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Gait in Parkinson's disease: a visuo-cognitive challenge

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Abstract

Vision and cognition have both been related to gait impairment in Parkinson's disease (PD) through separate strands of research. The cumulative and interactive effect of both (which we term visuo-cognition) has not been previously investigated and little is known about the influence of cognition on vision with respect to gait. Understanding the role of vision, cognition and visuo-cognition in gait in PD is critical for data interpretation and to infer and test underlying mechanisms. The purpose of this comprehensive narrative review was to examine the interdependent and interactive role of cognition and vision in gait in PD and older adults. Evidence from a broad range of research disciplines was reviewed and summarised. A key finding was that attention appears to play a pivotal role in mediating gait, cognition and vision, and should be considered emphatically in future research in this field.

Highlights

- Impaired vision and cognition contribute independently to gait deficit in PD
- Visual and cognitive interaction during gait has not been robustly examined
- Combined visuo-cognitive processes impact on gait
- Attention plays a pivotal role in visuo-cognitive control of gait in PD

Keywords: Parkinson's disease, older adults, vision, cognition, gait, saccades, visuo-cognition, attention

Contents

Introduction

Parkinson's disease (PD) is a common neurodegenerative disorder characterised by cardinal motor symptoms such as rigidity, bradykinesia, tremor, postural instability and gait deficit (Jankovic, 2008). Gait impairments in PD include both continuous (constantly present) and episodic (freezing of gait (FOG)) (Nutt *et al.*, 2011). Continuous gait impairment typically manifests as reduced velocity, step length, swing times, arm swing, increased gait variability and reduced automaticity. While episodic impairments emerge with increasing disease severity and are seen as hesitations when turning, a 'freezing' block in a small spaces such as doorways and difficulty with gait initiation (Giladi *et al.*, 2013). Gait impairments underpin difficulty walking in real-world environments such as maintaining a straight trajectory during gait (veering) (Davidsdottir *et al.*, 2008), negotiating obstacles (Vitorio *et al.*, 2013), and navigation (e.g. difficulties with narrow spaces such as doorways (Cowie *et al.*, 2010) and misjudgement of object distance (Davidsdottir *et al.*, 2005)). Moreover these problems are common and linked to falls (Paul *et al.*, 2014). Although these problems emphasise the motor complications of PD, it is widely recognised that gait impairment is complex and reflects input from multiple systems that include both motor and non-motor systems (Grabli *et al.*, 2012). For example, there is abundant evidence of the role of cognition in gait and increasing evidence of the role of vision. Understanding their respective contributions is critical in order to inform the mechanisms that drive gait impairment and to contribute to targeted therapeutic development to improve gait, independent mobility and falls risk.

A large body of evidence supports a robust relationship between cognition and gait, highlighting that gait is underpinned by cognitive functions (Lord *et al.*, 2014). Cognitive impairments are common in PD with an estimated 40% of patients presenting with mild cognitive impairment (MCI) at diagnosis (Yarnall *et al.*, 2014) and up to ~75% with dementia at ten years (Aarsland and Kurz, 2010). Previous studies have extensively investigated the relationship between gait and cognition (Amboni *et al.*, 2013) using two methodological approaches. Associative protocols measure gait and cognition as separate behaviours and explore their relationship to identify links between them (Lord *et al.*, 2014). Online protocols on the other hand, manipulate cognition particularly attention during walking through the use of dual-

task protocols which show in real-time the contribution of cognition to gait (Kelly *et al.*, 2012b). Such protocols demonstrate gait deficit such as reduced velocity and step length are associated with impaired cognition (Lord *et al.*, 2014), and exacerbated using dual-tasks in PD (Kelly *et al.*, 2012b).

Visual impairments are also common with up to 75% of people with PD experiencing at least one symptom such as blurred vision (Davidsdottir *et al.*, 2005; Collerton *et al.*, 2012; Urwyler *et al.*, 2013). The relationship between vision and gait in PD has also been investigated either by exploring relationships between separate visual functions and gait or use of online protocols where vision is manipulated during gait (i.e. light or dark rooms) (Azulay *et al.*, 1999; Almeida *et al.*, 2005). Selective gait impairments are associated with deficits in visual functions (Moes and Lombardi, 2009), and exacerbated by visual manipulation in PD (Cowie *et al.*, 2012). Studies have shown that visual functions contribute to gait control in PD (Azulay *et al.*, 1999; Azulay *et al.*, 2002; Khattab *et al.*, 2012).

To date the relationship between gait, cognition and vision has received scant attention and is poorly understood. Cognition, vision and gait potentially interact in a selective but overlapping manner in order to plan routes and make ongoing modifications appropriate to changing environments. Static and more recently dynamic test protocols have been used to examine the interplay between cognition and vision. Static protocols range from simple associations between separate cognitive and visual outcomes, to more complex neuro-imaging or computerised saccadic (fast, jump-like) eye-movement assessment. Evidence from static tests supports an interaction between cognition and vision (Lee *et al.*, 2015), and vice versa (Bertone *et al.*, 2007; Toner *et al.*, 2012). This interaction is encompassed by the term *visuo-cognition*, which is a global descriptor of interaction between cognitive and visual functions across multiple levels of information processing (Antal *et al.*, 1998; Bandini *et al.*, 2002). Visuo-cognition is therefore distinct from limited terms such as *visuo-spatial* function, which refers to the cognitive ability of the posterior parietal cortex to perceive the spatial relationship of objects (Benton and Tranel, 1993; Possin, 2010). Deficits in visual functions impact visuo-spatial ability due to their interaction (Stoerig and Cowey, 1997), but this exhibits only one aspect of visuo-cognition. Recent technological advances in mobile eye-tracking devices have facilitated measurement of saccadic eye movements during dynamic protocols

(Land, 2006), which serve as a proxy measure of visuo-cognition during gait in PD (Stuart *et al.*, 2014). To provide a detailed account of the role of vision and cognition during gait in PD there is first a need to understand the relationship and interactions between these two systems, and from these draw inferences about their potential impact on gait.

