Locomotion and Posture in Older Adults

The Role of Aging and Movement Disorders

Fabio Augusto Barbieri
Rodrigo Vitório
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Preface

The world population is getting older, and, as a consequence, pathologies associated with aging, such as movement disorders, are becoming more common. Both natural aging and movement disorders impair structures and systems used for daily activities involving locomotion and posture. Therefore, understanding how these impairments affect motor control during walking and posture is critical for the design of innovative therapies and interventions to enable older adults with and without movement disorders to navigate home and community environments safely and independently.

This book was conceived to bring together the most updated knowledge in the area of posture and gait research, with special focus on the alterations caused by aging and movement disorders. Top researchers from all around the world were then invited to cover specific topics mastered by them, with enough freedom for them to include whatever they felt appropriate about the invited topics. Authors have different backgrounds which characterize a multidisciplinary approach for the content presented. The final result is absolutely fantastic, and we are truly delighted to introduce *Locomotion and Posture in Older Adults: The Role of Aging and Movement Disorders* to the literature.

The book includes four distinct parts. First, readers will find an introductory part that presents background concepts to motor control in aging and movement disorders. The following two parts cover specific topics related to walking and posture, respectively. The final part is dedicated to technology and rehabilitation designed to manage the impairments caused by aging and movement disorders on gait and posture.

Individual chapters are independent, and therefore, readers can have the choice of not following the chapter order presented in the book. It is even possible that, depending on the level of expertise, readers can choose to skip chapters or jump from chapter to chapter. We hope that all those interested in posture and gait research and/or rehabilitation and physical intervention, including undergraduate and graduate students, researchers, and professionals, will find helpful content in the book. Most importantly, we hope that readers will be able to transfer this knowledge to their daily practice.

We acknowledge the effort and high-quality work of all authors listed in the contributors. A huge “thank you” for helping us complete this book.

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Fabio Augusto Barbieri Assistant Professor at São Paulo State University (Unesp), Faculty of Science, Department of Physical Education, Campus Bauru – Brazil since April 2014. Bachelor in Physical Education from Unesp - Brazil (2004). Master of Science in Human Movement Science from Unesp - Brazil (2007). Double Ph.D. in Human Movement Science from Unesp – Brazil (2012) and Vrije University Amsterdam (VU) - The Netherlands (2013). Post-doctorate in Physical Education (2015) from Unesp – Brazil. Leads a research group at the Human Movement Research Laboratory – MOVI-LAB – Unesp. The main aim of the group is to understand neuromotor mechanisms underlying how human movement, mainly gait and posture, are planned and controlled. The research approach consists of a combination of experimental and clinical studies aimed at unraveling the interplay between organismic, task-related, and environmental constraints examining gait and posture in neurologically healthy individuals and people with neurodegenerative diseases, such as Parkinson's disease (PD) and Alzheimer's disease (AD).

Rodrigo Vitório Postdoctoral Fellow and supervisor of the graduate program in Human Movement Science at Universidade Estadual Paulista (Unesp, Rio Claro, Brazil). Visiting associate researcher at the Institute of Neuroscience and member of the Brain and Movement Research Group (Newcastle University, United Kingdom). Bachelor in Physical Education (2006), Master of Science (2009) and PhD (2015) in Human Movement Science. Has worked as Research Assistant at the Movement Disorders Research and Rehabilitation Centre at Wilfrid Laurier University (Waterloo, Canada—2013). Main research interests are focused on (1) how visual information is used for the control of locomotion in patients with Parkinson's disease, (2) how Parkinson's disease and its progression affect the ability to avoid obstacles while walking, (3) the prediction of falls in patients with Parkinson's disease and older adults, and (4) ageing and Parkinson's disease-related changes on brain cortical activity (EEG and fNIRS) during complex walking.

Part I

Background Concepts to Motor Control in Aging and Movement Disorders

Complex Systems Approach to the Study of Posture and Locomotion in Older People

1

Renato Moraes and Eliane Mauerberg-deCastro

Abstract

Traditionally, the aging process has been viewed as something negative, a phenomenon that eventually results in frailty. However, the development and adaptive processes involved with the aging process are now understood as part of a nonlinear, systemic reorganization of the body. This view sees the aging body—a biological system—as having subsystems that interact at different levels and with different time scales. In the present chapter, we discussed some concepts associated with the aging process. We began by using a dynamic systems perspective to discuss changes in movement patterns of older individuals. According to this perspective, effects of the aging process are not due to isolated changes in different structures, but to collective changes in interacting subsystems. We then presented the role of functional variability, in particular addressing postural control by illustrating that increases in body sway can be the result of an active exploration of the limits of stability. In the last two sections, we discussed in more detail how the application of the dynamic systems approach can help to explain older adults' postural control strategies, as well as the flexibility of their gait patterns. We employed general concepts of a dynamic systems perspective to discuss the aging process, using the notions of *variability*, *adaptive potential*, and complex *multi-scale levels* or *dimensions* and *multi-time sources* of constraints. Specifically, we included the functional role of aging on posture and locomotion control.

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Aging • Posture • Locomotion • Variability • Haptic information • Dynamic systems

1.1 Introduction

In the field of movement science, discussions about development and adaptive processes during aging often reflect a biased or negative perspective [1]. It is difficult not to focus on losses, since the progression of age ultimately marks the end of the cycle of life. The process of aging, as revealed by numerous research findings [e.g., [2]], affects structures and functions of the neuro-muscular-skeletal system in varying degrees. However, according to Sleimen-Malkoun et al. [3], aging also can be viewed as a *nonlinear systemic reorganization* of a biological system and subsystems (an individual), at complex *multi-scale levels* or *dimensions* and in *multiple* time scales.

Age-associated "frailty" is, in fact, a phenomenon that dynamically integrates the effects of an irreversible biologic process, a sedentary lifestyle, and comorbidities (previous and/or cumulative diseases or injuries). Although aging can be healthy, functional alterations occur in the relatively long time scale of the human life cycle, providing contexts and opportunities for the system to assume new and unique organizations. In general, changes due to aging result from interactions with the lifestyle an individual adopted in past years, as well as the incidence of deterioration associated with diseases. Interactions between aging and lifestyle are particularly noticeable in older individuals who are engaged in active athletic performance. They are found to be at low risk to cardiovascular diseases and obesity, for example [4, 5].

In the motor behavior literature, researchers commonly investigate older individuals' skills as potential factors that negatively affect adaptation and often lead to loss of independence in daily life. Mobility, dexterity, postural control of the body, and the countless activities of daily living are dynamically integrated as functions that reflect the complex behavioral possibilities of the human body [6]. These possibilities evolve, become more or less sophisticated, and then decline with changes in the adaptive capabilities of the inevitable aging process.

The most prevalent themes of such investigations in motor behavior include posture and gait. Besides maintaining the upright position in humans, posture ensures that other motor skills can functionally exist and evolve. Postural control typically is investigated in quiet or standing positions and then compared to tasks in which perturbation or other demands are applied. Postural control interacts with gait performance continuously, and this is particularly evident in transitional skills (e.g., turning and twisting the body, shifting to backward locomotion, sitting to lay down, sitting to walking, and others). Bipedal locomotion allows humans not only to explore exterior spaces, surfaces, and

surrounding objects (e.g., avoiding obstacles, going over objects, passing through apertures) but also to explore alternatives with regard to their own intrinsic dynamics. The architecture of legs, muscles, and neurons guarantees that walking has an invariant pattern among healthy humans, including older adults. Such structures undergo changes in time scales that range from an instant up to periods of years (i.e., multiple time scales). Furthermore, they integrate a *multi-scale dimension* (spatial) influence that limits behavior under perceived (i.e., informative) task constraints.

Balance and locomotion behaviors are extremely complex and, in addition to the coordination of multiple muscles and joints, require the use of multiple sensory information that help control the movements and that influence adaptations. In the older individual, the functional status of the muscles and joints, which helps detect information for control and adaptation to the environment, undergoes progressive changes. Falls are one of the main concerns for older people [7]. Studies in motor behavior attempt to identify risk factors that predict falls.

About one-third of noninstitutionalized older adults above age 65 experience a fall during a one-year period [8, 9], and most of these falls occur during locomotion [10, 11]. Among institutionalized older persons, this number increases to about two-thirds, or 61 % [12]. Traditionally, falls are related to increased variability in posture and gait [13]. However, variability also can be functional to older adults, and this is one of the topics we address in this chapter. Additionally, we discuss concepts from the *dynamic systems approach* and how these relate to changes in the posture and gait of older individuals. This chapter starts with a general overview of dynamic systems concepts and their application to the aging process. From a dynamic systems perspective, we discuss the notion of *variability* and its functional role on posture and locomotion control of older people.

1.2 The *Dynamic Systems Approach* and How It Can Explain Changes in Movement Patterns in Older Individuals

One of enduring challenges in aging research is to interpret the changes in performance in physical and mental activities that older people undergo. We often refer to a "decline" in order to explain the way in which our aging brain controls our limbs and movements. Research studies [2, 6, 8, 10] present much evidence about the extent of losses due to aging and of profile changes in bodily functions (e.g., control of the upright body position, mobility, and instrumental motor skills). Researchers continue to identify differences in older adults as compared to young adults. However, conceptual paradigms have begun to emphasize new ways in which we can discuss adaptation: not just a comparative approach, but those that consider multiple factors that are unique to the process of aging. One such paradigm is the *dynamic systems approach* [3].

Complex systems theories[1] emphasize the *heterarchical* importance of multiple subsystems and their contributions to emergent behaviors [19, 20], whether these occur early in life's development or throughout the lifespan, including the process of aging. These theoretical perspectives provide a unified approach to studying patterned behavior and, according to various scholars [21], can explain behavior patterns in a wide range of organisms and categories of actions with diverse constituent subsystems. When we apply dynamic systems principles to human development, some of the fundamental issues we need to address are: (1) How do changes in patterns emerge? (2) How do behaviors become more stable or less stable? (3) How do task constraints affect the stability and geometry of patterns of movements? (4) And how can diversity in patterns improve our understanding about the meaning of adaptation? For instance, the traditional understanding of motor learning and the development of movement skills is that emergent behavior associates with decreased variability. Conversely, increased variability is often discussed as a cause of impaired or limited outcomes and is associated with aging, disease, or impairment. However, variability is not a parameter outcome that consistently expresses the direction of development [22]. Low variability sometimes has been found among older individuals as a factor that limits a behavior's flexibility and, therefore, affects adaptation.

Research on aging typically has explained declines in separate systems: *neural*, *cognitive*, *sensorimotor*, and *muscular* [3]. Sleimen-Malkoun et al. [3] have argued in favor of a conceptual framework that would help us understand "the general principles of age-related reorganization of the neuro-muscle-skeletal system" based on the phenomena of *dedifferentiation* and *loss of complexity*. They define *dedifferentiation* as "a process by which structures, mechanisms of behavior that were specialized for a given function, lose their specialization and become simplified, less distinct or common to different functions" [3] (p. 2). *Loss of complexity* is a transition marker for aging and is observed when there is a tendency toward more regular fluctuations in physiological parameters [23]. In other words, the system exhibits "less complex patterns of variability" during aging [3].

Vaillancourt and Newell [24] reviewed *loss of complexity* relative to the notion that complexity has been examined using individual parameters (e.g., less variable heart rate in older individuals). They observed that there are many sources of constraints that influence the biological system—those that sometimes increase, and those that sometimes reduce, variability. In fact, the contexts in which many behavioral as well as physiological events occur are the result of numerous

[1]Michaels and Beek [14] identified three perspectives related to the study of information and action that currently prevail in studies of movement science and ecological psychology. The first perspective is *direct perception*, originally centered in Gibson's [15] assumptions. The second is related to the *kinetic theory* (thermodynamics) developed by Kugler, Kelso, and Turvey [16, 17]. The third is the *dynamic systems approach*, which employs tools and the analytical application of nonlinear dynamics to the study of movement coordination. Haken's [18] *synergetics* is a conceptual view in dynamic systems that deals with spontaneous pattern formation (i.e., self-organization) arising from systems far from their equilibrium point.

interactions between intrinsic and extrinsic parameters that constrain the functional outcome. In the case of aging, the so-called *limitation* can be the result of more complex mechanisms and choices of strategies available to the organism. Therefore, the outcome pattern can be less flexible to task contexts because biological constraints could include an (already) established skill that has been interacting with progressive physical changes. For example, Ko and Newell [25] demonstrated recently that center of pressure (COP) complexity, as measured using multi-scale entropy and detrended fluctuation analysis in young and old adults, is dependent on task demand. In a constant task, participants were asked to match COP position over a target line displayed on a monitor by leaning their bodies forward. In this task, COP complexity was lower for older adults as compared to the young adults. On the other hand, for the dynamic task (target moved following a sine-wave pattern), COP complexity was higher for the older than for the young adults.

Thus, even though invariant patterns can be found in motor behavior repertories due to a system's inherit flexibility, rapid behavior modulations can take place in response to subtle environmental changes (or new task demands). When these modulations go too far beyond a system's preferred behavioral mode, transitions to new behavioral patterns can occur, whether resilient to context challenges or not. These concepts may help us to understand how motor behavior modifies with the aging process, showing regular and, at the same time, flexible patterns. Yet, the common understanding about adaptive phenomena reinforces our notion that older individuals are less adaptive than younger ones.

1.3 Variability and the Control of Posture

The study of postural control in older adults started with Sheldon [26], who was the first to investigate the effect of age in the control of body sway. He found that children swayed more than young adults and that body sway started to progressively increase after the age of 60. These findings have been systematically replicated in hundreds of studies about body sway in older adults since this seminal work. However, there is a great deal of controversy about the meaning of this increase in body sway. For some researchers, this increase is indicative of imbalance in older adults, and this could be related to incidence of falls in this population [10]. Others argue that this increase in body sway is not necessarily a problem and, through a dynamic systems perspective, could be seen as an exploratory phenomenon [22].

Posture is understood as the relative position of various body segments in relation to each other and relative to the environment. Thus, humans can take an infinite number of positions during daily and sport activities, such as standing, walking, running, and throwing an object, among others. The posture in which both feet are in contact with the ground indicates the position commonly known as *bipedal upright posture*. The control of posture is essential for successfully achieving motor actions and implies the control of body position in space, with the double purpose of orientation and stability [27, 28]. Postural orientation involves the

proper positioning of the body segments relative to each other and to the environment. Stability during quiet standing involves the maintenance of the center of mass (COM) projection within the boundaries of the base of support.

Body sway is traditionally assessed based on the displacement of the COP obtained by force platforms. The resulting force applied to the ground due to the body's weight and internal forces generates the ground reaction force (GRF, vector equal and opposite to the force applied to the ground). The resulting GRF originates at a point called COP. The COP and GRF reflect the effect of the postural muscles, activated by the control system to actively control the position of COM and, therefore, stability [28]. During upright standing, the control of COP displacement in the anterior-posterior (AP) direction is due to the activity of the plantar flexor and dorsiflexor muscles of the ankle joint. In the medial-lateral (ML) direction, the control of COP displacement is achieved by the loading and unloading mechanism related to the action of hip adductors/abductors [29].

The ground reaction force opposes the force of gravity, which, in turn, affects the acceleration of the COM to maintain support against gravity. At the same time, the maintenance of balance during quiet standing requires, also, the maintenance of the COM projection within the limits established by the base of support. In quiet standing, the body continually oscillates both in the AP and ML directions, which is called spontaneous sway [30]. Interestingly, the COP is in phase with the COM, indicating a stable temporal relationship between these two variables. Furthermore, the amplitude of displacement of the COP is greater than the COM. This relationship between COP and COM has led to the suggestion that the COM is the variable to be controlled by the postural control system, while the COP is the variable that controls the position of the COM [29]. According to this interpretation and based on an inverted pendulum model, the increase in the COP displacement is deliberate, as this allows an individual to change the direction of the COM movement, moving it away from the limits of the base of support. Winter et al. [31] showed that the difference between COP and COM position correlates negatively with COM acceleration, indicating that the position of the COP ahead of the COM, for example, accelerates the COM backward. In this way, COP keeps the COM within the limits of the base of support.

The prevalent idea is that an increase in body sway represents postural instability [32]. In this traditional approach, feedback provided by the different sensory systems is essential to control body sway and, consequently, reduce it [30]. Additionally, increase in body sway is seen as a consequence of increase in noise within the system. In a study that employed this traditional background, Maurer and Peterka [30] developed a feedback model of postural control in humans. Their model was able to reproduce sway behaviors that resembled those of young and older adults. The model is based on the assumption that any angular deviation from the upright reference position, detected by sensory systems, should be corrected by the body's generation of appropriate corrective torque. Particularly important to this model is the addition of disturbance torque in the form of Gaussian noise to simulate spontaneous body sway. In this sense, noise would be responsible for the

increase in nonfunctional aspects of the variability in COP excursions. Interestingly, in order to resemble spontaneous body sway in older adults, the noise level would have to increase by 50 % as compared to young adults. As suggested by the authors, this increase in noise level could be due to a degradation of the sensory system, as observed during the aging process. This would result in impaired information about body sway in this population. Thus, the increase in COP sway observed with aging is seen as a result of increased noise within the system and, consequently, as an unstable system. This traditional perspective treats increase in sway (or variability) as detrimental to the individual [22].

Although the results of the model developed by Maurer and Peterka [30] are consistent with a continuous feedback regulation control mechanism, they admit that their results do not prove the model is necessarily used by the control system. In fact, a dynamic systems approach based on nonlinearity and complex systems argues in favor of the possibility that variability is functional to the postural control system [22, 33].

Carpenter et al. [34] designed a study to test the relationship between COP and COM during quiet standing. They developed an apparatus to artificially "lock" movements of the COM without the participants' awareness. They postulated that locking the COM would decrease COP displacement, which supports the traditional approach. However, their results showed a consistent and surprising increase in COP displacement after locking the COM. This finding contradicts the traditional approach, and the authors interpreted this increase in COP displacement as an exploratory role of postural sway. Even in the presence of visual feedback of COP or COM excursions in the locked condition, COP displacement increased [35]. Similarly, the provision of an explicit verbal cue that the participants would be "locked" did not reduce COP displacement [36]. In fact, COP displacement was again greater in the locked as compared to the unlocked condition, independent of the cue about being "locked" or not.

These authors argued that the central nervous system actively sways the body—using this as an exploratory mechanism to acquire sensory information. Thus, the information obtained through body sway would be essential to detect the position of the body relative to the limits of stability. This strategy would avoid adaptation of the sensory receptors while at the same time stimulate a wider range of receptors. Similarly, the use of stochastic resonance can boost the activation of muscle spindles [37, 38]. In a recent study, the use of stochastic resonance with participants who lightly touched their fingertip on a rigid surface resulted in additional reduction in body sway as compared to the traditional light touch paradigm without stochastic resonance [39]. According to Carpenter et al. [34], movement variability through postural sway can stimulate more receptors and an increased variety of sensory receptors (slow and fast adapting receptors). Yet, postural sway also can ensure that different sensory systems will be stimulated and provide converging and reliable information.

1.4 Older Adults and Their Strategies for Postural Control

As for the advocates of reduction in physiological complexity [23], the explanation about the effects of biological aging and/or disease on the postural system is that a particular parameter reflects the system's reduced physiological complexity, and, therefore, its limited adaptive capacity [40]. In their study, older participants with poor visual acuity and impaired foot sole sensation exhibited the lowest complexity in postural sway.

An alternative explanation—that is, in dynamic systems terms—of why older people undergo so many changes as the years advance is that the complexity of a system is determined by both *deterministic* and *stochastic* influences. These influences interact from moment to moment (multiple time scales) with the ever-changing state (aging) of the system [24].

The context of a particular behavior can illustrate how older individuals respond to perturbations to their balance. When performing balance tasks, healthy older individuals can use exploratory strategies that compensate for disruptions as long as meaningful contexts create the need for adaptation and as long as the system has resources to resolve them (the perturbations). For example, haptic cues during the use of tools can be integrated into a balance task—as in *haptic* tasks[2]—in order for these individuals to exploit the context of perturbation. The extent of postural perturbation is crucial for individuals to exploit the haptic task. In Fig. 1.1, a blindfolded 60-year-old adult reduces sway during a haptic anchoring task only when her foot position is sufficiently disruptive to her balance (i.e., the tandem position). When her feet are in parallel position, the COP path length reduces ~21 % during the anchoring task. However, when her body is anchored in tandem position, a reduction of 56 % is observed.

In a preliminary study about effects of haptic information embedded in a postural task (called "haptic anchoring"), Mauerberg-deCastro and Magre[3] reported that an older group of blindfolded adults (mean age 69) showed a larger amount of sway when the balance task included a tandem foot position in comparison to a parallel foot position. As shown in Fig. 1.2, anchoring provided haptic cues to reduce body sway in older participants in all task conditions, though to a much greater extent when the feet were in a *tandem* position (50 % decrease when compared to the baseline) than when parallel (23 % decrease when compared to the baseline).

[2]Haptic anchoring tasks require the manipulation of two strings, one in each hand, with an attached load mass for the purpose of reducing postural sway. In such tasks, an individual is required to pull the load just enough to keep the string tight, but without lifting the load from the surface of support. The tension of strings and loads' resistance during these exploratory actions (haptic anchoring) results in reduction of postural sway [41, 42].

[3]Unpublished study with data extracted from a research report: Mauerberg-deCastro E, Magre FL. (2015). Ancoragem háptica e controle postural em idosos [Haptic anchoring and postural control in older adults]. Research report. São Paulo State University, Brazil.

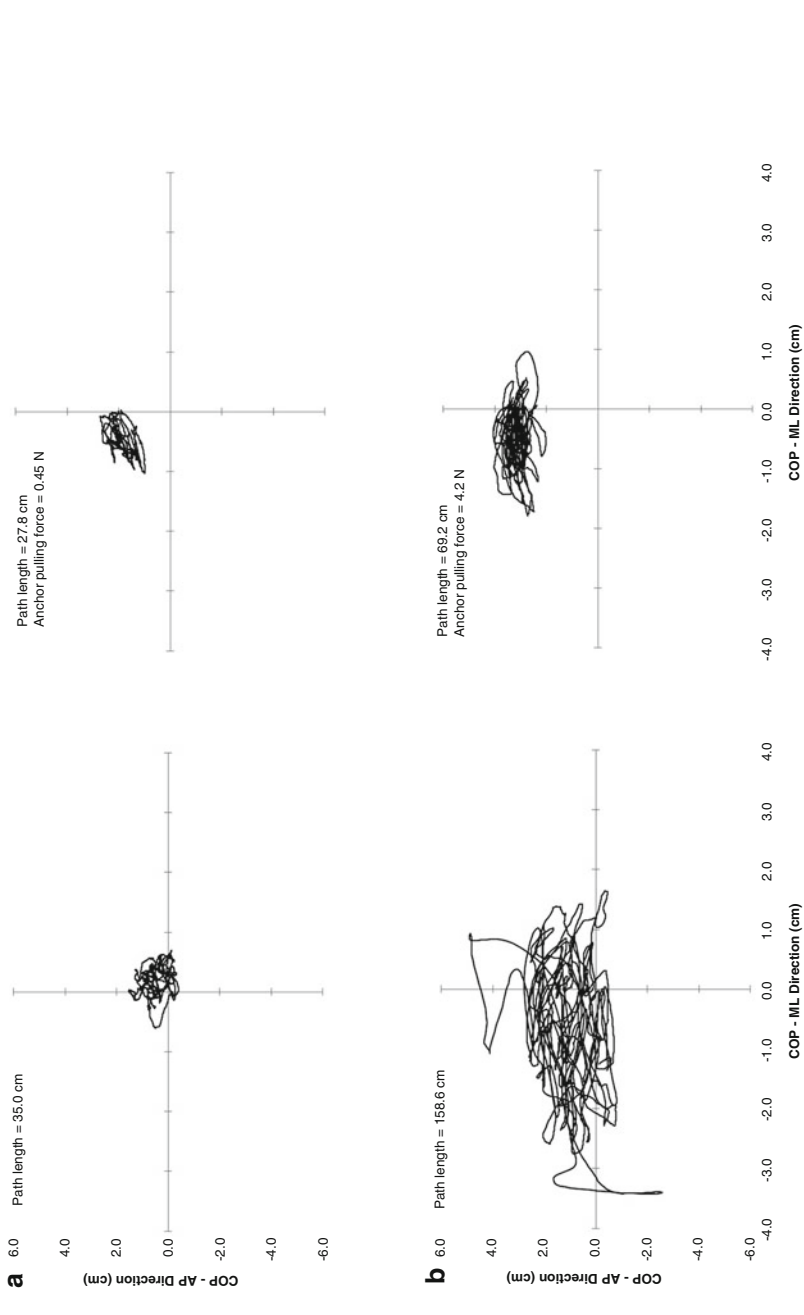

Fig. 1.1 COP displacement of an older blindfolded individual during a balance task while standing on a soft surface. *Left side* depicts baseline condition and *right side* the anchoring task in two-foot positions: parallel (**a**) and tandem position (**b**)

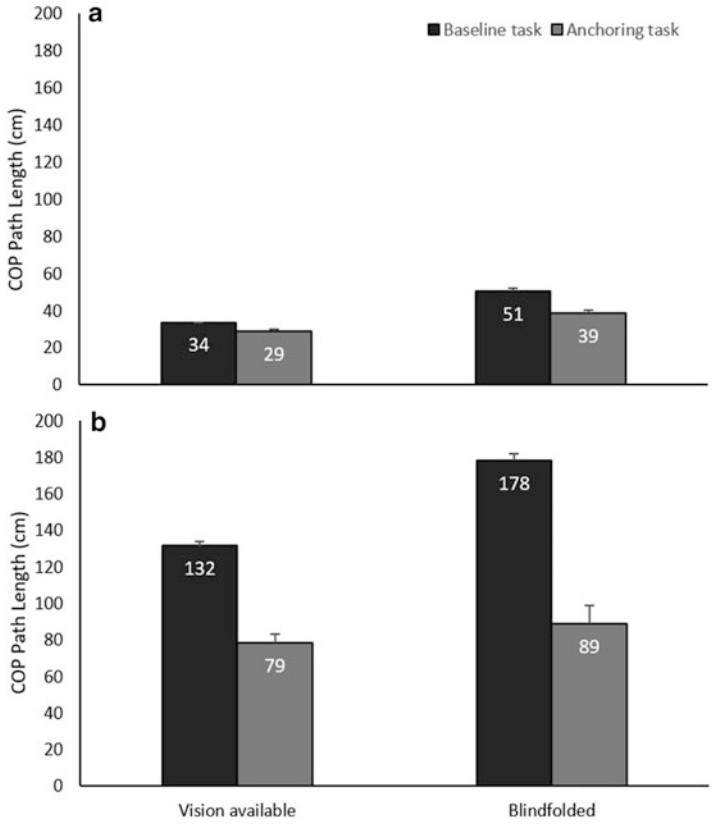

Fig. 1.2 Mean and standard error of COP's path length during a balance task on a soft surface, under baseline and anchoring task conditions, with vision available and blindfolded, in parallel foot position (**a**) and tandem position (**b**)

The task demands of controlling the anchors while keeping the body position stable revealed the extent of adaptability of the older group. The reduced ability to significantly alter the dynamics from one task to another appeared to depend on the degree of the task demand. The older group exploited the anchor context when the point of support on the surface was narrower (i.e., tandem foot position), more prominently reducing the magnitude of sway. This ability to adaptively weigh and select sensory references integrates task constraints. For older people, the exploitation of such a solution in order to maintain a relatively stable posture is a matter of safety [43]. While young adults generally have less traumatic consequences with falls, they can expand instability of the COM to a greater boundary. Cabe and Pittenger [44] considered the range of excursions of an object's COM to be limited by a *toppling point*, conceptualized as a *haptic angular tau*. As the postural control strategies of older adults include biomechanical parameters of the body (i.e., different anatomical structures and their lengths, mass, degrees of freedom) and

perceptual assemblies (i.e., information embedded in a particular task), the *toppling point* becomes the unstable equilibrium point that marks an impending moment for falling, or a phase transition to another behavioral pattern.

Concerning the notion of variability in postural control, as discussed above, the increase in postural sway can be seen as a functional aspect of motor control in older adults. Carpenter et al. [34] argued that the central nervous system intentionally increases COP sway "to ensure a certain quality and quantity of sensory information." Because of increased thresholds for sensory detection, older adults increase their body sway as a way to gain enhanced information about the limits of stability. On the other hand, the reduction in body sway with the addition of haptic cues reduces the need to sway the body to actively determine the limits of the base of support. Interestingly, Freitas et al. [45] showed, in a training study, that the intermittent use of an anchor system was the only condition that resulted in long-term effects on COP sway (i.e., reduced sway). These authors argued that the task variability embedded in the intermittent use of the anchor system was more beneficial to the postural control system of older adults than the repetitive use of the anchors.

1.5 Gait Patterns Are Flexible During Locomotion, Yet Regularity Is Task Context-Dependent

At any age, and for any open biological system, constraints set the boundaries of the system's behavior, leading to the emergence of semi-predictable, stable patterns. Even though the aging process is associated with losses, this adaptive phenomenon should not necessarily be interpreted as something bad or inefficient. Humans demonstrate multiple levels of flexibility in their behaviors [46]. At early ages, novice, immature infants demonstrate highly disorganized movement patterns, yet these patterns seem to provide opportunities for the infants to seek stable coordination solutions. This also is the case for older adults. Adaptation arises as a common, yet flexible, solution, which then results in movement patterns that appear similar to, and as stable as, those used by individuals in a wide range of ages [46].

Patterns of movements observed during our younger years versus our older years represent only an apparent difference. Spatial-temporal parameters of walking comparisons between older and young adults reflect an increase or decrease in parameters rather than in qualitative changes of movement patterns, as observed in comparisons between very young walkers and adults [47]. For example, novice walkers change the degrees of freedom of the lower limbs by freezing them as they acquire experience (e.g., limiting range of motion in the medial-lateral plane of walking step cycles) or releasing them (e.g., increasing range of motion in the knee and ankle joints to improve dampening of forces).

As for older adults who have experienced falls, changes in walking often include a significant decrease in speed and step length [48]. Such adjustments often are related to challenges in the environment (e.g., inclination of terrain, slippery surfaces, and presence of obstacles) and in the task requirements (e.g., performing

a simultaneous cognitive and motor task) and to the fact that humans use a variety of shoes to perform daily locomotion. For instance, shoes can be a risk factor for falls in older people [13]. While these authors found no differences between young and older adults' walking patterns with a variety of shoes, both adopted a conservative walking pattern when wearing elevated heel shoes. Rinaldi and Moraes [49] found that older adults with a history of falls (i.e., fallers) were unable to perform walking and prehension movements concomitantly. Fallers reduced their gait speed, especially when the prehension task was more difficult, which helped them to increase their margin of stability when grasping an object. Therefore, changes in behavioral outcomes often are a measure of compounded parameters, those that represent interplay of constraints rather than simple, individual variables (e.g., getting old).

Because comparative studies commonly use older individuals who are sedentary or potentially unhealthy, variable or unstable patterns of walking reflect particular strategies of the aging system, as well as physical limitations. In older adults with various health and physical conditions, Cozzani and Mauerberg-deCastro [50] found several common strategies in walking and stepping over obstacles. Sedentary and active community-dwelling older adults and institutionalized older adults slowed down while walking just prior to crossing over a 15-cm high obstacle, although the active group walked faster than the two other groups. These groups all exaggerated the vertical displacement of the clearing foot when crossing over a 15-cm obstacle, with the active group reaching a height of 20 cm and the sedentary group a height of 18 cm. The institutionalized group cleared the obstacle by only 2 cm, which, in this case, could have been the result of muscle weakness of the legs, which likely limited the amount of vertical foot elevation. The typical vulnerability (e.g., weak muscle strength of lower limbs) of older individuals who are institutionalized likely reflects their limited ability to exaggerate the movement. When crossing over obstacles or irregular surfaces, this weakness exposes them to risks for tripping and falling. All groups crossed the 2-cm obstacle height with a similar vertical foot elevation (~13-cm vertical displacement). To compensate for physical limitations, the sedentary and institutionalized individuals varied the horizontal distance of the last step according to the obstacle's height. The active individuals maintained a constant step length across obstacle heights. The exaggeration of vertical elevation imposes a high demand in the maintenance of balance—standing on one foot while the other swings to cross over the obstacle—but it likely serves as a compensation strategy to prevent collisions with obstacles. Is this exaggeration of movement a sign of awareness and fear of the potential danger of collisions leading to falls, even by physically active older individuals? The literature shows that, indeed, older individuals slow their locomotion patterns when a challenge is perceived to be a risk for falls [51]. Healthy and non-healthy older people equally perceive that they are at risk for falls.

Sources of constraints during the aging process are diverse enough to maintain a system's sensitivity to its own changes as well as to changes in the task context [52]. Whether older individuals misjudge or exaggerate their perceptions, a single explanation is not sufficient for us to understand everything about the aging process. For

example, research shows a contrast between age groups with regard to sensitivity to movement and actual physiological condition [53]. Moraes and Mauerberg-deCastro [54] found that older individuals perceived levels of difficulty of sitting and standing up tasks, from different chair heights, in a similar way as do young adults. However, the older individuals slowed down their motion, particularly during the descent phase of sitting down. Their movement patterns reflect instability and irregularity as the height of the chairs was lowered less than the typical 90° sitting position. One potential concern is that older adults may be unaware of their motor instabilities, and, by erroneously perceiving all chair heights as equal, they expose themselves to falls.

As sitting down and standing up can be integrated tasks in daily living activities, locomotion is a contingency of mobility and, therefore, a criterion for independence. Although locomotion is a relatively simple skill, older individuals' locomotion can reflect risks for falls, especially in the presence of obstacles. Thus, the immediate adaptations due to the demands of the environment and the task have direct implications with the functional conditions of the perceptual-motor systems [55]. For example, changes in locomotion patterns that are originally caused by limitations in postural control include reduction of speed, changes in the durations of the phases of stepping, and the addition of new control strategies (e.g., dragging the feet), among others.

Task constraints such as speed, direction, and movement magnitude are important variables that impact movement performance. This can be illustrated, for example, when healthy and older individuals with Down syndrome walk on a padded surface, a 15-cm thick mattress [46]. The older individuals attempted to dampen their steps, which they perceived were too fast and unsafe. If a person has balance problems, changes in their locomotion behavior may increase risk for falls as soon as the parameters escalate (i.e., speed, cadence). Older people become quite cautious and do not go beyond their perceived safe limits with regard to a particular behavior or skill. Figure 1.3 illustrates the gait pattern (i.e., phase portrait) of a young adult and an older adult with Down syndrome. The young adult shows a smooth, regular kinematic pattern of the shank displacements during walking. To the contrary, the walking topology of the older adult with Down syndrome is marked by irregular trajectories and limited excursions of the attractor in the state space [46].

During locomotion, older individuals maximize their biomechanical possibilities, whether in the presence of a disability condition or physical deteriorations (e.g., loss of vision acuity), and they respond to task and environmental perturbations by adjusting their skill parameters (e.g., slowing down the movement). In order to explain the adaptability of the locomotion behavior, we need to consider that the irregular, controlled, and slow motions are *solutions* to the perturbation, caused by the unstable surface. When locomotion slows down drastically, there is a higher demand on the postural control system, and ballistic portions of the motion have to be suppressed by an increased dampening of the body.

