extra-basal ganglia pathways, e.g., A11 hypothalamo-spinal dopaminergic pathways etc, might have a role in suppressing detrusor overactivity in PD. In contrast, 18% of patients with PD show both detrusor hyperreflexia during the filling phase and impaired contraction (a weak detrusor) in the voiding phase (so-called DHIC)[3], which seems to be caused by multiple factors. The wide range of pathologies in PD includes the micturition-facilitatory area (A10 ventral tegmental area (VTA)-mesolimbic dopaminergic pathway[4], D2-subthalamic *indirect pathway*, glutamatergic pontine micturition center (PMC) adjacent to the locus ceruleus, and sacral cholinergic preganglionic cells)[5]. From a clinical perspective, PD patients with advanced gait disorder may not only have overactive bladder but also a weak detrusor and large post-void residuals. Therefore, we should perform ultrasound echography of post-void residual repeatedly in those patients.

Concluding message

The close relation between the Watts factor and motor disorders in the present study suggests that, though clinically mild, a weak detrusor in PD might have a central origin. We should follow post-void residual volume carefully in PD patients with advanced gait disorder, since post-void residual volume might increase in such patients.

A



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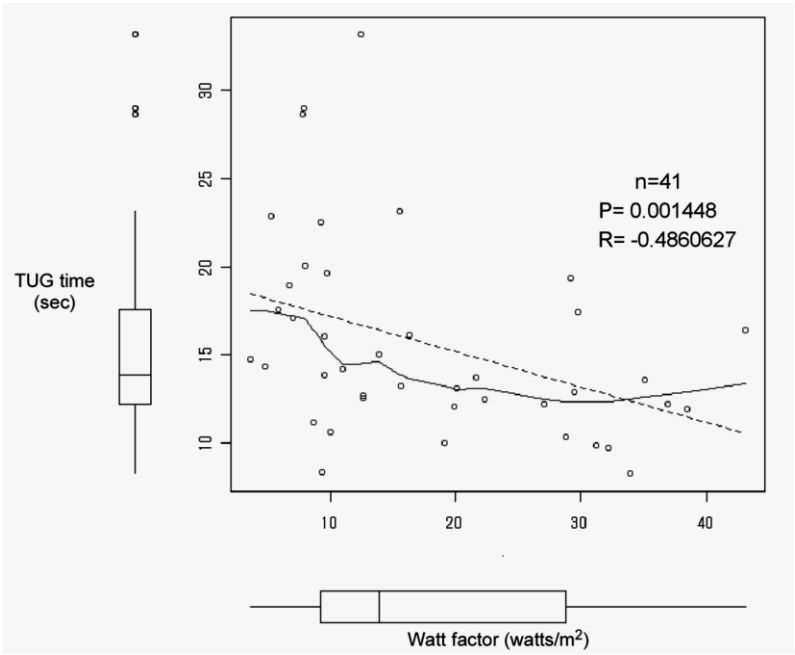

Figure 1 Relationship between urodynamic voiding phase parameter (Watts factor) and gait analysis parameters (A: time of strides for 5 m walk, and B: time for TUG).

References

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Disclosures

Funding: We do not have specify source of funding. **Clinical Trial:** No **Subjects:** HUMAN **Ethics Committee:** Ethics Committee of Toho univercity sakura medical center **Helsinki:** Yes **Informed Consent:** Yes