Study aims

A narrative review was undertaken to explore the diverse range of literature which was necessary to inform these complex interactive features. We adopt a model to explore the independent and interacting roles of vision and cognition in gait impairment in PD (Figure 1) and highlight relevant literature pertaining to the role of cognition in gait (Figure 1(A)); the role of vision in gait (Figure 1(B)); the relationship between visual function and cognition (visuo-cognition) (Figure 1(C)); and finally the role of visuo-cognition in gait (Figure 1(D)). We explore evidence in PD and older adults, and make recommendations for future work in this complex and developing area.

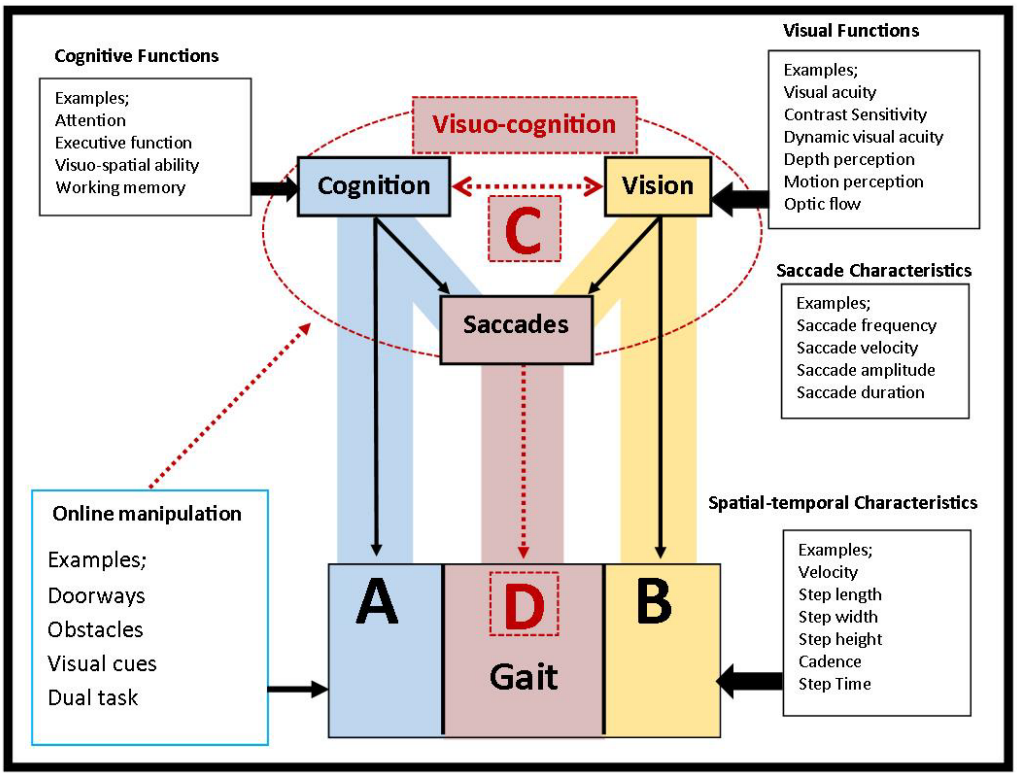

Figure 1 - A model of online relationships between vision, cognition and gait in Parkinson's disease

Four main pathways are involved; A) Cognition and gait, B) Vision and gait, C) Interaction between vision and cognition (visuo-cognition), and finally D) Visuo-cognition (measured through saccades) and gait.

***Recognised pathways** that have been assessed using both associative and online protocols are represented by black lines.*

***Unclear pathways** that have not been assessed using both associative and online protocols are represented by dashed red lines.*

A. The role of cognition in gait

Cognition is a multi-dimensional construct represented by interdependent systems, such as those described in Table 1. Attention is one of the most complex cognitive functions and is often considered to have overarching capacity (Lückmann *et al.*, 2014) as a 'gatekeeper' or 'supervisory system' that allocates resources to competing processes (Posner and Petersen, 1990; Baddeley, 1992; Engle, 2002). Therefore if attentional deficit is present, other cognitive functions are also compromised (Posner and Petersen, 1990). For example, working memory is dependent on attentional processes to determine capacity and allocation (Kane *et al.*, 2006).

Interpretation is complicated by the lack of a single and clear-cut definition of attention (Yogev-Seligmann *et al.*, 2008). As a result, different theoretical and neuroanatomical models of attention exist which are in turn selectively applied to different areas of research (Posner and Petersen, 1990; Baddeley, 1992; Baluch and Itti, 2011; Petersen and Posner, 2012). However, most neuroanatomical models are consistent in describing attentional processes that originate from the pre-frontal cortex (PFC) which are associated with executive function (Aleman and van't Wout, 2008) and extend to include broader cortical networks including those with BG input (McNab and Klingberg, 2008). Attentional processes are also influenced by sub-cortical noradrenaline and cholinergic projections (Delaville *et al.*, 2011; Müller and Bohnen, 2013), involving structures such as the locus coeruleus, thalamus, pedunculopontine nucleaus (PPN) and nucleus basalis of Meynert (Bohnen and Albin, 2011; Delaville *et al.*, 2011; Gratwicke *et al.*, 2015; Picillo *et al.*, 2015). Dysfunction in any of these cortical or sub-cortical attentional networks with age or pathology may impact cognitive, visual or gait processes.