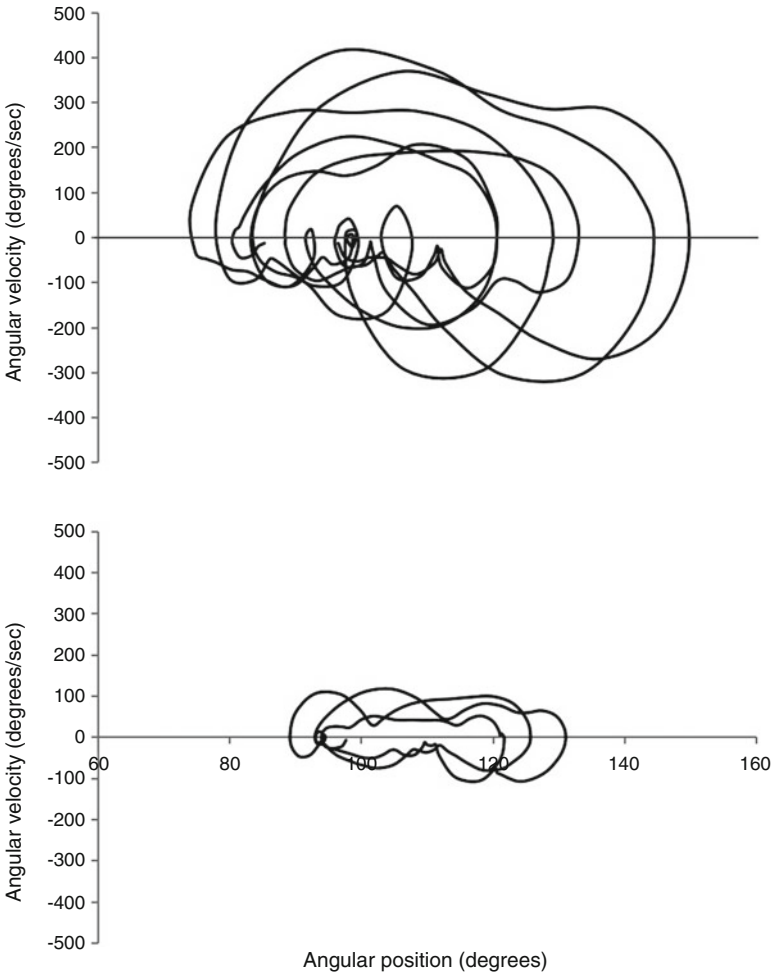

Fig. 1.3 Phase portraits of the shank segment of a young adult (*top*) and an older adult (*bottom*) with Down syndrome walking on a padded surface (adapted from data published in Mauerberg-deCastro and Angulo-Kinzler [46])

In a similar way as above, the addition of haptic cues (i.e., dragging an anchor system on the floor while performing various walking tasks) shows the adaptability of the walking behavior in older adults. During tandem walking, older adults who dragged the anchors showed reduced trunk sway acceleration in the frontal plane [41]. The haptic cues provided by the anchors allow an individual to expand his or her possibilities for exploring the adjacent environment and, consequently, to use this information about his or her body position relative to the ground to adaptively reduce body sway while walking.

1.6 Final Remarks

In this chapter, we employed general concepts of a dynamic systems perspective to discuss the aging process. We used the concepts of *variability*, *adaptive potential*, *complex multi-scale and multi-level*, and different *time-varying sources of constraints* that challenge the traditional notion of *frailty* in aging. Specifically, we approached these discussions with regard to the functional role of aging on posture and locomotion control.

While individual differences exist and provide potential for adaptive success, the variability of behavior outcome does not necessarily rely on the notion of "good" or "bad" control solutions. Stability, or regularity, in movement patterns is found to be complex, and it reflects diverse strategies in individuals of various ages. Often, older people experience the combined effects of aging and health deterioration, and, therefore, their systems and subsystems organize solutions with different levels of complexity.

Sometimes a solution is the result of an organism's lack of flexibility, sometimes not. Older individuals may resort to a few motor behavior solutions that can be considered effective. Exploitation of a task context must take into account a number of factors, including those that are not directly relevant to a function (e.g., unrealistic fear of falling when there is no explicit postural perturbation). The dynamic systems perspective provides a useful lens from which to reexamine the discontinuous process of aging and reinterpret change not as the body's inevitable journey to "frailty," but as a transition to possibilities for adaptation.

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References

1. Hong SL, James EG, Newell KM. Coupling and irregularity in the aging motor system: tremor and movement. Neurosci Lett. 2008;433:119–24. doi:10.1016/j.neulet.2007.12.056.
2. Spirduso WW, Francis KL, MacRae PG. Physical dimensions of aging. 2nd ed. Champaign: Human Kinetics; 2005.
3. Sleimen-Malkoun R, Temprado JJ, Hong SL. Aging induced loss of complexity and dedifferentiation: consequences for coordination dynamics within and between brain, muscular and behavioral levels. Front Aging Neurosci. 2014;6:140. doi:10.3389/fnagi.2014.00140.
4. Leyk D, Rüther T, Wunderlich M, Sievert A, Eßfeld D, Witzki A, et al. Physical performance in middle age and old age: good news for our sedentary and aging society. Dtsch Arztebl Int. 2010;107:809–16. doi:10.3238/arztebl.2010.0809.
5. Rittweger J, Kwiet A, Felsenberg D. Physical performance in aging elite athletes--challenging the limits of physiology. J Musculoskelet Neuronal Interact. 2004;4:159–60.
6. Van Lummel RC, Walgaard S, Pijnappels M, Elders PJM, Garcia-Aymerich J, Van Dieën JH, Beek PJ. Physical performance and physical activity in older adults: associated but separate domains of physical function in old age. PLoS One. 2015;10, e0144048. doi:10.1371/journal.pone.0144048.

7. Iezzoni LI, McCarthy EP, Davis RB, Siebens H. Mobility difficulties are not only a problem of old age. J Gen Intern Med. 2001;16:235–43.
8. Lord S, Sherrington C, Menz H, Close J. Falls in older people: risk factors and strategies for prevention. 2nd ed. Cambridge: Cambridge University Press; 2007.
9. Siqueira FV, Facchini LA, Piccini RX, Tomasi E, Thumé E, Silveira DS, et al. Prevalência de quedas em idosos e fatores associados [Prevalence of falls and associated factors in the elderly]. Rev Saude Publica. 2007;41:749–56. doi:10.1590/S0034-89102007000500009.
10. Berg WP, Alessio HM, Mills EM, Tong C. Circumstances and consequences of falls in independent community-dwelling older adults. Age Ageing. 1997;26:261–8.
11. Norton R, Campbell AJ, Lee-Joe T, Robinson E, Butler M. Circumstances of falls resulting in hip fractures among older people. J Am Geriatr Soc. 1997;45:1108–12.
12. Tinetti ME. Factors associated with serious injury during falls by ambulatory nursing home residents. J Am Geriatr Soc. 1987;35:644–8.
13. Menant JC, Perry SD, Steele JR, Menz HB, Munro BJ, Lord SR. Effects of shoe characteristics on dynamic stability when walking on even and uneven surfaces in young and older people. Arch Phys Med Rehabil. 2008;89:1970–6. doi:10.1016/j.apmr.2008.02.031.
14. Michaels C, Beek P. The state of ecological psychology. Ecol Psychol. 1995;7:259–78.
15. Gibson JJ. The senses considered as perceptual systems. Boston: Houghton Mifflin; 1966.
16. Kugler PN, Kelso JAS, Turvey MT. On the concept of coordinative structures as dissipative structures: I. Theoretical lines of convergence. In: Stelmach GE, Requin J, editors. Tutorials in motor behavior. Amsterdam: North-Holland; 1980. p. 3–47.
17. Kugler PN, Kelso JAS, Turvey MT. On the control and co-ordination of naturally developing systems. In: Kelso JAS, Clark JE, editors. The development of movement control and co-ordination. New York: Wiley; 1982. p. 5–87.
18. Haken H. Synergetics, an introduction: non-equilibrium phase transitions and self-organization in physics, chemistry, and biology. 3rd ed. Berlin: Springer; 1983.
19. Abernethy B, Sparrow WA. The rise and fall of dominant paradigms in motor behaviour research. In: Summers JJ, editor. Approaches to the study of motor control and learning. Amsterdam: Elsevier Science Publishers; 1992. p. 3–45.
20. Mauerberg-deCastro E. Abordagens teóricas do comportamento motor. Conceitos dinâmicos aplicados aos processos adaptativos e à diversidade do movimento [Theoretical approaches in motor behavior. Dynamic concepts applied to adaptive processes and diversity of movement]. In: Guedes MG, editor. Aprendizagem motora [Motor learning]. Lisboa: Edições FMH; 2001. p. 105–25.
21. Thelen E, Smith L. A dynamic system approach to the development of cognition and action. Cambridge: MIT Press; 1994.
22. Van Emmerik REA, Van Wegen EEH. On the functional aspects of variability in postural control. Exerc Sport Sci Rev. 2002;30:178–83. doi:10.1097/00003677-200210000-00007.
23. Lipsitz LA, Goldberger AL. Loss of "complexity" aging. Potential applications of fractals and chaos theory to senescence. JAMA. 1992;267:1806–9.
24. Vaillancourt DE, Newell KM. Changing complexity in human behavior and physiology through aging and disease. Neurobiol Aging. 2002;23:1–11.
25. Ko JH, Newell KM. Aging and the complexity of center of pressure in static and dynamic postural tasks. Neurosci Lett. 2016;610:104–9. doi:10.1016/j.neulet.2015.10.069.
26. Sheldon JH. The effect of age on the control of sway. Geront Clin. 1963;5:129–38.
27. Horak F, MacPherson JM. Postural orientation and equilibrium. In: Rowell LB, Shepherd JT, editors. Handbook of physiology - section 12. Exercise: regulation and integration of multiple systems. New York: Oxford University Press; 1996. p. 255–92.
28. MacPherson JM, Horak FB. Posture. In: Kandel ER, Schwartz JH, Jessell TM, Siegelbaum SA, Hudspeth AJ, editors. Principles of neural science. New York: McGraw Hill; 2013. p. 935–59.
29. Winter DA. Anatomy, biomechanics and control of balance during standing and walking. Waterloo: Waterloo Biomechanics; 1995.

30. Maurer C, Peterka RJ. A new interpretation of spontaneous sway measures based on a simple model of human postural control. J Neurophysiol. 2005;93:189–200. doi:10.1152/jn.00221.2004.
31. Winter DA, Patla AE, Prince F, Ishac M, Gielo-Perczak K. Stiffness control of balance in quiet standing. J Neurophysiol. 1998;80:1211–21.
32. Van Wegen EEH, Van Emmerik REA, Riccio GE. Postural orientation: age-related changes in variability and time-to-boundary. Hum Mov Sci. 2002;21:61–84.
33. Van Emmerik REA. Functional role of movement variability in movement coordination and disability. In: Davis WE, Broadhead GD, editors. Ecological task analysis and movement. Champaign: Human Kinetics; 2007. p. 25–52.
34. Carpenter MG, Murnaghan CD, Inglis JT. Shifting the balance: evidence of an exploratory role for postural sway. Neuroscience. 2010;171:196–204. doi:10.1016/j.neuroscience.2010.08.030.
35. Murnaghan CD, Horslen BC, Inglis JT, Carpenter MG. Exploratory behavior during stance persists with visual feedback. Neuroscience. 2011;195:54–9. doi:10.1016/j.neuroscience.2011.08.020.
36. Murnaghan CD, Squair JW, Chua R, Inglis JT, Carpenter MG. Are increases in COP variability observed when participants are provided explicit verbal cues prior to COM stabilization? Gait Posture. 2013;38:734–8. doi:10.1016/j.gaitpost.2013.03.012.
37. Collins JJ, Imhoff TT, Grigg P. Noise-enhanced tactile sensation. Nature. 1996;383:770.
38. Cordo P, Inglis JT, Verschueren S, Collins JJ, Merfeld DM, Rosenblum S, et al. Noise in human muscle spindles. Nature. 1996;383:769–70.
39. Magalhães FH, Kohn AF. Vibratory noise to the fingertip enhances balance improvement associated with light touch. Exp Brain Res. 2011;209:139–51. doi:10.1007/s00221-010-2529-3.
40. Manor B, Costa MD, Hu K, Newton E, Starobinets O, Kang HG, Peng CK, Novak V, Lipsitz LA. Physiological complexity and system adaptability: evidence from postural control dynamics of older adults. J Appl Physiol. 2010;109:1786–91. doi:10.1152/japplphysiol.00390.2010.
41. Costa AAS, Manciopi PAR, Mauerberg-deCastro E, Moraes R. Haptic information provided by the "anchor system" reduces trunk sway acceleration in the frontal plane during tandem walking in older adults. Neurosci Lett. 2015;609:1–6. doi:10.1016/j.neulet.2015.10.004.
42. Mauerberg-deCastro E, Moraes R, Tavares CP, Figueiredo GA, Pacheco SM, Costa TDA. Haptic anchoring and human postural control. Psychol Neurosci. 2014;7:301–18. doi:10.3922/j.psns.2014.045.
43. Johnson CB, Mihalko SL, Newell KM. Aging and the time needed to reacquire postural stability. JAPA. 2003;11:459–69.
44. Cabe PA, Pittenger JB. Time-to-topple: haptic angular tau. Ecol Psychol. 1992;4:241–6.
45. Freitas MBZ, Mauerberg-deCastro E, Moraes R. Intermittent use of an "anchor system" improves postural control in healthy older adults. Gait Posture. 2013;38:433–7. doi:10.1016/j.gaitpost.2013.01.004.
46. Mauerberg-deCastro E, Angulo-Kinzler R. Locomotor patterns of individuals with Down syndrome: effects of environmental and task constraints. In: Elliot D, Chua R, Weeks D, editors. Perceptual-motor behavior in Down syndrome. Champaign: Human Kinetics; 2000. p. 71–98.
47. Halverson LE. Development of motor patterns in young children. Quest. 1966;6:44–53.
48. Giladi N, Herman T, Reider II G, Gurevich T, Hausdorff JM. Clinical characteristics of elderly patients with a cautious gait of unknown origin. J Neurol. 2005;252:300–6.
49. Rinaldi NM, Moraes R. Older adults with history of falls are unable to perform walking and prehension movements simultaneously. Neuroscience. 2016;316:249–60. doi:10.1016/j.neuroscience.2015.12.037.
50. Cozzani M, Mauerberg-deCastro E. Estratégias adaptativas durante o andar na presença de obstáculos em idosos: impacto da institucionalização e da condição física [Adaptive strategies during walking over obstacles by elderly: effects of institutionalization and physical condition]. Rev Bras Educ Fis Esp. 2005;19:49–60.

51. Callisaya ML, Blizzard L, McGinley JL, Srikanth VK. Risk of falls in older people during fast-walking – The TASCOG study. Gait Posture. 2012;36:510–5. doi:10.1016/j.gaitpost.2012.05.003.
52. Saltzman EL, Kelso JAS. Skilled actions: a task-dynamic approach. Psychol Rev. 1987;94:84–106.
53. Delbaere K, Close JC, Brodaty H, Sachdev P, Lord SR. Determinants of disparities between perceived and physiological risk of falling among elderly people: cohort study. BMJ. 2010;341:c4165. doi:10.1136/bmj.
54. Moraes R, Mauerberg-deCastro E. Relação entre percepção e ação durante os movimentos de sentar e levantar em indivíduos idosos [The relation between perception and action during the stand-to-sit and sit-to-stand movements in elderly individuals]. Psic Teor e Pesq. 2010;26:253–64. doi:10.1590/S0102-37722010000200007.
55. Goldfield EC. Emergent forms. New York: Oxford University Press; 1995.

Clinical Aspects of Movement Disorders: Effects on Walking and Posture

2

Henrique Ballalai Ferraz and Roberta Arb Saba

Abstract

Movement disorders are a group of syndromes which includes parkinsonism, chorea, dystonia, myoclonus, tremor, and tics. Many of the diseases of this group manifest disturbances of gait and posture along with other abnormalities. Parkinsonism is characterized by rest tremor, muscular rigidity, bradykinesia, flexed posture of the trunk, and loss of postural reflexes. Parkinson's disease (PD) is the most prevalent of the diseases of this group and patients usually manifest problems of gait and posture in late phases. On the other hand, atypical parkinsonism diseases (multiple-system atrophy, progressive supranuclear palsy, dementia with Lewy bodies, and corticobasal ganglionic degeneration) manifest gait problems and falls very early and are much more incapacitating than PD. Vascular parkinsonism manifests itself with lower limb rigidity and bradykinesia and affects gait very early. Huntington's disease is a genetic disorder characterized by behavioral and cognitive disturbances associated with chorea, dystonia, and loss of equilibrium. Sometimes, especially in subjects with the juvenile form of Huntington's disease, it may manifest with a very severe parkinsonism.

Keywords

Movement disorders • Parkinsonism • Parkinson's disease • Parkinson-plus syndromes • Multiple-system atrophy • Progressive supranuclear palsy • Dementia with Lewy bodies • Corticobasal ganglionic degeneration • Vascular parkinsonism • Huntington's disease

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2.1 Introduction

Some of the diseases of the spectrum of movement disorders are associated with abnormalities of posture and locomotion. We can mention the multiple forms of parkinsonism, Huntington's disease, and generalized dystonia, among others. Posture and walking may be affected by the involuntary movements per se or by degeneration or lesions of neural pathways involved in tonus and balance. In this chapter we will present some of the diseases associated with abnormal involuntary movements and impairment of posture and gait.

2.2 Parkinsonism

Parkinsonism is a syndrome characterized by the combination of bradykinesia, also called akinesia, with any of the three: muscular rigidity, rest tremor, or impairment of postural reflexes.

Bradykinesia is the reduction of the global repertoire of automatic movements, and the decrease of the amplitude and the speed of the voluntary movements. We can detect bradykinesia in a parkinsonian patient either by observing the lack of facial expression while talking or listening with the examiner or by the reduced arm and hand gestures during a conversation. In other words, bradykinetic patients tend to have a poor repertoire of body language [1].

Muscular rigidity is observed by a resistance while manipulating a joint of a limb. Resistance is present both during passive flexion and extension of a limb. We can also feel the sensation of being manipulating a lead pipe, and sometimes this is associated with the clinical sign called "cogwheel rigidity." This is perceived as an intermittent and very short-lived release of the resistance during a full extension or flexion movement [2].

Rest tremor can be observed in any part of the body. Hand tremor manifests as a rhythmic flexion and extension of the fingers and it is called "pill-rolling tremor." Tremor can also affect the lower limbs, trunk, chin, head, and larynx. It may cycle in a fashion varying from 4 to 6 Hz. Sometimes, parkinsonian patients may present with a postural and kinetic hand tremor associated or not with the resting tremor [1].

Impairment of postural reflexes is characterized by an inability to correct sudden shifts of the normal upright position. People with this kind of impairment usually have frequent falls and assume a stooped posture. The more the trunk is bent forward, the higher the risk of having falls [2].

The combination of the four cardinal symptoms results in the typical parkinsonian gait and posture. Patients tend to flex the trunk forward and the arms are flexed close to the level of the hip. The arms do not harmonically sway while the body is moving. The turning of the body is made by slow and fragmented successive short movements. The length of the steps is very short, and heels drag to the floor. Sometimes, due to the flexed posture, parkinsonian people tend to fall forward, and to avoid it they shift the slow steps to a running pace. This is called festination or festinating gait and when present is a classical manifestation of parkinsonism.

Too many clinical conditions may precipitate parkinsonism. The most frequent is idiopathic Parkinson's disease (PD). Other situations are pharmacologically induced parkinsonism (mainly antipsychotics), metal or poison exposure (manganese, pesticides, carbon monoxide), vascular lesions of the central nervous system (CNS), hereditary diseases, some dementia syndromes (Alzheimer's disease and frontotemporal dementia), and other degenerative diseases called atypical parkinsonism or Parkinson-plus syndromes (progressive supranuclear palsy, multiple-system atrophy, dementia with Lewy body, and corticobasal ganglionic degeneration). We will discuss some of the specific diseases associated with parkinsonism.

2.2.1 Parkinson's Disease

PD is characterized by motor and non-motor symptoms and is associated with a progressive degeneration of neurons of the CNS. The degenerative process seems to start in the medulla and pons and progresses to a wider distribution in the encephalon. Motor symptoms (parkinsonism) begin when dopaminergic neurons of the substantia nigra are affected. Nigral neurons become progressively scarce and there is the presence of Lewy bodies, an eosinophilic intraneuronal inclusion, the classical pathological hallmark of PD [3].

PD may affect adult individuals of both gender and at any age, although most frequently those with 50 or more years of age. Unilateral symptoms are the most common way of starting the disease, and the other side tends to be affected in the following months or years. Patients may manifest rest tremor in one arm or feel difficulty in moving one limb. In a few months, the symptoms tend to become more and more intense and eventually will interfere with daily activities. Difficulty in arising from chair or bed is also common complaints in the early phase of the disease. Gait and postural impairment are usually present after a few years of onset [4].

Fully manifested PD includes bradykinesia, rigidity, and postural abnormalities with gait and balance impairment. PD patients tend to have a shuffling gait and a tendency to have falls. Festinating gait is a classic parkinsonian sign and results from the combination of the flexed forward posture, the short steps, and the loss of postural reflexes. In this case, patients tend to accelerate the steps in order to catch up the center of the gravity abnormally placed forward.

Freezing of gait may be present in many patients and is characterized by a difficulty in taking out the feet off the floor while attempting to start the gait. Freezing of gait is more frequent when patients try to turn around or during the passage of narrow spaces. The "pull test" is the clinical way to detect impairment of postural reflexes. Patients are put standing with feet comfortably apart. The doctor stands backward and pulls him or her briskly and forcefully. The balance is considered impaired if the patient has to move backward for at least three steps of recovery. In severely affected patients, the examiner has to catch the patients to prevent the fall.

Considering only the clinical presentation of PD, there are two main subtypes: a tremor-dominant form and a postural impairment with gait disturbance (PIGD). The first one is characterized by a more benign progression, with excellent response to levodopa and late development of cognitive decline. The PIGD form has a faster progression, with less response to levodopa and early development of falls and cognitive decline [5].

Non-motor symptoms include mood disorders (depression), cognitive decline (in late stages of the disease), olfaction impairment, and disturbances of autonomic function (also in late stages). Sleep disorders, especially rapid eye movement behavioral disorder, are quite common and may be present years before the development of motor symptoms.

Treatment is based in dopaminergic reposition. Levodopa is the mainstay of pharmacological treatment. The drug is taken by mouth and in the brain, particularly in neuron and glial cells, is converted into dopamine. If the conversion to dopamine is made by a nigral dopaminergic neuron, it can be stored in vesicles inside the synaptic terminal and can be released afterward. If the conversion is made in glial cells or in non-dopaminergic neurons, dopamine cannot be stored and is immediately released to the postsynaptic receptor. Long-term use of levodopa may be associated with motor fluctuations and dyskinesias. These complications are more common if higher doses of levodopa are used (more than 500 mg a day) and if patients are more severely affected. Dopamine agonists may be also prescribed and act directly at the postsynaptic receptors of striatum. Most commonly prescribe dopamine agonists are pramipexole, ropinirole, and rotigotine. Other drugs are entacapone (catechol-o-methyltransferase inhibitor), rasagiline or selegiline (monoamine oxidase inhibitor), amantadine (glutamate receptor inhibitor), and anticholinergics [6].

Surgery treatment is indicated when patients are optimized receiving levodopa combined with other adjuvant medications but present uncontrolled motor fluctuations and dyskinesias. There are two main surgery procedures: ablative lesions and deep brain stimulation (DBS). The targets indicated are thalamic nuclei, subthalamic nucleus, and globus pallidus interna [7].

Physical and speech therapy are useful for any phase of the disease, but are particularly indicated in patients experiencing balance disturbances and fall (physical therapy) and those with swallowing difficulties and significant dysarthrophonia (speech therapy).

2.2.2 Atypical Parkinsonism

Atypical parkinsonism is also called Parkinson-plus syndromes since most of the patients of this group manifest motor symptoms similar to PD. The difference between PD and these diseases is that atypical parkinsonian patients manifest other neurological abnormalities, such as cerebellar ataxia, autonomic dysfunction, paralysis of the vertical gaze, early-phase cognitive decline, and behavioral disturbances.

All the diseases of this group are neurodegenerative in origin and display neuronal loss in different structures of the central nervous system. None of the diseases of this group have a curative or evolution-modifying pharmacotherapy. Levodopa rarely induces some benefit to this group of patients. Alpha-synuclein inclusions are seen in multiple-system atrophy and dementia with Lewy bodies, while tau protein fibrils are seen in progressive supranuclear palsy and corticobasal ganglionic degeneration.

2.2.3 Multiple-System Atrophy (MSA)

MSA is characterized by the combination of parkinsonian signs with autonomic, pyramidal, and cerebellar signs. There are two forms: MSA-P (parkinsonism), with a predominance of parkinsonian signs, and MSA-C (cerebellar), where there is a predominance of cerebellar signs. The most common form is the MSA-P. The most common signs are dysautonomic: postural hypotension, constipation, urinary incontinence, erectile dysfunction, and cold and cyanotic extremities ("cold blue hands"). Parkinsonian signs are bilateral from the start with the absence of rest tremor. Gait ataxia and balance disturbances can arise from the very beginning, as well as frequent falls. Pyramidal signs as the Babinski sign are present; dysphagia and dysarthria also manifest in the early stage. Other symptoms such as laryngeal stridor, trunk dystonia, and cerebellar signs may be observed. Neuroimaging studies show atrophy of the pons and the cerebellum and usually the presence of the "hot-cross bun signal," a characteristic hyperintensity on FLAIR of the transverse fibers of the pons and cerebellar peduncle with normal sign of the corticospinal tract and pontine tegmentum [8].

2.2.4 Progressive Supranuclear Palsy (PSP)

It was described in the 1960s by Steele, Richardson, and Olszewski. Patients manifest the first symptoms around 50 to 60 years which are characterized by gait disturbance block and trunk hyperextension, postural instability with falls, lower vertical gaze palsy, and cognitive impairment and do not respond to levodopa treatment. The classic form or Richardson syndrome occurs in about 55 % of subjects and presents the peculiar symptoms of PSP, as parkinsonism, paralysis of vertical gaze, bilateral bradykinesia, frequent falls, and cognitive decline. PSP-parkinsonism (PSP-P) is the way of presentation in 30 % of the subjects and the clinical picture is very similar to PD, and only on late stages patients develop gaze palsy and prominent cognitive decline. Fifteen percent of PSP patients present atypical forms. On imaging techniques, particularly in magnetic resonance imaging (MRI), enlargement of the third ventricle and atrophy of midbrain are prominent in late phases of the disease [9].

2.2.5 Dementia with Lewy Bodies (DLB)

DLB starts between 60 and 70 years and is characterized by the presence of parkinsonian signs closely resembling DP associated with cognitive decline. Cognitive symptoms precede or follow up to 12 months after parkinsonism starts. The patient may present, in addition to parkinsonism and dementia, behavioral symptomatology such as hallucinations and delusions, with fluctuation of these symptoms (worse at night). It is quite common patients manifest great sensitivity to the exposure to neuroleptics (rapid development of severe stiffness and tremor) and rapid eye movement (REM) sleep behavioral disorder [10].

2.2.6 Corticobasal Ganglionic Degeneration (CBGD)

CBGD is characterized by progressive and asymmetric stiffness associated with upper limb apraxia. The phenomenon of "alien limb" is commonly present and characterized by involuntary movements of the upper limb, with levitation. Most patients present aphasia, unilateral dystonia, myoclonus, and rapidly progressive dementia of the frontotemporal type. Asymmetric atrophy of the cerebral hemisphere, mainly in the parietal region, is usually seen on MRI studies [1].

2.2.7 Vascular Parkinsonism

Vascular parkinsonism (VP) accounts for 2.5–5 % of the total cases of parkinsonism. VP patients exhibit lower disease duration, older age at onset, and higher frequency of cardiovascular risk factors. This disease can be clinically distinguished from PD based on sudden onset of parkinsonism at older age, characterized by lower body predominance, urinary incontinence, pyramidal signs, postural instability with freezing of gait and falls, and dementia. The upper limbs in VP are less symptomatic in terms of parkinsonism throughout the course of disease. This is in clinical contrast to PD, in which patients generally present with tremor, rigidity, or slowness of the upper limbs. Thus, VP is also called "lower body or lower half" parkinsonism. VP patients developed cognitive impairment in comparison with same age individuals. These patients had a global pattern of cognitive impairment, including executive function, verbal memory, and language [11].

The main differential diagnosis of VP is PD, frontal lobe tumors, obstructive hydrocephalus, normal pressure hydrocephalus, and progressive supranuclear palsy.

Imaging studies serve two purposes in a VP patient. First, it rules out other causes of parkinsonism, both degenerative (progressive supranuclear palsy) and secondary (tumors, hydrocephalous). Second, it may show ischemic lesions to support the clinical diagnosis. MRI or computerized tomography (CT) is supportive, but not conclusive, of the diagnosis of VP because there are no characteristic features of VP in CT/MRI. However, white matter lesions and lacunes are also common observations among the older people who do not have parkinsonism.

This causes difficulty in determining the etiological mechanisms of VP. A study showed that most patients with VP had brain MRI changes with multiple lacunar infarcts (66.7 %) or extensive white matter (WM) disease (26.7 %) [12]. Others investigated the WM microstructure using diffusion tensor imaging in patients with VP, and specific fiber tract involvement, with respect to clinical severity, showed disruption of the microstructural organization of frontal lobe WM is associated with the severity of VP [13]. These findings are in accordance with the frontal lobe disconnection hypothesis for gait problems and reinforce the paradigm that the involvement of fibers related to the prefrontal cortex is crucial for the core features of VP [13].

Clinical features of VP phenotype are early postural instability and gait difficulty (PIGD). PIGD as the initial symptom is significantly more common in VP than in PD. Gait disturbance was reported as the initial symptom in 90 % of patients with VP as compared to 7 % of patients with PD [14]. The gait in VP is like a "parkinsonian-ataxic" gait. The stance is wide based. The posture is upright at least in the earlier stages and a much lesser degree of flexion is seen at the knees and hips than in PD. Steps are slow, short, and with a tendency to shuffle, particularly at the start and while turning the corners. Freezing of gait can be seen, but festination is absent. In severe cases, the patient is unable to initiate a step and the feet remained fixed to the floor (magnetic gait). Postural instability and falls dominate the gait at this advanced stage and render the patient unable to walk or even stand without support. Another factor contributing to the impaired mobility in advanced stages is truncal ataxia. These patients also have truncal and axial immobility. Sideways turning of the head is limited and patients turn their heads, neck, and shoulder in block to be able to look sideways. The change of posture while lying in bed is also difficult and limited. An interesting feature in obvious PIGD, at least in the earlier stages, is that many patients can perform simple tasks with their legs, like imitating bicycling or walking movements, when seated or lying. However, these movements are also slow.

The treatment should be directed at secondary stroke prevention in an attempt to halt further disease progression although, understandably, it would not reverse or provide symptomatic relief to the existing clinical manifestations. The management of conventional vascular risk factors such as diabetes, hypertension, dyslipidemia, and smoking cessation would probably have the maximal impact in this approach to treatment. The levodopa response rates in VP vary from 20 to 40 %. A dose of up to 1000 mg/day should be given for at least 3 months before labeling the patient as a non-responder or poor responder. In good responders the duration of sustained response is typically less than in PD, with the drug becoming non-beneficial after one to two years [15].

2.3 Huntington's Disease

Huntington's disease (HD) is a progressive neurodegenerative disease, rare and fatal, characterized by a movement disorder mostly manifesting as choreic and dystonic movements, psychiatric abnormalities, and cognitive decline.

HD is a genetic disease of autosomal dominant inheritance. An expansion occurs in the number of repeats of CAG pair of nitrogenous bases (adenine, cytosine, guanine) in unstable regions of DNA HTT gene, which is located on the short arm of chromosome 4 (4p16.3). Twenty-seven or fewer repetitions of CAG have a normal phenotype. Intermediate alleles with 27 to 35 CAG repeats are unstable and may be transmitted as an expanded allele to offspring. However, the carriers of the normal intermediate allele also have a normal phenotype. These intermediate expanded alleles occur, particularly, during male gametogenesis, as the same is subjected to a larger number of division cycles and duplication of genetic material. Thus, there is a greater likelihood of an error in DNA replication during spermatogenesis, which increases the risk of mutated alleles to be paternal inheritance [16]. Alleles with 36–39 CAG repeats indicate incomplete penetrance and can generate both normal phenotypes with the disease. Alleles with more than 39 CAG repeats have complete penetrance and inevitably cause, in any phase of life, the phenotype of HD.

Symptoms usually start between ages 35 and 55 and duration of disease can vary from 15 to 20 years. In some cases, it may manifest after 80 years of age or earlier, in adolescents or children, due to anticipation phenomenon, which occurs in 20 % of cases. Due to the variability of the clinical signs and symptoms, the molecular test is required for the diagnosis.

The most frequent isolated initial complaint is the lack of "coordination" and occasional involuntary tremors in several body segments, which usually can be attributed to the presence of chorea. Other early motor abnormalities include saccadic eye movements, interrupted or slow, inability to sustain tongue protrusion, and difficulty in performing rapid alternating movements. Behavioral changes are often reported to precede movement disorder over a decade. Symptoms of emotional nature or personality changes may antedate these movements in at least half of patients with DH. Patients are excessively irritable, impulsive, unstable, and aggressive. The most common early symptoms are mental depression. In some of the cases, the opening frame can be a frank psychosis.

The most striking feature of HD is the presence of choreic movements seen in approximately 90 % of the affected individuals. At first, chorea may be mild, affecting only the face or distal portions of limbs, but, with time, it becomes widespread, interfering with any voluntary movement [17]. Gradually, with the progress of the disease, the patients are becoming slower in nature, taking an athetoid character, and, subsequently, dystonic [18]. Other involuntary movements, such as tremors and intention myoclonus, can rarely be present. Dysarthria is another prominent symptom that appears early in the course of disease, as slowing or hesitancy in speech, which gradually becomes explosive, irregular, and interspersed with long silences, and an increase of chances for a complete disorganization and mutism. Dysphagia, common in the advanced stages of HD, is often responsible for choking and aspiration, bronchopneumonia aspiration, being reported in up to 85 % of mortality. The causes of death are related complications of immobility, such as food or trauma, and asphyxia aspirations. The patient's gait

is characterized, constantly parasitized, by choreic movements, taking uncoordinated character in appearance, and it can become impossible. Patients also exhibit postural instability and primary changes in the balance, with frequent falls and fractures. Bradykinesia, with or without stiffness, occurs in the later stages of disease with extreme slowness of all movements. This symptom is directly related to the disability of patients and it occurs regardless of chorea [17]. Sphincter incontinence, both urinary and anal, is common, especially in the terminal stages of HD. There is evidence of autonomic dysfunction as hyperhidrosis and changes in blood pressure regulation. Generalized tonic-clonic seizures may occur in up to 3 % of adults with DH and in approximately 40 % of patients with early disease onset (under 20), as other types of seizures can happen [18].

