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Disturbances of automatic gait control mechanisms in higher level gait disorder

Mary Danoudis [*,a], Ganga Ganesvaran [a], Robert Iansek [b]

[a] Clinical Research Centre for Movement Disorders and Gait, The National Parkinson Foundation Center of Excellence, Kingston Centre, Monash Health, Cheltenham, Victoria 3192, Australia
[b] Department of Medicine, Monash University, Clayton 3800, Australia

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ABSTRACT

The underlying mechanisms responsible for the gait changes in frontal gait disorder (FGD), a form of higher level gait disorders, are poorly understood. We investigated the relationship between stride length and cadence (SLCrel) in people with FGD ($n = 15$) in comparison to healthy older adults ($n = 21$) to improve our understanding of the changes to gait in FGD.

Gait data was captured using an electronic walkway system as participants walked at five self-selected speed conditions: preferred, very slow, slow, fast and very fast. Linear regression was used to determine the strength of the relationship (R^2), slope and intercept.

In the FGD group 9 participants had a strong SLCrel (linear group) ($R^2 > 0.8$) and 6 a weak relationship ($R^2 < 0.8$) (nonlinear group). The linear FGD group did not differ to healthy control for slope ($p > 0.05$) but did have a lower intercept ($p < 0.001$). The linear FGD group modulated gait speed by adjusting stride length and cadence similar to controls whereas the nonlinear FGD participants adjusted stride length but not cadence similar to controls. The non-linear FGD group had greater disturbance to their gait, poorer postural control and greater fear of falling compared to the linear FGD group.

Investigation of the SLCrel resulted in new insights into the underlying mechanisms responsible for the gait changes found in FGD. The findings suggest stride length regulation was disrupted in milder FGD but as the disorder worsened, cadence control also became disordered resulting in a break down in the relationship between stride length and cadence.

1. Introduction

Gait disorders due to neurological or non-neurological causes are common in the elderly [1,2]. Gait changes can also present in the absence of any clinical symptoms associated with a specific neurological disease [1,3,4]. Gait impairments that present without obvious cause, previously considered 'normal' in older adults, are now recognised as being associated with central neurologic changes [3–6]. Attempts have been made to classify these gait disorders resulting in the use of various terminologies, including lower half parkinsonism, vascular parkinsonism and gait apraxia [4,7]. To address the clinical confusion caused by the use of these varying terms, the term higher level gait disorders (HLGD) was adopted to categorise the varying gait patterns [4]. The initial division of HLGD into five subcategories [4] has been changed to two–anterior or frontal HLGD and posterior or parieto-temporo-occipital HLGD [7]. This current study investigated people who had frontal HLGD, which we refer to as frontal gait disorder (FGD) [7].

Higher level gait disorders, including FGD, are typically diagnosed by clinical presentation and by the systematic elimination of other causes [4,7,8]. Recent investigations using imaging techniques have shown HLGD to be associated with increasing white matter changes in the brain, including the brainstem, periventricular and frontal regions, the basal ganglia and cerebellar locomotor regions [5,9]. Even with the advances in imaging the underlying mechanisms that cause the gait changes presenting in FGD still remain unclear. This uncertainty on the pathophysiology of FGD limits the development of interventions to address the gait deficits. The gait changes present in FGD disrupt mobility, increase falls risk, lead to a loss of function and independence, and a greater need for supported care [2].

Gait speed is modulated by changing stride length and cadence. Studies of healthy adults of differing age groups showed a consistently strong positive relationship between stride length

* Corresponding author. Tel.: +61 3 92651360; fax: +61 392651577.
E-mail addresses: Mary.Danoudis@monashhealth.org (M. Danoudis), ganga.ganesvaran@monashhealth.org (G. Ganesvaran), robert.iansek@monash.edu (R. Iansek).

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and cadence (SLCrel) (determined by linear regression analysis and expressed as R^2) when walking at a range of self-selected gait speeds [10,11]. In addition the ratio of stride length to cadence (reported as slope) and intercept were maintained within and between healthy adult groups [10,11]. The SLCrel has been used to investigate the changes to the central control mechanisms of gait in Parkinson's disease (PD), progressive supra nuclear palsy (PSP) and Huntington's disease (HD) [12–14]. Findings from these studies showed that in all three disorders slope did not differ to that of healthy controls however intercept was lower or scaled down compared to controls [12–14]. It was concluded that step length was reduced in these gait disorders due to a scaling deficit [12–14].

The aim of this current study was to improve our understanding of the changes to gait in FGD by investigating the SLCrel when participants walked at a range of self-determined gait speeds, and to compare with the SLCrel of healthy older adults (HOA). We hypothesised that the strength of the relationship, R^2, and intercept would both be lower for the FGD group compared with controls but slope would not differ between groups.