Visuo-spatial ability also shares a complex relationship with attention particularly in PD (Crucian and Okun, 2003; Crucian *et al.*, 2010). Standard visuo-spatial assessments require attentional input from an early stage of visual processing to select focal areas of interest (Finton *et al.*, 1998; Baluch and Itti, 2011; White *et al.*, 2013). One study demonstrated that visuo-spatial deficits with PD disappeared when controlling for attention (Bondi *et al.*, 1993), indicating need for a cautious approach to interpretation.

Cognition in Parkinson's disease

Cognitive impairment in PD is diverse, with severity and progression to dementia (classified according to criteria from the Movement Disorders Society taskforce (Litvan *et al.*, 2012)) selective to PD phenotype (Pagonabarraga and Kulisevsky, 2012). Cognitive deficits most commonly present in attention, executive function, working memory and visuo-spatial ability (Caccappolo and Marder, 2010) (summarised in Table 1), whereas other processes such as language are usually less affected (Barone *et al.*, 2011). Such deficits occur early and insidiously (Pfeiffer *et al.*, 2014; Yarnall *et al.*, 2014), and are dominated by attentional impairment (Taylor *et al.*, 2008; Svenningsson *et al.*, 2012).

Progression of cognitive impairment relates to genetic factors and pathological changes in different substrates (Svenningsson *et al.*, 2012), such as fronto-striatal (Jokinen *et al.*, 2013) and posterior-cortical dysfunction (Pagonabarraga and Kulisevsky, 2012). Dopaminergic fronto-striatal deficits relate to slow cognitive decline (Emre *et al.*, 2014), and primarily impact attention, executive function and working memory. Whereas posterior-cortical deficits relate to rapid cognitive decline, perhaps determined by degeneration of cholinergic projections from the basal forebrain (Pagonabarraga and Kulisevsky, 2012). Age-related cognitive deficits (Table 1) which are typically more amnestic and represent increased cholinergic burden also contribute to PD cognitive impairment (Petersen *et al.*, 1999; Bohnen *et al.*, 2006), especially in more advanced disease (Bohnen and Albin, 2011).

Cognition and gait

The relationship between gait and cognition in PD (Figure 1(A)) is particularly strong and supported by mechanistic and imaging work (Grabli *et al.*, 2012; Maillet *et al.*, 2012). Various relationships between selective gait characteristics and cognitive functions have been found, however attention has a central role in gait in PD (Yogev-Seligmann *et al.*, 2008).

Recent work from our group examined the association between gait and cognition in older adults and PD (Lord *et al.*, 2014), using a comprehensive battery of cognitive and gait measures. We found a strong relationship between attention and the 'pace' domain of gait (comprising gait velocity, step length and step time). Similarly, online studies utilising dual task protocols which manipulate attention in real-time demonstrate an increase in gait variability, reduced velocity, swing time and step length in older adults (Hollman *et al.*, 2007; Verghese *et al.*, 2007; Hausdorff *et al.*, 2008) and PD (Yogev *et al.*, 2005; Rochester *et al.*, 2008; Kelly *et al.*, 2012a). However dual task interpretation is challenging because of the complex intertwined nature of attention, executive function and working memory (Yogev-Seligmann *et al.*, 2008; Rochester *et al.*, 2014), which have overlapping influences on dual task performance (Kelly *et al.*, 2012b).

Executive dysfunction is related to gait deficit in PD, particularly in those who report FOG (Amboni *et al.*, 2008; Heremans *et al.*, 2013) and people with the Postural Instability and Gait Disturbance (PIGD) phenotype (Lord *et al.*, 2014), who present

with greater frontal impairment (Burn *et al.*, 2006; Maidan *et al.*, 2015). Associations between gait and cognition have shown that executive dysfunction is related to reduced gait velocity, increased variability, step time and swing time in older adults (Ble *et al.*, 2005; Springer *et al.*, 2006; van Iersel *et al.*, 2008; Liu-Ambrose *et al.*, 2010; Holtzer *et al.*, 2012) and PD (Plotnik *et al.*, 2009; Lord *et al.*, 2010; Lord *et al.*, 2014). Interpretation is complicated by the intimate relationship between executive function and attention (Kudlicka *et al.*, 2011), which has prompted these functions to be discussed both separately and as a unitary domain (i.e. executive-attention) (Holtzer *et al.*, 2006; Verghese *et al.*, 2008; MacAulay *et al.*, 2014). Discerning their individual role in gait is therefore challenging, and highlights a need for precise cognitive assessment and outcome reporting.

As another closely related cognitive function, working memory is also associated with gait deficit in older adults, for example with gait velocity (Holtzer *et al.*, 2006; Soumare *et al.*, 2009), step time (Holtzer *et al.*, 2012), step time variability, double support time and step length (Holtzer *et al.*, 2006; Martin *et al.*, 2013). The relationship in PD is less clear with research showing contradictory results (Amboni *et al.*, 2012; Lord *et al.*, 2014; Stegemoller *et al.*, 2014). Inconsistencies in PD associations are possibly due to the use of subtly different working memory assessments (i.e. digit span forward or backward, or Rey Auditory Verbal Learning Test) and limited consideration for features that potentially sensitise the relationship such as disease phenotype, as reported by Lord *et al.* (2014).