Psychiatric and cognitive changes may precede the motor manifestations, accompany them, or occur after [19]. Dementia is considered one of the cardinal signs of the disease. Patients with DH are more impaired in learning, mental flexibility, and attention. Executive function is compromised and is noted early, even in the absence of complaints from patients or family members. It includes difficulty in planning, organizing, and scheduling activities. The executive functions are related to the frontal lobe and its connections. Cognitive functions are more strongly correlated with the functional capacity of the motor symptoms.

The psychiatric symptoms occur in about 35–70 % of cases. Patients may present frameworks of personality change, mood disorders, and psychosis. The most commonly found personality changes are psychiatric and include irritability, apathy, emotional lability, impulsivity, and aggressiveness. Depression may precede motor symptoms. The frequency of suicides in HD is four to six times higher than that in the general population. Suicidal ideation is present in approximately 10 % of individuals diagnosed with HD. Episodes of mania can also be found, less commonly, and schizophreniform psychosis occurs in 6–25 % of cases; this is characterized by paranoid tables with persecutory delusions, jealousy, and auditory hallucinations. Other psychiatric conditions include irritability, agitation and aggression, disorder and obsessive-compulsive behavior, psychosomatic complaints, anxiety, sexual paraphilias, and behavioral changes [20].

Symptomatic treatment reduces motor and behavioral symptoms and thus maximizes functional capacity of the affected individual. The treatment is done with chorea antagonists dopaminergic with high affinity for D2 receptors. Among the drugs which may be used to control abnormal movements, there are risperidone and olanzapine (typical neuroleptic), and haloperidol (atypical neuroleptic). Tetrabenazine is also indicated for the control of choreic movements. However, due to the high incidence of depression in patients treated with this drug, its use is limited. The dose should be individualized, starting always with low doses and increasing until the control of involuntary movements is satisfactory from a functional point of view [21]. Patients generally tolerate high doses of conventional neuroleptics, showing no symptoms of impregnation. The inhibitors of N-methyl-D-aspartate (NMDA) glutamate receptors, such as amantadine, can be used for the motor's movement control. Depression usually responds to classical antidepressants such as selective inhibitors or serotonin reuptake dual inhibitors.

Excessive irritability can be treated with benzodiazepines. Aggressive patients can benefit from both antidepressants and neuroleptics. There is not a specific treatment for HD dementia [21].

2.4 Final Remarks

Gait and balance are usually impaired in many neurodegenerative diseases, but parkinsonism and Huntington's disease are the most frequent, with the higher impact on daily activities of patients. Although these conditions usually do not have a definite treatment to control axial symptoms, pharmacological and rehabilitation approaches are essential to improve quality of life of the patients.

References

1. Fahn S, Jankovic J. Principle and practice of movement disorders. Philadelphia: Churchill Livingstone; 2007. p. 652.
2. Campbell WW. DeJong's. The neurologic examination. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2013.
3. Jellinger KA. Neuropathological spectrum of synucleinopathies. Mov Disord. 2003;18 suppl 6:S2–12.
4. Kalia LV, Lang AE. Parkinson's disease. Lancet. 2015;386:896–912.
5. Jankovic J, McDermott M, Carter J, et al. Variable expression of Parkinson's disease: a baseline analysis of the DATATOP cohort. Neurology. 1990;41:1529–34.
6. Connolly BS, Lang AE. Pharmacological treatment of Parkinson's disease. A review. JAMA. 2014;311:1670–83.
7. Wagle Shukla A, Okun MS. Surgical treatment of Parkinson's disease: patients, targets, devices, and approaches. Neurotherapeutics. 2014;11:47–59.
8. Fanciulli A, Wenning GK. Multiple-system atrophy. N Engl J Med. 2015;372:249–63.
9. Golbe LI. Progressive supranuclear palsy. Semin Neurol. 2014;34:151–9.
10. Walker Z, Possin KL, Boeve BF, Aarsland D. Lewy body dementias. Lancet. 2015;386:1683–94.
11. Benítez-Rivero S, Lama MJ, Huertas-Fernández I, et al. Clinical features and neuropsychological profile in vascular parkinsonism. J Neurol Sci. 2014;345:193–7.
12. Vale TC, Caramelli P, Cardoso F. Clinicoradiological comparison between vascular parkinsonism and Parkinson's disease. J Neurol Neurosurg Psychiatry. 2015;86(5):547–53.
13. Wang HC, Hsu JL, Leemans A. Diffusion tensor imaging of vascular parkinsonism: structural changes in cerebral white matter and the association with clinical severity. Arch Neurol. 2012;69:1340–8.
14. FitzGerald PM, Jankovic J. Lower body parkinsonism: evidence for vascular etiology. Mov Disord. 1989;4:249–60.
15. Constantinescu R, Richard I, Kurlan R. Levodopa responsiveness in disorders with parkinsonism: a review of the literature. Mov Disord. 2007;22:2141–8.
16. Wheeler VC, Persichetti F, McNeil SM, et al. Factors associated with HD CAG repeat instability in Huntington disease. J Med Genet. 2007;44:695–701.
17. Tabrizi SJ, Langbehn DR, Leavitt BR, TRACK-HD Investigators, et al. Biological and clinical manifestations of Huntington's disease in the longitudinal TRACK-HD study: cross-sectional analysis of baseline data. Lancet Neurol. 2009;8:791–801.
18. Cardoso F. Huntington disease and other choreas. Neurol Clin. 2009;27:719–36.

19. Andrew SE, Goldberg YP, Kremer B, Telenius H, et al. The relationship between trinucleotide (CAG) repeat length and clinical features of Huntington's disease. Nat Genet. 1993;4:398–403.
20. Morreale MK. Huntington's disease: looking beyond the movement disorder. Adv Psychosom Med. 2015;34:135–42.
21. Bonelli RM, Hoffman P. A systematic review of the treatment studies in Huntington's disease since 1990. Expert Opin Pharmacother. 2007;8:141–53.

Neuropsychological Aspects of Gait Disorders

3

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Abstract

In movement disorders, neuropsychological factors play an important yet overlooked role in locomotion. Given that walking is almost always driven by function and goal-directed behaviors (e.g., in something as simplistic as a turn), aspects of perception, planning, processing capacity, and even emotion/anxiety drive need to be carefully considered and potentially treated for their great potential to influence locomotor behaviors in gait disorders.

Keywords

Gait • Parkinson's disease • Perception • Planning • Cognition • Basal ganglia • Anxiety

3.1 Introduction

Movement disorders are neurodegenerative conditions clinically defined as being specific to basal ganglia dysfunction, most often associated with progressive dopamine depletion. Clinical motor symptoms of these progressive disorders include resting tremor, rigidity, bradykinesia, flexed posture, and gait deficits [1]. Specific to gait, movement disorders are typically associated with a slower (even cautious) and more variable gait pattern, although it has been debated whether these deficits may be more related to spatial or temporal aspects of gait control. This debate has been fueled by the fact that step length appears to be a characteristic positively responsive to dopaminergic replacement during self-paced walking, while temporal aspects of gait (including temporal variability) may be negatively associated with

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dopamine replacement, and this is notable in cognitively demanding situations [2]. Thus, from a planning perspective, goal-directed locomotion (i.e., walking to a target or destination) can involve many neuropsychological aspects, that if impaired could influence movement control. Potential impaired neuropsychological elements could include perception to recognize and judge proximity of the target, and attention, to maintain focus on target goals while not being distracted by environmental distractors, and also to make appropriate use of sensory feedback while making online adjustments to a movement plan which can be critical neuropsychological factors that can alter our ability to locomotor efficiently and effectively.

If we look at the most common movement disorder, Parkinson's disease (PD) gait is a distinctive feature of the disease and is often characterized by a limited arm swing, slow walking speed, and small shuffling steps [3], all exemplary features of bradykinesia. Thus, from a theoretical point of view, bradykinesia may be a neural adaptation in which a "need to put on the brakes" is employed so that more time is available to perceive, plan, and make online adjustments while walking. Even the limited arm swing may be an attempt to mobilize an arm for its ability to steady balance or to interact with objects in the environment. As the disease progresses, gait deficits have been shown to become more stereotyped, making it difficult for these individuals to adjust gait in new environmental conditions [4]. This may be one reason why patients often report difficulty during turning tasks [5], where such spatiotemporal gait adaptations and higher-level planning are necessary to negotiate changes in direction, velocity, and balance. Since locomotor deficits have been closely linked to fall and serious injury, it may be equally important to consider how these control deficits have been closely linked to falling and injury in PD [6, 7], and this may be the result of limited cognitive resources and/or neuropsychological decline. Thus, this chapter will examine neuropsychological factors that may contribute to both spatial and temporal aspects of locomotion in movement disorders and then further examines what impact these factors may have on freezing of gait (FOG) and other severe gait deficits.

We first examine the reduced step length as a central gait problem associated with movement disorders (i.e., stride length was reported to be 0.96 m in PD on average compared to 1.13 m in healthy participants) [8, 9]. Along with a decreased ground clearance and slower than average gait velocity (PD = 49.7 m/min, controls = 73.7 m/min), researchers have attributed this reduced step amplitude (15 % smaller stride length than healthy control participants) to be a general hypometric movement deficit resulting from a lack of dopamine and its role in the basal ganglia to prepare and execute movement [9]. While locomotion in healthy individuals is fairly automatic, it is important to recognize that some attentional and central processing resources are still necessary in neurodegenerative populations to allow movements that were once automatic [10]. As previously mentioned, perhaps during more complex tasks such as turning or multitasking, these preplanned movements are disrupted even further. However, previous studies have suggested that individuals with PD overcome this deficit by focusing on each step discretely [9, 11], requiring more conscious and cognitively demanding control. Visual cueing rehabilitation studies have supported this theory as

improvements in step length were achieved when patients were asked to focus on the stepping process by walking on transverse lines in the range 9–35 % step length improvement [9, 11–14]. This improvement in step length ultimately improved overall walking performance in these individuals, but even the effects of rehabilitation may be to reduce cognitive processing demands through practice.

Although spatial aspects of gait can be easily identified, gait timing deficits are more difficult to detect through visual inspection and are often just as problematic. PD gait studies have often used cadence as a measure to assess timing control, suggesting that a faulty cadence (cadence of PD and healthy participants was 112.04 steps/min and 106.47 steps/min, respectively) has been suggested to be partially the result of a defective internal rhythm within the basal ganglia [15]. However, Morris and colleagues found that when cadence was "fixed" using visual cueing, stride length and velocity remained abnormally low, revealing that cadence modulation remained intact in PD [3]. Importantly however, Almeida et al. [2] argued that this was only applicable for certain situations as his group discovered that gait timing control may be implicated when a more goal-directed task that may require higher levels of conscious or cognitive control is involved during walking (e.g., modulating cadence relative to different speeds of a metronome). This study also demonstrated that dopamine replacement therapy leads to increased temporal variability, which may be concerning since variability has been linked to increased risk of falls. Nonetheless, the lack of consensus on cadence modulation suggests that cadence may not be a sensitive enough measurement to reflect the faulty internal rhythm found in individuals with PD. However, there is some evidence that rhythmicity as manifested in the stride-to-stride variations is more precise and useful in measuring timing [16]. Work by Hausdorff et al. [6, 17] suggests that gait timing is influenced by the basal ganglia and is therefore impaired in individuals with PD. Therefore, utilizing a more sensitive measure such as step time variability may be warranted when evaluating gait timing control.

Overall, gait variability has been a common measure used to reflect step-to-step fluctuations during walking, indicative of both conscious neural control of locomotion [18] and stability of gait. A high step length variability in PD has been suggested to be a consequence of attempting to voluntary control an unsteady gait pattern by increasing the sampling of interoceptive (proprioceptive) feedback [19]. Alternatively, step time variability has been argued to be a sensitive measure of gait rhythmicity and coordination [20]. Dual-task paradigms have previously been implemented in PD gait research to draw out step time variability deficits (step time increased from 1.23 (0.18) s to 1.33 (0.17) s, coefficient of variation of step time increased from 3.61 to 4.41) [21], revealing that walking while cognitively challenged impairs the ability of patients with PD to maintain a steady gait [20], and these effects are exacerbated in movement disorder patients with greater neuropsychological deficit. Another situation that requires steady gait timing is turning. It may be argued that turning (while walking) requires a similar control of gait rhythmicity, as it helps maintain locomotion when a change in direction is necessary. Although this area has yet to be fully explored, investigating gait timing during turning may be valuable in understanding its contribution in controlling

functional aspects of walking, and in fact the inability to turn may be an important area to help identify cognitive deficits in movement disorders since, in comparison to straight line walking, turning requires the central nervous system (CNS) to reorient the body toward a new travel direction while maintaining stability and preserving the ongoing step cycle [22].

While gait variability may be seen in early PD, work by Hausdorff [17, 23] and more recent findings by Almeida and Lebold [19] identified both step time variability and step length variability were significantly greater in individuals with PD that experience freezing of gait. Moreover, research has shown that an increased stride-to-stride variability was a precursor to freezing episodes [20] and a significant predictor of future falls in PD [6].

Given the gait deficits faced by individuals living with PD, it is critically important to continuously measure spatiotemporal gait parameters in settings where patients find walking most difficult. Moreover, large fluctuations during the stepping process are a strong predictor of both falling episodes and freezing of gait instances; therefore, when conducting walking studies, the focus should be placed on gait variability rather than solely temporal or spatial gait measures.

3.2 Freezing of Gait

The freezing of gait (FOG) phenomenon is one of the most debilitating gait symptoms affecting people with PD, and so it is important to consider how neuropsychological issues may contribute. Previous studies have identified FOG episodes to occur in specific situations, which "trigger" the onset of the freeze. Some of the most common situations that provoke a freezing episode require increased levels of cognitive demand and planning including turning, gait initiation, and passing through narrow doorways, in confined spaces, during stressful situations, or when approaching a final destination [5]. Although FOG remains a relevant area of study in PD research, capturing a freeze in the experimental setting can be extremely difficult [19, 24]. Therefore, it is important to utilize freezing triggers within experimental paradigms that increase the likelihood of a freezing episode, so that it may provide insight into the underlying mechanisms that cause FOG. Moreover, freezing episodes seem to manifest in variety of forms, from "trembling in place" to a total akinesia (lack of movement) type of FOG [25], and can often be confused with festination (as described below). While FOG occurs in the more advanced stages of the disease, it can be reported in the early stages as well, often characterized as mild and short in duration [26]. This inconsistency may also be suggestive of an association between the level of cognitive dysfunction rather than pure stage of motor disease progression. Thus, with the various subtypes and inconsistency of freezing occurrences, it is important to think carefully about the underlying neuropsychological (and other) mechanisms that contribute to FOG episodes.

Part of this issue surrounding FOG is the inconsistencies in its very definition. In one of the most early attempts at defining, Giladi and Fahn [27] described the

phenomenon as a motor block that usually lasts seconds, in which the motor activity being attempted by the individual is halted. Other researchers refer to the stepping process within the FOG episode, describing FOG as an episodic inability to generate effective stepping in the absence of any known cause other than Parkinsonism or higher-level gait disorders [28]. This definition would suggest that potentially known causes such as neuropsychological decline should be ruled out be clinical research, unless the definition is reconceptualized. Thus, although these definitions seem suitable, many of these working definitions are largely based on subjectivity. Some researchers have alluded to triggers of FOG within the definition, describing freezing to occur when individuals are unable to start locomotion, turn, or pass through a doorway [29]. However, previous research has shown freezing to occur in open space [5, 25] and literally where there may be no environmental triggers or distractors that appear to precede the FOG episode; therefore, identifying triggers within the definition seems inappropriate. From a neuropsychological perspective, it may be possible that, in addition to environmental triggers, something as simple as losing one's focus of attention (or so-called mind-wandering) may also lead to FOG. Therefore, one must carefully consider the neuropsychological processes that might be ongoing while walking that may not have any obvious environmental triggers. For example, it may be possible that an interruption in thought, as simple as forgetting why you were headed in the selected direction of travel or recalling that you may have forgotten to turn off the stove (even if while walking in an uncluttered open space), could be enough to lead to an interruption to the forward progression of gait. Thus, Almeida et al. [30] suggested a freezing episode to be any period of at least one second in which one or both limbs display no movement. This was the first definition to include the potential for both the upper and lower limbs to freeze, and importantly the suggested definition may be more applicable when studying FOG as it encompasses all manifestations of freezing episodes, including both upper and lower limbs, while recognizing that higher-level neuropsychological issues might contribute to freezing in any motor effector (and not just the lower limbs).

3.3 Other Mechanistic Explanations for Severe Gait Deficits

It is important to recognize that there is generally a higher degree of neuropsychological impairment and severity of gait and balance impairment that accompanies more severe gait deficits, yet the underlying mechanisms are poorly understood. A study by Lamberti et al. [31] revealed that FOG is highly correlated with disease duration and identified FOG episodes to occur most often in individuals that were diagnosed with PD for 9 years or more. While it is well known that cognitive impairments worsen with disease progression, some researchers have suggested that freezing may be a manifestation that arises from late stages of disease, similar to other motor symptoms [32]. However, work by Giladi [27] refuted this theory and found FOG to be more strongly associated with speech and balance problems rather than bradykinesia or rigidity, and it should be noted that both balance and

speech control requires continuous monitoring and planning from a neuropsychological perspective.

Several researchers have linked an inability to produce normal step length (step length of freezers, non-freezers and healthy controls was found to be 37.76 (18.18) cm, 64.40 (5.86) cm, and 76.13 (8.62) cm, respectively) amplitude to be linked with freezing during walking [33, 34]. Chee et al. [34] suggest that PD freezers experience a "sequence effect," a gradual step-to-step reduction combined with an overall reduced step length that eventually leads to a freezing episode. Others might argue that this sequence effect might simply be a lack of cognitive effort dedicated toward gait, and even the failure to incorporate feedback as a result of limited attentional or processing resources. It is also possible that freezers have difficulty set shifting between maintaining what should be an automatic continuation of gait and simultaneous monitoring of what is approaching in the environment. This could be part of a depth perception problem, a sensory feedback processing issue, or overall cognitive load issue. Either way, it has been shown that by systematically decreasing participant's step length using visual stepping cues (50 % and 25 % of preferred step length), more freezing episodes were elicited. Individuals with PD have been shown to implement a decreased step length in freeze-provoking situations, such as walking through a narrow doorway [19] and turning [4]. Importantly, dual-tasking studies would suggest that limited cognitive abilities are linked to more severe gait deficits in movement disorders, such as FOG [35]. Supporters of the "sequence effect" suggest that an alteration in step length may be the reason freezing manifests so often in these situations. However, it should be noted that this reduction in step length is simply a behavior describing FOG, rather than the underlying mechanism that causes the phenomenon.

Alternatively, some researchers propose that there is a gait rhythmic asymmetry motor deficit in PD freezers [36] and that there is a lack of bilateral coordination in the lower limbs which causes gait to become disturbed to the point of freezing. It is possible that turning is a large provoker of FOG episodes due to its high demand in coordination. However, the threshold of freezing has yet to be clearly defined, making it difficult to identify when exactly during an asymmetric movement freezing emerges.

The "asymmetric" gait theory and "sequence effect" have been suggested to be a product of the faulty link between the basal ganglia and supplementary motor area [34]. However, the "sequence effect" describes freezing as a step-to-step amplitude reduction, while supporters of the "asymmetric" gait theory suggest that freezing is linked to an inability to control timing of step movements. As reviewed in Lancet Neurology, it has been suggested that both deficits might be more thoroughly examined by considering a sensorimotor integration malfunction in PD and its contributions to freezing [37]. Nevertheless, examining turning in PD is ideal in understanding which of these two deficits is linked to FOG. Due to its inherent nature, turning is often accompanied by a reduction in step length (stride length decreased from 1.33 (0.102) m to 1.250 (0.085) m from straight line walking to turning) [4, 38]. Therefore, if freezers show a similar step length reduction pattern as non-freezers, but an inability to maintain proper step timing, it may be argued

that amplitude production is not a contributor to FOG. Contrarily, if step timing is intact in both PD groups, but step length production is impaired, it may be suggested that freezing is not associated with a gait timing impairment. Therefore, concurrently examining spatial and temporal variables during turning in healthy control participants, freezers and non-freezers can provide greater insight into the deficits associated with FOG and eventually contribute to understanding the pathogenesis of this phenomenon. Furthermore, considering that turning is a functional aspect of locomotion that has been shown to be problematic in PD, this type of investigation may be more informative (than straight line walking) of gait deficits faced by patients during everyday locomotion.

In conclusion, it is continuously being recognized that many non-motor mechanisms may lie at the route of motor impairments. Neuropsychological factors are important to consider in movement disorders, as they can contribute to the underlying spatial and temporal deficits observed commonly in the gait of populations with movement disorders. Moreover, neuropsychological factors may have an important impact underlying the cause for more severe movement deficits such as freezing of gait. Identifying the primary mechanisms may be key to identifying appropriate therapeutic interventions in movement disorder populations such as PD.

References

1. Marsden CD. Parkinson's disease. J Neurol Neurosurg Psychiatry. 1994;57(6):672–81.
2. Almeida QJ, Frank JS, Roy EA, Patla AE, Jog MS. Dopaminergic modulation of timing control and variability in the gait of Parkinson's disease. Mov Disord. 2007;22(12): 1735–42.
3. Morris ME, Iansek R, Matyas TA, Summers JJ. Ability to modulate walking cadence remains intact in Parkinson's disease. J Neurol Neurosurg Psychiatry. 1994;57(12):1532–4.
4. Huxham F, Baker R, Morris ME, Iansek R. Footstep adjustments used to turn during walking in Parkinson's disease. Mov Disord. 2008;23(6):817–23.
5. Rahman S, Griffin HJ, Quinn NP, Jahanshahi M. The factors that induce or overcome freezing of gait in Parkinson's disease. Behav Neurol. 2008;19(3):127–36.
6. Hausdorff JM, Rios DA, Edelberg HK. Gait variability and fall risk in community-living older adults: a 1-year prospective study. Arch Phys Med Rehabil. 2001;82(8):1050–6.
7. Olanow CW, Stern MB, Sethi K. The scientific and clinical basis for the treatment of Parkinson disease (2009). Neurology. 2009;72(21 Suppl 4):S1–136.
8. Suteerawattananon M, Morris GS, Etnyre BR, Jankovic J, Protas EJ. Effects of visual and auditory cues on gait in individuals with Parkinson's disease. J Neurol Sci. 2004;219 (1-2):63–9.
9. Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms. Brain. 1996;119(Pt 2):551–68.
10. Shumway-Cook A, Woollacott MH. Moto control: translating research into clinical practice. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2007.
11. Lebold CA, Almeida QJ. An evaluation of mechanisms underlying the influence of step cues on gait in Parkinson's disease. J Clin Neurosci. 2011;18(6):798–802.
12. Martin JP. The basal ganglia and postural mechanisms. Agressologie. 1977;18:75–81.
13. Almeida QJ, Bhatt H. A manipulation of visual feedback during gait training in Parkinson's disease. Parkinson's disease: Special Issue on Rehabilitation 2012:508720, doi:10.1155/2012/508720: 1-7.

14. Sidaway B, Anderson J, Danielson G, Martin L, Smith G. Effects of long-term gait training using visual cues in an individual with Parkinson disease. Phys Ther. 2006;86(2):186–94.
15. Rubinstein TC, Giladi N, Hausdorff JM. The power of cueing to circumvent dopamine deficits: a review of physical therapy treatment of gait disturbances in Parkinson's disease. Mov Disord. 2002;17(6):1148–60.
16. Hausdorff JM. Gait dynamics in Parkinson's disease: common and distinct behavior among stride length, gait variability, and fractal-like scaling. Chaos. 2009;19(2):026113.
17. Hausdorff JM, Cudkowicz ME, Firtion R, Wei JY, Goldberger AL. Gait variability and basal ganglia disorders: stride-to-stride variations of gait cycle timing in Parkinson's disease and Huntington's disease. Mov Disord. 1998;13(3):428–37.
18. Lord S, Baker K, Nieuwboer A, Burn D, Rochester L. Gait variability in Parkinson's disease: an indicator of non-dopaminergic contributors to gait dysfunction? J Neurol. 2011;258 (4):566–72.
19. Almeida QJ, Lebold CA. Freezing of gait in Parkinson's disease: a perceptual cause for a motor impairment? J Neurol Neurosurg Psychiatry. 2010;81(5):513–8.
20. Hausdorff JM, Balash J, Giladi N. Effects of cognitive challenge on gait variability in patients with Parkinson's disease. J Geriatr Psychiatry Neurol. 2003;16(1):53–8.
21. Lamoth CJ, van Deudekom FJ, van Campen JP, Appels BA, de Vries OJ, Pijnappels M. Gait stability and variability measures show effects of impaired cognition and dual tasking in frail people. J Neuroeng Rehabil. 2011;8:2.
22. Patla AE, Adkin A, Ballard T. Online steering: coordination and control of body center of mass, head and body reorientation. Exp Brain Res. 1999;129(4):629–34.
23. Hausdorff JM, Schaafsma JD, Balash Y, Bartels AL, Gurevich T, Giladi N. Impaired regulation of stride variability in Parkinson's disease subjects with freezing of gait. Exp Brain Res. 2003;149(2):187–94.
24. van Wegen E, Lim I, de Goede C, Nieuwboer A, Willems A, Jones D, et al. The effects of visual rhythms and optic flow on stride patterns of patients with Parkinson's disease. Parkinsonism Relat Disord. 2006;12(1):21–7.
25. Schaafsma JD, Balash Y, Gurevich T, Bartels AL, Hausdorff JM, Giladi N. Characterization of freezing of gait subtypes and the response of each to levodopa in Parkinson's disease. Eur J Neurol. 2003;10(4):391–8.
26. Okuma Y, Yanagisawa N. The clinical spectrum of freezing of gait in Parkinson's disease. Mov Disord. 2008;23 Suppl 2:S426–30.
27. Giladi N, McDermott MP, Fahn S, Przedborski S, Jankovic J, Stern M, et al. Freezing of gait in PD: prospective assessment in the DATATOP cohort. Neurology. 2001;56(12):1712–21.
28. Giladi N, Nieuwboer A. Understanding and treating freezing of gait in parkinsonism, proposed working definition, and setting the stage. Mov Disord. 2008;23 Suppl 2:S423–5.
29. Amboni M, Cozzolino A, Longo K, Picillo M, Barone P. Freezing of gait and executive functions in patients with Parkinson's disease. Mov Disord. 2008;23(3):395–400.
30. Almeida QJ, Wishart LR, Lee TD. Disruptive influences of a cued voluntary shift on coordinated movement in Parkinson's disease. Neuropsychologia. 2003;41(4):442–52.
31. Lamberti P, Armenise S, Castaldo V, de Mari M, Iliceto G, Tronci P, et al. Freezing gait in Parkinson's disease. Eur Neurol. 1997;38(4):297–301.
32. Factor SA, Jennings DL, Molho ES, Marek KL. The natural history of the syndrome of primary progressive freezing gait. Arch Neurol. 2002;59(11):1778–83.
33. Nieuwboer A, Dom R, De Weerdt W, Desloovere K, Fieuws S, Broens-Kaucsik E. Abnormalities of the spatiotemporal characteristics of gait at the onset of freezing in Parkinson's disease. Mov Disord. 2001;16(6):1066–75.
34. Chee R, Murphy A, Danoudis M, Georgiou-Karistianis N, Iansek R. Gait freezing in Parkinson's disease and the stride length sequence effect interaction. Brain. 2009;132 (Pt 8):2151–60.

35. Spildooren J, Vercruysse S, Desloovere K, Vandenberghe W, Kerckhofs E, Nieuwboer A. Freezing of gait in Parkinson's disease: the impact of dual-tasking and turning. Mov Disord. 2010;25(15):2563–70.
36. Plotnik M, Giladi N, Balash Y, Peretz C, Hausdorff JM. Is freezing of gait in Parkinson's disease related to asymmetric motor function? Ann Neurol. 2005;57(5):656–63.
37. Nutt JG, Bloem BR, Giladi N, Hallett M, Horak FB, Nieuwboer A. Freezing of gait: moving forward on a mysterious clinical phenomenon. Lancet Neurol. 2011;10(8):734–44.
38. Courtine G, Schieppati M. Human walking along a curved path. II. Gait features and EMG patterns. Eur J Neurosci. 2003;18(1):191–205.

Part II

Walking

Gait Stability and Aging

4

Luis Mochizuki and Sandra Aliberti

Abstract

Stability is an important concept in biomechanics. It means that a mechanical system in equilibrium should resist changing its motion properties after an external perturbation. Gait is one of the most common ways of locomotion in humans. Walking is a motor action achieved by interaction of the nervous system, muscles, joints, and bones with the environment and its external forces. Moreover, several unhealthy conditions are prone to change how people walk. Therefore, the comprehension of how someone deals with an external perturbation to maintain its walking pattern, i.e., how to control the body to keep it stable during walking, is important. The aim of this chapter is to describe the tools to evaluate the stability on walking, the main concerns to apply those tools and concepts of dynamical systems in gait analysis, and how the most common biomechanical variables applied on gait analysis should be prepared for that.

Keywords

Dynamical systems • Biomechanics • Gait • Equilibrium • Aging • Vector coding

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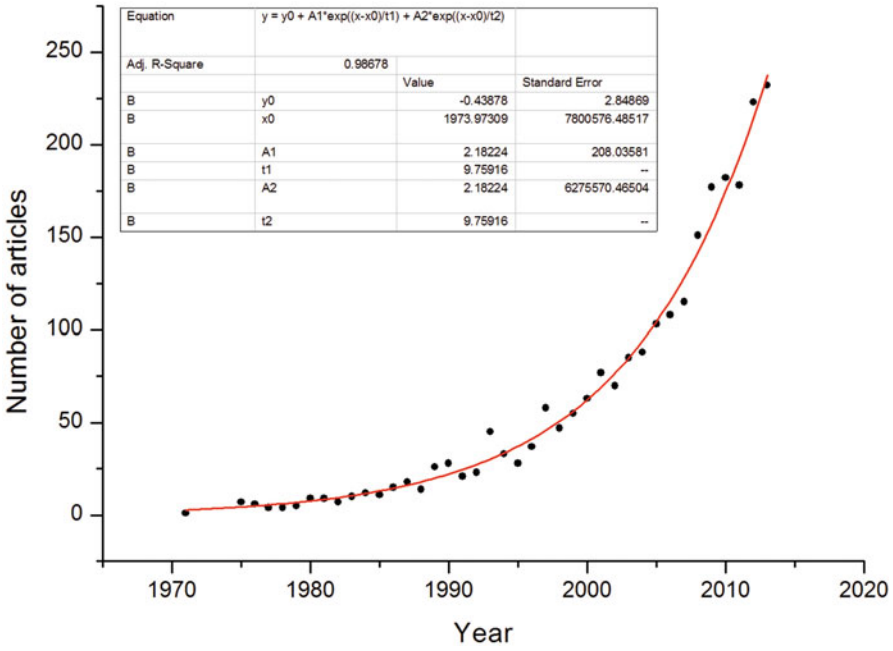

Fig. 4.1 Number of articles indexed in PUBMED from the search (gait or running) and dynamics

4.1 Introduction

In 1901, Ferrier [1] stated that the stretching of the foot is a physiological mechanism that neutralizes body vibration during walking, increases foot contact to ground, and increases the body stability to support the body weight. Several mechanics are available to stabilize the body during walking. Moreover, the analysis of stability during walking or running has been increasing in the last half century. According to PUBMED data, since the 1970s, there is an exponential growth in the number of papers about envolving (stability AND gait OR walking OR running) (Fig. 4.1).

Stability can be understood under different points of view. For mechanics, stability is a condition that classifies the body's equilibrium in stable and unstable. Stable equilibrium occurs when any external perturbation does not change its final potential energy. Also for the mechanics, direction stability deals with the perpendicular forces applied to body that are moving along a path. For fluid mechanics, stability deals with the effects of turbulence to the motion of the body. In mathematics, stability is the analysis of partial equations and dynamical systems and their tendency to converge or diverge. In motor control, stability is usually related to balance and equilibrium. Some authors propose that in order to maintain balance it

is necessary to stabilize the projection of the position of center of mass inside of the limits of basis of support. For motor control, stability is not only associated with balance. It is associated to maintain a certain body position during a motor action, to every situation which is necessary. The most common situation is, indeed, the standing position. Other examples are maintaining shoulder position while doing hand activities or hip position while walking.

The dynamical systems can be evaluated based on the behavior of their trajectories under small perturbations of their initial conditions. The sensibility to small changes in initial conditions defines if a dynamical system can be fully predicted or it has some specific conditions on which its behavior becomes very complicated. There are several examples of dynamical systems in nature, but we shall focus on those related to motor behavior.

Walking can be modeled and studied with the dynamical systems approach [2]. Therefore, its states, in time, can be described, analyzed, and predicted, in many conditions. The time series of stride intervals during walking presents allometric properties [3], which suggests that its fluctuations exhibit a long-range correlation and are not related to short-range correlation nor any random processes. Stride frequency or gait cadence is the result of the interaction between internal system and the external forces and constrains. The combination among ground reaction forces, stance time, stride frequency, and length changes gait velocity. If the stride interval fluctuations are not random, what provokes such variability?

Walking is a motor action achieved by interaction of the nervous system, muscles, joints, and bones with the environment and its external forces. Moreover, several unhealthy conditions are prone to change how people walk. On the other hand, evaluating how someone walks gives important information about its health status. The coordination between body's structures under different types of stress might lead to affect how they are coupled. To explore the natural restrain of the anatomical structures, different couplings between joints are tested during motor development and learning. Therefore, the comprehension of how someone deals with an external perturbation to maintain its walking pattern, i.e., how to control the body to keep it stable during walking, is important. The aim of this chapter is to describe the tools to evaluate the stability on walking, the main concerns to apply those tools and concepts of dynamical systems in gait analysis, and how the most common biomechanical variables applied on gait analysis should be prepared for that.

4.2 The Tools to Evaluate the Stability on Walking

Physiological systems are open systems (changes energy-mass with the environment) and nonlinear, and their periodicity is triggered by several external and internal factors. Mathematical concepts to analyze the nonlinear behavior of dynamical systems are available to be applied to understand the physiological systems. Most of nonlinear tools are designed for low-dimension deterministic chaos. It means that such dynamical systems are determinist (predictable) and very sensitive to initial

conditions (chaotic behavior). But, the dimension of physiological systems is not a priori known. Besides, the nature of noise in physiological systems is hard to understand because it can come from any interaction with the world.

What variables should we use to apply dynamical system tools in order to evaluate the dynamics of walking? Descriptive variables obtained from gait analysis usually used the dynamical systems approach, such as stride-to-stride distance, joint angles, or body position and body acceleration. Time series of those variables are commonly used to calculate the Lyapunov exponents, the dynamical systems approach's variable related to stability.

Aleksandr Mikhailovich Lyapunov was born on 6 June 1857 at Yaroslavl, Russia [4]. In 1880, he graduated in mathematics at St. Petersburg University, where he was very influenced by Professor P. L. Chebyshev. In Moscow, 1892, he defended his doctoral thesis "The general problem of the stability of motion," and one member of his doctoral thesis's committee members was N. E. Zhukovski (the father of Russian aviation). In his thesis, Lyapunov have discussed the fact that the motion of a particle can be stable in relation to one variable, but it can be unstable in relation to another variable. Besides, he suggest that, given a function $x(t)$, there is a critical number which is the characteristic number of this function. This characteristic number or Lyapunov exponent has been used to analyze if two points $x_1(t)$ and $x_2(t)$, in the state space of a dynamical system which are initially very close, remains together in time. This notion of motion stability can be measured with the Lyapunov exponent; therefore, this exponent can be used to understand chaotic motion. Chaos relates to aperiodic dynamics in deterministic equations in which there is a sensitivity to initial conditions [5]. In a chaotic motion [4], long-term prediction is seriously affected because those two points move apart as time increases.

