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Peering through the FoG: Visual manipulations shed light on freezing of gait

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Freezing of gait (FoG) – the intermittent and usually brief interruption of forward progression despite intention to walk – is poorly understood, in part because its episodic and unpredictable nature makes it notoriously difficult to study in the laboratory.^{1, 2} This issue of *Movement Disorders* includes descriptions of two promising new methods for eliciting and quantifying FoG or the slowing that may be a precursor to FoG in people with Parkinson's disease (PD). Both methods exploit the known association of FoG with visual stimuli, most classically the precipitation of FoG by doorways and narrow spaces.³

In fact, vision plays multiple, important roles in normal gait. One role is through automatic interpretation of self-motion feedback, called *optic flow*, which helps stabilize the head and trunk to prevent falling and contributes to perception of walking speed and direction.^{4, 5} In healthy subjects, the effect of optic flow is not very large, due to relatively greater reliance on proprioceptive information for gait.⁶ However, evidence suggests that individuals with PD are more dependent on vision than healthy subjects, probably because of impaired use of proprioception,⁷ and thus they are more affected by changes in the visual environment.⁸ Therefore, the manipulation of optic flow is a promising avenue for studying gait problems in PD.⁹

Vision is also essential for navigating complex environments. A person approaching an obstacle fixates on it visually at least 3 steps in advance in order to adjust stride length, cadence, and body orientation to successfully avoid the obstacle using feedforward, predictive control.¹⁰ Passing through a narrow doorway is a similar challenge, in that it depends on visual perception of the passability of the doorway. Studies of healthy subjects have shown that passage through a narrow doorway induces shoulder rotation when the aperture is 130% of shoulder width,¹¹ and that imagined walking through a narrow gate takes longer than imagined walking than through a wide one.¹² Likewise, a narrow path

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slows both actual and imagined gait.^{13, 14} Thus, slowing when passing through a narrow door is normal. However, this tendency is exaggerated in PD subjects,¹⁵ especially those with FoG.^{16, 17} Therefore, studying how subjects with PD pass through actual or simulated narrow doorways is a promising method for investigating FoG.¹⁸

In this issue, van der Hoorn et al⁹ describe the effects on gait speed of a virtual reality display of optic flow while the subject walked on a treadmill. The visual stimuli consisted of white dots that moved smoothly from the middle to the outside of a screen to create an illusion of forward body motion. By abruptly changing the trajectories of the dots, the experimenters could create the illusion that the space through which the subject was moving had become suddenly narrower. This manipulation led to brief slowing of gait. This slowing was more prominent in subjects with left-sided PD (LPD) and in those who performed poorly on the Block Design subtest of the Wechsler Adult Intelligence Scale (WAIS), a spatial cognitive test that is particularly sensitive to right parietal function.¹⁹ These results are consistent with those of Davidsdottir and colleagues²⁰ in which a visual display of optic flow caused perception of the midline to shift to the right for LPD subjects but not for healthy controls and right-sided PD (RPD) subjects.

If perception of optic flow relates to FoG, FoG might be expected to occur earlier, or with greater frequency, in subjects with LPD than in subjects with RPD. To our knowledge, this has not been investigated. The van der Hoorn group studied only mild PD subjects, and no information is provided about the movement parameters, such as stride length, cadence, variability, double-support time and trembling of knees, so it is unknown whether the slowing they observed was accompanied by gait features associated with FoG. Generally, the literature examining cognitive and motor differences in subjects with LPD vs. RPD is inconclusive.²¹

One caveat about the experimental paradigm used by van der Hoorne et al is that it combines a strong visual stimulus from the display of moving dots with relatively weak vestibular and proprioceptive information from walking on a treadmill instead of a solid surface. This experimental paradigm thus forces vision to be weighted more highly than it is during over-ground walking, which may exaggerate the effects of visual flow on gait.

Another paradigm using vision to investigate FoG is described by Cowie et al¹⁸ in this issue. Gait kinematics were measured while subjects walked toward and through a doorway that could be adjusted to different body-scaled widths. The changes in gait speed and kinematics induced by narrowing the doorway provided information about visuomotor contributions to gait dysfunction and the effects of levodopa and deep brain stimulation (DBS) on the gait abnormalities. An important contribution of this publication is the proposal of two methods to objectively quantify “freeze-like events”: a decrease in velocity below 10% of average walking speed when no doorway is present and double-support time more than 3.1 standard deviations above the subject’s mean double-support time when walking is unimpeded by the doorway. Although the agreement between these objective measures and two clinical raters was only moderate, the correlation was as strong as the agreement between the two raters. This highlights the problem with clinical ratings as the “gold standard” for detection of brief (<500 ms) freezing episodes and the importance of developing more sensitive measures of

FoG. The value of these objective measures of FoG is demonstrated by their ability to discern a difference between the effects of levodopa and DBS on FoG, a controversial question due to in part to difficulty measuring FoG.^{22, 23}

Because the effects of optic flow and narrow doorways on gait are not unique to PD, it is likely that the strong effect of the visual environment on gait and FoG in PD is caused by difficulty in appropriately scaling a motor response to the visually-perceived environmental affordance. PD reduces central drive from the supplementary motor area, impairs endogenously generated movements, and forces increased reliance on the external cues mediated by the premotor cortex.²⁴ Thus, people with PD have increased susceptibility to the influence of visual cues²⁵ and may show exaggerated visuomotor scaling.^{26, 27} However, the alternative suggestion provided by the authors,¹⁸ that the doorway captures attention and thus interferes with voluntary compensation for underlying short stride length, is also worth considering.²⁸

The publications of Cowie et al and van der Hoorne et al. demonstrate new ways to study the influence of vision on gait and to quantify the exaggerated effects of vision on gait in subjects with PD. We expect that these tools will facilitate laboratory investigations of neural mechanisms underlying FoG, bringing us closer to understanding this important problem.

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