Visuo-spatial ability has been related to Parkinsonian gait, possibly due to impairment of attentional networks common to visuo-spatial function and gait control (Menant *et al.*, 2014). Amboni et al. (2012) reported an association in PD between impaired visuo-spatial ability and deficits in their ‘stability’ gait domain. Correspondingly, deficits are implicated in falls in older adults (Reed-Jones *et al.*, 2013) and PD (Davidsdottir *et al.*, 2005; Allen *et al.*, 2013). Visuo-spatial impairment with age and PD also relates to reduced step length (Nadkarni *et al.*, 2010), gait velocity (Beurskens and Bock, 2011), and increased double support time, stride time variability (Menant *et al.*, 2014), step length variability (Martin *et al.*, 2013) and reduced timed up and go speed (Donoghue *et al.*, 2012). Findings are however contradictory (Soumare *et al.*, 2009; Plotnik *et al.*, 2011), at least partly due to lack of comprehensive and rigorous visuo-spatial assessment (Lord *et al.*, 2014). Again, the

relationship may also depend on disease severity, as reported previously for the PIGD phenotype (Domellof *et al.*, 2011) and in those who experience FOG (Nantel *et al.*, 2012; Heremans *et al.*, 2013) (Table 1). A recent study involving a large number of people with PD (n=783) found that visuo-spatial ability was significantly related only with FOG severity (Kelly *et al.*, 2015), possibly due to greater frontal and right posterior-parietal cortex deficits in those with FOG (Velu *et al.*, 2013; Handojoseno *et al.*, 2015). Understanding of visuo-spatial contribution to gait is further limited by lack of online studies (Kelly *et al.*, 2012b). For example, a recent study by Ricciardi *et al.* (2014) manipulated visuo-spatial ability during gait in a small cohort of PD using a dual task (i.e. completion of a visuo-spatial assessment shown on a projector screen while walking), but did not report gait characteristics during the task which limited findings. Test paradigms are not always considered with respect to other cognitive (i.e. attention) and visual functions which are not routinely assessed. A further issue is that laboratory manipulations may also be unrepresentative of real-world environments (Dowiasch *et al.*, 2015; Ottosson *et al.*, 2015).

Evidence from imaging

Imaging the brain while walking is impossible as the head has to remain still. To overcome this, protocols have used motor imagery or assays of gait in an attempt to understand the neural correlates of gait. Imaging studies generally demonstrate that gait involves a widely distributed neural network (Maillet *et al.*, 2012; Bohnen and Jahn, 2013; Herman *et al.*, 2013; Holtzer *et al.*, 2014). Although most studies have focussed on motor control, more recent work demonstrates overlap with neural networks associated with cognitive function such as the pre-frontal and frontal cortex (Seidler *et al.*, 2010; Shine *et al.*, 2013a). More recent work has used techniques such as functional near infra-red spectroscopy (fNIRs) that allow activity in the frontal cortex to be measured while a person is walking (Ferrari and Quaresima, 2012). These studies have shown that episodic gait impairment and postural control in PD are associated with online changes in frontal cortex activation (cerebral oxygenation: HbO_2) levels (Mahoney *et al.*; Maidan *et al.*, 2015). Similarly, fNIRs studies have shown increased PFC activation during dual task gait in older adults (Holtzer *et al.*, 2011; Doi *et al.*, 2013; Beurskens *et al.*, 2014). Also, studies exploring network functions and connectivity have shown a breakdown in connectivity between regions related to gait, attention, executive function (Fasano *et al.*, 2015; Sarasso *et al.*,

2015) and visuo-spatial ability (Nantel *et al.*, 2012), accompanied by greater right hemisphere dysfunction (Tessitore *et al.*, 2012; Fling *et al.*, 2013; Shine *et al.*, 2013b; Peterson *et al.*, 2014). To date, limitations to this emerging area of research include recruitment of mostly advanced cohorts and test protocols using techniques such as motor imagery or virtual reality, which may only partially represent online execution and therefore require cautious application (Cohen *et al.*, 2011).

Table 1 – Brief overview of Cognitive Deficits in Parkinson's disease and Older Adults