Stability can be measured with Lyapunov exponents and recurrence quantification analysis (RQA). In this chapter, we will focus on local dynamic behavior by Lyapunov exponents. First, entropy and variability can be found in some articles as measures of stability. Second, it is not unusual to find Lyapunov exponents as a measure of variability or combined with entropy. Entropy determinates the complexity of a system. Variability relates to how variable is the behavior/response of a system. Thus, even entropy nor variability truly measures of stability, but they access phenomena that might be connected to stability. From RQA, it is possible to measure repeatability (absolute agreement among repetitions, how often a dynamical system shows a similar behavior by percent recurrence), predictability (how predictable is a dynamical system by percent determinism), complexity (entropy), and stationarity (how stationary is a dynamical system in time by trend). Repeatability and predictability are measures associated to stability.

Dynamical systems approach to understand local dynamical stability calculates Lyapunov exponents to generate gait information. Most of studies we will present are based on accelerometers which were attached to the trunk, thigh, or shank. Oscillations due to different gait phases are analyzed under the dynamical systems approach.

4.3 Stability, Development, and Aging

There are some concerns about the use of Lyapunov exponents or local dynamical stability. Should we use local dynamical stability to understand the changes in walking across ages? Local dynamical stability changes due to aging. First, toddlers, young, and older adults do not have similar local dynamical stability during walking [6]; however, those changes might not be observed when directional components of acceleration are used to calculate Lyapunov exponents. Bisi et al. [6] compared walking in toddlers and young and older adults. The authors applied short-term Lyapunov exponents and RQA to compare strides among participants. Short-term Lyapunov exponents calculated from all combined three orthogonal trunk acceleration components and RQA were able to discern between strides from toddlers and young adults and from young adults and the elderly. Besides, stability has no a monotonic relation to aging. Terrier and Reynard [7] compared Lyapunov exponents calculated from participants aging from 20 to 69 years old. They walked on a treadmill for five minutes wearing a 3D accelerometer on the trunk. The authors found a quadratic association between age and gait instability in the mediolateral direction. Gait instability likely begins to increase at an accelerated rate as early as age 40–50. Local dynamical stability might be used to compare the effects of aging in walking.

Another comparison across ages was performed with amputees. To identify how amputees control the pelvis when they have to walk in different conditions, Howcroft et al. [8] evaluated unilateral transtibial amputees with very different ages, but they have found similar behaviors across ages. Unilateral transtibial amputees aging from 40 to 90 years old have similar pelvis stability when they have done level-ground walking and simulated uneven ground walking. Howcroft et al. [8] evaluated those amputees walked with inertial sensors on the pelvis during level and uneven conditions. The maximum Lyapunov exponent was similar for those two conditions.

Stability in walking can be also associated with risk to fall. Young and old adults do not have the same consequences after an accidental falls. Old adults have higher mortality after a fall than young adults, and the elderly is more prone to have more health conditions related to fall than young adults. Therefore, risk to fall in the elderly is a public health problem. The presence of risk factors (loss of muscle power and strength, balance diseases, visual problems, and others) are used to define the risk to fall. Functional tests have been also designed to determinate the risk to fall in the elderly. Although it is quite known which are risk factors and functional tests, both fail to prevent falls. Therefore, researchers are still looking for variables or tests to clearly find the ones with more risk to fall. Lockhart and Liu [9] designed an experiment to test whether largest Lyapunov exponent is sensitive enough to differentiate people prone to accidental falls. Healthy young adults, healthy old adults, and fall-prone old adults walked on a treadmill for 1 min and the kinematics of the trunk and foot were recorded for further analysis. Maximum Lyapunov exponent was the highest for the fall-prone old adults group.

Another study about falls in elderly evaluated how older adults recover from a fall on a slippery surface. About 100 older adults participated into a gait-slip

experiment. Yang and Pai [10] asked older adults to walk on a pathwalk with a sliding device. The trajectory of whole body center of mass was measured and used to calculate the Lyapunov exponent. Short- and long-term Lyapunov exponents were not different between fallers and those who did not fall; although, Lyapunov exponent presented more sensitivity to detect falls than specificity to detect recoveries from falling.

To understand the risk to fall, studies have compared the stability during walking of fallers and non-fallers [9] and how older adults recover stability when they were walking under an unexpected slippery condition [10]. Although Lyapunov exponents were able to differentiate fallers and non-fallers, this measure was not sensible enough to discriminate when older adults slipt. Another risk to fall is to perform a dual task. Howcroft et al. [11] proposed to elderly participants to walk saying words starting with A, F, or S in a random order. These authors showed that such a cognitive load did not affect the maximum Lyapunov exponent calculated from pelvis acceleration.

Those three studies suggest that stability measured with Lyapunov exponent has not a simple association with the risk to fall. Although Lyapunov exponent can be used to discriminate fallers and non-fallers, when elderly people and perform a task, the changes due to the task did not affect stability. It is important to emphasize that participants performed the task during those studies, which was walking. Even though older adults present more difficulties to perform more dangerous or complex tasks, they have preserved the same level of stability.

Lyapunov exponents are sensitive to find changes in gait stability not only due to fall. Unstable walking behaviors might be observable when someone rumbles or claudicates during walking. Claudication can be induced by peripheral arterial disease (PAD). Myers et al. [12] compared elderly with PAD and matched healthy controls when they were walking on a treadmill. PAD participants had higher largest Lyapunov exponent compared to control group. Another health condition that changes gait pattern is orthopedic conditions. Such conditions induce pain or lead to physical constraints in joint movements. Osteoarthritis is one example of such condition. Alkjaer et al. [13] showed that older women with osteoarthritis and matched controls have similar stability in joint kinematics. Alkjaer et al. [13] evaluated the ankle and knee kinematics during 6 min walking test on a treadmill and used those data to calculate the largest Lyapunov exponent. Those women with osteoarthritis presented similar knee and ankle largest Lyapunov exponents compared with matched controls. In order to avoid pain, it is common to observe that people change how to move. This pain avoidance was studied by Hamacher et al. [14] using visual feedback. Visual feedback was applied to change gait patterns in order to avoid pain in different parts of the body. Hamacher et al. [14] applied this approach in 60-year-old women. Those authors found that visual feedback of the frontal plane pelvis and trunk movements have increased largest Lyapunov exponent.

4.4 Stability and Gait Speed

When researchers design an experimental protocol for gait analysis, one typical concern is walking speed. Two major branches appear: studies with self-selected walking speed and studies with fixed walking speed. Any walking speed can be achieved by a combination of stride length and stride frequency. For fast running, athletes usually do not change stride frequency but increase stride length [15]. Russel and Haworth [16] compared Lyapunov exponents calculated during preferred walking stride frequency (PSF) higher and lower stride frequencies in young adults. The authors found that local dynamic stability was maximal at the PSF, becoming less stable for higher and lower stride frequencies.

England and Granata [17] examined gait instability in relation to walking velocity. When the data from every stride were time-normalized to 100 data points per stride, the authors found that Lyapunov exponents were linearly associated to walking velocity. Temporal variation per stride introduces a more quadratic trend. Smaller Lyapunov exponents represent a more stable system. England and Granata [17] found that every lower limb joint was more stable at lower velocities.

4.5 Sampling Frequency and Stability

In order to calculate the Lyapunov exponents, phase space portraits are necessary to identify the nearest neighbors. Important requirement to calculate local dynamical stability is the minimum number of data points. As a general rule, 10^d is necessary, where d is the data dimension [18]. To achieve such a requirement, it is important to use the adequate sampling frequency and collect data for time enough to have enough data. England and Granata [17] investigated the effect of halving the number of data samples per stride. They compared results from data that were resampled at 3000 samples per 30 strides versus results from the same walking trials that were resampled at 1500 samples per 30 strides; both resample rates retained the stride-to-stride temporal variation of the original kinematic data. Although, shorter data set lengths reduced the mean value of λMax by 17.2 %.

Riva et al. [19] measured variability and stability (standard deviation, coefficient of variation, Poincaré plots, maximum Floquet multipliers, short-term Lyapunov exponents, recurrence quantification analysis, multiscale entropy, harmonic ratio, and index of harmonicity) with stride duration and trunk acceleration data during walking. Those authors verified the effect of sampling frequency and directional changes in walking (young adults should walk for 6 min along a 30 m pathway) to variability and stability measures. They found that multiscale entropy, short-term Lyapunov exponents, and recurrence quantification analysis were generally not affected by directional changes nor by sampling frequency (100 and 200 Hz).

Riva et al. [20] calculated short-term/long-term Lyapunov exponents and RQA in ten young adults. Participants walked in a straight line at self-selected speed on a 250 m long dead-end road (about 180 strides). The acceleration of the trunk and shank was measured. The authors have studied minimum number of strides

required to use those measures. Ten strides were enough for Lyapunov exponents and RQA to depict walking behaviors.

Reynard and Terrier [21] calculated the local dynamical stability (LDS) (Lyapunov exponents) walking tests, and they found that:

1. The intrasession repeatability of gait LDS was about 0.50 for long-term LDS and 0.85 for short-term LDS and the intersession repeatability was around 0.6 for both short- and long-term LDS.
2. Long-term LDS estimated from short-duration measurements (35 strides) exhibited low repeatability, while that of short-term LDS was around 0.75.
3. Long-term LDS exhibited discrepancy between the predicted repeatability and the actual repeatability, while it was not the case for short-term LDS.
4. Taking into account both intracorrelation coefficient and standard error mean, short-term LDS measured over one step tended to have better intra- and inter-session reliability than short-term LDS measured over one stride.

Therefore, the authors have recommended to normalize sample length before computing LDS to thwart the trend to higher LDS estimates with longer measurements. For long-term LDS, it is highly recommended to record long duration walking tests with treadmill; its use should be restricted to group-level assessment with a sufficient sample size to lower type II error risk. For short-term LDS, it is recommended to use λ 0.5; it may be sufficient to detect moderate changes (around 20 %) at individual level. However, due to the lower intersession repeatability, one should aggregate several measurements taken on different days to better approximate the true LDS.

4.6 Algorithms to Calculate Lyapunov Exponent

Local dynamic stability or largest (maximum) Lyapunov exponent depicts how rate close trajectories converge or diverge infinitesimally within a *n*-dimensional phase space [22]. Mostly, two algorithms are applied to calculate the largest Lyapunov exponents: Wolf [23] and Rosenstein [24] algorithms.

Wolf et al. [23] presented a technique to calculate the nonnegative Lyapunov exponents from finite amounts of experimental data, but this technique is limited to systems where a well-defined, one-dimensional (l-D) map can be recovered. This approach is based on a spectral estimation of Lyapunov exponents. Wolf et al. [23] remind that it is necessary to attend three factors to calculate Lyapunov exponents: the number of points necessary to provide an adequate number of replacement points (between 10^d and 30^d, where d is the embedded dimension), the number of orbits of data necessary to probe stretching (but not folding) within the attractor (10–100), and the number of data points per orbit that allow for proper attractor reconstruction with delay coordinates ($d \times 10^{d-1}$ to $d \times 100^{d-1}$).

Rosenstein et al. [24] proposed another technique to calculate the largest Lyapunov exponents, claiming its reliability for small data sets. Those authors

have used the method of delays to develop a fast and easily implemented algorithm. The matrix of reconstructed trajectories of the data is used to reconstruct the dynamics of the system. Then, Rosenstein's algorithm locates the nearest neighbor of each point on the trajectory. The authors have used time series as short as only 100–1000 data points successfully.

It is important to emphasize that more accurate Lyapunov exponents are observable with larger data sets (Cignetti et al. [22]). One thing that must be avoided, in order to have the necessary data length, is to indiscriminately increase the sampling rate. Higher sampling rate does augment the length of a time series but not necessarily increases information. Parsimonious relation between information and sampling frequency exists (Nyquist frequency) and must be taken into account to decide the adequate sampling frequency.

4.7 Conclusion

Many parameters based on dynamical systems or variability are used to study gait stability. Definitions of most of those parameters tell that different issues might be accessed during gait stability studies that do not have a relation to stability. Important concerns about sampling, minimum number of strides, data normalization, and confiability were presented. Lyapunov exponents can be used to compare persons with different ages.

References

1. Ferrier JF. The stretching of the foot during walking. Comptes Rendus des Seances de la Societe de Biologie et de ses Filiales. 1901;53:721–2.
2. Hamill J, van Emmerik RE, Heiderscheit BC, Li L. A dynamical systems approach to lower extremity running injuries. Clin Biomech. 1999;14(5):297–308.
3. Hausdorff JM, Peng CK, Ladin Z, Wei JY, Goldberger AL. Is walking a random walk? Evidence for long-range correlations in stride interval of human gait. J Appl Physiol. 1995;78(l):349–58.
4. Parks PC. A. M. Lyapunov's stability theory - 100 years on. IMA J Math Cont Inf. 1992;9:275–303.
5. Glass L. Synchronization and rhythmic processes in physiology. Nature. 2001;410:277–84.
6. Bisi MC, Riva F, Stagni R. Measures of gait stability: performance on adults and toddlers at the beginning of independent walking. J NeuroEng Rehab. 2014;11:131–9.
7. Terrier P, Reynard F. Effect of age on the variability and stability of gait: a cross-sectional treadmill study in healthy individuals between 20 and 69 years of age. Gait Posture. 2015;41:170–4.
8. Howcroft J, Lemaire ED, Kofman J, Kendell C. Understanding dynamic stability from pelvis accelerometer data and the relationship to balance and mobility in transtibial amputees. Gait Posture. 2015;41(3):808–12.
9. Lockhart TE, Liu J. Differentiating fall-prone and healthy adults using local dynamic stability. Ergonomics. 2008;51(12):1860–72.
10. Yang F, Pai YC. Can stability really predict an impending slip-related fall among older adults? J Biomech. 2014;47(16):3876–81.

11. Howcroft JD, Lemaire ED, Kofman J, McIlroy WE. Analysis of dual-task elderly gait using wearable plantar-pressure insoles and accelerometer. Conf Proc IEEE Eng Med Biol Soc. 2014;2014:5003–6.
12. Myers SA, Johanning JM, Stergiou N, Celis RI, Robinson L, Pipinos II. Gait variability is altered in patients with peripheral arterial disease. J Vasc Surg. 2009;49(4):924–31.
13. Alkjaer T, Raffalt PC, Dalsgaard H, Simonsen EB, Petersen NC, Bliddal H, Henriksen M. Gait variability and motor control in people with knee osteoarthritis. Gait Posture. 2015;42 (4):479–84.
14. Hamacher D, Hamacher D, Schega L. Does visual augmented feedback reduce local dynamic stability while walking? Gait Posture. 2015;42(4):415–8.
15. Rabita G, Dorel S, Slawinski J, Sàez-de-Villarreal E, Couturier A, Samozino P, Morin JB. Sprint mechanics in world-class athletes: a new insight into the limits of human locomotion. Scand J Med Sci Sports. 2015;25:583–94.
16. Russell DM, Haworth JL. Walking at the preferred stride frequency maximizes local dynamic stability of knee motion. J Biomech. 2014;47:102–8.
17. England SA, Granata KP. The influence of gait speed on local dynamic stability of walking. Gait Posture. 2007;25(2):172–8.
18. Rossler OE. Continuous chaos - four prototype equations. Ann NY Acad Sci. 1979;316:376–92.
19. Riva F, Grimpampi E, Mazzà C, Stagni R. Are gait variability and stability measures influenced by directional changes? BioMed Eng OnLine. 2014;13:56–65.
20. Riva F, Bisi MC, Stagni R. Gait variability and stability measures: minimum number of strides and within-session reliability. Comput Biol Med. 2014;50:9–13.
21. Reynard F, Terrier P. Local dynamic stability of treadmill walking: intrasession and week-to-week repeatability. J Biomech. 2014;47:74–80.
22. Cignetti F, Decker LM, Stergiou N. Sensitivity of the Wolf's and Rosenstein's algorithms to evaluate local dynamic stability from small gait data sets. Ann Biomed Eng. 2012;40:1122.
23. Wolf A, Swift JB, Swinney HL, Vastano JA. Determining lyapunov exponents from a time series. Physica D. 1985;16:285–317.
24. Rosenstein MT, Collins JJ, De Luca CJ. A practical method for calculating largest Lyapunov exponents from small data sets. Physica D. 1993;65:117–34.

Visual Control of Adaptive Locomotion and Changes Due to Natural Ageing

5

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Abstract

This chapter will review current theories of how adults use vision to guide adaptations to the basic locomotor rhythm in order to cope with environmental demands, e.g. to step over an obstacle in our path or to guide the foot to specific safe areas in the terrain or to cope with the challenges to balance posed by descending stairs. A finding common to different gait adaptations is that they are usually, by necessity, programmed in advance to ensure stability and maximise efficiency. Thus visual cues are predominantly sampled and used in a feedforward or anticipatory manner to make gait adjustments. Recent evidence suggests that older adults, particularly those characterised as being at higher risk of tripping and falling, show changes in visuomotor control during adaptive locomotion and that the resulting changes to visual behaviour may be causally related to lower limb movement inaccuracies, compromised balance control and associated increased falls risk. The putative neural and biomechanical mechanisms underlying these changes will be discussed alongside the potential applications of current knowledge to falls prevention and rehabilitation in older adult populations.

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5.1 Introduction

In the United Kingdom, around 30 % of adults over the age of 65 living in the community fall each year. Furthermore, falls are the main cause of disability and the leading cause of death from injury among people aged over 75 in the United Kingdom. Older adults who survive a fall often suffer ongoing problems such as injury and infection, increasing risk for loss of mobility, depression, anxiety and an increased mortality risk. The associated healthcare costs of older adult falls in Britain alone are estimated at around £1 billion per year [1]. Therefore, understanding the mechanisms underlying falls in older adults has become an important target in ageing research, with the aim to develop effective prevention strategies.

The majority of falls result from slips or trips [2, 3] which represent a failure to place the feet appropriately to meet environmental demands usually specified by visual information describing the locations of features such as stair tread edges, the location and dimensions of obstacles and safe areas in the terrain. Within this chapter we will summarise our current understanding of how vision is used to guide three common walking tasks: (1) stepping onto targets, (2) ascending and descending stairs and (3) crossing obstacles. Throughout the chapter we will also review the literature demonstrating that there are age-related differences in the visual control of these common walking tasks and explore the putative neural and physiological mechanisms underlying age-related decline in function. A final aim is to discuss the relevance of these findings to the problems of falls in older adults and offer suggestions for possible interventions to restore or compensate for age-related dysfunction in the visual control of walking.

5.2 Visually Guided Target Stepping

This section aims to summarise our current understanding of how visual information is used to guide accurate lower limb movements to specific locations on the ground during walking and will provide evidence that compromised eye movement control or maladaptive gaze strategies contribute to gait deficits in patient and older adult populations. Other objectives are to explore the mechanisms underlying suboptimal gaze and stepping behaviour and to suggest potential intervention strategies to improve eye and stepping coordination and reduce falls risk. Since much of our understanding of visual control of limb movements and the role of eye movements is based on investigations of the upper limb, this review will begin with a brief overview of the eye-hand coordination literature before considering the similarities and differences in eye-foot coordination.

The majority of everyday visually guided limb movements (e.g. reaching to pick up a cup of tea) are characterised by an initial saccadic eye movement to the target of interest [4] which usually results in the image of the target landing on the fovea, the most sensitive part of the retina. This behaviour is often termed visual fixation. Usually, only a single fixation is made for any single action, and gaze is often redirected away from the object before the limb reaches it [4]. Although there are very few studies that describe gaze behaviour during everyday activities, there is a growing body of evidence from the sport science literature that the duration of the final fixation of a target (e.g. a basketball hoop or a corner of a soccer goal) prior to movement initiation is causally related to the accuracy of the action. Joan Vickers carried out much of the seminal work in this area and coined the term 'quiet eye duration' to describe this parameter [5]. It has been demonstrated that skilled athletes show significantly longer quiet eye durations than less-skilled athletes [6–8] and that successful actions are preceded by longer quiet eye durations than unsuccessful actions [9]. It has also been shown that training individuals to increase quiet eye duration has a positive impact on accuracy [10]. In combination, these studies demonstrate that visually fixating a target is beneficial for producing accurate visually guided actions and that movement accuracy is dependent on optimal timing of eye movements to fixate targets.

Although the specific roles of visual fixation are not fully understood, it is likely that the main function of directing gaze at a target is to provide information about the position of the target with respect to the body which is used by the motor system (or systems) controlling the limb to plan and execute the required amplitude and direction of movement in an open-loop or ballistic manner [4]. It is important to note, however, that pointing the eyes at a target provides not only visual information about target properties (e.g. direction and distance) but also has the potential to provide non-visual information such as eye muscle proprioception and efference copy of eye movement commands which signal eye direction with respect to the head [11]. This information may provide additional cues as to where the target is located with respect to the limb being controlled. Support for this notion can be found in primate neurophysiological studies. There are single neurones in the posterior parietal cortex of monkeys that are most active when the monkeys fixate targets at a specific combination of depth and eccentricity (e.g. one neurone may be preferentially active when eyes are directed 15° to the left and 10 cm in front of the head, and a different neurone may be preferentially active when eyes are directed at a target laying straight ahead at a distance of 5 cm) [12]. These findings clearly demonstrate that signals derived from eye position can describe the location of the target being fixated in three-dimensional space. Although there is not yet any direct evidence of similar neural mechanisms in humans, there is evidence from behavioural studies that pointing the eyes at target locations is useful even when they are not visible [13] and that final hand location when stepping and reaching to invisible targets is geometrically related to the direction in which gaze is aligned [14]. If the hypothesis is correct that pointing the eyes at a target provides a combination of visual and non-visual information that can tell the CNS where the target is in 3D space, then this might help explain the large extent to which eye and

hand movements are coordinated by the CNS during reaching and pointing. For example, the latency of both eye and hand movements to look and point at targets varies greatly depending on the context in which the target is presented [15]. However, the relative timing between the eye and hand movement initiation is relatively invariant suggesting that the instructions to move them are generated synchronously. It has also been shown that, when looking at and reaching to targets that dissapear at movement onset, if the eyes are initially aimed at a more eccentric target location than the hand (and so move through a larger distance) the hand tends to overshoot the target [16]. Conversely, it has been shown that eye kinematics are influenced by hand movement characteristics [17, 18]. There are many more examples of studies which provide evidence that eye and hand motor systems interact to produce coordinated movements when looking and pointing to targets. However, there are surprisingly few studies which have studied the coordination of eye and foot movements during walking tasks.

Hollands et al. [19] were the first to document where and when individuals look during locomotion in a study of the timing relationships between participants' eye and stepping movements as they walked along a pathway of irregularly placed stepping targets [19]. The results showed that participants invariably fixated a target just prior to initiating a step towards it and that there was a close timing relationship between eye and foot movement initiation similar to that seen in eye-hand coordination studies. The same authors subsequently demonstrated that eye-stepping coordination was also seen when targets were temporarily rendered invisible [20]. Despite delays in step initiation resulting from the temporary removal and subsequent reinstatement of visual information, the mean interval between the start of the eye movement and the start of the swing towards a target did not vary significantly. These findings support the notion that eye and stepping movements are not programmed independently but rather are planned simultaneously as part of a coordinated eye-stepping movement similar to well-documented eye-hand coordination evident during reaches to targets. Although it is important to know when vision is sampled, this does not tell us how it is used to guide limb movements, e.g. for planning the stepping trajectory in an open-loop control manner or for online guidance of the limb onto the target via a closed-loop control process. Hollands and Marple-Horvat [21] showed that intermittently removing vision of stepping targets at different gait phases only affected behaviour when the limb was in stance phase (i.e. did not affect the limb in swing phase) and only when vision was unavailable during late stance [21]. Other studies have subsequently confirmed that removing vision during the stance phase of the targeting foot results in increased variability and reduced accuracy of subsequent foot placement, whereas removing vision during the ongoing swing phase has no effect [22]. These studies suggest that vision of a target is normally used in an open-loop or feedforward way prior to movement initiation and is not usually needed to guide the foot onto a target. There is evidence that this behaviour represents an energy-efficient biomechanical strategy resulting in preprogrammed ballistic limb movements [22–24]. However, other studies have showed that it is possible for young adults to make online adjustments to stepping trajectories as late as mid-swing phase in

order to track a target that moves [25, 26] and that removing vision of the lower limb relative to a target results in reduced stepping accuracy [27]. Therefore the strategy of fixating a stepping target prior to step initiation and maintaining gaze on a target until late swing allows for optimal use of visual information to both plan and guide ongoing stepping actions. It should be noted that other studies have subsequently shown that the extent to which participants look ahead at stepping targets is dependent on the characteristics of the targets, e.g. individuals tend to look two steps ahead when walking on regularly placed targets [28] but tend to look three steps in advance when walking on irregularly placed targets surrounded by differently coloured distractor targets [29]. Therefore the timing of gaze fixation is dependent on the complexity or precision of the task. Despite this context-dependent variability in the relative timing of eye and stepping movements, in most situations, gaze is directed to the future footfall target at some time point in advance of initiation of the step onto the target suggesting that directing gaze to a location on the ground ahead is useful to the motor control system responsible for programming a step to that location. If this hypothesis is correct, then individuals who have problems making accurate eye movements should show problems in performing target stepping tasks.

The importance of making accurate eye movements in the control of accurate step generation was demonstrated in a study of cerebellar patients who exhibit both eye movement problems (including saccadic dysmetria resulting in the need for multiple saccades to fixate a target) and walking deficits (ataxic gait—including stepping inaccuracies) [30]. The authors compared stepping performance according to the extent of saccadic inaccuracy (i.e. whether patients made one, two, three or more than three saccades to fixate a target during a particular step) and found significant delays in step initiation which increased with the extent of eye movement inaccuracies. A similar approach was presented by Di Fabio et al. [31] who studied the step climbing ability of progressive supranuclear palsy (PSP) patients, a patient group who has problems generating vertical eye movements. The authors scored the patients on the extent of their oculomotor dysfunction and showed that those patients with the most severe eye movement symptoms showed diminished trailing limb step clearances putting them at an increased risk of tripping on the stair edge [32]. The results of these studies suggest that problems making eye movements may be causally linked to problems making stepping movements and raise the possibility that interventions aimed at improving eye movement function may have a positive effect on functional mobility. Indeed preliminary evidence suggests that eye movement training has a positive effect on performance in both cerebellar [33] and PSP patients [32]. These clinical studies show that stepping inaccuracies in patients with gait disorders may be caused, at least in part, by a reduced ability to fixate environmental features at appropriate times in the action sequence. Could the increased incidence of trips and falls in older adults also be partially explained by age-related changes in eye movements?

Chapman and Hollands [34, 35] studied the gaze and stepping characteristics of older adults asked to make different combinations of target and obstacle stepping and compared their data to that collected from young adults. The older adults were

separated into two groups, based on their performance in various clinical tests of functional mobility, representing those individuals deemed to have a low risk of falling and those deemed to have a higher risk of falling. Comparisons between the three groups showed that on average, the high-risk group looked away earlier from the current target than the other groups but only in the more complex tasks requiring additional future obstacles or targets [34, 35]. The extent of premature redirection of gaze correlated with increased target stepping errors. A causal link between early gaze transfer and stepping inaccuracies was demonstrated in a further experiment that showed that simply instructing participants to maintain target fixation until after the foot had landed resulted in increased stepping accuracy [36]. However, this advice may not be appropriate in real-world contexts, and therefore in order to address this problem, we need to know what is causing older adults to look away early.

Young et al. [25, 37] provided evidence that anxiety about upcoming obstacles is one factor that causes high-risk older adults to look away prematurely from targets [37] and that this anxiety can result from experiencing actual falls [25]. It is well established that anxiety and fear of falling are significant predictors of falls prevalence [38]. These findings suggest that stepping inaccuracies resulting from anxiety-induced changes to gaze behaviour may offer a mechanistic explanation for the link between anxiety and falls risk. Interventions aimed at increasing self-confidence and reducing anxiety may well lead to improvements in gaze and stepping behaviour.

Other evidence suggests that age-related decline in cognitive function and the ability to appropriately switch attention between environmental hazards may underlie maladaptive gaze behaviour [39]. There is a large body of evidence demonstrating a link between cognitive decline and general falls risk in older adults and also between anxiety and cognitive decline [38]. A number of target stepping experiments have shown age-related decline in stepping performance is associated with cognitive function [37, 40–43] and that delays in the timing and accuracy of step adjustments are related to, or predictive of, falls risk [37, 40].

Finally, laboratory studies have shown age- and falls risk-related differences in visuomotor ability. High-risk older adults need to view stepping targets for longer than younger adults and low-risk older adults in order to make medial-lateral stepping adjustments [44] presumably because it takes longer to generate coordinated eye and stepping actions [25]. Both Nonnekes et al. [45] and Young and Hollands [37] have proposed that delays in, possibly subcortical, visuomotor processing with ageing may be responsible for disruption to timing and magnitude of stepping adjustments to visual cues in these populations.

In combination these findings suggest that maladaptive gaze behaviour which contributes towards stepping inaccuracies and increased falls risk results from age-related changes to a number of interrelated CNS functions. Interventions aimed at addressing this problem should therefore target each of these factors. Encouragingly, a recent randomised controlled trial studying the effects of multiple target stepping exercises in combination with a multicomponent exercise programme led to improved scores on functional mobility tests, increased target stepping accuracy, improved gaze behaviour and reduced fall rates in community-dwelling older adults compared to a control group experiencing comparable

exercise intensity [46]. Although the specific mechanisms that drove the improvement in walking and associated reduction in falls risk could not be fully elucidated, it is possible that exercises aimed at improving visually guided stepping actions will prove effective in improving cognitive, visuomotor and physical function and reducing falls risk. One visually guided walking task that is particularly hazardous for older adults is stair walking. The next section will summarise our current understanding of the roles of vision in stair negotiation and the implications of visual decline on the problem of stair falls in older adults.

5.3 Visual Control of Stair Walking

Falling on stairs is the leading cause of accidental death in older adults accounting for 60 % of fall-related deaths [47]. Falls occur most often during stair descent [48–50] and often result from a trip on a stair edge or tread surface [49, 51] which needs to be visually identified by the walker. Accordingly, as with other visually guided tasks requiring precise foot placement, it is likely that appropriate direction of gaze to relevant environmental features at appropriate times is crucial for safe stair negotiation. Anecdotal evidence that supports the notion that gaze direction is important for safe stair walking comes from security video footage showing that falls are more likely to occur when an individual does not appear to look down prior to walking up or down stairs [52]. Zietz and Hollands [53] studied gaze behaviour of both younger and older adults walking up and down the middle eight stairs of a twelve-step staircase [53]. They found that both groups spent the majority of the time during both stair ascent and descent looking at the edges of stairs with an average time lead of around two to four steps. Interestingly, unlike gaze behaviour observed during target stepping described earlier in this chapter, participants did not generally look towards future foot placement locations on the stairs, but rather gaze was constrained to the central portion of the stairway suggesting that gaze may serve another function than identifying locations on the steps to guide foot placement. Den Otter et al. [54] provided further evidence for this hypothesis by showing that 28–34 % of treads were never fixated during stair walking, and when participants travelled two stairs per step rather than one, they still commonly (>35 % of occasions) fixated the stair treads they did not subsequently step on [54]. These studies raise the possibility that visual descriptors of the stair edges may be used for other purposes than guiding foot trajectory to clear stair edges or to land in particular locations on the stairs.

Zietz et al. [55] studied the stepping characteristics and posture of older adults descending stairs under combinations of ambient light levels and stair edge contrast conditions in order to further elucidate the roles of vision describing tread edges in controlling stair walking [55]. They found that increased tread edge contrast led to greater postural stability during stepping in the older adult groups. However, they found no effects of step edge characteristics on foot placement parameters. These results suggest that stair edges may provide visual cues that maximise perception of self-motion (derived from optic flow) to aid in balance maintenance in addition to the spatial properties of the stairs.

It is noteworthy that the role of vision when negotiating stairs seems to differ markedly from that during overground target stepping as evidenced by the differences in gaze behaviour and effects of manipulating vision on stepping and balance characteristics. One possible reason for this difference is the predictability of the required step adjustments under comparison. Staircases usually have uniform stair dimensions, and therefore the role of vision in describing their spatial properties will be understandably reduced compared to a task that required large and variable changes in step characteristics to achieve. Also, stair walking, and in particular stair descent, places much greater demands on postural and balance control than level walking; therefore it is, perhaps, unsurprising that visual cues that aid the control of balance are preferentially attended. Arguably the greatest challenge to balance associated with stair walking occurs during the transitions from floor to stairs and vice versa. Studying the visual control of such transitions is important since the three steps at the bottom and at the top of stairs are reported as the most common location for missteps and stair accidents [48, 56, 57].

Miyasike-daSilva et al. [58] studied the gaze behaviour of young adults ascending and descending stairs and included analysis of behaviour during the stair transitions [58]. Contrary to their predictions, they did not find that participants made additional or longer fixations during transition steps than during mid-steps and concluded that participants may rely more on peripheral than foveal vision to guide step transitions. This finding is consistent with the results from other studies showing that occluding the lower visual field during walking leads to various compensatory changes to head posture and gait parameters [59, 60] and that bifocal spectacles that distort the lower visual field have negative effects on stair walking characteristics [61] and increase falls risk [62]. The observed gaze strategy of maintaining a relatively constant angle between gaze and staircase may be advantageous to participants by not only simplifying the extraction of pertinent information from retinal flow fields for maintaining heading and guiding balance but also for providing peripheral vision of the lower limbs and stairs. Since visual information is crucial for safe stair walking both for identifying step edges and for controlling balance, what then are the implications for the increased prevalence of falls on stairs in older adults?

The visual system undergoes specific age-related changes which affect an individual's ability to see relevant stair features. For example, the ability of the pupil to dilate under low to moderate levels of illumination is greatly reduced with increasing age, resulting in limitations to amount of light reaching the retina [63]. Indeed, it has been shown experimentally that reducing the levels of ambient lighting results in older adults exhibiting greater variability and average reduction in the size of the clearance between the toe and the stair during a step [55]. There is also evidence that restricted visual conditions adversely affect the control of locomotion in transitions between floor level and stairs [64]. Age-related changes in contrast sensitivity, which are pronounced at low levels of lighting, may hinder the detection of stair edges and transition regions. Indeed, visually identifying the tread edge is particularly problematic for older adults when the ambient illumination is low, the step covering is patterned or if they are visually impaired resulting in decreased stability, more cautious stepping, reduced foot clearance with the step

and increased foot clearance variability [48, 65–68]. Reduced foot/heel clearances over the tread edge, greater clearance variability and misjudgements in foot placement when descending steps or stairs are factors that are all reported to increase falls risk [68, 69]. As mentioned above multifocal eyeglasses that correct eyesight problems experienced by the majority of older adults distort the lower peripheral visual field resulting in impaired step negotiation and accuracy of foot placement when stepping on to a raised surface or negotiating an obstacle course [70–72]. Finally, Zietz and Hollands [53] identified significant differences in the length of time older adults fixated the stairs prior to stepping and a reduced variability in the extent to which older adults looked ahead [53]. These differences in looking strategies might be a by-product of age-related changes to visuomotor control. However, the authors did not measure body kinematics and so could not directly relate differences in gaze behaviour to stepping or balance performance. Nevertheless, this study suggests that visual information describing the tread edges is important for safe stair walking and that there are age-related differences in when this information is sampled which might contribute towards altered stepping and balance control and associated increased falls risk.