2. Methods

2.1. Participants

Fifteen participants with FGD were recruited from a movement disorders clinic and diagnosed by movement disorder neurologists. Classification of a participant's gait disorder as FGD was based on [8]: (1) observation of the gait abnormalities; (2) clinical tests for gait and balance to help distinguish between clinically similar gait disorders; and (3) clinical neurological examination. Key gait characteristics included shortened steps, wider base of support, possible hesitation or difficulty initiating stepping and having arm swing. Participants had a MRI to confirm the presence of white matter changes and to eliminate other pathologies, such as tumours, normal pressure hydrocephalus. Healthy older adults (HOA) were recruited from local community groups. Inclusion criteria for all participants were: a Mini Mental State Examination (MMSE) score $\geq$24, the ability to walk over the 12 m testing zone without assistance and having sufficient English to follow instructions. Exclusion criteria included the presence of other medical conditions that impacted on gait, such as musculoskeletal and neurological disorders. The Southern Health Research Ethics Committee approved the study and participants provided written informed consent before testing.

2.2. Gait assessment

Participants were tested on one occasion. Gait was quantified using an 8 m long electronic walkway system, GAITRite®, validated with healthy older adults [15] and pathological gait disorders [16]. Participants walked at five self-selected speeds: preferred, very slow, slow, fast and very fast. Preferred speed was recorded first to minimise any impact from the other conditions. To control for the influence of fatigue, the remaining speed conditions were counterbalanced. Self-selected speeds were used to minimise the need for attention when walking [10,11]. Three trials were recorded for each condition, with trials two and three being used in the analysis. To capture participants' gait at their steady state walking speed, walking commenced a minimum of 1 metre in front of the GAITRite®. Participants walked without gait aids or assistance. Standardised instructions were used, which were to walk at their normal/preferred pace, and slow, very slow, fast and very fast pace compared to their normal pace. Gait data extracted included gait speed, stride length, cadence, double stance time and step width. Participants' speed and stride length were normalised by dividing the mean values by their leg length.

2.3. Outcomes

Demographic data recorded included sex, age, height, leg length, and MMSE scores [17]. Fear of falling, using the modified falls efficacy scale [18], freezing of gait, using the Freezing of Gait questionnaire (FOGQ) [19] and the Pastor shoulder tug were additional measures used with the FGD group.

The primary outcome was the SLCrel which was determined for each participant using the mean normalised stride length and cadence data from two trials for all five speed conditions. Using linear regression analysis, stride length was plotted against cadence for each participant and their relationship inspected for linearity. Only participants whose regression lines were found to be linear (a linear model $R^2 \geq 0.80$) were included in the SLCrel analysis. Based on our previous study in healthy adults [10], participants demonstrating a quadratic relationship most often had a reduced step length and high cadence in the very fast speed condition, possibly due to biomechanical constraints. When trials with high cadences (>150 steps/minute) were removed, the SLCrel was linear. This method was applied to the current study. After removing trials with a mean cadence greater than 150 steps/min linear regression analysis was repeated and if this resulted in a $R^2 \geq 0.80$, the adjusted data was included in the SLCrel group comparison. The intercept was reported at a cadence of 100 steps/min in accordance with methods reported in earlier studies by our group [10]. This method was considered more robust than reporting the intercept at a cadence of zero. Cadence was set at *cadence-100* in the calculation of intercept and slope.

The ability to modulate stride length and cadence in response to changing gait speed was explored by comparing the mean values of these variables from the slow and fast conditions to that of the preferred speed condition and by determining the minimum, maximum and range of these variables for each participant at each speed condition.

Participants with FGD had MRIs. A radiologist blinded to study aims independently scored all MRIs and classified changes in the white matter signals according to the revised Fazekas scale [20]. The Fazeka scale is reliable for cross-sectional assessment of white matter signal abnormalities [21] and is used to rate the signal abnormalities around the ventricles (periventricular hyperintensity, PVH) and in the deep white matter (the deep white matter hyperintensity, DWMH). The PVH and DWMH scoring was from 0 (absent) to 3 (large confluent areas) and the scores summed to give a total score [20].

2.4. Statistical analysis

Independent *t*-tests were used to analyse differences in demographics, preferred gait speed measures and the SLCrel between the FGD and HOA groups and between FGD sub-groups. One way ANOVA analyses were used to compare the mean ranges, maximum and minimum values for stride length and cadence between the FGD sub-groups and HOA. Partial correlation analyses was used to determine the relationship between Fazeka's scores and gait measures (speed, stride length and cadence) and SLCrel in the FGD group while statistically controlling for the effect of age on these variables. Results when controlling for age, (partial correlations), were then compared to those when not controlling for age (Pearson's or zero-order correlations). Data were analysed using SPSS®. Level for statistical significance was set at $p \leq 0.05$.

3. Results

3.1. Characteristics

The characteristics of both groups are reported in Table 1. The FGD group walked slower, with a correspondingly shorter stride