Cognitive Function	Definition/Background	Older adults	Parkinson's disease
Attention	An overarching cognitive function (Lückmann *et al.*, 2014). Ability to focus, select information (inhibition) and mediate parallel processes, allocating limited central processing capacity where relevant (Noudoost *et al.*, 2010).	Declines with age • Declines more rapidly than other cognitive functions (Sweeney *et al.*, 2001) • Deficits impact various aspects of attentional control such as inhibition seen in a number of tests such as the Stroop test (West and Alain, 2000)	Impaired • Commonly impaired even in those without dementia (Palavra *et al.*, 2013) • Relates to dysfunctional fronto-striatal and fronto-parietal pathways (Gerrits *et al.*, 2015) • Cholinergic dysfunction is also involved via nucleus basalis of Meynert and pedunculo-pontine nucleus input to the thalamus and cerebral cortex (Yarnall *et al.*, 2011) • Shown via neuropsychological tests and prolonged P3 latencies (Suna *et al.*, 2014) which increase with disease severity (Lopes *et al.*, 2014; Tang *et al.*, 2015)
Executive Function	Ability to plan and execute goal-directed behaviours (Ding *et al.*, 2015).	Declines with age • Linked to age-related frontal-striatal deterioration (Buckner, 2004) • Impairments impact on intention, initiation, inhibition and switching performance (Hull *et al.*, 2008)	Impaired • Sensitive to neuropsychological tests such as the Trail Making Test (Lewis *et al.*, 2003) • Early impairment which primarily involves the pre-frontal cortex and fronto-striatal pathway (Zgaljardic *et al.*, 2006) • Reflected by impairment in a range of cognitive skills such as poor inhibitory response (Ding *et al.*, 2015) • Linked to increased motor slowing and difficulties in planning (Weintraub *et al.*, 2005)
Working Memory	Ability to maintain and manipulate information over short time periods, which is linked to attentional control (Baddeley, 1992; Awh *et al.*, 2006).	Declines with age • Decline related to deterioration of attention (Gazzaley *et al.*, 2005) • Related to inhibition and decreased functional connectivity within large-scale brain networks (Fabiani *et al.*, 2015)	Impaired • Impairment is related to fronto-striatal (Robbins and Cools, 2014) and right hemisphere dysfunction (Foster *et al.*, 2013) • Not always apparent without the use of sensitive neuropsychological tests (Possin *et al.*, 2008)
Visuo-spatial ability	Ability to visually perceive the spatial relationships of objects. It is linked to attention and memory (Richards *et al.*, 1993).	Declines with age • Declines more than verbal cognitive tasks (Jenkins *et al.*, 2000) • Declines related to changes in underlying neural mechanisms (Klencklen *et al.*, 2012), which involve altered fronto-parietal projections (Drag *et al.*, 2015)	Impaired • Can be less impaired than other cognitive domains (Possin, 2010; Caproni *et al.*, 2014) • Associated with increased motor severity and freezing of gait (Nantel *et al.*, 2012) • Related to frontal and parietal lobe deterioration (Biundo *et al.*, 2013), with right hemisphere dysfunction implicated (Karádi *et al.*, 2015; Seichepine *et al.*, 2015) • Underlying structural changes of grey matter in frontal and temporal-parietal cortices impact this function (Pereira *et al.*, 2009; Rektorova *et al.*, 2014)

(Unless otherwise stated reported older adult impairments relate to comparison between older adults (>50 years old) and either younger adults or pathological groups, and reported Parkinson's disease impairments relate to comparison to a control group)

B. Role of vision in gait

Vision is a complex sensory system, involving integration of multiple structures and levels of information processing (Kaas, 2008). Critically vision relies on creation of various components (i.e. form, colour and movement) to allow interpretation of complex visual scenes (Cavanagh, 2011). Visual processes begin at the retina where photoreceptors absorb light and visual functions begin to break down the retinal image into its components (Itti and Koch, 2001) before sending the information to high-level areas for further processing (Wolfe, 1994) (Table 2). Integrity of these low-level visual functions is therefore vital for adequate vision.

Visual function in Parkinson's disease

Visual impairment is common in PD and is associated with gait dysfunction, although methodological issues (summarised in Table 2) necessitate cautious interpretation. The impact of visual impairment on gait has primarily been investigated in healthy young and older adults, with limited evidence in PD. Such studies demonstrate that age-related deficit in visual function is associated with reduction in activities of daily living, quality of life, mobility and is an independent risk factor for falls (Reed-Jones *et al.*, 2013; Uiga *et al.*, 2015). Visual pathology, such as glaucoma, cataracts and macular de-generation are a common and often under-reported problem in older adults. However these visual problems are seen in PD along with a wide range of other visual impairments, from impairment of basic functions such as visual acuity (VA) and contrast sensitivity (CS) to more complex processes such as depth perception, motion perception and optic flow (Armstrong, 2011), as shown in Table 2. Associations between visual impairments and gait in older adults may be stronger in PD especially as visual deficits increase with disease progression.

Vision and gait

Paradigms that explore the association between visual function and gait characteristics or manipulate vision in real-time while the participant is walking (e.g. navigating narrow doorway, lines on the floor, light and dark rooms) provide some understanding of the contribution of vision to gait in PD, as depicted in Figure 1(B). Impaired visual functions such as VA have been associated in PD and older adults with reduced step length (Spaulding *et al.*, 1994; Hallemans *et al.*, 2010) and gait velocity (Shin *et al.*, 2015), although this finding is not consistent (Klein *et al.*, 2003).

In PD, VA is the most commonly and often only assessed visual function. Changes in vision may not be adequately represented by VA alone (Geldmacher, 2003). CS is considered more applicable to real-world vision during gait, where the contrast of light and shade is critical. Indeed, impaired CS has been associated with reduced step width (Wood *et al.*, 2009), step length (Wood *et al.*, 2009; Swigler *et al.*, 2012), gait velocity (Moes and Lombardi, 2009; Wood *et al.*, 2009), physical activity levels (Black *et al.*, 2011), and fear of falling (Wang *et al.*, 2012). Other functions related to real-world vision such as dynamic VA have also been associated with falls (Honaker and Shepard, 2011). This indicates a need for comprehensive visual function assessment and more stringent methodological consideration. More complex assessments involving depth perception have been associated with increased obstacle contacts during gait (Menant *et al.*, 2010), likely due to impairment of obstacle height perception (Yamaji *et al.*, 2011). Motion perception (described in Table 2) has been associated with reduced functional task (e.g. driving) performance (Owsley, 2011), however despite obvious ties to gait it has largely been overlooked (Armstrong and Kergoat, 2015).