Encouragingly, most of the age-related problems listed above can be easily addressed by manipulating environmental conditions, e.g. ensuring stairwells are adequately illuminated and that tread edges are highlighted appropriately. Advice can be given when eyeglasses are prescribed as to when it is appropriate to wear multifocal lenses. It is also possible that the observed age-related differences in gaze behaviour may contribute to stepping inaccuracies and instability in older adults walking on stairs and, if so, may be amenable to intervention, but further studies of frail older adults are needed in order to establish that link. Finally, Elliott et al. [73] showed that it is possible to manipulate the perceived height of a step using a simple visual illusion (a striped pattern on the stair riser) [73]. This altered perception led to the adoption of a safer stepping strategy in terms of greater foot clearance over a step edge. These studies raise the possibility of designing optimal environments to ameliorate age-related changes to stepping and balance resulting from visual and other sensory decline.

5.4 Visual Control of Obstacle Crossing

Stepping over obstacles, such as door thresholds and broken sidewalks, is a common requirement of community mobility [74]. The observation that trips account for up to 53 % of falls in older adults [2, 3] indicates that older adults are not very successful at avoiding obstacles in their everyday lives. This lack of success could result from a number of age-related factors, including a compromised ability to visually detect the obstacle height and position, reduced ability to lift the limb adequately to clear the obstacle and/or an inability to recover balance after the foot contacts an obstacle. A systematic review of reactive responses to trips in older adults was recently completed, and the reader is referred to Galna et al. [75]. This reactive mechanism should be considered the ‘backup’ defence when the

proactive mechanism fails [76]; avoiding the trip in the first place is the more effective means of ensuring stability. Thus, this section provides an overview of the literature regarding visual control of stepping over stationary, visible obstacles.

When walking up to a stationary obstacle, young participants visually sampled the obstacle, with obstacle fixations occurring for about 20 % of the approach phase [77]. The frequency of obstacle fixations increased as a function of obstacle height [77], reflecting increased visual attention on obstacles which have a higher risk of tripping [78]. The information gained from obstacle fixations is used to control movement in a feedforward mode, rather than online, as young adults fixated on the obstacle during approach, but not during crossing [77]. Pontecorvo et al. [79] have demonstrated that obstacle fixations are related to success in crossing the obstacle in young adults. When stepping over stationary obstacles in the lab, participants inadvertently contact the obstacle in 1–2 % of trials [80, 81]. While these trials are typically discarded, Heijnen et al [80] demonstrated that these failures provide important insight into the behaviours that result in a trip and possible fall. Using this methodology, it was found that young participants who never contacted the obstacle had longer and more frequent fixations on the obstacle than those who contacted the obstacle at least twice during data collection (approximately 1.5 hours duration) [79].

Changes in locomotor performance were also observed in young adults during modification of visual information of the foot relative to the obstacle (termed exproprioception) and when visible characteristics of the obstacle (termed exteroception) were reduced. When exproprioception is reduced with lower visual field occlusion goggles, young adults demonstrate increased toe clearance, toe clearance variability, foot placement farther away from the obstacle and increased contacts [78, 82–84]. The increased variability indicates that the limb trajectory is modified in an online manner as the limb crosses the obstacle. Since participants do not foveate the obstacle during crossing [77], the participants are relying on peripheral visual cues during obstacle crossing for this online control. However, Rietdyk and Rhea [83] demonstrated that increased visual cues in the environment can guide foot placement when foot-obstacle exproprioception is obstructed. This group also demonstrated that when exteroceptive information was systematically reduced by decreasing the visual characteristics of the obstacle, obstacle contact rates increased [78]. In particular, when the interface of the ground and the bottom edge of the obstacle was not visible, such as observed when an obstacle is created with a dowel suspended across two vertical supports, contact rates were higher than when the bottom edge of the obstacle was clearly demarcated [78]. It is likely that the visibility of the obstacle-ground interface improves knowledge of obstacle position, leading to optimal foot placement around an obstacle, which in turn decreases contact risk as discussed below. Thus, it is clear that young adults use visual information in both a feedforward and online manner to ensure successful clearance of an obstacle.

Gaze behaviour in older adults during obstacle approach and crossing has been examined concurrently with various manipulations, such as dual tasking or responding to a limb-selection cue during approach [31, 85]. Older fallers looked away from the obstacle sooner than older non-fallers in a dual task condition; this early transfer of gaze was accompanied by increased foot placement variability

[85]. When older adults were presented with a limb-selection cue during obstacle approach, relatively longer saccade/foot lift latency and prolonged gaze fixation time were observed relative to young adults [31]. Thus, differences in gaze behaviour are observed both as a function of age and falls risk. However, a direct comparison of gaze behaviour between young and older adults without various instructions or manipulations has not been examined. Muir et al. [86] used head angle to infer gaze behaviour and found that older adults (65–79 years) had a lower head angle than young adults during the approach phase, consistent with increased visual fixation on the obstacle. Reduced visual acuity did not drive this behaviour as all participants had corrected-to-normal vision. Rather, these changes likely reflect factors such as compromised visuomotor transformation or other vision factors, such as depth perception [87]. Next, we describe the age-related changes in gait behaviour that inform visual control of locomotion.

When older adults walk over a clear walkway or step over obstacles, they demonstrate slower gait speed and shorter step lengths than young adults, likely due to increased caution (e.g. [81, 86, 88, 89]). However, the slower speed would also provide more time to gather visual information, which would facilitate control of locomotion. The interpretation that the slower gait speed allows the older adult more time to gather visual information is consistent with the lower head angle observed when older adults approach the obstacle (Fig. 5.1) [86]. The observation

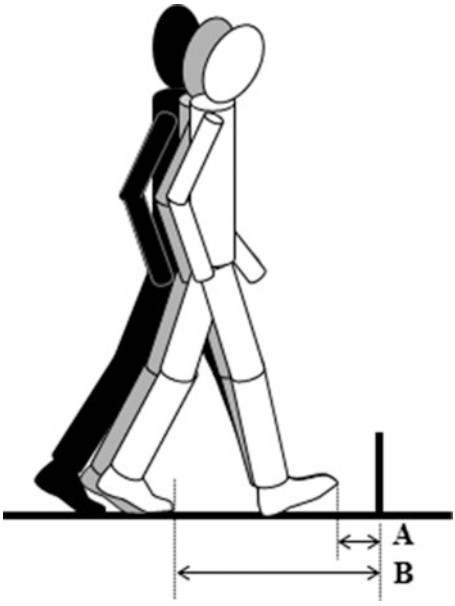

Fig. 5.1 Representative body positions for young (20–25 years, *black figure*), older (65–79 years, *grey figure*) and oldest adults (80–91 years, *white figure*). The head angle is tilted down more with advancing age, consistent with increased foveation on the obstacle during approach. The step length and lead approach distance (*B*) are progressively shorter with advancing age. However, trail foot placement before obstacle (*A*) was not different as a function of age (results from [81, 86, 88])

that adults older than 80 years walked slower and looked down more than adults aged 65–79 years indicates that these strategies become more prevalent with advancing age [86]. While slower speed appears to be an appropriate strategy to maintain stability and optimise visual information, the shorter step length increases the likelihood of contacting an obstacle during obstructed gait [88]. Thus, it's important to examine how older adults manage this contact risk.

The shorter step length when crossing obstacles results in closer foot placement to the obstacle before and/or after crossing, increasing the risk of obstacle contact [88]. To ensure safe placement of the foot, both young and older adults visually fixate on the landing area after the obstacle [31, 77]. This observation is further supported by the higher frequency of fixations on the landing area by young participants who never contacted the obstacle when compared to participants who inadvertently contacted the obstacle at least twice [79]. A remarkably consistent behaviour in the foot placement before the obstacle has been observed [81, 86, 88] (Fig. 5.1). Despite the change in step length, it is clear that the same foot placement in the ultimate step is selected. Thus, healthy ageing older adults have maintained the ability to transform the visual information of obstacle position to an appropriate placement of the trail foot (second foot to cross the obstacle). This visuomotor transformation is likely facilitated by looking down more (Fig. 5.1) [86]. Trail foot placement is critical because a closer foot placement corresponds to a greater risk of obstacle contact with the trail limb in young adults [80, 90]. Thus, there is converging evidence that indicates that healthy older adults have adequate visuomotor transformation to maintain an invariant trail foot placement, in order to reduce the likelihood of obstacle contact with the trail limb.

A consistent trail foot placement coupled with a shorter step length necessitates that the lead foot is placed closer to the obstacle after the obstacle is cleared. This closer foot placement leads to increased likelihood of contact with the rear region of the foot during lead limb crossing. Indeed, this has been observed experimentally by examining the inadvertent obstacle contacts. Only older adults contacted the obstacle in Chen et al. [88], and all participants were described as having stepped on the obstacle—consistent with inadequate distance between the heel of the lead limb and the obstacle at crossing. Further, older adults had higher proportion of lead limb contacts than young adults [86]. The majority of inadvertent contacts occur with the trail limb for young adults with either full vision or lower visual field obstruction [78, 80, 83, 84, 91–93]. More frequent trail limb contacts are attributed to the fact that the trail limb is not visible during crossing, so online visual control is not available. Conversely, lead limb contacts were more frequent when vision was distorted or fully obstructed [71, 82, 93, 94]. Thus, the higher proportion of lead limb contacts in older adults may reflect impairment of the online visual guidance of the lead limb trajectory. Higher lead limb contacts in older adults are relevant for falls risk due to the higher likelihood of a fall for a lead limb versus trail limb contact [86].

It is traditionally assumed that lower foot clearances are associated with increased trip risk, so foot clearances are typically quantified in adaptive locomotor research. Heijnen and Rietdyk [95] provided preliminary evidence that supports this

intuitive assumption: falls in the real world are related to lower foot clearances when crossing an obstacle in the lab in young adults [95]. However, age-related changes in clearance during obstacle crossing include increased clearance [96], decreased clearance [97, 98] and no significant change in clearance [81, 86, 88, 99]. Since obstacles and lab environments are different across studies, and clearance measures are sensitive to visible obstacle characteristics [78] and visual structure in environment [83], as described earlier, foot clearance may not provide a reliable indicator of age-related changes unless different labs adopt uniform obstacles and lab environments.

Finally, we further examine the relationship between vision and obstacle contact rates. Older adults contact more obstacles under the following conditions: wearing multifocal lenses [71, 72] and when provided with visual samples of 16 ms duration at a rate of three samples per second during the approach (using liquid crystal goggles) [100]. Further, the depth perception of older adults was a significant determinant of obstacle contacts [87].

The findings reported here lead to a series of potential intervention strategies. First, older adults should be assessed for depth perception and should be encouraged to wear single-distance lenses when walking (as opposed to multifocal lenses). Second, optimal gaze behaviour for reducing trip and falls risk should be confirmed for older adults, and interventions that promote this gaze behaviour should be empirically tested. Therapeutic interventions to promote safe obstacle crossing should consider not only limb elevation but also optimal foot placement. The environment can also be modified to optimise safe clearance of fixed obstacles. For example, kerbs should be painted with high-contrast paint to increase the visibility of the location of the rising edge, which may reduce trip risk. We have had older adults indicate that the sidewalk kerbs with a curved rising edge, as opposed to a vertical rising edge, are especially problematic. This is likely due to the inability to determine the ideal foot placement before and after the rising edge, as there isn't a clear demarcation of the position of the rising edge. Further, adding visual structure around obstacles may be helpful, such as using a high-contrast paint colour around doorways with a threshold.

5.5 Conclusions

This chapter has reviewed our current understanding of the visual control of three common adaptations to the basic locomotor pattern to avoid obstacles and guide the feet to safe locations in the environment. Although the review has highlighted variations in the types of visual and non-visual information used to guide balance and stepping movements in different contexts, it is clear that appropriate alignment of the eyes with respect to environmental features at particular points in the gait cycle is important for both programming and guiding safe and efficient stepping adjustments. The chapter has also reviewed evidence that age-related changes in gaze behaviour due to visual, visuomotor and cognitive decline have been causally linked to increased falls risk in older adults, and, therefore, it is clear that these

impairments should be targeted when designing intervention strategies aimed at reducing falls risk. Encouragingly, there is a growing body of evidence to suggest that interventions aimed at improving eye and stepping coordination, whether through training or through built-environment design, have a positive effect on stepping control and reducing falls risk. However, further work is still needed to fully understand the mechanisms underlying age-related changes in visual and sensorimotor processing so that effective interventions can be designed and implemented.

References

1. Scuffham P, Chaplin S, Legood R. Incidence and costs of unintentional falls in older people in the United Kingdom. J Epidemiol Commun Health. 2003;57(9):740–4.
2. Berg WP, Alessio HM, Mills EM, Tong C. Circumstances and consequences of falls in independent community-dwelling older adults. Age Ageing. 1997;26(4):261–8.
3. Blake A, Morgan K, Bendall M, Dallosso H, Ebrahim S, Arie T, et al. Falls by elderly people at home: prevalence and associated factors. Age Ageing. 1988;17(6):365–72.
4. Land MF, Hayhoe M. In what ways do eye movements contribute to everyday activities? Vision Res. 2001;41(25-26):3559–65.
5. Vickers JN. Visual control when aiming at a far target. J Exp Psychol Human. 1996;22 (2):342–54.
6. Vickers JN, Adolphe RA. Ball tracking gaze behaviour of elite volleyball players during the serve reception and pass. J Sport Exercise Psychol. 1998;20:S119.
7. Vickers JN. Gaze control in putting. Perception. 1992;21(1):117–32.
8. Vickers JN, Martell SG. How elite ice hockey players read tactical plays as they defend against opponents. Res Q Exercise Sport. 2004;75(1):A129–30.
9. Wilson MR, Miles CAL, Vine SJ, Vickers JN. Quiet eye distinguishes children of high and low motor coordination abilities. Med Sci Sport Exercise. 2013;45(6):1144–51.
10. Harle SK, Vickers JN. Training quiet eye improves accuracy in the basketball free throw. Sport Psychol. 2001;15(3):289–305.
11. Balslev D, Himmelbach M, Karnath HO, Borchers S, Odoj B. Eye proprioception used for visual localization only if in conflict with the oculomotor plan. J Neurosci. 2012;32 (25):8569–73.
12. Hadjidimitrakis K, Breveglieri R, Placenti G, Bosco A, Sabatini SP, Fattori P. Fix your eyes in the space you could reach: neurons in the macaque medial parietal cortex prefer gaze positions in peripersonal space. Plos One. 2011;6(8).
13. Enright JT. The nonvisual impact of eye orientation on eye-hand coordination. Vision Res. 1995;35(11):1611–8.
14. Flanders M, Daghestani L, Berthoz A. Reaching beyond reach. Exp Brain Res. 1999;126 (1):19–30.
15. Sailer U, Eggert T, Ditterich J, Straube A. Spatial and temporal aspects of eye-hand coordination across different tasks. Exp Brain Res. 2000;134(2):163–73.
16. vanDonkelaar P. Eye-hand interactions during goal-directed pointing movements. Neuroreport. 1997;8(9-10):2139–42.
17. van Donkelaar P, Siu KC, Walterschied J. Saccadic output is influenced by limb kinetics during eye-hand coordination. J Motor Behav. 2004;36(3):245–52.
18. Engel KC, Soechting JE. Interactions between ocular motor and manual responses during two-dimensional tracking. Prog Brain Res. 2003;142:141–53.
19. Hollands MA, Marplehorvat DE, Henkes S, Rowan AK. Human eye-movements during visually guided stepping. J Motor Behav. 1995;27(2):155–63.

20. Hollands MA, Marple-Horvat DE. Coordination of eye and leg movements during visually guided stepping. J Motor Behav. 2001;33(2):205–16.
21. Hollands MA, MarpleHorvat DE. Visually guided stepping under conditions of step cycle-related denial of visual information. Exp Brain Res. 1996;109(2):343–56.
22. Matthis JS, Barton SL, Fajen BR. The biomechanics of walking shape the use of visual information during locomotion over complex terrain. J Vision. 2015;15(3).
23. Matthis JS, Fajen BR. Visual control of foot placement when walking over complex terrain. J Exp Psychol Hum Percept Perform. 2014;40(1):106–15.
24. Matthis JS, Fajen BR. Humans exploit the biomechanics of bipedal gait during visually guided walking over complex terrain. Proc Biol Sci. 2013;280(1762):20130700.
25. Young WR, Hollands MA. Evidence for age-related decline in visuomotor function and reactive stepping adjustments. Gait Posture. 2012;36(3):477–81.
26. Reynolds RF, Day BL. Visual guidance of the human foot during a step. J Physiol. 2005;569 (Pt 2):677–84.
27. Rietdyk S, Drifmeyer JE. The rough-terrain problem: accurate foot targeting as a function of visual information regarding target location. J Mot Behav. 2010;42(1):37–48.
28. Patla AE, Vickers JN. How far ahead do we look when required to step on specific locations in the travel path during locomotion? Exp Brain Res. 2003;148(1):133–8.
29. Yamada M, Higuchi T, Mori S, Uemura K, Nagai K, Aoyama T, et al. Maladaptive turning and gaze behavior induces impaired stepping on multiple footfall targets during gait in older individuals who are at high risk of falling. Arch Gerontol Geriat. 2012;54(2):E102–8.
30. Crowdy KA, Hollands MA, Ferguson IT, Marple-Horvat DE. Evidence for interactive locomotor and oculomotor deficits in cerebellar patients during visually guided stepping. Exp Brain Res. 2000;135(4):437–54.
31. Di Fabio RP, Greany JF, Zampieri C. Saccade-stepping interactions revise the motor plan for obstacle avoidance. J Mot Behav. 2003;35(4):383–97.
32. Zampieri C, Di Fabio RP. Balance and eye movement training to improve gait in people with progressive supranuclear palsy: quasi-randomized clinical trial. Phys Ther. 2008;88 (12):1460–73.
33. Crowdy KA, Kaur-Mann D, Cooper HL, Mansfield AG, Offord JL, Marple-Horvat DE. Rehearsal by eye movement improves visuomotor performance in cerebellar patients. Exp Brain Res. 2002;146(2):244–7.
34. Chapman GJ, Hollands MA. Evidence for a link between changes to gaze behaviour and risk of falling in older adults during adaptive locomotion. Gait Posture. 2006;24(3):288–94.
35. Chapman GJ, Hollands MA. Age-related differences in stepping performance during step cycle-related removal of vision. Exp Brain Res. 2006;174(4):613–21.
36. Young WR, Hollands MA. Can telling older adults where to look reduce falls? Evidence for a causal link between inappropriate visual sampling and suboptimal stepping performance. Exp Brain Res. 2010;204(1):103–13.
37. Young WR, Wing AM, Hollands MA. Influences of state anxiety on gaze behavior and stepping accuracy in older adults during adaptive locomotion. J Gerontol B Psychol. 2012;67 (1):43–51.
38. Yogev-Seligmann G, Hausdorff JM, Giladi N. The role of executive function and attention in gait. Mov Disord. 2008;23(3):329–42.
39. Stanley J, Hollands M. A novel video-based paradigm to study the mechanisms underlying age- and falls risk-related differences in gaze behaviour during walking. Ophthalmic Physiol Opt. 2014;34(4):459–69.
40. Yamada M, Higuchi T, Tanaka B, Nagai K, Uemura K, Aoyama T, et al. Measurements of stepping accuracy in a multitarget stepping task as a potential indicator of fall risk in elderly individuals. J Gerontol A Biol. 2011;66(9):994–1000.
41. Mazaheri M, Roerdink M, Bood RJ, Duysens J, Beek PJ, Peper CE. Attentional costs of visually guided walking: effects of age, executive function and stepping-task demands. Gait Posture. 2014;40(1):182–6.

42. Peper CE, Oorthuizen JK, Roerdink M. Attentional demands of cued walking in healthy young and elderly adults. Gait Posture. 2012;36(3):378–82.
43. Lindemann U, Klenk J, Becker C, Moe-Nilssen R. Assessment of adaptive walking performance. Med Eng Phys. 2013;35(2):217–20.
44. Chapman GJ, Hollands MA. Age-related differences in visual sampling requirements during adaptive locomotion. Exp Brain Res. 2010;201(3):467–78.
45. Nonnekes JH, Talelli P, de Niet M, Reynolds RF, Weerdesteyn V, Day BL. Deficits underlying impaired visually triggered step adjustments in mildly affected stroke patients. Neurorehabil Neural Repair. 2010;24(4):393–400.
46. Yamada M, Higuchi T, Nishiguchi S, Yoshimura K, Kajiwara Y, Aoyama T. Multitarget stepping program in combination with a standardized multicomponent exercise program can prevent falls in community-dwelling older adults: a randomized. Controlled Trial J Am Geriatr Soc. 2013;61(10):1669–75.
47. Dowswell T, Towner E, Cryer C, Jarvis S, Edwards P, et al. Accidental falls: fatalities and injuries. An examination of the data sources and review of literature on preventative strategies. UK: Department of Trade and Industry. 1999.
48. Templer J. The staircase: studies of hazards falls and safer design. Cambridge: MIT Press; 1992.
49. Cohen HH, Templer J, Archea J. An analysis of occupational stair accident patterns. J Saf Res. 1985;16:171–81.
50. Svanstrom L. Falls on stairs: an epidemiological accident study. Scand J Soc Med. 1974;2(3):113–20.
51. Cohen HH. A field study of stair descent. Ergon Des. 2000;8:11–5.
52. Archea J. Guidelines for stair safety. Washington: National Bureau of Standards; 1979.
53. Zietz D, Hollands M. Gaze behavior of young and older adults during stair walking. J Motor Behav. 2009;41(4):357–65.
54. Den Otter AR, Hoogwerf M, Van der Woude LH. The role of tread fixations in the visual control of stair walking. Gait Posture. 2011;34(2):169–73.
55. Zietz D, Johannsen L, Hollands M. Stepping characteristics and centre of mass control during stair descent: effects of age, fall risk and visual factors. Gait Posture. 2011;34(2):279–84.
56. Sheldon JH. On the natural history of falls in old age. Br Med J. 1960;2(5214):1685–90.
57. Wild D, Nayak USL, Isaacs B. Description, classification and prevention of falls in old-people at home. Rheumatol Rehabil. 1981;20(3):153–9.
58. Miyasike-daSilva V, Allard F, McIlroy WE. Where do we look when we walk on stairs? Gaze behaviour on stairs, transitions, and handrails. Exp Brain Res. 2011;209(1):73–83.
59. Timmis MA, Bennett SJ, Buckley JG. Visuomotor control of step descent: evidence of specialised role of the lower visual field. Exp Brain Res. 2009;195(2):219–27.
60. Marigold DS, Patla AE. Visual information from the lower visual field is important for walking across multi-surface terrain. Exp Brain Res. 2008;188(1):23–31.
61. Timmis MA, Johnson L, Elliott DB, Buckley JG. Use of single-vision distance spectacles improves landing control during step descent in well-adapted multifocal lens-wearers. Invest Ophthalmol Vis Sci. 2010;51(8):3903–8.
62. Lord SR, Dayhew J, Howland A. Multifocal glasses impair edge-contrast sensitivity and depth perception and increase the risk of falls in older people. J Am Geriatr Soc. 2002;50(11):1760–6.
63. Owsley C. Aging and vision. Vision Res. 2011;51(13):1610–22.
64. Cavanagh PRH JS. What is the role of vision during stair descent? In: Harvey LO, Jeffrey OD, editors. Visual perception: the influence of H W Leibowitz decade of behaviour. Washington: American Psychological Association; 2003. p. 213–42.
65. Buckley JG, Heasley K, Scally A, Elliott DB. The effects of blurring vision on medio-lateral balance during stepping up or down to a new level in the elderly. Gait Posture. 2005;22(2):146–53.

66. Buckley JG, Heasley KJ, Twigg P, Elliott DB. The effects of blurred vision on the mechanics of landing during stepping down by the elderly. Gait Posture. 2005;21(1):65–71.
67. Buckley JG, MacLellan MJ, Tucker MW, Scally AJ, Bennett SJ. Visual guidance of landing behaviour when stepping down to a new level. Exp Brain Res. 2008;184(2):223–32.
68. Hamel KA, Okita N, Higginson JS, Cavanagh PR. Foot clearance during stair descent: effects of age and illumination. Gait Posture. 2005;21(2):135–40.
69. Simoneau GG, Cavanagh PR, Ulbrecht JS, Leibowitz HW, Tyrrell RA. The influence of visual factors on fall-related kinematic variables during stair descent by older women. J Gerontol. 1991;46(6):M188.
70. Johnson L, Buckley JG, Harley C, Elliott DB. Use of single-vision eyeglasses improves stepping precision and safety when elderly habitual multifocal wearers negotiate a raised surface. J Am Geriatr Soc. 2008;56(1):178–80.
71. Johnson L, Buckley JG, Scally AJ, Elliott DB. Multifocal spectacles increase variability in toe clearance and risk of tripping in the elderly. Invest Ophthalmol Vis Sci. 2007;48 (4):1466–71.
72. Menant JC, St George RJ, Sandery B, Fitzpatrick RC, Lord SR. Older people contact more obstacles when wearing multifocal glasses and performing a secondary visual task. J Am Geriatr Soc. 2009;57(10):1833–8.
73. Elliott DB, Vale A, Whitaker D, Buckley JG. Does my step look big in this? A visual illusion leads to safer stepping behaviour. Plos One. 2009;4(2).
74. Shumway-Cook A, Patla AE, Stewart A, Ferrucci L, Ciol MA, Guralnik JM. Environmental demands associated with community mobility in older adults with and without mobility disabilities. Phys Ther. 2002;82(7):670–81.
75. Galna B, Peters A, Murphy AT, Morris ME. Obstacle crossing deficits in older adults: a systematic review. Gait Posture. 2009;30(3):270–5.
76. Patla AE. Understanding the roles of vision in the control of human locomotion. Gait Posture. 1997;5(1):54–69.
77. Patla AE, Vickers JN. Where and when do we look as we approach and step over an obstacle in the travel path? Neuroreport. 1997;8(17):3661–5.
78. Rietdyk S, Rhea CK. The effect of the visual characteristics of obstacles on risk of tripping and gait parameters during locomotion. Ophthalmic Physiol Opt. 2011;31(3):302–10.
79. Pontecorvo SM, Heijnen MJH, Muir BC, Rietdyk S. Relationship between gaze behavior and failure to cross a stationary, visible obstacle. 2015 International Society for Posture and Gait Research World Congress; Seville, Spain. 2015.
80. Heijnen MJH, Muir BC, Rietdyk S. Factors leading to obstacle contact during adaptive locomotion. Exp Brain Res. 2012;223(2):219–31.
81. Lowrey CR, Watson A, Vallis LA. Age-related changes in avoidance strategies when negotiating single and multiple obstacles. Exp Brain Res. 2007;182(3):289–99.
82. Patla AE. How is human gait controlled by vision. Ecol Psychol. 1998;10(3-4):287–302.
83. Rietdyk S, Rhea CK. Control of adaptive locomotion: effect of visual obstruction and visual cues in the environment. Exp Brain Res. 2006;169(2):272–8.
84. Rhea CK, Rietdyk S. Visual exteroceptive information provided during obstacle crossing did not modify the lower limb trajectory. Neurosci Lett. 2007;418(1):60–5.
85. Yamada M, Tanaka H, Mori S, Nagai K, Uemura K, Tanaka B, et al. Fallers choose an early transfer gaze strategy during obstacle avoidance in dual-task condition. Aging Clin Exp Res. 2011;23(4):316–9.
86. Muir B, Haddad J, Heijnen M, Rietdyk S. Proactive gait strategies to mitigate risk of obstacle contact are more prevalent with advancing age. Gait Posture. 2015;41(1):233–9.
87. Menant JC, St George RJ, Fitzpatrick RC, Lord SR. Impaired depth perception and restricted pitch head movement increase obstacle contacts when dual-tasking in older people. J Gerontol A Biol Sci Med Sci. 2010;65(7):751–7.
88. Chen HC, Ashton-Miller JA, Alexander NB, Schultz AB. Stepping over obstacles: gait patterns of healthy young and old adults. J Gerontol. 1991;46(6):M196–203.

89. Winter DA. Biomechanics and motor control of human gait: normal, elderly and pathological. 2nd ed. Waterloo: University of Waterloo Press; 1991.
90. Chou LS, Draganich LF. Placing the trailing foot closer to an obstacle reduces flexion of the hip, knee, and ankle to increase the risk of tripping. J Biomech. 1998;31(8):685–91.
91. Berard JR, Vallis LA. Characteristics of single and double obstacle avoidance strategies: a comparison between adults and children. Exp Brain Res. 2006;175(1):21–31.
92. Rhea CK, Rietdyk S. Influence of an unexpected perturbation on adaptive gait behavior. Gait Posture. 2011;34(3):439–41.
93. Mohagheghi AA, Moraes R, Patla AE. The effects of distant and on-line visual information on the control of approach phase and step over an obstacle during locomotion. Exp Brain Res. 2004;155(4):459–68.
94. Patla AE, Greig M. Any way you look at it, successful obstacle negotiation needs visually guided on-line foot placement regulation during the approach phase. Neurosci Lett. 2006;397 (1-2):110–4.
95. Heijnen MJH, Rietdyk S, editors. Falls in the real world are related to obstacle crossing behaviors in a lab setting for young adults. 2015 International Society for Posture and Gait Research World Congress; Seville, Spain. 2015.
96. Lu T-W, Chen H-L, Chen S-C. Comparisons of the lower limb kinematics between young and older adults when crossing obstacles of different heights. Gait Posture. 2006;23(4):471–9.
97. McFadyen BJ, Prince F. Avoidance and accommodation of surface height changes by healthy, community-dwelling, young, and elderly men. J Gerontol A Biol Sci Med Sci. 2002;57(4):B166–74.
98. Soma M, Masuda T, Shimamura R, Abiko T, Uematu H, Kawama K. Influence of a dual-task on toe clearance of the young and elderly while stepping over an obstacle. J Phys Ther Sci. 2010;22(1):75–9.
99. Barbieri FA, dos Santos PCR, Vitório R, van Dieën JH, Gobbi LTB. Effect of muscle fatigue and physical activity level in motor control of the gait of young adults. Gait Posture. 2013;38 (4):702–7.
100. Vitorio R, Lirani-Silva E, Barbieri FA, Raile V, Stella F, Gobbi LTB. Influence of visual feedback sampling on obstacle crossing behavior in people with Parkinson's disease. Gait Posture. 2013;38(2):330–4.

6 The Walking Ability in Healthy Older Adults: The Role of Aging and Physical Activity and Its Interface with Agility, Balance, Cognition, and Risk of Falls

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Abstract

Walking is the most basic and common means of locomotion. Walking requires integration and good levels of different physical and mental components and processes. A number of factors, including an inverse relationship with aging and the level of physical activity, can modulate the level of functionality of these components. In addition, the presence of chronic conditions such as sarcopenia, cognitive deficits, and specific changes mainly in frontal brain structures associated with the aging process may affect an individual's walking pattern. Moreover, walking performance demands integration with the environment and requires reasonable levels of balance, agility, and cognitive skills. Furthermore,

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walking demands performance in a multiple-task manner which is affected during the aging process due to impaired sensorimotor integration. Impairment in walking pattern along with reduced levels of agility and balance leads to an increased risk of falls. Despite the negative effects of aging on the human organism, regular exercise has been recommended as a means of reducing the rate of decline and maintaining functionality in older adults. Acknowledging the role that aging, physical activity, agility, balance, and cognitive functioning play in walking and also in the risk of falls, this chapter (i) describes how aging and physical activity interfere with walking pattern (stride length, speed, variability), agility, balance, and cognitive functioning and (ii) describes how these components contribute to either increasing or reducing the risk of falls in older adults. To accomplish these goals, scientific literature was reviewed, analyzed, and summarized.

Keywords
Ambulation • Cognition • Elderly • Falls • Falls risk • Locomotion • Mobility • Older adults • Physical fitness • Physical activity

6.1 Introduction

The aging process is often defined and characterized by progressive and irreversible changes in biologic, morphologic, and functional parameters that often result in the decline of performing physical abilities [1]. Although physical changes are more visible in the human body over time, the impact of time on mental processes (cognition) is also experienced and includes negative consequences late in life. The degree of decline in those domains negatively affects one's ability to perform simple or more complex activities of daily living. It is known that an active lifestyle through regular exercise is important in reducing the rate of decline in different human functional systems and in maintaining good levels of functional capacity, thus extending lifetime independence and autonomy in older adults. Population aging is a phenomenon being experienced by almost all countries in the world. However, this human success comes with several challenges to society. The aging process associated with an inactive lifestyle has led to accelerated morphological and functional changes that negatively impact independence and autonomy in older individuals and consequently the quality of life by drastically reducing functional capacity. Functional capacity is defined as the ability to perform activities of daily living in a safe and effective fashion [2]. Functional capacity is composed of a set of components such as strength, flexibility, agility, and dynamic balance and coordination that are highly required, for example, during walking tasks.

Walking is the most basic and common means of locomotion. Erect bipedal walking is not a luxury of modern human beings and in fact it is not a recent phenomenon. The species *Australopithecus afarensis*, our human ancestor, was

able to walk in an upright position in a pattern similar to ours approximately 3.7 million years ago [3]. It is thought that a bipedal walking position offered several advantages and benefits to that species and undoubtedly is highly important to *Homo sapiens sapiens* in order to succeed in their sociocultural environment once standing and walking became inherent to most human daily activities. However, walking is not as simple as it appears, and certainly the ability to walk becomes more challenging late in life. Arguably, the reasons for this could be divided in two main aspects: (1) age-related decline in functional and mental capacity components involved in the walking task and (2) lack of regular exercise. These reasons will be better explained throughout this chapter.

Generally speaking, walking requires not only good levels of certain functional capacity components but also good levels of cognition [4, 5]. A number of factors, including an inverse relationship with aging and physical activity level, can certainly modulate those fitness levels. Furthermore, the presence of chronic conditions such as sarcopenia, cognitive deficits, and specific changes mainly in the frontal brain structure associated with the aging process may affect an individual's walking pattern [6, 7]. As a means of locomotion, it is important to keep in mind that walking does not present a single movement pattern. The reasons for this lie in the fact that the way individuals walk demands integration with the environment and requires reasonable levels of agility, balance, and cognitive skills to be performed adequately. Additionally, walking demands performance in a multitask fashion, which is affected during the aging process due to impaired sensorimotor integration [7]. Impairment in walking pattern along with reduced levels of agility and balance and declines in mental processes has been associated with increased risk of falls [8, 9]. Falling is a public health concern because it is a major threat to health and independence of older adults. Falls in older adults normally result in serious injuries most often requiring hospitalization. Falls are not an inevitable consequence of aging, but they are more common in older adults because of risk factors, such as a lack of exercise or medication. They increase with age and are usually related to poor health and aging conditions [10].