Optic flow is a similar concept to motion perception, and has predominantly been studied using online manipulation. Manipulation of optic flow while walking is carried out using video or projection based visual input (i.e. projected dots on a screen) shown at varying velocities to provide a sense of depth. In PD, significant gait impairments are found in velocity and step length (Lebold and Almeida, 2010) as well as increased veering (Davidsdottir *et al.*, 2008), with dysfunction of the right parietal cortex implicated (Davidsdottir *et al.*, 2008; Putcha *et al.*, 2014). Optic flow protocols however require intact depth perception (Simpson, 1993) and a limitation of these studies is that they do not control for visual deficits, as noted in Table 2. As a consequence it is unclear if gait impairment is a result of impaired depth perception (Lord *et al.*, 2002; Menant *et al.*, 2010) or indeed optic flow as suggested. Lack of an appropriate control group (older adults) in optic flow studies in PD (Lebold and Almeida, 2010; Almeida and Bhatt, 2012) and use of attentional tasks (such as lines on the floor to step on) which alter optic flow without consideration of cognitive processes further confound interpretation of findings.

Other studies with simple visual manipulations such as doorways (Cowie *et al.*, 2010; Cowie *et al.*, 2012) have shown reduction in gait velocity and step length, and

increased step time in PD (Lebold and Almeida, 2010; Pieruccini-Faria *et al.*, 2014). These studies suggest that people with PD become reliant on vision for gait (Azulay *et al.*, 1999; Azulay *et al.*, 2002; Khattab *et al.*, 2012). However many previous studies have involved visual occlusion (e.g. walking in a dark room) (Azulay *et al.*, 1999; Adamovich *et al.*, 2001; Almeida *et al.*, 2005), which merely provides a comparison of the contribution of proprioception compared to vision during gait (Stuart *et al.*, 2014). When vision is occluded, visual processing still occurs with visuo-spatial information obtained from working memory (Jackson *et al.*, 1995), which adds unnatural cognitive load during gait. Mimicking real-world environments with more subtle visual manipulations (such as adding a doorway) may provide insight into real-world impairments (Jackson *et al.*, 1995).

Table 2 – Brief overview of Visual Deficits in Parkinson's disease and Older Adults

Visual Function	Definition	Older adults	Parkinson's disease	Key Methodological Issues
Visual acuity (VA)	The ability to distinguish small details and shapes of objects (Kaiser, 2009).	Declines with age • Susceptible to decline from changes in ocular media (Sjostrand *et al.*, 2011), and changes in neural processing (Hennelly *et al.*, 1998)	Impaired • Associated with subjective reports of blurred vision (Jones *et al.*, 1992; Archibald *et al.*, 2011; Armstrong, 2011) • Linked to dopamine depletion in the retina (Archibald *et al.*, 2009)	Often non-significant impairment in PD compared to controls reported due to small sample sizes e.g. Galna *et al.* (2012). Often only visual function assessed.
Contrast sensitivity (CS)	The ability to differentiate between objects and their background (Evans and Ginsburg, 1985).	Declines with age • Susceptible to decline from changes in ocular media (Ross *et al.*, 1984), and changes in neural processing (Sloane *et al.*, 1988)	Impaired • Seen via standard visual chart assessment (Galna *et al.*, 2012) • Specific losses for spatial frequencies (Bodis-Wollner *et al.*, 1987; Price *et al.*, 1992; Swigler *et al.*, 2012) • Significant deficit in orientation discrimination for horizontal but not for vertical gratings (Mestre *et al.*, 1990)	Often non-significant impairment in PD compared to controls reported due to small sample sizes e.g. Galna *et al.* (2012).
Dynamic visual acuity	The ability to perceive an object when there is motion between the observer and the target (Ishigaki and Miyao, 1994).	Declines with age • Under all luminance, velocity, and duration conditions (Long and Crambert, 1990)	Impaired • Under all luminance, velocity, and duration conditions (Uc *et al.*, 2005; Taweekarn *et al.*, 2009)	Not often assessed.
Depth perception	The ability to perceive the world in three dimensions (3D) and the distance of an object (Omoto *et al.*, 2010).	Declines with age • Common in the absence of ocular morbidity (Wright and Wormald, 1992) • Decline is marked in those >60 years old (Garnham and Sloper, 2006)	Impaired • Common in drug naïve patients (Kim *et al.*, 2011) • Linked to reduction in gray matter volume in the right extra-striate visual cortex (Koh *et al.*, 2013)	Some studies limited by not assessing for nor excluding patients with vision affecting eye conditions e.g. Goodale and Haffenden (1998).
Motion perception	The process of inferring the speed and direction of elements in a scene (Ehrenstein, 2003).	Declines with age • Motion perception thresholds shown to be approximately two times higher in those 70-80 years old than individuals under thirty (Trick and Silverman, 1991)	Impaired • Motion perception thresholds significantly elevated (Trick *et al.*, 1994) • Linked to VA and CS impairment (Uc *et al.*, 2005)	Not often assessed.
Optic flow	Refers to the motion of the environment projected on the retina during movement in the world (Kelly *et al.*, 2005).	Declines with age • Decline in ability to localise and detect optic flow patterns (Berard *et al.*, 2009) • Affects navigation and steering control (Berard *et al.*, 2011)	Impaired • Linked to gait impairments such as veering and navigation issues (Davidsdottir *et al.*, 2008; Lin *et al.*, 2014) • Relates to impaired neural processing in visuo-vestibular (Putcha *et al.*, 2014) and feed-forward visuo-motor regions (van der Hoorn *et al.*, 2014)	Many studies use artificial assessment devices which require depth perception, but do not control for or exclude based on depth perception deficits.

(Unless otherwise stated reported older adult impairments relate to comparison between older adults (>50 years old) and either younger adults or pathological groups, and reported Parkinson's disease impairments relate to comparison to a control group)

C. The interaction between visual and cognitive function: Visuo-cognition

To date the interaction between visual and cognitive functions (Tables 1 and 2) during gait has not been considered in PD or older adults, but are instead distinguished from each other (Figure 1(C)). This distinction is somewhat contrived, given that evidence from static studies indicate an association between each function (Harris, 1998; Lin *et al.*, 2004) and gait in older adults and PD.

A recent review by Archibald *et al.* (2009) supported the notion that cognitive and visual functions interact in PD. Indeed, foveal retinal dopaminergic depletion (Bodis-Wollner, 2009) and structural changes (Bodis-Wollner, 2013) such as retinal thinning (Adam *et al.*, 2013; Bodis-Wollner *et al.*, 2013) can distort signals from visual functions which in turn influence subsequent cognitive processes. Abnormal visual processing within BG loops is also suggested to cause people with PD to become reliant on attentional compensation (Redgrave *et al.*, 2010). Imaging data demonstrates that attention can compensate for visual function deficits in healthy adults (Meppelink *et al.*, 2009), a mechanism which may be intact in early PD. Attention has been shown to influence stimulus appearance (Carrasco *et al.*, 2004) which can improve spatial resolution (Yeshurun and Carrasco, 1998; Carrasco *et al.*, 2002), and enhance contrast (Carrasco *et al.*, 2000; Pestilli and Carrasco, 2005; Carrasco, 2006) and salience via V4 neurons by up to 51% (Reynolds *et al.*, 2000). Attention is also related to increased visual processing speed in neurons as early as V1 (Carrasco and McElree, 2001; Pestilli and Carrasco, 2005). However, despite attentional compensation and the ability for levodopa to sustain dopamine within the retina (Archibald *et al.*, 2009) visual deficits such as slow visual processing persist in PD (Woollacott and Shumway-Cook, 2002). Importantly, compensation via attention is constrained because it is also impaired due to pathology, as noted above. Of further interest is the attenuation of visual control during gait when attentional demands increase, for example when walking under dual task conditions.

Cognitive and visual functions share the same neural resources and BG-cortical loops, and these overlap in striatal regions that have greater dopaminergic activity (e.g. ventral striatum) (Helmich *et al.*, 2010), which further implicates a role for PD pathology in visuo-cognitive interactions during gait. However, these interactions in PD are complex and the processes that underpin them remain unclear (Figure 1(C)). Cognitive functions, particularly attention activate and inhibit many structures during

visual processing (Buhmann *et al.*, 2015), giving rise to an internal priority (saliency) map (Baluch and Itti, 2011). Executive processes at the PFC signal an initial 'estimate' at the main visual priority (based on task goals) and project back via attentional circuits to the temporal cortex where selection is integrated into further automatic visual processing (Bar *et al.*, 2006). Therefore early cognitive biasing of visual input selection occurs before the automatic (bottom-up) visual processing cascade (Baluch and Itti, 2011), and would indicate that even though the two systems (vision and cognition) work in unison, cognitive functions may underpin basic visual functions (Borji *et al.*, 2011), especially during goal-orientated tasks such as gait.

Saccades during static tasks

Investigation of saccades during static tasks is one methodology that has allowed study of visuo-cognition in older adults and PD (van Stockum *et al.*, 2012). Saccades provide an online behavioural measure of visuo-cognition (Land, 2006) due to their links to both visual (Bridgeman *et al.*, 1981; Hernandez *et al.*, 2008) and cognitive functions, particularly attention (van Stockum *et al.*, 2011) (Figure 1). Saccades are integral to accurate task completion, as they align areas of interest in the environment with our fovea to produce high quality visual information (Bodis-Wollner, 2013; Bodis-Wollner *et al.*, 2013) for further cognitive processing.

Visuo-cognitive deficits in older adults are evidenced by ineffective visual search strategies (Becic *et al.*, 2008) and impaired saccades (Ridderinkhof and Wijnen, 2011) during static testing. Similarly people with PD demonstrate saccadic impairment when compared to older adults (Chan *et al.*, 2005; Mosimann *et al.*, 2005), with impaired voluntary (cognitively activated) and to a lesser extent reflexive (visual stimuli activated) saccades (Terao *et al.*, 2013). Voluntary saccades have been shown to be impaired more in advanced PD than early or moderate PD (Blekher *et al.*, 2000). Similarly Briand *et al.* (2001) and Terao *et al.* (2013) demonstrated that reflexive saccades are relatively preserved in early PD but worse in advanced PD. Other specific PD saccadic impairments have been highlighted in several recent reviews (Anderson and MacAskill, 2013; Srivastava *et al.*, 2014; Antoniades and Kennard, 2015), such as; hypometric saccades, initiation deficits including increased errors during anti-saccade tasks, reduced gain, increased

latency of voluntary saccades, reduced latency of reflexive saccades and abnormal facilitation during inhibition of return tasks.