Acknowledging the role of aging; the lack of exercise; important components of physical and mental capacity, like agility; and balance and cognitive functioning all having a part in walking as well as in the risk of falls, this chapter:

1. Describes how aging and physical activity interfere with walking pattern, agility, balance, and cognitive functioning. In terms of walking pattern, stride length, speed, and variability are explored.
2. Describes how all the abovementioned components modulate locomotion (walking) and contribute to either increased or reduced risk of falls in older adults.

To accomplish these goals, the current scientific literature was reviewed, analyzed, and summarized. This chapter is organized into four distinct but interconnected subsections: (1) agility and aging, (2) gait and cognitive function, (3) exercise and the walking task, and (4) mechanisms of postural control during aging. Therefore, by the end of this chapter, the reader will be able to understand

how aging affects walking as well as the complex relationship between physical (with emphasis on agility) and mental components that make walking possible in older adult and how regular exercise may help in the walking task and reduce the risk of falls.

6.2 Agility and Aging: Parameters to Perform a Motor Intervention

Agility is defined as the ability to perform rapid movements with a good degree of precision at high intensity, employing direction changes or fluctuations in the center of gravity and in situations requiring acceleration and deceleration [11]. Among the functional capacity components related to aging, less attention has been given to the impact of agility. This could be due to the fact that this component is often viewed as a performance indicator. However, considering the activities of daily living (ADL) requiring locomotion, several unpredictable daily actions demand movements involving speed, quick direction change, and adjustments in the center of gravity (e.g., detour from an object or another person, overcome an obstacle during gait). To this end, agility is highly important. For instance, persons with good levels of agility walk with more self-assurance and confidence [12] and respond better to the demands of the surround environment [13]. Therefore, neglecting the development of this component may negatively affect one's functionality and autonomy. It could have a potential impact on increasing the risk of falls, which often results in fractures and greater risk of morbidities. The development of agility during the lifespan is necessary in order to keep it at good levels during adulthood and senescence.

The aging process is characterized by progressive and irreversible changes in biologic, morphologic, and functional parameters. These changes often result in a decline in the performance of physical abilities [1] which could negatively affect the execution of ADL. In general older adults are the least active age group to engage in regular physical activity programs [14]. Several epidemiologic studies have demonstrated that living a sedentary lifestyle intensifies the process of incapacity and dependence resulting from the normal degeneration of the aging process. Moreover, a sedentary lifestyle increases the risk of chronic diseases which contribute to autonomy loss and consequently diminished quality of life in this population [15].

The set of functional capacity components ensures independence and autonomy during aging. Agility is a complex component that depends upon the integration of the visual, vestibular, and proprioceptive systems as well as the processing centers. Additionally, it also depends upon the centers for central superior information and neuromuscular responses, mainly muscle strength and reaction time. Among the most important losses experienced by older adults, postural instability which occurs due to changes in sensorial and motor systems is highlighted [16]. These changes result in a reduction in the ability to perform self-shifting during information processing, slowing down the execution of tasks that meet the demands of the

environment. A reduction in the sensorial system efficacy, especially visual acuity as well as proprioceptive changes and cognitive deficits that harm the integration and information processing which resulted from aging, often has a negative impact on agility performance and mobility. Additionally, deficits in neuromuscular function originating from lean mass loss during aging (sarcopenia) also contribute to intensifying the responses of this dysfunction.

Falls in older adults are a major mobility impairment factor [17]. Falls are commonly accompanied by fractures, hospitalization, and an increased risk of death in this population. However, engagement in regular physical activity has been recommended as an efficient and effective preventive mean to reduce risk of falls [18], and falls are one of the main causes of mortality in older adults [19]. Therefore, maintaining good levels of physical fitness has proven a significant association with the reduction of frail phenotypes in advanced ages.

Initially, the use of instruments/tools able to identify and determine the prevalence of falls, functional deficits, and incapacities positively related to progressive loss of agility and balance may become potential predictors of functional impairment, as well as a guide for the development of appropriate strategies to minimize the effects of aging on this functional component. Although agility is a specific component, it depends upon the integrity of other components, such as strength, speed, flexibility, and, mainly, balance. Taking into account the progressive loss of all these components during aging, especially those related to the reduction in the number of rapid muscle contraction fibers and reduction in the number of motor units, studies have demonstrated the efficacy of resistance exercise training as an efficient alternative to promote improvements in agility [20, 21]. This type of training is associated with improvement in movement precision that demands agility by increased recruitment of motor units, which consequently improve the execution of movements that require speed and accuracy.

Multimodal programs require individuals to perform activities that involve movements that stimulate multiple components of functional capacity. Several studies have demonstrated that multimodal programs also promote positive changes in functional mobility and agility indexes [22]. Because of the interdependent relationship between agility and other components, these types of interventions have been shown to be effective. Intervention of this nature should be developed with the aim of optimizing information processing and stimulating the superior centers. This could be done by using challenging activities that involve performing dual tasks [23]—a task requiring one to perform a primary task, concurrent to a secondary task, normally cognitive task (e.g., standing upright while counting backward), especially those mimicking daily activities. In addition, interventions that promote significant improved flexibility have the potential to make it easier for individuals to perform movements with higher amplitude and better efficacy. This will optimize the lever systems required to perform high-speed movements and facilitate the capacity of muscle contraction and relaxation of main motor muscles involved in daily actions.

Within exercise programs for older adults aiming to improve agility, it is important to consider the development of coordination, which will allow better speed response of information processing and efficiency in the self-shifting system.

This will promote positive reflexes for performing activities quickly. It is also important to highlight the development of muscular strength to intensify the recruitment and synchronization of new motor units as well as neural adaptations to provide fast drive impulse of afferent and efferent pathways. This will ensure performance with greater safety and effectiveness while activities requiring agility are executed.

The evaluation of agility performance related to walking of older adults should take into consideration the full body, as full body is engaged during walking. The evaluation should further consider the multifactorial changes in lifestyle behaviors (e.g., reduced physical activity) common during the aging process that negatively impact agility and balance. It is parallel important to considering many of the risk factors as potential intervention targets. Mainly, such tests should ensure the safety of the individual during assessment procedures because they require some degree of speed. The agility and dynamic balance test, part of the battery of functional tests of the American Alliance for Health, Physical Education, Recreation and Dance (AAHPERD) [24], offers a good parameter to assess this component by requiring functional movements present in daily situations of older people. It allows researchers and health professionals to quantify this component. The test involves locomotion, sitting, standing, and making turns and consists of starting from a sitting position, and when signaled by the evaluator, the individual should cover a preestablished route as quickly as possible.

Along with neuromuscular and metabolic changes, locomotor system alterations are also observed during the aging process. Taken together, these changes cause impairment to the functions involved ambulation and mobility. Moreover, they are often related to the risk of falls. Interventions designed to develop maximum speed can be a risk for older adults because of the high demand imposed on the structures involved in the execution of these types of activities. Therefore, developing protocols aimed at stimulating multiple components may have a positive impact on specific abilities and is being recognized as a safe and efficient alternative to improve the relationship between older individuals and their socio-environmental context. It is expected that these programs will not provide improvements related to high levels of performance but rather improvements related to functionality and mobility of the participants. Engaging in exercise has been recommended as a means to achieve better functionality that makes individuals abler and adapted to facing challenging and unpredictable situations posed by daily activities, including safer locomotion.

6.3 Gait and Cognitive Function

The understanding of the association between mobility/locomotion and cognition is evolving. In the past, it was believed that walking was an entirely automatic process. However, gait control requires a well-organized integration of the motor, sensorial, and cognitive systems. It is known that gait is mediated by frontal subcortical circuits, and those are responsible for mediating executive functions

[25, 26]. Such a function comprises planning, abstraction, strategy development, organization, self-control, and mental flexibility [27].

Several studies have shown that gait can be affected by cognitive tasks. This interaction between gait and cognition can be proven by the dual-task paradigm. In the dual-task paradigm, performance of motor and cognitive tasks is assessed simultaneously (e.g., having people walk while performing a task that requires cognitive activation) [28, 29]. Furthermore, studies have established the importance of cognitive control for the older adult gait. Executive functions and attention are altered in the normal aging process, and this cognitive deficit may affect the gait execution either in dual-task conditions or in situations where there are environmental changes [26, 30]. A reduction in the gait speed and increased step variability are observed in older adults who are asked to walk and perform a cognitive task simultaneously [31]. Gait variability is found to be higher in older adults possibly because of deficits in both executive functions and attention [32]. Investigations that have been conducted using dual-task methodology have focused on cinematic parameters of the gait such as stride length, step time, and speed and gait variability. These studies reported alterations in gait parameters in older adults [33, 34].

According to Yogev-Seligmann and colleagues [9], some domains of the executive functions such as mental flexibility, behavior self-control, and planning have important function in gait performance. For instance, mental flexibility allows individuals to detect errors and modify motor behavior, if necessary. Behavior self-control allows individuals to bypass irrelevant stimulus and leach distractions. The planning domain allows individuals to identify and organize elements needed to perform an intention. A deficit in the previous cognitive domains may engender risky choices and failures in decision-making. A simple example of this is when subtle changes occur in the walking surface, and the individual is not able to adopt an efficient strategy and ends up slipping or stumbling because of the obstacle. The activities of daily living encompass several situations where individuals perform motor and cognitive tasks simultaneously, such as walking and talking, walking and thinking, and walking and listening to music. Besides these dual-task situations, other conditions require cognitive demands. These include but are not limited to walking on slippery surfaces, performing a new motor skill, walking and overcoming obstacles, or walking and facing diminishing light in the environment. In the last situations, cognitive demand is higher, which means that gait requires a higher level of the executive functions and attention [35].

Gait disturbance, such as decreased walking speed, reduced stride length, and increased gait variability, results in increased risk of falls in older adults [36]. On the other hand, more recent studies consider that cognitive decline and falls related to the aging process are two geriatric syndromes connected by a common neural substratum [37]. Falls have been associated to aging-related changes in the prefrontal cortex [38]. Therefore, older adults with better performance in executive functions present lower risk of falls. Even a subtle executive dysfunction may impair the capability of planning and organizing the thinking for gait stages. Thus, older adults facing either situation of dual tasks or environmental demands

have difficulties to perform an efficient gait and to take appropriate precautions. This results in an increased risk of falls [39].

A study conducted in a population-based sample of older adults (n = 658) found significant associations between falls and cognitive measurements (i.e., executive functions, reaction time, work memory, speed processing, and attention) [40]. In all examined variables, the fallers had the worse cognitive performance. The findings of this study suggest that the falls which occurred late in life may be associated with age-related changes in the prefrontal cortex and consequently failure in executive control. On the other hand, the recurrent falls may be a result of marked aging of the cerebral cortex and general cognitive decline [40]. In this sense, the risk of falls is increased in cognitive disorders, such as mild cognitive impairment and in Parkinson's and Alzheimer's diseases.

Several studies have demonstrated that physical exercise has an effect on gait of older people. A recent systematic literature review demonstrated that physical exercise can improve single-task gait speed and dual-task gait speed in older adults [41]. The nature of the physical exercise interventions varied across the studies in the previous systematic review, but most of the studies included interventions with dual-task component. Cognitive activities used in the dual-task interventions included naming words in a particular category, spelling backward, counting forward or backward, and reciting letters of the alphabet among others. There is further evidence that multimodal intervention also reduces variability of spatiotemporal gait parameters during dual-task walking in older adults [42, 43].

Based on the above, we noted that both attention and executive functions play a decisive role in gait regulation in older people, especially in complex and challenging conditions. Moreover, we noted that deficits in these cognitive components might contribute to changes in the gait and therefore an increased risk of falls in this population.

6.4 Exercises to Help Older Adults Master Walking Ability in Different Environments

Maintaining reasonable levels of physical function is important for older adults in order to remain independent and autonomous. With this in mind, being physically active has proven to be beneficial [44]. To be independent it is highly important that mobility be preserved. Mobility is defined not only as the capacity of one to walk and move, but it also takes into consideration the environment and the individual capability to adapt to it [45]. Lifestyle-related factors and/or pathological or even idiopathic factors that may come along with the aging process may decrease one's physical function levels, affecting mobility engendering what is called functional incapacity. Functional incapacity can be defined as the difference between the physical function of an individual and environmental challenges to which he/she is subjected [46]. An example of environmental challenge could be as simple as walking outdoors on a flat surface [46]. Functional incapacity ranges from preclinical to severe. In the preclinical range, there are limitations only in highly

challenging environments, whereas in the severe range, individuals cannot get out of bed [47].

Many risk factors for reduced mobility have been investigated; however none can accurately predict mobility limitations. Like other geriatric syndromes, the determinants are probably multifactorial [48]. The risk factors often identified as related to low-mobility capacity include but are not limited to advanced age, low level of physical activity, obesity, balance impairments, and chronic diseases (e.g., diabetes, arthritis, etc.) [49]. Depressive symptoms or cognitive impairment, being female, history of hospitalization, tobacco and alcohol consumption, and Parkinson's disease were also found to be related to low-mobility capacity [50].

Regarding low levels of physical activity, interventions employing physical exercise have the potential to improve mobility in older adults. A relative new intervention activity that could be highlighted in this matter is the *square-stepping exercise* (SSE). This intervention was developed in Japan [51] with the main purpose being the promotion of balance and reducing the risk of falls in older adults. To achieve these goals, different step sequences were developed to be performed on a gridded mat (1 m wide by 2 m long). The sequences increased progressively in terms of complexity and number of steps needed to be completed [51]. The first findings from studies using this intervention were promising. The studies showed benefits in different functional capacity components highly required during a walking task such as balance, agility, and reduced risk of falls [52, 53]. Moreover, studies conducted in Brazil observed that the SSE intervention required refined cognitive components from which concentrated attention, memory, and executive functions can be highlighted [54]. Like the physical components, these cognitive components are also activated during walking.

In terms of mobility and in line with the above, studies have shown that SSE intervention was able to improve balance and walking ability in community dwellings and with frail older adults living in a day care center for older adults [55, 56]. The groups undertaking the SSE intervention improved in both time and amount of steps required to complete the task (*Timed Up and Go Test)*, which assessed balance and walking ability. In this sense, interventions using walking may also be an interesting option for mobility maintenance. For instance, a study using *Nordic Walking* as intervention observed that mobility and other functional capacity components such as strength endurance and balance had improved at the end of the program [57]. Li et al. [58] used two different types of training in older adults: (a) traditional walking and (b) walking on a cobblestone mat (mats with hard plastic replicas of stones that require more balance and attention for foot positioning). A 16-week intervention period was enough to observe improvements in mobility in both groups. However, the traditional walking training group perceived more benefits in terms of daily functional measurements.

On the other hand, interventions using weight training had also proven to be beneficial to mobility. Vincent et al. [59] compared the effects of high- and low-intensity weight training for mobility in older adults aged 63 to 80 years old. The authors observed that weight training was beneficial for mobility mainly in those participating in the low-intensity group. Difficulties in mobility as well as in

activities of daily living such as traveling, self-care, and moving in public places are especially difficult for obese older adults [60]. Therefore, interventions aiming to reduce weight are important tools for recovering mobility and improve other physiologic functions. Low-intensity aerobic activities have been recommended for these cases [61]. Thus, multidisciplinary approaches are needed to mitigate the impact of obesity in the deficiency of mobility in aging [60].

In general, regular exercise has been shown to be extremely beneficial to mobility maintenance. Furthermore, as mentioned by Ross, Schmidt, and Ball [62], other interventions such as cognitive training and educational approaches can also help. Roenker et al. [63] showed beneficial effects of a computer-based cognitive intervention in individuals at risk of mobility decline and [64] showed reduction on perceived walking difficulty due to educational orientation on physical activity. This suggests both physical and cognitive components can be the focus of interventions aiming to improve mobility in older adults.

6.5 Understanding the Mechanisms of Postural Control During the Aging Process: From Assessment to Professional Intervention

Among the wide range of changes occurring during the aging process that impact locomotion of older adults, it is highly important to understand mechanisms of postural control that contribute to gait initiation. Gait initiation is challenging to the motor control system because it is a volitional transition from a condition of a static stable support to a continuously unstable posture during locomotion [65]. Because of this, gait initiation has been used as an investigative tool to provide insight into postural control and the changes that occur with advancing age and disability [66].

An adequate postural control is able to promote innumerous motor skills and is an absolute prerequisite to safety and efficient performing activities of daily living. Postural control is defined in this chapter as the ability to maintain, achieve, or restore a state of balance during any posture or performing activity [67]. Horak [68] states that postural control is a complex ability based on a dynamic interaction of the sensorimotor process that involves several components. Among those components it is important to highlight a) biodynamics (range of motion, strength, and stability limit), b) sensory (integration and response), c) space orientation (perception, gravity, surface, and vision), d) strategic movements (reaction, anticipation, and volitional movements), and e) cognitive processing (attention and learning). The aging process has a negative impact on postural control either because of a specific pathology that affects a certain component of the sensory, biodynamic, and cognitive processing systems or as a result of age-related deterioration generally of the mechanisms of sensory and biodynamic control [69, 70].

The balance control during operation and during the change of a postural set to another requires a complex motor control of a center of mass (CM) in motion.

The center of mass of a body is the point where all its mass is concentrated. Unlike the static support during operation or during the transition from one set to another posture, a healthy individual, the CM is not within the support base [71]. The postural stability during operation results from the placement member for swing phase under the CM when the latter moves in the lower direction. However, lateral stability derives from the combination of the lateral trunk control and lateral foot placement [72]. Elderly at risk of falling tend to have higher CM side than normal and lateral placements of more irregular walking tours [73].

So for a better understanding of the relationship of postural control and gait, the postural control is usually studied by observing behavior, essentially body oscillation during a given task [74]. Performing an additional task while maintaining an upright posture may cause prejudice to posture, requiring appropriate adjustments and integration of the postural control components to avoid falls. Attention tends to be divided between postural control and cognitive processing, using dual task. In this case, the primary task is the upright posture, concurrent to a secondary task. Individuals are instructed to perform two different tasks at the same time using the best of their abilities. It is assumed that the attention priority is divided between the tasks. This requires a competition of processing resources [75]. Several studies observed that even in young individuals, postural control may be negatively affected during the execution of two tasks performed simultaneously. This puts a great load on working memory capacity [76, 77]. The age-related losses of the visual and proprioceptive sensibility and of the vestibular system require greater attention to maintain postural stability during the standing/upright position [78, 79]. In this sense, the responses to dual task could destabilize the motor activity [80, 81]. Moreover, the aging process is not only associated with motion dynamic affected by impairment but also may be associated with a disorientation of the cognitive processes. Thus, many reasons related to a reduction of postural control during the execution of a concurrent task can be identified. One of them is related to the fact that there is a higher necessity of conscious attention to maintain good postural control because of sensorial function and biodynamic system deficits. These reflect in reduced attention and worse performance.

Studies have shown that older adults presenting cognitive impairment inherent of a pathological condition such as Alzheimer's disease have lower performance on postural control when executing concurrent tasks compared to neurological healthy older adults [82, 83]. In line with this finding, older adults with motor impairment characteristic of a pathologic process such as Parkinson's disease perform worse on postural control compared to older adults without any neurodegenerative condition [84]. A possible explanation may be related to the fact that individuals without cognitive impairment may use cerebral mechanisms that compensate for minor changes in one or more components of postural control [85].

A common means of assessing postural control is through posturography. Assessed through a force platform, the static posturography allows quantifying the oscillation and balance and consequently predicting an increased risk of falls in the older adult population [86]. The force platform provides data on corporal

oscillation through the center of pressure (COP). COP data refers to two coordinates on the platform surface that are identified in relation to the individual orientation: anterior-posterior (AP) and mediolateral (ML) directions [23]. In fact, research has been conducted employing this measure during quiet stance to detect changes in gait and intervention outcomes [87].

In recent years, many interventions have been conducted aiming to minimize the risk of falls and to reduce fall rates. Intervention programs that include strength, balance, gait training, shoe adaptation, assessment and use of assistive devices, medication assessment, visual tests, and information about falls have been incorporated into clinical practices [88, 89]. Within these interventions it is important to highlight the fall prevention training program for older adults living in the community which includes balance, muscle strength, flexibility, and resistance exercises [90].

A positive effect on fall rates was observed in programs including a combination of a high volume of exercise (higher than 50 hours throughout the intervention period) and more challenging balance exercises [91]. Interventions adopting a bolder character in older adults such as the use of *exergames* (exercises plus games) have shown to be effective in improving balance in older adults [92]. Other interventions such as *tae kwon do* also have been shown to be beneficial in improving balance in older adults [93]. Researchers attribute such improvements to the dynamic movements present in these activities that contribute to reduce the rate of functional decline in older adults. The *Tai Chi* improved the mechanism by which forward momentum is generated and improved coordination during gait initiation, suggesting improvements in postural control [87].

A systematic review showed that there is weak evidence that some types of exercise (walking, balance, coordination and functional tasks, strengthening exercise, 3D exercise, and various types of exercises) are moderately effective immediately post action, improvement of clinical equilibrium results in older people [94]. There is limited evidence to draw conclusions for the general physical activity (walking or cycling) and exercises involving balance programs between computers or vibration plates. The paucity of solid evidence underscores the need for more research employing high methodological quality, and using core measures and adequate monitoring is required.

In the same way, another systematic review showed that interventions involving repetitive task-specific practice and/or auditory cueing appeared to be the most promising approaches to restore gait coordination. Because overall improvements in gait coordination coincided with increased walking speed lends support to the hypothesis cue targeting gait coordination may be the way of improving overall walking ability, especially in individuals poststroke [95].

Therefore, enrollment in physical exercise on a regular basis combined with multicomponent interventions that involve several physical components of functional capacity, especially balance, muscle strength, flexibility, and endurance may effectively contribute to postural control (i.e., stand and during walking) in older adults ensuring the execution of activities of daily living with greater safety and efficiency.

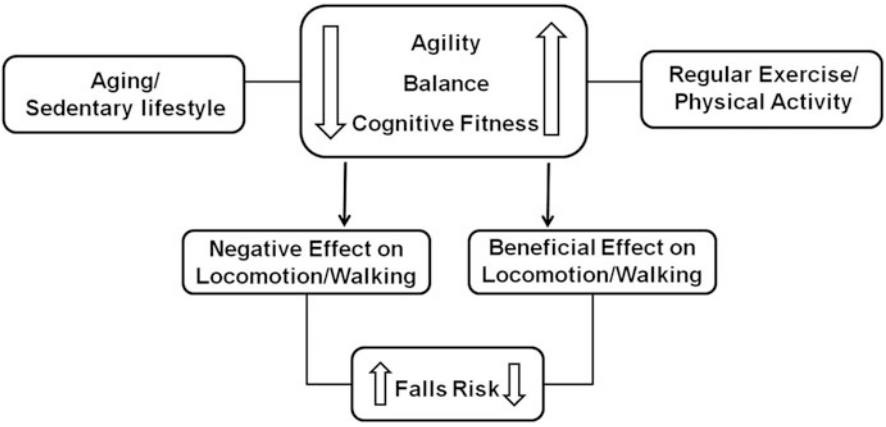

Fig. 6.1 Antagonistic modulation between aging/sedentary lifestyle (*left*) and physical exercise (*right*)

6.6 Final Considerations

This chapter described how aging and physical activity interfere with walking ability. Specifically, this chapter explored how physical and cognitive components required during walking such as agility, balance, and cognitive functioning change with aging and how these components are affected by regular exercise. Furthermore, this chapter described how the above components contribute to either increase or diminish the risk of falls in older adults and how regular exercise may be beneficial to this population. The following Fig. 6.1 summarizes the chapter content as a schematic model on how aging and exercise differently modulate the variables examined.

Considering Fig. 6.1 above, intervention programs focused on improving walking abilities in older adults should take into consideration not only physical but also cognitive components that are also involved and important to walking ability and in preventing falls in the older adult population. In closing, although the aging process poses several challenges to the organism that can significantly result in dependent-related walking in older adults, regular exercise has been shown to be extremely beneficial to mobility maintenance in older adults and to reducing the risk of falls.

References

1. Mazo GZ, Benedetti TB, Sacomori C. Association between participation in community groups and being more physically active among older adults from Florianopolis. Brazil Clin. 2011;66 (11):1861–6.
2. Clark BA. Testes for fitness in older adults – AAHPERD Fitness task force. JOPERD. 1989;60 (3):66–71.

3. Crompton RH, Pataky TC, Savage R, D'Août K, Bennet MR, Day MH, et al. Human-like external function of the foot, and fully upright gait, confirmed in the 3.66 Mya-old Laetoli hominin footprints by topographic statistics, experimental footprint-formation and computer simulation. J R Soc Interface. 2012;9(69):707–19.
4. Simões LA, Dias JMD, Marinho KC, Pinto CLLR, Britto RR. Relationship between functional capacity assessed by walking test and respiratory and lower limb muscle function in community dwelling elders. Rev Bras Fisioter. 2010;14(1):24–30.
5. Lowry KA, Brach JS, Nebes RD, Studenski SA, van Swearingen JM. Contributions of cognitive function to straight- and curved-path walking in older adults. Arch Phys Med Rehabil. 2012;93:802–7.
6. Viana JU, Silva SLA, Torres JL, Dias JMD, Pereira LSM, Dias RC. Influence of *sarcopenia* and functionality *indicators* on the frailty profile of community-dwelling elderly subjects: a cross-sectional study. Brazil J Phys Ther. 2013;17(4):373–81.
7. Beurskens R, Bock O. Age-related deficits of dual-task walking: a review. Neural Plast. 2012; Article ID 131608: 9 p.
8. Bird M-L, Pittaway JK, Cuisick I, Rattray M, Ahuja KDK. Age-related changes in physical fall risk factors: results from a 3 year follow-up of community dwelling older adults in Tasmania, Australia. Int J Environ Res Public Health. 2013;10(11):5989–97.
9. Yogev-Seligmann G, Hausdorff JM, Giladi N. The role of executive function and attention in gait. Mov Disord. 2008;23(3):329–42.
10. National Center for Injury Prevention and Control. Preventing falls: how to develop community-based fall prevention programs for older adults. Atlanta: Centers for Disease Control and Prevention; 2008.
11. Gobbi S, Villar R, Zago AS. Theoretical and practical bases of fitness [Portuguese]. 1st ed. São Paulo: Guanabara Koogan; 2005.
12. Hernandes ESC, Barros JF. Effects of a program of physical and educational activities for elderly people under the performance in daily activities tests. R Bras Ci e Mov. 2004;12 (2):43–50.
13. Miyasike-da-Silva V, Villar R, Zago AS, Polastri PF, Gobbi S. Agility levels in 42 to 73 years old people: effect of a generalized physical activity program of moderate intensity. Rev Bras Cienc Esporte. 2002;23(3):65–79.
14. Nascimento CMC, Gobbi S, Hirayama MS, Brazão MC. Level of physical activity and main barriers perceived by older people living in Rio Claro. Rev Educ Fís/UEM. 2008;19 (1):109–18.
15. Christensen U, Stovring N, Schultz-Larsen K, Schroll M, Avlund K. Functional ability at age 75: is there an impact of physical inactivity from middle age to early old age? Scand J Med Sci Sports. 2006;16(4):245–51.
16. Jorstad EC, Hauer K, Becker C, Lamb SE. Measuring the psychological outcomes of falling: a systematic review. J Am Geriatr Soc. 2005;53(3):501–10.
17. Gama ZA, Gomez-Conesa A. Risk factors for falls in the elderly: systematic review. Rev Saude Publica. 2008;42(5):946–56.
18. Pizzigalli L, Filippini A, Ahmaidi S, Jullien H, Rainoldi A. Prevention of falling risk in elderly people: the relevance of muscular strength and symmetry of lower limbs in postural stability. J Strength Cond Res. 2011;25(2):567–74.
19. Bucksch J. Physical activity of moderate intensity in leisure time and the risk of all cause mortality. Br J Sports Med. 2005;39(9):632–8.
20. Ferreira L, Gobbi S. General agility and upper limbs agility in trained and untrained third-aged women [Portuguese]. Rev Bras Cineantropom Desempenho Hum. 2003;5(1):46–53.
21. Silva AD, Almeida GJ, Cassilhas RC, Cohen M, Peccin MS, Tufik S, et al. Balance, coordination and agility of older individuals submitted to physical resisted exercises practice [Portuguese]. Rev Bras Med Esporte. 2008;14(2):88–93.
22. Nóbrega ACLD, Freitas EVD, Oliveira MABD, Leitão MB, Lazzoli JK, Nahas RM, et al. Official position of the Brazilian Society of Sports Medicine and the Brazilian Society of

Geriatrics and Gerontology: physical activity and health in older people [Portuguese]. Rev Bras Med Esporte. 1999;5(6):207–11.
23. Woollacott M, Shumway-Cook A. Attention and the control of posture and gait: a review of an emerging area of research. Gait Posture. 2002;16(1):1–14.
24. Osness WH, Adrian M, Clark B, Hoeger W, Raab D, Wiswell R. Functional fitness assessment for adults over 60 years (a field based assessment). Reston: American Aliance for Health, Physical Education, Recreation and Dance; 1990. P. 36.
25. Parihar R, Mahoney JR, Verghese J. Relationship of gait and cognition in the elderly. Curr Transl Geriatr Exp Gerontol Rep. 2013;2(3):1–10.
26. Holtzer R, Verghese J, Xue X, Lipton RB. Cognitive processes related to gait velocity: results from the Einstein Aging Study. Neuropsychology. 2006;20(2):215–23.
27. Yaari R, Bloom JC. Alzheimer's disease. Semin Neurol. 2007;27(1):32–41.
28. Montero-Odasso M. Dual-tasking and gait in people with mild cognitive impairment: the effect of working memory. BMC Geriatr. 2009;9(41):1–8.
29. Sheridan PL, Hausdorff JM. The role of higher-level cognitive function in gait: executive dysfunction contributes to fall risk in Alzheimer's disease. Dement Geriatr Cogn Disord. 2007;24(2):125–37.
30. Ble A, Volpato S, Zuliani G, Guralnik JM, Bandinelli S, Laruetani F. Executive function correlates with walking speed in older persons: the InCHIANTI study. J Am Geriatr Soc. 2005;53(3):410–5.
31. Springer S, Giladi N, Peretz C, Yogev G, Simon ES, Hausdorff JM. Dual-tasking effects on gait variability: the role of aging, falls, and executive function. Mov Disord. 2006;21:950–7.
32. Sheridan PL, Mat JS, Kowall N, Hausdorff JM. Influence of executive function on locomotor function: divided attention increases gait variability in Alzheimer's disease. J Am Geriatr Soc. 2003;51(11):1633–7.
33. Doi T, Shimada H, Makizako H, Tsutsumimoto K, Uemura K, Anan Y, et al. Cognitive function and gait speed under normal and dual-task walking among older adults with mild cognitive impairment. BMC Neurol. 2014;14(67):1–8.
34. Gillain S, Warzee E, Lekeu F, Wojtasik V, Maquet D, Croisier JL, et al. The value of instrumental gait analysis in elderly healthy, MCI or Alzheimer's disease subjects and a comparison with other clinical tests used in single and dual-task conditions. Ann Phys Rehabil Med. 2009;52(6):453–74.
35. Schaefer S. The ecological approach to cognitive–motor dual tasking: findings on the effects of expertise and age. Front Psychol. 2014;5:Article 1167.
36. Yogev-Seligmann G, Giladi N, Gruendlinger L, Hausdorff JM. The contribution of postural control and bilateral coordination to the impact of dual tasking on gait. Exp Brain Res. 2013;226(1):81–93.
37. Mirelman A, Herman T, Brozgol M, Dorfman M, Sprecher E. Executive function and falls in older adults: new findings from a five-year prospective study link fall risk to cognition. PLoS One. 2012;7(6):1–8.
38. Blahak C, Baezner H, Pantoni L, Poggesi A, Chabriat H, Erkinjuntti T, et al. Deep frontal and periventricular age related white matter changes but not basal ganglia and infratentorial hyperintensities are associated with falls: cross sectional results from the LADIS study. J Neurol Neurosurg Psychiatry. 2009;80(6):608–13.
39. Chapman GJ, Hollands MA. Evidence that older adult fallers prioritise the planning of future stepping actions over the accurate execution of ongoing steps during complex locomotor tasks. Gait Posture. 2007;26(1):59–67.
40. Anstey KJ, Wood J, Kerr G, Caldwell H, Lord SR. Different cognitive profiles for single compared with recurrent fallers without dementia. Neuropsychology. 2009;23(4):500–8.
41. Plummer P, Zukowski LA, Giuliani C, Hall AM, Zurakowski D. Effects of physical exercise interventions on gait-related dual-task interference in older adults: a systematic review and meta-analysis. Gerontology. 2016;62(1):94–117.
42. Theill N, Schumacher V, Adelsberger R, Martin M, Jancke L. Effects of simultaneously performed cognitive and physical training in older adults. BMC Neurosci. 2013;14:103.

43. Trombetti A, Hars M, Herrmann FR, Kressig RW, Ferrari S, Rizzoli R. Effect of music-based multitask training on gait, balance, and fall risk in elderly people: a randomized controlled trial. Arch Intern Med. 2011;171(6):525–33.
44. Pavol MJ, Owings TM, Foley KT, Grabiner MD. Mechanisms leading to a fall from an induced trip in healthy older adults. J Gerontol A Biol Sci Med Sci. 2001;56(7):M428–37.
45. Rivera JA, Fried LP, Weiss CO, Simonsick EM. At the tipping point: predicting severe mobility difficulty in vulnerable older women. J Am Geriatr Soc. 2008;56(8):1417–23.
46. Marko M, Neville CG, Prince MA, Ploutz-Snyder LL. Lower extremity force decrements identify early mobility decline among community-dwelling older adults. Phys Ther. 2012;92 (9):1148–59.
47. Fried LP, Bandeen-Roche K, Chaves PHM, Johnson BA. Preclinical mobility disability predicts incident mobility disability in older women. J Gerontol A Biol Sci Med Sci. 2000;55(1):M43–52.
48. Inouye SK, Studenski S, Tinetti ME, Kuchel GA. Geriatric syndromes: clinical, research, and policy implications of a core geriatric concept. J Am Geriatr Soc. 2007;55(5):780–91.
49. Brown CJ, Flood KL. Mobility limitation in the older patient: a clinical review. JAMA. 2013;310(11):1168–77.
50. Wannamethee SG, Ebrahim S, Papacosta O, Shaper AG. From a postal questionnaire of older men, healthy lifestyle factors reduced the onset of and may have increased recovery from mobility limitation. J Clin Epidemiol. 2005;58(8):831–40.
51. Shigematsu R, Okura T. A novel exercise for improving lower-extremity functional fitness in the elderly. Aging Clin Exp Res. 2006;8(3):242–8.
52. Shigematsu R, Okura T, Nakagaichi M, Tanaka K, Sakai T, Kitazumi S, et al. Square-stepping exercise and fall risk factors in older adults: a single-blind, randomized controlled trial. J Gerontol A Biol Sci Med Sci. 2008;63(1):76–82.
53. Shigematsu R, Okura T, Sakai T, Rantanen T. Square-stepping exercise versus strength and balance training for fall risk factors. Aging Clin Exp Res. 2008;20(1):19–24.
54. Teixeira CVL, Gobbi S, Pereira JR, Vital TM, Hernandez SSS, Shigematsu R, et al. Effects of square-stepping exercise on cognitive functions of older people. Psychogeriatrics. 2013;13 (3):148–56.
55. Teixeira CVL, Gobbi S, Pereira JR, Ueno DT, Shigematsu R, Gobbi LTB. Effect of square-stepping exercise and basic exercises on functional fitness of older adults. Geriatr Gerontol Int. 2013;13(4):842–8.
56. Pereira JR, Gobbi S, Teixeira CVL, Nascimento CMC, Corazza DI, Vital TM, et al. Effects of Square-Stepping Exercise on balance and depressive symptoms in older adults. Motriz. 2014;20(4):454–60.
57. Parkatti T, Perttunen J, Wacker P. Improvements in functional capacity from Nordic walking: a randomized-controlled trial among elderly people. JAPA. 2012;20(1):93–105.
58. Li F, Fisher KJ, Harmer P. Improving physical function and blood pressure in older adults through cobblestone mat walking: a randomized trial. J Am Geriatr Soc. 2005;53(8):1305–12.
59. Vincent KR, Braith RW, Feldman RA, Magyari PM, Cutler RB, Persin SA, et al. Resistance exercise and physical performance in adults aged 60 to 83. J Am Geriatr Soc. 2002;50 (6):1100–7.
60. Vincent HK, Vincent KR, Lamb KM. Obesity and mobility disability in the older adult. Obes Rev. 2010;11(8):568–79.
61. Miller GD, Nicklas BJ, Davis C, Loeser RF, Lenchik L, Messier SP. Intensive weight loss program improves physical function in older obese adults with knee osteoarthritis. Obesity. 2006;14(7):1219–30.
62. Ross LA, Schmidt EL, Ball K. Interventions to maintain mobility: what works? Accid Anal Prev. 2013;61:167–96.
63. Roenker DL, Cissell GM, Ball KK, Wadley VG, Edwards JD. Speed-of-processing and driving simulator training result in improved driving performance. Hum Factors. 2003;45 (2):218–33.

64. Mänty M, Heinonen A, Leinonen R, Törmäkangas T, Hirvensalo M, Kallinen M, et al. Long-term effect of physical activity counseling on mobility limitation among older people: a randomized controlled study. J Gerontol A Biol Sci Med Sci. 2009;64(1):83–9.
65. Polcyn AF, Lipsitz LA, Kerrigan DC, Collins JJ. Age-related changes in the initiation of gait: degradation of central mechanisms for momentum generation. Arch Phys Med Rehabil. 1998;79(12):1582–9.
66. Mbourou GA, Lajoie Y, Teasdale N. Step length variability at gait initiation in elderly fallers and non-fallers, and young adults. Gerontology. 2003;49(1):21–6.
67. Pollock AS, Durward BR, Rowe PJ, Paul JP. What is balance? Clin Rehabil. 2000;14:402–6.
68. Horak FB. Postural orientation and equilibrium: what do we need to know about neural control of balance to prevent falls? Age Ageing. 2006;35(s2):ii7–11.
69. Laughton CA, Slavin M, Katdare K, Nolan L, Bean JF, Kerrigan DC, et al. Aging, muscle activity, and balance control: physiologic changes associated with balance impairment. Gait Posture. 2003;18(2):101–8.
70. Horak FB, Shupert CL, Mirka A. Components of postural dyscontrol in the elderly: a review. Neurobiol Aging. 1989;10(6):727–38.
71. Winter DA. Human balance and posture control during standing and walking. Gait Posture. 1995;3(4):193–214.
72. Bauby CE, Kuo AD. Active control of lateral in human walking. J Biomech. 2000;33 (11):1433–40.
73. Prince F, Corrivecuy H, Herbert R, Winter A. Gait in elderly. Gait Posture. 1997;5(2):128–35.
74. Duarte M, Freitas SMSF. Revision of posturography based on force plate for balance evaluation [Portuguese]. Rev Bras Fisioter. 2010;14(3):183–92.
75. Weeks DL, Forget R, Mouchino L, Gravel D, Bourbonnais D. Interaction between attention demanding motor and cognitive tasks and static postural stability. Gerontology. 2003;49 (4):225–32.
76. Beauchet O, Dubost V, Herrmann FR, Kressig RW. Stride-to-stride variability while backward counting among healthy young adults. J Neuroeng Rehabil. 2005;2:26.
77. Lajoie Y, Teasdale N, Bard C, Fleury M. Attentional demands for static and dynamic equilibrium. Exp Brain Res. 1993;97(1):139–44.
78. Brown LA, Shumway-Cook A, Woollacott MH. Attentional demands and postural recovery: the effects of aging. J Gerontol A Biol Sci Med Sci. 1999;54(4):165–71.
79. Teasdale N, Simoneau M. Attentional demands for postural control: the effects of aging and sensory reintegration. Gait Posture. 2001;14(3):203–10.
80. Ljmker T, Lamoth CJC. Gait and cognition: the relationship between gait stability and variability with executive function in persons with and without dementia. Gait Posture. 2012;35(1):126–30.
81. Lamoth CJ, van Deudekom FJ, van Campen JP, van Appels BA, Pijnappels M. Gait stability and variability measures show effects of impaired cognition and dual tasking in frail people. J Neuroeng Rehabil. 2011;8:2.
82. Manckoundia P, Pfitzenmeyer P, D´athis P, Dubost V, Mourey F. Impact of cognitive task on the posture of elderly subjects with Alzheimer's disease compared to healthy elderly subjects. Mov Disord. 2006;21(2):236–73.
83. Rapp MA, Krampe RT, Bealtes PB. Adaptive task prioritization in aging: selective resource allocation to postural control is preserved in Alzheimer disease. Am J Geriatr Psychiatry. 2006;14(1):52–61.
84. Andrade LP, Rinaldi NM, Coelho FGM, Tanaka K, Stella F, Gobbi LTB. Dual task and postural control in Alzheimer's and Parkinson's disease. Motriz. 2014;20(1):78–84.
85. Franssen EH, Souren LEM, Torossian CL, Reisberg B. Equilibrium and limb coordination in mild cognitive impairment and mild Alzheimer's disease. J Am Geriatr Soc. 1999;47 (4):463–9.
86. Piirtola M, Era P. Force platform measures as predictors of falls among older people: a review. Gerontology. 2006;52(1):1–16.

87. Hass CJ, Gregor RJ, Waddell DE, Oliver A, Smith DW, Fleming RP, Wolf SL. The influence of Tai Chi training on the center of pressure trajectory during gait initiation in older adults. Arch Phys Med Rehabil. 2004;85(10):1593–8.
88. Kannus P, Sievänen H, Palvanen M, Järvinen T, Parkkari J. Prevention of falls and consequent injuries in elderly people. Lancet. 2005;366(9500):1885–93.
89. Doughty K, Lewis R, McIntosh A. The design of a practical and reliable fall detector for community and institutional telecare. J Telemed Telecare. 2000;6 Suppl 1:S150–4.
90. Gillespie LD, Robertson MC, Gillespie WJ, Lamb SE, Gates S, Cumming RG, et al. Interventions for preventing falls in older people living in the community. Cochrane Database Syst Rev. 2009;15(2), CD007146.
91. Sherrington C, Whitney JC, Lord SR, Herbert RD, Cumming RG, Close JC. Effective exercise for the prevention of falls: a systematic review and meta-analysis. J Am Geriatr Soc. 2008;56 (12):2234–43.
92. van Diest M, Lamoth CJC, Stegenga J, Verkerke GJ, Postema K. Exergaming for balance training of elderly: state of the art and future developments. J Neuroeng Rehabil. 2013;10:101.
93. Cromwell RL, Meyers PM, Meyers PE, Newton RA. Tae Kwon Do: an effective exercise for improving balance and walking ability in older adults. J Gerontol A Biol Sci Med Sci. 2007;62 (6):641–6.
94. Howe TE, Rochester L, Neil F, Skelton DA, Ballinger C. Exercise for improving balance in older people. Cochrane Database Syst Rev. 2011;11, CD004963.
95. Hollands KL, Pelton TA, Tyson SF, Hollands MA, van Vliet PA. Interventions for coordination of walking following stroke: systematic review. Gait Posture. 2012;35(3):345–59.

Cognition, Gait Disorders, and Fall Risk in Healthy Neurological Older Individuals

7

Manuel Montero-Odasso

Abstract

Clinicians and researchers have commonly performed gait and cognitive assessments separately when evaluating older adults. Evidence from clinical practice, epidemiological studies, and clinical trials shows that gait and cognition are interrelated. Quantifiable alterations in gait among older adults are associated with falls, dementia, and disability. At the same time, emerging evidence indicates that early disturbances in cognitive processes such as attention, executive function, and working memory are associated with slower gait and gait instability and also with future mobility loss, falls, and progression to dementia. This chapter reviews the importance of the gait–cognition interrelationship in aging and presents evidence that gait performance mirrors cognitive function and provides insights about how brain and gait control contributes to fall risk in older people. To this end, the benefits of dual-task gait assessments (e.g., walking while performing a cognitive-demanding task) as a marker of fall risk are presented. Finally, evidence from an emerging therapeutic approach for reducing the risk of falls by improving certain aspects of cognition through both non-pharmacological and pharmacological treatments is reviewed. Disentangling the relationship between gait disturbances and cognitive changes at their earlier stages may be helpful for identifying older adults at higher risk of experiencing mobility decline and falls.

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7.1 Introduction

7.1.1 Are Falls in Older People a Manifestation of Brain Failure?

Forty years ago, Professor Bernard Isaacs postulated that to only attribute falls in older people to muscular-articular and sensory impairments and their effect on balance was over simplistic, and rather, that a failure of our sophisticated mechanism of brain mobility control plays a capital role in triggering falls [1, 2].

Cognitive impairment and falls hold a very well-established relationship [3]. Importantly, dementia and falls often coexist in older adults; gait impairments and falls are more prevalent in patients with dementia than in normal aging and are related to the severity of cognitive impairment [4]. Gait and cognitive impairment are prominent independent risk factors for falls. Falls are a common geriatric syndrome affecting about a third of older adults each year, and a better understanding of the relationship between cognitive function and gait impairments may help clinicians and researchers to develop interventions and institute preventive measures to delay the transition to falls and promote disability-free life expectancy.

Fall syndrome is a major cause of morbidity and mortality among older adults, especially for those with cognitive problems. For instance, older adults with moderate to severe cognitive impairment have a higher risk of falls, with an annual incidence of around 60–80 %, which is twice the rate of that in cognitively normals [5]. The consequences of falls in this population are very serious; fallers with cognitive problems are approximately five times more likely to be admitted to institutional care than cognitively impaired people who do not fall [6]. They are also at high risk of major fall-related injuries, including fractures and head injuries, and mortality. In addition to indirect costs and caregiver burden due to fall syndrome, the direct costs of emergency care, acute rehabilitation, and long-term care are substantial and challenge sustainability in several healthcare systems [7].

The precise mechanisms underlying the increased fall risk in cognitively impaired older adults are not completely understood; however, several studies have shown that impaired cognitive abilities reducing attentional resource allocation compromise postural and gait stability [8]. Similarly, executive function, a set of cognitive processes, including attention, inhibitory control, working memory, and cognitive flexibility, is an essential cognitive resource required for normal walking, and impairments in this cognitive domain are associated with both dementia syndromes and fall risk [9]. One specific early change in gait performance seen among older adults with mild-to-moderate dementia is a decrease in gait velocity [4]. Mechanistically, the relationship between cognitive deficits and gait

disturbances has been attributed to specific brain networks such as the prefronto-parietal and cingulate areas that are selectively affected by diseases that accompany, but are not necessarily caused by, aging [10].

The interrelationship between cognitive and gait dysfunction has also been found in otherwise healthy older adults or in those who have subtle cognitive deficits that are not yet profound enough to be identified using global measures of cognitive status. Such findings challenge our understanding and definition of what is currently considered clinically relevant cognitive impairment. For instance, even among healthy older adults with "normal" cognition, low performance in executive function was prospectively associated with falls, as assessed using cutoff scores on global clinical cognitive tests such as the Mini Mental Status Exam.

A systematic review and meta-analysis of 27 prospective cohort studies with at least 1 year of follow-up among healthy community-dwelling older adults found that executive dysfunction was associated with an increased risk for any fall by 40 % and with falls associated with serious injury [12]. Similarly, early mobility decline, assessed as slowing of gait, has been found to coexist or even precede the onset of clinically demonstrable cognitive decline in older adults [13]. This slowing of gait may have its onset up to 12 years before the clinical presentation of cognitive changes in older adults who later convert to mild cognitive impairment (MCI) syndrome, a transitional state between normal aging and early dementia [14, 15]. Recent studies have also shown that older adults with MCI have a higher prevalence of gait impairments and higher risk of falling compared to cognitively normal older adults [15–17]. Thus, older adults with MCI can be seen as a population at risk, not only for future dementia, but also for falls, and should perhaps be specifically targeted for interventions to reduce fall risk.

Until recently, falls and dementia were studied and assessed as distinct geriatric syndromes (Fig. 7.1a). This may have led to a gap in our understanding of the motor–cognitive interactions that affects the pathways to future falls and fall-related disability in older adults. This gap may also explain why cognition has received little attention with regard to intervention strategies for falls prevention. New evidence supports these two geriatric syndromes as interrelated outcomes associated with aging. Figure 7.1b shows the merging view and conceptual framework to understand the interrelationship in the concurrent decline of cognition and mobility in aging.

7.1.2 Cognition and Gait Instability

Although walking has long been considered a primarily automatic motor task, emerging evidence suggests that this view is too simplistic [18]. Walking in the real world requires paying attention to various environmental features and recovering from postural perturbations to avoid stumbles or falls. Therefore, it is

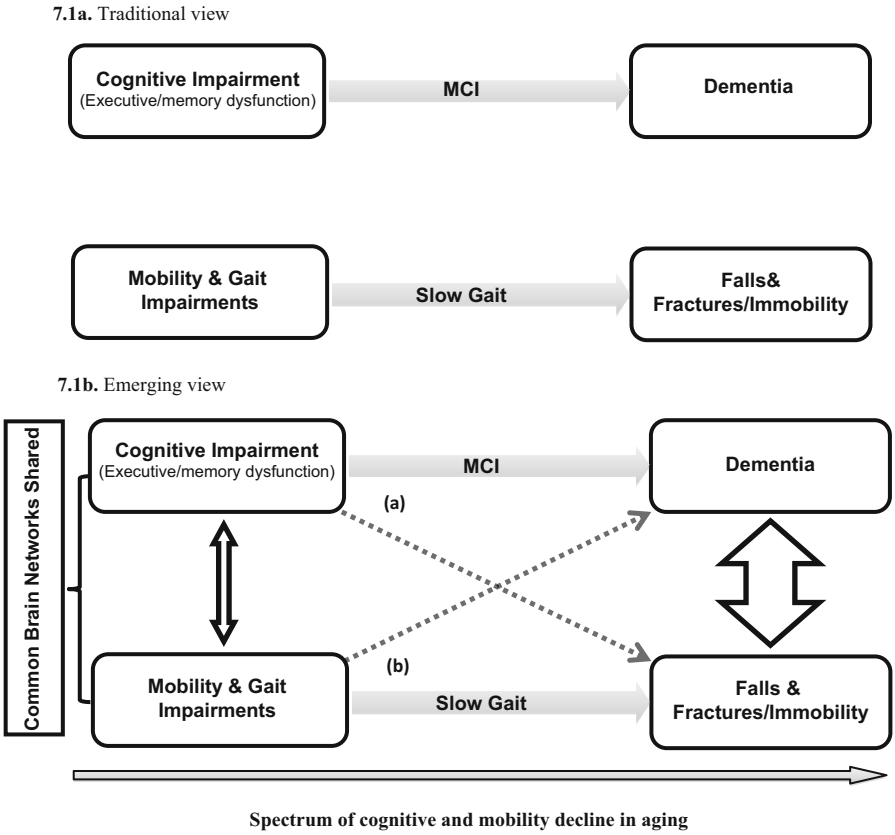

Fig. 7.1 (**a**) Traditional view of the parallel decline of gait and cognitive function with aging. Gait performance and cognitive function deteriorate with aging yielding two geriatric entities: falls and dementia. (**b**) Alternative, emerging view. Cognition predicts mobility decline and falls (a), and similarly, mobility decline and slow gait predict cognitive deterioration and future dementia (b). These phenomena occur in a concurrent manner. Modified from Montero-Odasso et al. [3]

not surprising that deficits in attention and executive function processes are independently associated with risk of postural instability, impairment in activities of daily living, and future falls [9].

The mechanistic evidence about mobility and cognition relationship is supported by brain imaging studies. Functional magnetic resonance imaging and near-infrared spectroscopy studies have demonstrated significant associations for basal ganglia and prefrontal motor regions with gait during walking and performing a cognitively demanding task, as dual-tasking [11]. These studies also pointed that shared cortical brain networks are involved in information processing, memory, and gait control and that abnormalities accumulating throughout these specific brain networks as we age could lead to mobility decline. For instance, diffusion tension imaging (DTI) studies, an imaging technique used to describe and detect brain tracts, have shown

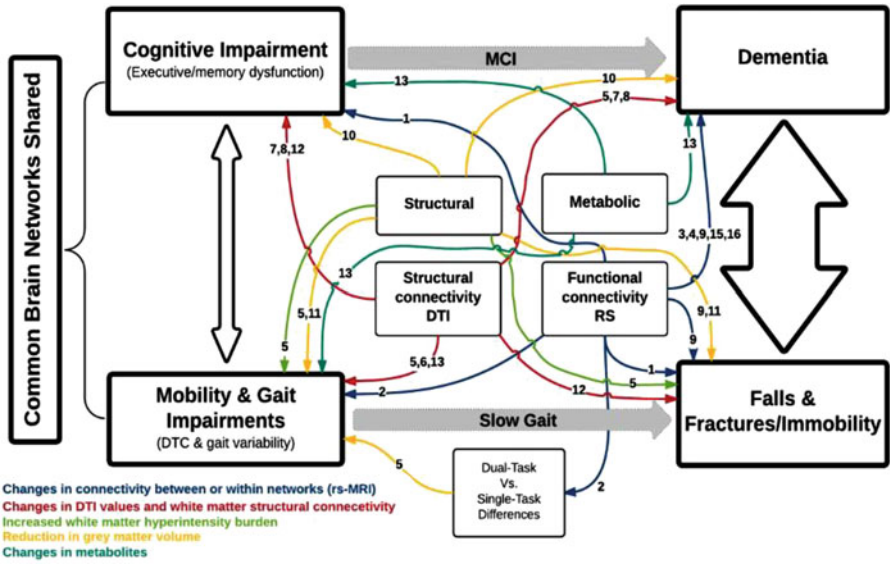

Fig. 7.2 Evidence from brain imaging studies supporting the relationship between cognitive and mobility decline, gait decline and future falls, and progression to dementia syndromes

that lower integrity of prefrontal–subcortical networks, including prefrontal cortex, basal ganglia, and medial temporal lobe, is related to slower gait and increased fall risk [9–11]. Figure 7.2 synthetizes the evidence of imaging studies supporting the association across cognition and mobility in the conceptual framework described above.

7.2 Mechanism and Pathophysiology of Falls

7.2.1 Basis of Posture Control

The human upright position is naturally unstable with a narrow base of support and a high center of body mass. To maintain this delicate equilibrium while walking or standing, the human body has a harmonious modulation and coordination of the trunk and ankle flexibility. This equilibrium modulation is challenged by motor impairments, either weakness, slowness, or poor coordination, that increase the risk of falling under physiological perturbations (e.g., body sway during standing or walking) or after extrinsic destabilizing factors (e.g., during tripping). The rapid succession of strategies aimed at preserving body stability after a perturbation includes first the "ankle strategy" for small perturbations and later the "hip strategy" for larger perturbations. The ankle strategy is a motor plan characterized by the relaxation of trunk muscles and stiffening of the ankle joint, and the hip strategy relaxes the hip muscles and stiffens the leg muscles with the head moving out of

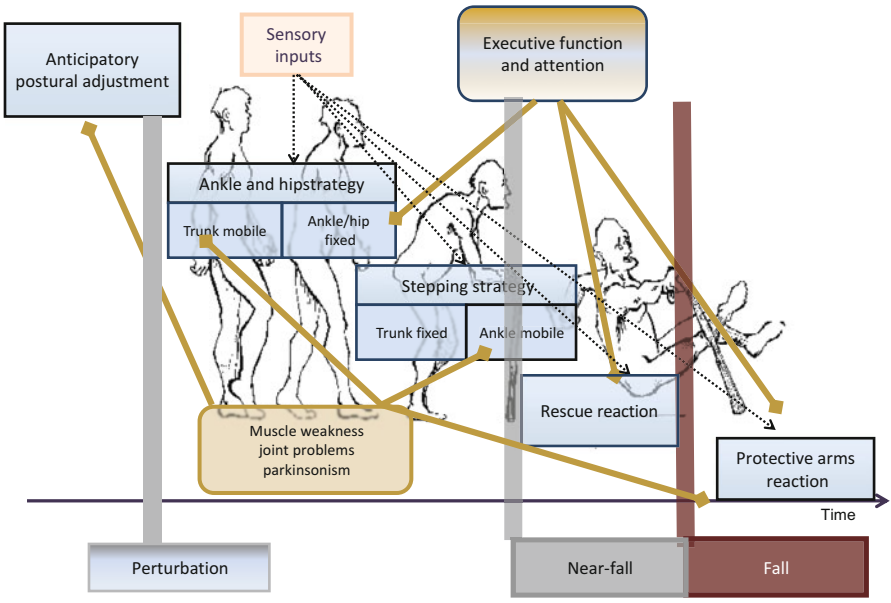

Fig. 7.3 Schematization of the rapid succession of strategies aimed at preserving body stability after a single perturbation. Note the role of cognitive function in the three rescue strategies. Modified from Fasano et al. [23]

phase and hip moving to maintain the center of body mass over the base of support [19–22]. When the perturbation is more severe and the ankle and hip strategies are not efficient enough, a third motor plan is the "stepping strategy," during which the ankle joint is released and the subject performs one or more steps to enlarge the base of support. If these motor acts fail to preserve stability, the upper limbs play a major role in performing rescue strategies (e.g., holding on some support) or protective reactions to limit the traumatic consequence of falling. This model helps to explain the pathophysiological link between trunk inflexibility, worsened by rigidity or fear of falling, instability (i.e., ankle strategy), and the mechanistically link between gait disorders and falling (i.e., "stepping strategy").

Figure 7.3 depicts the emerging role of cortical cognitive resources, attention and executive functions, in each of these strategies [23]. An adequate flow of information through visual, vestibular, and somatosensory afferents is required, paired with attentional and executive resources to effectively adapt to the environment and to the type of perturbation by rapidly switching from one strategy to the other [23, 24].

The motor determinants of falls are characterized by a disorder of either the base of support or the center of body mass [19]. A useful human model to study base of support disorders is Parkinson's disease (PD). Patients with Parkinson's manifest disorders of both the base of support and the center of body mass and therefore fall much more frequently than age-matched control individuals. Additional aging

processes not strictly confined to the dopaminergic systems play a major role in the pathogenesis of the axial motor impairment. In recent years, mild parkinsonian signs have been recognized in elderly subjects without PD. These patients present features recognized as risk factors for falling including involvement of gait and postural stability as well as executive cognitive function. These mild parkinsonian signs in older individuals without PD have been related to brain vascular lesions mainly involving the frontal regions [25] which support the hypothesis that cortical control of gait shares the same neural networks of important frontal lobe function included in executive function [25].

Postural stability can be viewed as a strategy per se. As such, the central nervous system adapting to the environmental constrains should rapidly select the appropriate stabilizing strategy for each circumstance which evolves from postural perturbation, including a protective reaction when fall cannot be avoided. Seniors with a higher rate of injuries show an impaired protective arm response during falling. The relevance of the protective arm response is also highlighted by the observation that elderly fallers with the combined fractures of distal radius and hip have a better prognosis than the peers with isolated hip fracture [26].

7.2.2 Cognitive Aspects of Fall Risk

Gait performance is a complex task that depends on the normal functioning of multiple systems working in a highly coordinated and integrated manner [27, 28]. As impairments in different domains can alter this delicate system, it has been hypothesized that different chronic conditions and risk factors for falls such as visual or hearing problems, muscular weakness, osteoarthritis, or peripheral neuropathy could be evidenced through gait performance [28]. Therefore, gait performance can be seen as a common pathway affected by different factors that can cause the fall syndrome. This fact may explain why gait problems are among the highest predictive risk factors for falls in older adults [27–30]. Walking also relies on cognition [3]; although it has long been considered a primarily automatic motor task, emerging evidence suggests that this view is overly simplistic [18]. Walking requires paying attention to various environmental features and recovering from postural perturbations to avoid stumbles or falls. Therefore, it is not surprising that deficits in attention and executive function processes are independently associated with risk of postural instability, impairment in activities of daily living, and future falls [9].

The research on dual-task gait, i.e., the ability to perform a secondary task simultaneous to walking, increased exponentially after a seminal study has shown that the incapacity to maintain a conversation while walking ("stop walking when talking") was a strong predictor of future falls in cognitively impaired older adults [31]. Patients with a neurological overt disease, such as stroke, PD, or mild cognitive impairment (MCI) and dementia present a marked deterioration during dual-tasking [17, 32, 33]. This evidence helps to understand why patients with cognitive impairments and dementia syndromes are vulnerable to dual-task

challenges and why falls are so common in this group of patients. Daily life activities involve many attention-demanding events which explains the high occurrence of falling while performing a secondary attentional demanding task. Current evidence supports that dual-task gait and balance abilities worsen due to the impairment of attentional-related cognitive resources, including executive function. Even during standing, postural sway increases when a cognitive task is performed, suggesting that constant dynamic control of postural adjustments during standing also requires certain levels of cognitive attentional resources.

Even among healthy older adults with "normal" cognition, low performance in attention and executive function was prospectively associated with falls [11]. A systematic review and meta-analysis found that executive dysfunction was associated with 1.44 times increased risk for any fall and falls associated with serious injury [12].

Finally, evidence for the role of attention and cognition in postural control is also provided by the fact that psychotropic drugs such as benzodiazepines and neuroleptics that impair cognition affect postural control and reaction times and increase risk of falls [34]. On the other hand, cognitive enhancers, including donepezil, which are usually used for the treatment of dementia, have been found to significantly reduce falls rather than near falls in patients with PD without cognitive impairment, thus indicating that the drug did not improve stability, but rather cognitive resources. Similarly, cognitive enhancers have been shown to improve gait and mobility in people with Alzheimer's disease (AD) [35, 36].

7.2.3 Dual-Task Gait Assessments

As explained above, dual-task gait performance is a sensitive measure of the role of cognitive deficits in gross motor performance, gait stability and navigation, and fall risk. Specifically, dual-task gait performance isolates the role of attention and executive function deficits in the regulation of brain gait control [3, 17, 32]. Emerging evidence is sustaining that "dual-task gait" can help to identify risk of falls in an individual without previous falls [3]. During the dual task, the subject performs an attention-demanding task while walking to assess any modifications, compared to the reference, single-task condition, in either the cognitive or the walking subtasks [8]. The underlying hypothesis is that two simultaneously performed tasks interfere and compete for brain cortical resources [9]. Therefore, dual-task gait can act as a stress test to the brain to detect impeding mobility problems and risk of fall. Gait changes during dual-tasking (also known as dual-task costs), such as slowing of gait, are interpreted as the increased cost of involvement of cortical attention processes while walking. The role of dual-task costs as a marker of future falls over simple gait assessments has been evaluated with mixed results in the literature due to the heterogeneity of studies, small sample sizes, limited prospective fall ascertainment, and the lack of standardization in dual-task procedures [37]. Although clinically meaningful, cutoff values of dual-task costs are still controversial, and other unanswered questions remain. A growing body of evidence supports

the potential clinical utility of this paradigm for fall prediction since it provides a valid and sensitive means of assessing motor–cognitive interactions and fall risk. Based on recent studies, a dual-task cost higher than 20 % may denote individuals at higher risk of falls when they sustain a gait velocity of 95 cm/s or faster, highlighting the sensitivity and predictive ability in older adults who have a relatively normal gait velocity [38].

7.2.4 Gait Variability and Risk of Falls

Gait is a complex motor behavior with many measurable facets. This growing field of research provides an interesting window for the study of the regulation of the locomotor and cognitive control. Gait velocity is easily measured and provides valuable information about physical and medical status in the complete functional spectrum of older people [29, 39]. Another way to quantify gait instability that has garnered much attention in the literature recently is gait variability.

Changes in gait velocity and variability are not mutually exclusive; however, they provide different information. For example, Herman et al. reported that gait variability during dual-tasking predicted future falls among community-living older adults, while gait velocity did not [11]. The variability of several spatial-temporal gait parameters has been studied, with stride-to-stride fluctuations in gait cycle timing (e.g., stride time) being the most widely reported [40]. In brief, it has been suggested that stride time reflects one of the final pathways of the outcomes regulated by the central nervous system. Stride time variability is becoming a relevant marker of gait stability, both in research and clinical settings, and can be measured easily and robustly [40]. The general assumption is that there is an inverse association between stride time variability and gait stability. Low stride time variability reflects automatic processes that require minimal higher cortical input and is associated with efficient and safe gait patterns [40]. Walking is one of the most repetitive and "hard wired" human movements; the normal fluctuations in stride time variability are usually below 3 % among healthy adults [40, 41]. However, higher gait variability has been described in older adults with frailty [42], PD [43], and AD [33, 44] and prospectively associated with a high risk of future falls and mobility decline [45, 46]. More interestingly, high stride time gait variability has been shown to predict future falls in community-dwelling older adults [11, 47], even when gait velocity failed to demonstrate an association. Evidence has shown that gait variability not only serves as a clinically relevant approach in the evaluation of mobility, but may also be responsive to fall prevention interventions [40, 41].

Variability in stride time and stride length are related to the control of the rhythmic stepping mechanism and should, therefore, be considered as markers of adaptability to the walking environment. Sheridan et al. reported that high gait variability could be a sensitive marker of dysfunction in the frontal cortical control of walking in subjects with moderate AD and executive dysfunction [44]. Hausdorff et al. showed that the degree of executive function efficiency was correlated to the degree of stride time variability [18]. These and other studies have shown that there

is interplay between gait variability, cognitive dysfunction, in particular executive function and attention, and the risk of falls.

7.3 Emerging Therapeutical Approaches

7.3.1 Learning from Cognitively Impaired Populations

Deficits in cognitive function and mobility coexist in older adults even in the early stages of aging. Older people with MCI may have impairments in either memory (amnestic) or non-memory (non-amnestic) cognitive domains [18], as well as impairments in fine and complex motor skills, equilibrium, and limb coordination. Quantitative testing has revealed gait dysfunction in subjects with both the amnestic and non-amnestic MCI subtypes compared to healthy controls [16]. Neurological gaits, such as hemiparetic, and frontal or parkinsonian gaits were almost twice as common in amnestic MCI as in normal controls in this study. Additionally, subjects with MCI and gait abnormalities (defined as either having slow gait or a neurological gait) were more disabled than subjects with MCI without gait abnormalities [16].

Low performance in three related cognitive domains (i.e., attention, executive function, and working memory) was associated with slowing of gait velocity specifically under dual-task conditions, suggesting that these specific cognitive domains are relevant for maintaining a normal gait pattern in the presence of a cognitive load [17]. It has been proven that increased variability of time-related gait parameters was significantly different under dual-task testing in older adults with MCI compared to age-matched normal controls, but less affected in people with mild AD [33]. The same group compared the effect of dual-task of different complexities between cognitively normal controls and older adults with MCI [48]. A significant dual-task cognitive status interaction was found for gait variability, but not for gait velocity, further demonstrating that gait variability may be especially sensitive to dual-tasking and to the complexity of the task given. These findings suggest that cognitive control of gait performance is impaired in people with MCI and in the early clinical stages of dementia. Interestingly, these disturbances were not evident during the single-task test condition. This finding highlights the ability of tests that challenge brain function to reveal subtle or early gait problems. In summary, several studies have shown that gait is altered in people with early cognitive problems, most prominently under dual-task conditions, and may provide an explanation for the high risk of falls in people with MCI and with executive dysfunction [15, 25–28, 49].

7.3.2 Strategies to Prevent Falls and Enhance Mobility Targeting Cognition

Fall prevention trials in cognitively normal older adult populations have demonstrated, that both multifactorial (e.g., review of medications, strength and

balance training, visual and hearing correction and environmental modifications) and single interventions (e.g., resistance exercises or progressive balance training) can be effective in preventing falls. In contrast, intervention studies targeting fall prevention in people with cognitive problems have been inconsistent or have only had modest success [50]. Older adults with cognitive problems, while at high risk for falls, represent a group who may be less responsive to the interventions that are effective among subjects that are cognitively intact [51]. There are several potential explanations for the lack of benefit of fall prevention programs in the cognitively impaired population including different underlying mechanisms for falls in those with cognitive problems and failure to address cognitive deficits adequately. Although much is known about the multifactorial nature of falls in cognitively normal individuals, the emerging knowledge about the nature and interrelationship of risk factors in those with cognitive problems can help to understand how cognitive control of balance and gait can trigger falls in cognitive healthy individuals.

Current preliminary evidence from interventional studies support that targeting cognition may reduce falls. Specifically, it has been postulated that improving certain aspects of cognition, as a complementary intervention, can help to prevent falls in older adults. The following two sections review recent studies addressing the role of improving cognition to improve mobility and reduce falls in older adults. Figure 7.4 summarizes the effects of four different forms of interventions targeting cognition with the goal of improving mobility, specifically gait velocity and dual-tasking gait velocity.

7.3.2.1 Non-pharmacological Approaches

Cognitive remediation interventions have been shown to improve attention and executive function as well as memory in older adults without dementia. Table 7.1 summarizes recent studies that have specifically evaluated the effects of interventions to improve cognition on gait and fall risk. Verghese et al. [52] conducted a pilot study in 24 older adults who were randomly assigned to either a computerized cognitive remediation intervention or a wait list. The ten participants who completed the cognitive remediation showed improvement on gait velocity during normal walking and walking while talking (dual task) compared to baseline (Fig. 7.3a). While the initial findings of this pilot trial need confirmation in larger-scale trials, they indicate that a non-pharmacological cognitive intervention can positively modify gait performance, especially during dual-task testing.