Static studies have provided insight into underlying mechanisms involved in saccadic impairment in PD. Voluntary saccades are controlled by interaction between the frontal cortex, BG and brain stem (Javaid *et al.*, 2012; Matsumoto *et al.*, 2012). Recent investigations have shown that frontal pathology rather than motor severity is linked to saccadic deficits in PD (Perneczky *et al.*, 2011; Macaskill *et al.*, 2012; Tommasi *et al.*, 2015). However, dysfunction of the BG in PD also causes deficits in voluntary (top-down) saccades due to impairment of cortico-BG loops (Tommasi *et al.*, 2015). The BG inhibit and disinhibit information based on attentional signals from the PFC. Excessive inhibition on the superior colliculus (SC) by the BG with PD can impair voluntary and reflexive (bottom-up) saccades, as seen by increased errors in pro and anti-saccade tasks (Armstrong, 2011). Reflexive saccades are primarily controlled by the parietal cortex (posterior-parietal cortex and posterior eye-field) and the brain stem cholinergic system rather than the dopaminergic reward system (Terao *et al.*, 2013), and as such they are relatively spared in early PD. However the ability to inhibit reflexive saccades degrades with PD progression. In early disease, BG impairment can be circumvented with inhibition elicited via direct top-down influence from the PFC to the SC (Pierrot-Deseilligny *et al.*, 2004). Progressive dopamine depletion in the striatum with PD reduces the PFC inhibitory effect (Tommasi *et al.*, 2015). Therefore reduced PFC activity and disruption of the BG-thalmo-cortical loops results in an inability to suppress reflexive saccades (Deijen *et al.*, 2006). Combined voluntary saccade impairment and increased distractibility in PD during static tasks has implications for gait, as such visuo-cognitive impairment likely impacts gait control.

D. The role of visuo-cognitive processes in gait

As noted above, investigation of the role of vision and cognition as separate entities with respect to gait has led to some understanding of the mechanisms involved (see Figure 1 (A&B)). However because vision and cognition interact (Figure 1(C)) this is likely to have important implications for gait in PD (Figure 1(D)). Knowledge of visuo-cognitive processes during gait is therefore important and critical to fully understand mechanisms underlying gait impairment.

Saccades during gait and other real-world tasks

Visuo-cognitive processes during gait in PD have largely been investigated by monitoring saccades during gait (Figure 1). Only one previous study (Galna *et al.*, 2012) explored the association between vision, cognition and saccades, finding that saccade frequency during gait had greater association with attention than visual functions (VA and CS). To date no one has examined the relationship between saccadic and gait characteristics in PD (Figure 1(D)), but online studies have revealed important findings.

A recent structured review (Stuart *et al.*, 2014) summarised studies that report deficits in saccades and fixations (pauses between saccades) in PD during real-world tasks (i.e. gait, reaching, obstacle crossing etc.), which indicated the task-dependent nature of saccadic impairments. For example; during gait a reduction in saccadic frequency was found in PD compared to older adults, which although did not reach significance (Galna *et al.*, 2012), suggests impairment of voluntary saccadic initiation caused by dopaminergic depletion (van Stockum *et al.*, 2011). In contrast, saccade frequency was increased in PD during turning in place (Anastasopoulos *et al.*, 2011; Lohnes and Earhart, 2011), which may reflect impairment of smooth pursuit in PD and use of 'catch up' saccades to compensate (Helmchen *et al.*, 2012). Several other studies have also showed reduction in saccade and fixation activity during real-world tasks in PD (Uc *et al.*, 2006; Heremans *et al.*, 2012), including increased fixation durations (Sacrey *et al.*, 2009; Sacrey *et al.*, 2011) and reduced saccade latencies (Anastasopoulos *et al.*, 2011; Lohnes and Earhart, 2011). Similar differences in saccadic activity during gait in older adults are suggested to reflect compensatory adaptations (Uiga *et al.*, 2015), and the same could be true for those with PD. For example; a change in saccadic activity may help maintain online control of real-world tasks despite visual and cognitive impairment.

Saccadic activity differences during gait could also be an attempt to compensate for underlying visual, cognitive and motor deficits associated with PD. For example; reduced saccade latencies and longer fixation durations during gait in PD (Anastasopoulos *et al.*, 2011; Lohnes and Earhart, 2011) may be needed due to increased visual processing time required for motor programming, which attention is unable to expedite due to resources being preferentially allocated to maintaining gait

(Lee *et al.*, 2003). However such differences are possibly due to a number of underlying visuo-cognitive interactions yet to be fully investigated, such as; imbalance between dopaminergic and cholinergic systems, abnormal frontal processes involved in saccade facilitation influencing the SC, fluctuations of inhibitory mechanisms or facilitation from other regions such as the frontal and supplementary eye-fields (Terao *et al.*, 2011; van Stockum *et al.*, 2011; van Stockum *et al.*, 2012; Terao *et al.*, 2013; van Stockum *et al.*, 2013). Further investigation is required to understand specific saccadic impairments, and how these relate to cognitive function, visual function, and gait in PD.

Limitations

Given the broad topic of inquiry and the exploratory nature of this review which drew on inter-related but independent areas of research, we choose a narrative rather than a structured review process. As such, our critique of the literature was confined. However, this review was able to identify and draw attention to a research area that is currently not considered within gait research.

Summary and Conclusions

This narrative review covered a substantial body of literature and used a theoretical model to explore the contribution of vision, cognition and visuo-cognition to gait in PD. The use of associative and online protocols revealed a complex interdependence of these functions with evidence suggesting that attention may play a pivotal role. Exacting research is required to illuminate the field of inquiry and enhance our understanding of this relationship. This consolidated knowledge will then be used to optimise the management of gait dysfunction in PD, and thereby enhance overall function and quality of life.

With this in mind we make the following recommendations for future research:

- Assess vision, attention and cognition separately for independence and interaction
- Static and dynamic test protocols yield different but complimentary information
- A combination of methodological approaches (associative and online) provide a comprehensive understanding of the relative contribution of vision, cognition and visuo-cognition to gait impairment in PD

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Conflicts of Interest

No conflicts of interest are declared.

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