Silsupadol et al. [53] conducted an intriguing but small study among 21 older adults with balance and gait impairment. Participants were randomly assigned to one of three interventions: single task (active control), fixed-prioritization dual-task training, and dual-task training with variable prioritization. Improvements in balance and gait velocity were found in all groups after training (Fig. 7.3b). However, when a cognitive, dual task was added during testing, only participants who received dual-task training exhibited significant improvements in gait velocity ($p < 0.001$). Furthermore, the group that trained with variable-priority instructions demonstrated a dual-task training effect that was retained after 12 weeks of follow-

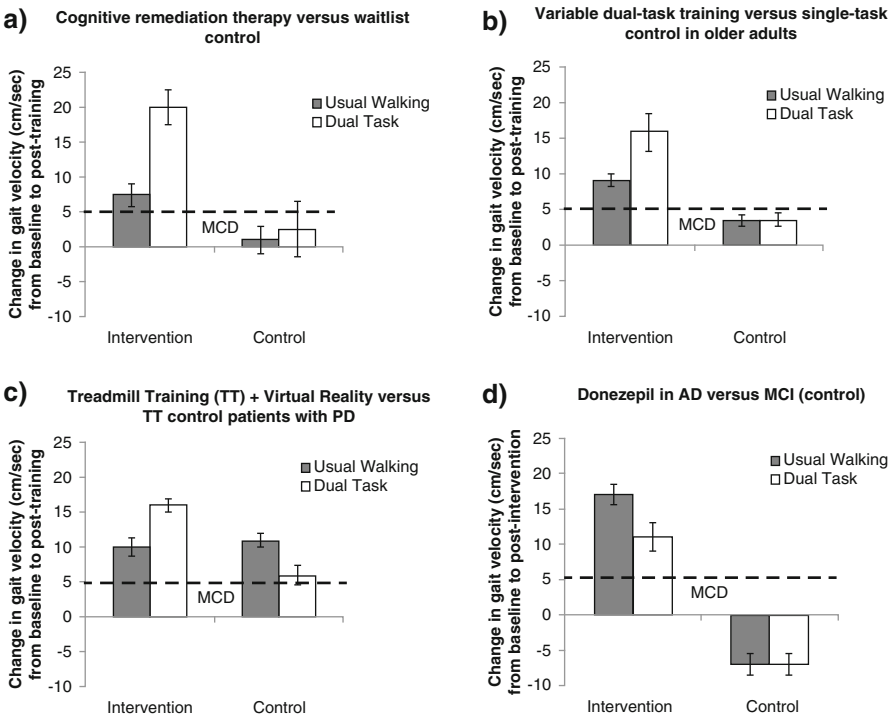

Fig. 7.4 Summary effects of four different forms of cognitive therapy on usual-walking gait velocity and dual-tasking gait velocity. Values shown are changed with respect to baseline. Adapted from Montero-Odasso et al. [3, 67]. Note that 5 cm/s has been identified as the minimal clinical difference (MCD) and 10 cm/s as a substantial difference. (**a**) Effects of 8 weeks of computerized cognitive remediation training (while seated) in sedentary older adults, compared with wait-list controls. Data from [52]. (**b**) Effects of dual-task training during walking on gait speed in older adults with balance impairment, compared with subjects who only practiced walking. Data from [53]. (**c**) Effects of 6 weeks of TT augmented with VR among patients with PD, compared with an active control comparison of 6 weeks of TT alone. Usual-walking gait speed increased in both the TT alone and TT + VR groups; however, DT gait speed only improved among the patients who participated in TT + VR. Data from [52, 56]. (**d**) Effects of 4 months of donepezil use on gait speed in patients with Alzheimer's disease and compared with control patients with mild cognitive impairment. *AD* Alzheimer's disease, *DT* dual-tasking, *MCD* minimal clinical difference, *MCI* mild cognitive impairment, *PD* Parkinson's disease, *TT* treadmill training, *VR* virtual reality

up [53]. These results suggest that varying focus during training between the cognitive and motor tasks apparently had more benefits than training that required constant focus of attention on both tasks.

Schwenk et al. [54] conducted a randomized controlled trial (RCT) that evaluated the efficacy of a 12-week dual-task training program in 61 seniors with dementia. The intervention group received dual-task training, using progressively more complex activities such as walking while throwing and catching a ball or walking while performing mental arithmetic, during a 2 hour exercise session

Table 7.1 Cognitive training interventions for balance, gait, and fall risk

Study (year)	Study design	Type of intervention	Duration of training intervention	Participants	Summary of findings
You et al. (2009)	RCT	Dual-task CGI: simultaneous motor (walking 30 m) and cognitive (memory recall) task ($n = 8$), whereas the control group ($n = 5$) received a placebo treatment (walking while listening to simple music)	18 sessions, 30 min per *session* over 6 weeks	13 older adults (mean age, 68.3 ± 6.5 years) with a history of falls	Working memory performance under the dual-task condition improved ($p < 0.05$), but significant changes in gait velocity and stability were not observed
Silsupadol et al. (2009)	RCT	Participants were randomly assigned to one of three interventions: single-task training ($n = 7$), dual-task training with fixed-priority instructions ($n = 8$), and dual-task training with variable-priority instructions ($n = 6$)	12 sessions; 45 min individualized sessions, three times a week for 4 weeks	21 older adults (aged 65 years and older) with balance impairment	Improvement in balance and gait velocity was found in all groups. Only the DT training with variable-priority instructions group demonstrated a DT training effect at the second week maintained at 12-week follow-up
Yogev-Seligmann et al.	Open-label, pilot	A 4-week program of one-on-one training included walking while performing several distinct cognitive tasks	12 sessions; three times a week for 4 weeks	Seven patients with PD (mean age, 63.8 ± 8.4 years)	Gait velocity and gait variability during DT significantly improved. Untrained DT also improved and was retained 1 month after the end of the training
Verghese et al. (2010)	RCT	Computerized "MindFit" program ($n = 10$). Each training session included a mixture of 21 visual, auditory, and cross-modality tasks compared with wait list ($n = 10$)	24 sessions; 45–60 min/session, three times a week for 8 weeks, compared with wait-list controls	Sedentary older adults (mean age, 77.4 ± 7.0 years; MMSE, 29 ± 0.3) at training and wait list (mean age, 79.9 ± 7.5 years; MMSE, 29.1 ± 0.4)	Gait velocity improved during normal walking ($p < 0.05$) and "walking while talking" ($p = 0.002$) only in study group. Speed of processing improved significantly in the training group ($p = 0.03$)

(continued)

Table 7.1 (continued)

Study (year)	Study design	Type of intervention	Duration of training intervention	Participants	Summary of findings
Schwenk et al. (2010)	RCT	Intervention group ($n = 20$) underwent dual-task-based exercise training (motor, throwing or catching a ball; cognitive, arithmetic tasks, repeating names of animals). The control group ($n = 29$) performed low-intensity exercise	24 sessions over 12 weeks, 1 h twice a week	49 participants (mean age, 81.9 ± 7.5 years) with confirmed mild-to-moderate dementia (MMSE 17–26, 21.4 ± 2.9)	DT training significantly improved gait velocity under dual task ($p < 0.001$), cadence ($p = 0.007$), stride length ($p = 0.001$), and single support ($p = 0.003$) under complex three-step backward calculation conditions compared with the control, but not under the less challenging DT two-step forward calculation conditions
Mirelman et al. (2010)	Repeated measures design	TT with virtual obstacles. The VR simulation required obstacle negotiation in two planes while continuing to walk on the treadmill. Comparison was made to a historical active control group who followed similar protocol of TT but without VR	18 sessions (three per week for 6 weeks)	20 participants with PD (mean age, 67.1 ± 6.5 years) moderately impaired but were able to walk unassisted for at least 5 min	Improvements in gait velocity and stride length in usual walking. However, dual-tasking, gait velocity ($p = 0.003$), and stride length ($p < 0.001$) were significantly higher after training with TT + VR compared with TT alone Dual-task gait variability decreased and trail-making test improved after TT + VR training

Adapted from Montero-Odasso et al. [3]

Note: *CGI* cognitive–gait intervention, *DT* dual-tasking, *MMSE* mini mental state examination, *RCT* randomized controlled trial, *TT* treadmill training, *VR* virtual reality

carried out twice a week. The control group received low-intensity exercise for 1 hour once per week over the same period of follow-up. After the 12 weeks of training, the intervention group performed significantly better on gait in a complex dual-task condition ("walking while doing serial three subtractions") compared with the control group. Despite some limitations, including that the training in the intervention group included repeated exposures to the same dual-task test conditions that served as the main outcomes, this study shows RCT evidence [55] that dual-task training improves gait in people with mild-to-moderate dementia.

Finally, Mirelman et al. [56] reported an improvement in gait using a program of treadmill training enhanced with virtual reality in patients with PD. After 6 weeks of training, gait velocity, stride time, and stride length significantly improved in usual and dual-tasking conditions as well as during overground obstacle negotiation (Fig. 7.3c). In addition, gait variability decreased (i.e., improved) under dual-task conditions [56]. Moreover, the improvements in dual-task walking were much larger than that seen in a previous study that followed an almost identical treadmill training program, without the virtual reality component. This finding suggests that a complex motor–cognitive training may be more effective at improving dual-tasking and functional gait, compared to a program that focuses exclusively on motor function.

While the effects of these preliminary cognitive-based therapies to mediators of fall risk are promising, studies have not yet examined the impact of these interventions on fall frequency. Large-scale RCT trials are needed to generate critical evidence-based results before this approach can be widely recommended.

7.3.2.2 Pharmacological Approaches

Pharmacological interventions targeting attention and executive function have also improved gait performance in older adults [57, 58]. Table 7.2 summarizes studies that specifically evaluate the effects of cognitive pharmacological interventions to improve gait and fall risk in older individuals. Methylphenidate (MPH) has long been used to improve attention in children with attention-deficit hyperactivity disorder; however, its potential to modify motor function is less known. In a pilot study, Auriel et al. [57] evaluated the effect of a single dose (20 mg) of MPH on cognitive function, gait performance, and markers of fall risk in 21 patients with PD who were receiving L-Dopa. A single dose of MPH was associated with a significant improvement in attention, executive function, gait velocity, stride time variability, and the Timed Up and Go Test, i.e., validated markers of abnormal gait and fall risk. Ben-Itzhak et al. tested the effect of MPH in 26 community-living older adults without dementia using a randomized, double-blind, crossover design. MPH improved executive function and gait velocity and reduced gait variability in this sample of older adults as well [58]. Additionally, a single dose of MPH reduced the detrimental effect of dual-task testing on gait variability. These initial findings suggest that MPH, and potentially other drugs designed to enhance

Table 7.2 Cognitive pharmacotherapy for balance, gait, and fall risk

Study (year)	Study design	Type of intervention	Duration of intervention	Participants	Summary of findings
Auriel et al. (2006)	Open-label, before–after design	Before and 2 h after taking a single dose of 20 mg of MPH	2 h	21 patients with PD who receive L-Dopa, (mean age, 70.2 ± 9.2 years; MMSE, 28.8 ± 1.7)	Improvement in attention ($p = 0.025$) and EF index showed only a trend ($p = 0.09$). Improvements in Timed Up and Go ($p = 0.001$), gait velocity ($p = 0.005$), and stride time variability ($p = 0.013$)
Ben-Itzhak et al. (2008)	RCT, double-blind, placebo-controlled	Before and 2 h after taking 20 mg MPH or a placebo	2 h	26 older adults without dementia with subjective memory complaints (mean age, 73.8 ± 1.2 years; mean MMSE, 27.8 ± 1.4)	MPH improved Timed Up and Go ($p = 0.004$), stride time variability ($p = 0.03$), and EF ($p = 0.03$); effects not observed after treatment with the placebo
Montero-Odasso et al. (2009)	Open-label study with controls	5 mg/day of donepezil for 1 month and another 3 months with 10 mg/day MCI group with no treatment	4 months	Six patients with mild AD (mean age, 79.9 ± 4.8 years; MMSE, 22.3 ± 1.2; MoCA, 15 ± 1.4) compared with eight patients with MCI (mean age, 75.6 ± 6.2 years; MMSE, 27.9 ± 1.7; MoCA, 22.9 ± 1.7)	AD patients increased their gait velocity ($p = 0.045$) and dual-task gait velocity ($p = 0.047$) after 1 month. Stride time variability decreased (improved) during follow-up. Changes were maintained for 4 months. MCI (control) group declined in gait velocity and variability
Assal et al. (2008)	Before–after design	Galantamine mean dose of 17.8 ± 3.5 mg/day	6 months	Nine patients with mild-to-moderate AD (mean age, 77.9 ± 2.1 years; mean MMSE, 26.4 ± 5.2) compared with 18 no-treatment control subjects without dementia (mean age, 78.1 ± 1.0 years; mean MMSE, 29.4 ± 0.8)	Stride time was shorter under dual-tasking after treatment ($p = 0.01$). There was no change in the controls

Chung et al. (2010)	Randomized, crossover, double-blind	Donepezil versus placebo. In each drug phase, subjects were instructed to take 5 mg/day of donepezil or placebo for 3 weeks and to increase to 10 mg/day for the remaining 3 weeks	6 weeks, 3 weeks washout 6 weeks of placebo	23 patients with PD who reported falling or nearly falling (mean age, 68.3 ± 10.8 years; MMSE, 27.6 ± 4.5)	Less falls with donepezil than placebo ($p = 0.049$). Subjects with the most falls at baseline tended to show the largest improvements. No differences in Activities of Balance Confidence Scale, Berg Balance Scale, UPDRS III, or MMSE scores
Beauchet et al. (2011)	Before–after design	Memantine mean dose of 20 mg/day, titrated in 5 mg increments over 4 weeks	4.4–8 months	17 patients with AD (mean age 83.8 ± 5.8 years; 52.9 % women; MMSE:, 14.5 ± 4.2) and 32 age- and gender-matched control patients with AD without any anti-dementia drug (mean age 80.0 ± 6.6 years; 56.3 % women; MMSE, 23.2 ± 5.3)	Stride time variability decreased (improved) during follow-up in the memantine group (6.3 ± 6.1 versus 3.6 ± 1.3, $P = 0.038$)
Montero-Odasso et al. (2014)	Phase II trial	5 mg/day of donepezil for 1 month and 5 months with 10 mg/day	5 months	43 patients with mild AD (mean age, 76.9 ± 8 years; MMSE, 24.63 ± 2; MoCA, 18.5 ± 4)	Gait velocity improved from 108.4 ± 18.6 to 113.3 ± 19.5 cm/s, $p = 0.01$; dual-task gait velocity from 80.6 ± 23.0 to 85.3 ± 22.3 cm/s, $p = 0.03$. Stride time variability showed a not statistically significant improvement. Trail-making tests A ($p = 0.030$), B ($p = 0.001$), and B-A ($p = 0.042$) improved after intervention
Henderson et al. (2015)	RCT, phase II	Rivastigmine titrated from 3 mg/day (or equivalent as placebo) and incremented every	8 months	130 patients with mild PD (mean age, 71.9 ± 4.8 years; 1.2; MoCA, 25 ± 1.4); half in the intervention groups and half in the placebo group	Intervention group (rivastigmine) was associated with a small but significant improvement in gait velocity (greatest effect) and dual-

(continued)

Table 7.2 (continued)

Study (year)	Study design	Type of intervention	Duration of intervention	Participants	Summary of findings
		4 weeks to a maximum of 12 mg per day at week 13 onward			task gait velocity. Stride time variability decreased (improved) during follow-up but difference was not significant

Modified and updated from Montero-Odasso et al. [3]
Note: *AD* Alzheimer's disease, *EF* executive function, *MCI* mild cognitive impairment, *MMSE* mini mental state examination, *MoCA* Montreal cognitive assessment, *MPH* methylphenidate, *PD* Parkinson's disease, *UPDRS* unified Parkinson's disease rating scale

attention, may have a role as a therapeutic option for reducing fall risk in older adults.

Cognitive function and the brain control of gait also share several neurotransmitters that may serve as additional potential targets for pharmacological interventions. Dopamine deficits have been associated with fall risk in patients with PD; levodopa typically improves many gait features in patients with this neurological disease. Interestingly, however, a recent study that assessed the role of dopamine neurotransmission in fall risk [59] demonstrated that dopaminergic denervation is apparently not associated with falls in older adults with PD. Similarly, the neurotransmitter acetylcholine has shown to have an important role in cognitive function and in controlling gait and balance [60]. Specific regions within the brain with cholinergic tracts include the hippocampus, nucleus basalis of Meynert, basal ganglia, thalamus, and peduncle pontine nucleus. The thalamic cholinergic activity, which derives mainly from terminals of brain stem peduncle pontine nucleus neurons, plays a central role in the generation of the elementary movement patterns and gait and balance control [61]. The cholinergic system is also an important and specific controller of selective attention, likely an important factor in the dual-task decrement in walking that occurs in cognitively impaired adults. Therefore, correcting the cholinergic loss in dementia may improve attention and subsequently gait performance.

Cholinesterase inhibitors (ChEIs), e.g., donepezil, galantamine, and rivastigmine, are the currently approved symptomatic treatments for AD and vascular dementia. The molecular mechanism of action for ChEI is achieved by increasing cortical and hippocampal acetylcholine, regulators of memory and synaptic plasticity [62]. However, the mechanisms of clinical improvements in function in patients with dementia are not thoroughly understood. Possible explanations may relate to the cognitive action of the drug and to subtle improvements in motor function. For instance, there may be cognitive-related and non-cognitive-related enhancement mechanisms by which ChEIs might improve gait performance and potentially reduce fall risk [60]. Improving executive function and attention may affect gait control, and cholinergic enhancement of the peduncle pontine nucleus may improve stride-to-stride variability. Both of these mechanisms have the theoretical potential to reduce the occurrence of falls. Whether by a direct effect or mediated through cognition, motor function improvement would consequently serve to stabilize mobility, reduce falls, and delay functional decline.

A few studies have evaluated the effect of ChEI in motor performance in older adults. These are generally pilot investigations or phase II trials, but they are, nonetheless, worthy of consideration given their novelty and potential importance to our understanding of therapeutic possibilities. Two pilot studies evaluated the effect of ChEI on gait performance [35, 63]. Assal et al. tested the effect of galantamine on gait in nine participants with moderate AD. Their gait was compared with ten controls without dementia. Controls suffered a significant dual-task decrement in stride time, though there was no decrement among the participants with AD on galantamine. This finding suggests a galantamine-associated

enhancement of the ability to adapt gait patterns to tasks that require attention. Montero-Odasso et al. assessed the effect of donepezil over 4 months of treatment on gait velocity and gait variability in six individuals with AD (Fig. 7.3d). Increases in gait velocity and a reduction in gait variability were seen at 1 month following treatment with the 5 mg of donepezil. These benefits were further improved after 4 months of treatment when the full dose of 10 mg was achieved, suggesting a dose-response pattern. The control group, composed of eight individuals with MCI, experienced an expected reduction of gait velocity and an increase in gait variability over time [35].

A phase II clinical trial involving 53 participants with mild Alzheimer's disease found that after 4 months of treatment, mean gait velocity and dual-task gait velocity significantly improved in the range of 5–8 cm/s. Changes in stride time variability were in the expected direction, although not statistically significant. Participants also showed significant improvements in trail-making tests A, B, and B-A [36].

A randomized crossover study involving 23 older participants with PD conducted by Chung et al. observed that fall frequency was almost 50 % lower when the patients were on donepezil than when they were taking a placebo [64]. A recent RCT in PD patients conducted by Henderson et al. enrolled 130 patients and randomly assigned them to rivastigmine (3–12 mg/day) versus placebo group. After 8 months of treatment, participants assigned to rivastigmine (55 assessed) had improved step time variability for normal walking (or 0.72, 95 % CI 0.58–0.88; $p = 0.002$) and the simple dual task (0.79, 0.62–0.99; $p = 0.045$) [65].

Memantine is another symptomatic treatment option for patients with moderate to severe AD. Beauchet et al. reported a memantine-related decrease in stride time variability among AD outpatients followed in a memory clinic, suggesting that decrease in gait variability may be explained by the combined dopaminergic and glutamatergic effects of memantine [66].

These preliminary interventional studies provide a basis for testing a variety of approaches in larger clinical trials. If cognitive enhancers can change gait in a meaningful way, it could reduce the risk of falls. Currently, there are clinical trials under way to explore these interesting possibilities [60].

7.4 Conclusions and Future Directions

A fresh look at existing data on gait and mobility impairment and falls in cognitive healthy and cognitively impaired populations set the stage for a novel approach and some practical conclusions. Cognitive impairment should be considered as a continuum from normal aging to advanced dementia and, similarly, mobility decline, and slowing of gait is a continuum that coexists with or even precedes the declines in cognition. Cognitive impairment, gait decline, and falls singly and together have a sufficiently high prevalence in older adults to constitute significant population health problems and important causes of disability. Gait variability is an objective

measure that reflects cognitive dysfunction and may prove useful prognostically for mobility, cognition, and fall outcomes. Gait assessment under dual-tasking apparently can be used in the clinical encounter as a window into brain function to help detect the impact of cognitive deficits in motor and gait regulations. Dual-task gait seems to predict fall risk, particularly in those with normal gait velocity. Disentangling the relationship between early gait and cognitive impairments may help better understand fall risk in aging.

Finally, improving certain aspects of cognition, specifically attention and executive function, in older adults can be a complementary way to treat mobility decline and risk of falls. In the cognitively impaired, this may be critical to reducing fall risk and its attendant disability. Preliminary studies have shown that both non-pharmacological and pharmacological interventions targeting cognition may improve gait performance and, consequently, have the potential to reduce fall risk. These interventions may be effective for improving mobility and reducing falls. It remains to be seen whether the pharmacologic and cognitive-training approaches would have a synergistic effect, whether one can substitute for the other, or whether one would eclipse the other. While important questions need to be addressed in depth, these initial investigations suggest that a complementary approach to fall prevention can be incorporated into current fall intervention strategies.

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References

1. Isaacs B. Are falls a manifestation of brain failure? Age Ageing. 1978;Suppl:97–111.
2. Montero-Odasso M, Bherer L, Studenski S, Gopaul K, Oteng-Amoako A, Woolmore-Goodwin S, et al. Mobility and Cognition in Seniors. Report from the 2008 Institute of Aging (CIHR) Mobility and Cognition Workshop. Can Geriatr J. 2015;18(3):159–67.
3. Montero-Odasso M, Verghese J, Beauchet O, Hausdorff JM. Gait and cognition: a complementary approach to understanding brain function and the risk of falling. J Am Geriatr Soc. 2012;60(11):2127–36.
4. van Iersel MB, Hoefsloot W, Munneke M, Bloem BR, Olde Rikkert MG. Systematic review of quantitative clinical gait analysis in patients with dementia. Z Gerontol Geriatr. 2004;37(1):27–32.
5. Tinetti ME, Speechley M, Ginter SF. Risk factors for falls among elderly persons living in the community. N Engl J Med. 1988;319(26):1701–7.
6. Morris JC, Rubin EH, Morris EJ, Mandel SA. Senile dementia of the Alzheimer's type: an important risk factor for serious falls. J Gerontol. 1987;42(4):412–7.

7. the Canadian Patient Safety Institute, the Canadian Institute for Health Information, Accreditation Canada. Preventing Falls: From Evidence to Improvement in Canadian Health Care. 2016.
8. Woollacott M, Shumway-Cook A. Attention and the control of posture and gait: a review of an emerging area of research. Gait Posture. 2002;16(1):1–14.
9. Yogev-Seligmann G, Hausdorff JM, Giladi N. The role of executive function and attention in gait. Mov Disord. 2008;23(3):329–42.
10. Rosano C, Brach J, Longstreth Jr WT, Newman AB. Quantitative measures of gait characteristics indicate prevalence of underlying subclinical structural brain abnormalities in high-functioning older adults. Neuroepidemiology. 2006;26(1):52–60.
11. Herman T, Mirelman A, Giladi N, Schweiger A, Hausdorff JM. Executive control deficits as a prodrome to falls in healthy older adults: a prospective study linking thinking, walking, and falling. J Gerontol A Biol Sci Med Sci. 2010;65(10):1086–92.
12. Muir SW, Gopaul K, Montero Odasso MM. The role of cognitive impairment in fall risk among older adults: a systematic review and meta-analysis. Age Ageing. 2012;41(3):299–308.
13. Buracchio T, Dodge HH, Howieson D, Wasserman D, Kaye J. The trajectory of gait speed preceding mild cognitive impairment. Arch Neurol. 2010;67(8):980–6.
14. Petersen RC, Doody R, Kurz A, Mohs RC, Morris JC, Rabins PV, et al. Current concepts in mild cognitive impairment. Arch Neurol. 2001;58(12):1985–92.
15. Liu-Ambrose TY, Ashe MC, Graf P, Beattie BL, Khan KM. Increased risk of falling in older community-dwelling women with mild cognitive impairment. Phys Ther. 2008;88 (12):1482–91.
16. Verghese J, Robbins M, Holtzer R, Zimmerman M, Wang C, Xue X, et al. Gait dysfunction in mild cognitive impairment syndromes. J Am Geriatr Soc. 2008;56(7):1244–51.
17. Montero-Odasso M, Bergman H, Phillips NA, Wong CH, Sourial N, Chertkow H. Dual-tasking and gait in people with mild cognitive impairment. The effect of working memory. BMC Geriatr. 2009;9:41.
18. Hausdorff JM, Yogev G, Springer S, Simon ES, Giladi N. Walking is more like catching than tapping: gait in the elderly as a complex cognitive task. Exp Brain Res. 2005;164(4):541–8.
19. Maki BE, McIlroy WE. Postural control in the older adult. Clin Geriatr Med. 1996;12 (4):635–58.
20. Maki BE, McIlroy WE. The role of limb movements in maintaining upright stance: the "change-in-support" strategy. Phys Ther. 1997;77(5):488–507.
21. McIlroy WE, Maki BE. Preferred placement of the feet during quiet stance: development of a standardized foot placement for balance testing. Clin Biomech (Bristol, Avon). 1997;12 (1):66–70.
22. McIlroy WE, Maki BE. The control of lateral stability during rapid stepping reactions evoked by antero-posterior perturbation: does anticipatory control play a role? Gait Posture. 1999;9 (3):190–8.
23. Fasano A, Plotnik M, Bove F, Berardelli A. The neurobiology of falls. Neurol Sci. 2012;33 (6):1215–23.
24. Amboni M, Barone P, Hausdorff JM. Cognitive contributions to gait and falls: evidence and implications. Mov Disord. 2013;28(11):1520–33.
25. Montero-Odasso M, Hachinski V. Preludes to brain failure: executive dysfunction and gait disturbances. Neurol Sci. 2014;35(4):601–4.
26. Berner Y, Shabat S, Mann G, Stern A, Sagiv P, Nyska M. Comorbidity of distal radius (Colles' type) and hip fractures implies a better outcome in older patients. J Am Geriatr Soc. 2002;50 (5):976–7.
27. Alexander NB. Gait disorders in older adults. J Am Geriatr Soc. 1996;44(4):434–51.
28. Montero-Odasso M, Schapira M, Varela C, Pitteri C, Soriano ER, Kaplan R, et al. Gait velocity in senior people. An easy test for detecting mobility impairment in community elderly. J Nutr Health Aging. 2004;8(5):340–3.

29. Montero-Odasso M, Schapira M, Soriano ER, Varela M, Kaplan R, Camera LA, et al. Gait velocity as a single predictor of adverse events in healthy seniors aged 75 years and older. J Gerontol A Biol Sci Med Sci. 2005;60(10):1304–9.
30. Tinetti ME, Speechley M. Prevention of falls among the elderly. N Engl J Med. 1989;320 (16):1055–9.
31. Lundin-Olsson L, Nyberg L, Gustafson Y. "Stops walking when talking" as a predictor of falls in elderly people. Lancet. 1997;349(9052):617.
32. Montero-Odasso M, Muir SW, Speechley M. Dual-task complexity affects gait in people with mild cognitive impairment: the interplay between gait variability, dual tasking, and risk of falls. Arch Phys Med Rehabil. 2012;93(2):293–9.
33. Muir SW, Speechley M, Wells J, Borrie M, Gopaul K, Montero-Odasso M. Gait assessment in mild cognitive impairment and Alzheimer's disease: the effect of dual-task challenges across the cognitive spectrum. Gait Posture. 2011;35(1):96–100.
34. Leipzig RM, Cumming RG, Tinetti ME. Drugs and falls in older people: a systematic review and meta-analysis: I. Psychotropic drugs. J Am Geriatr Soc. 1999;47(1):30–9.
35. Montero-Odasso M, Wells J, Borrie M. Can cognitive enhancers reduce the risk of falls in people with dementia? An open-label study with controls. J Am Geriatr Soc. 2009;57 (2):359–60.
36. Montero-Odasso M, Muir-Hunter SW, Oteng-Amoako A, Gopaul K, Islam A, Borrie M, et al. Donepezil improves gait performance in older adults with mild Alzheimer's disease: a phase II clinical trial. J Alzheimers Dis. 2015;43(1):193–9.
37. Beauchet O, Annweiler C, Dubost V, Allali G, Kressig RW, Bridenbaugh S, et al. Stops walking when talking: a predictor of falls in older adults? Eur J Neurol. 2009;16(7):786–95.
38. Yamada M, Aoyama T, Arai H, Nagai K, Tanaka B, Uemura K, et al. Dual-task walk is a reliable predictor of falls in robust elderly adults. J Am Geriatr Soc. 2011;59(1):163–4.
39. Studenski S, Perera S, Patel K, Rosano C, Faulkner K, Inzitari M, et al. Gait speed and survival in older adults. JAMA. 2011;305(1):50–8.
40. Hausdorff JM. Gait variability: methods, modeling and meaning. J Neuroeng Rehabil. 2005;2:19.
41. Hausdorff JM. Gait dynamics, fractals and falls: finding meaning in the stride-to-stride fluctuations of human walking. Hum Mov Sci. 2007;26(4):555–89.
42. Montero-Odasso M, Muir SW, Hall M, Doherty TJ, Kloseck M, Beauchet O, et al. Gait variability is associated with frailty in community-dwelling older adults. J Gerontol A Biol Sci Med Sci. 2011;66(5):568–76.
43. Hausdorff JM, Cudkowicz ME, Firtion R, Wei JY, Goldberger AL. Gait variability and basal ganglia disorders: stride-to-stride variations of gait cycle timing in Parkinson's disease and Huntington's disease. Mov Disord. 1998;13(3):428–37.
44. Sheridan PL, Solomont J, Kowall N, Hausdorff JM. Influence of executive function on locomotor function: divided attention increases gait variability in Alzheimer's disease. J Am Geriatr Soc. 2003;51(11):1633–7.
45. Brach JS, Studenski SA, Perera S, VanSwearingen JM, Newman AB. Gait variability and the risk of incident mobility disability in community-dwelling older adults. J Gerontol A Biol Sci Med Sci. 2007;62(9):983–8.
46. Herman T, Giladi N, Gurevich T, Hausdorff JM. Gait instability and fractal dynamics of older adults with a "cautious" gait: why do certain older adults walk fearfully? Gait Posture. 2005;21 (2):178–85.
47. Hausdorff JM, Rios DA, Edelberg HK. Gait variability and fall risk in community-living older adults: a 1-year prospective study. Arch Phys Med Rehabil. 2001;82(8):1050–6.
48. Montero-Odasso M, Muir SW, Speechley M, Borrie M. Gait variability and mild cognitive impairment: the effect of increasing task complexity. Arch Phys Med Rehabil. 2011;93 (2):293–9.

49. Camicioli R, Majumdar SR. Relationship between mild cognitive impairment and falls in older people with and without Parkinson's disease: 1-Year Prospective Cohort study. Gait Posture. 2010;32(1):87–91.
50. Oliver D, Connelly JB, Victor CR, Shaw FE, Whitehead A, Genc Y, et al. Strategies to prevent falls and fractures in hospitals and care homes and effect of cognitive impairment: systematic review and meta-analyses. BMJ. 2007;334(7584):82.
51. Hauer K, Becker C, Lindemann U, Beyer N. Effectiveness of physical training on motor performance and fall prevention in cognitively impaired older persons: a systematic review. Am J Phys Med Rehabil. 2006;85(10):847–57.
52. Verghese J, Mahoney J, Ambrose AF, Wang C, Holtzer R. Effect of cognitive remediation on gait in sedentary seniors. J Gerontol A Biol Sci Med Sci. 2010;65(12):1338–43.
53. Silsupadol P, Shumway-Cook A, Lugade V, van Donkelaar P, Chou LS, Mayr U, et al. Effects of single-task versus dual-task training on balance performance in older adults: a double-blind, randomized controlled trial. Arch Phys Med Rehabil. 2009;90(3):381–7.
54. Schwenk M, Zieschang T, Oster P, Hauer K. Dual-task performances can be improved in patients with dementia: a randomized controlled trial. Neurology. 2010;74(24):1961–8.
55. Verghese J, Holtzer R. Walking the walk while talking: cognitive therapy for mobility in dementia? Neurology. 2010;74(24):1938–9.
56. Mirelman A, Maidan I, Herman T, Deutsch JE, Giladi N, Hausdorff JM. Virtual reality for gait training: can it induce motor learning to enhance complex walking and reduce fall risk in patients with Parkinson's disease? J Gerontol A Biol Sci Med Sci. 2011;66(2):234–40.
57. Auriel E, Hausdorff JM, Herman T, Simon ES, Giladi N. Effects of methylphenidate on cognitive function and gait in patients with Parkinson's disease: a pilot study. Clin Neuropharmacol. 2006;29(1):15–7.
58. Ben-Itzhak R, Giladi N, Gruendlinger L, Hausdorff JM. Can methylphenidate reduce fall risk in community-living older adults? A double-blind, single-dose cross-over study. J Am Geriatr Soc. 2008;56(4):695–700.
59. Bohnen NI, Muller ML, Koeppe RA, Studenski SA, Kilbourn MA, Frey KA, et al. History of falls in Parkinson disease is associated with reduced cholinergic activity. Neurology. 2009;73 (20):1670–6.
60. Montero-Odasso M, Wells JL, Borrie MJ, Speechley M. Can cognitive enhancers reduce the risk of falls in older people with mild cognitive impairment?A protocol for a randomised controlled double blind trial. BMC Neurol. 2009;9:42.
61. Devos D, Defebvre L, Bordet R. Dopaminergic and non-dopaminergic pharmacological hypotheses for gait disorders in Parkinson's disease. Fundam Clin Pharmacol. 2010;24 (4):407–21.
62. Drever BD, Riedel G, Platt B. The cholinergic system and hippocampal plasticity. Behav Brain Res. 2011;221(2):505–14.
63. Assal F, Allali G, Kressig RW, Herrmann FR, Beauchet O. Galantamine improves gait performance in patients with Alzheimer's disease. J Am Geriatr Soc. 2008;56(5):946–7.
64. Chung KA, Lobb BM, Nutt JG, Horak FB. Effects of a central cholinesterase inhibitor on reducing falls in Parkinson disease. Neurology. 2010;75(14):1263–9.
65. Henderson EJ, Lord SR, Brodie MA, Gaunt DM, Lawrence AD, Close JC, et al. Rivastigmine for gait stability in patients with Parkinson's disease (ReSPonD): a randomised, double-blind, placebo-controlled, phase 2 trial. Lancet Neurol. 2016;15(3):249–58.
66. Beauchet O, Launay C, Fantino B, Annweiler C, Allali G. Does memantine improve the gait of individuals with Alzheimer's disease? J Am Geriatr Soc. 2011;59(11):2181–2.
67. Segev-Jacubovski O, Herman T, Yogev-Seligmann G, Mirelman A, Giladi N, Hausdorff JM. The interplay between gait, falls and cognition: can cognitive therapy reduce fall risk? Expert Rev Neurother. 2011;11(7):1057–75.

Visual Control of Locomotion in People with Parkinson's Disease

8

Rodrigo Vitório and André Macari Baptista

Abstract

Vision is intricately linked with action and plays an essential role in guiding safe locomotion in humans. However, vision can influence locomotion of people with Parkinson's disease (PD) in two distinct manners. While visual information such as doorways can trigger "freezing" episodes during which the patient feels as if the feet are glued to the floor, visual cues (transverse lines placed on the ground) can increase stride length and release freezing episodes. These paradoxical responses to visual information have motivated researchers to investigate the role of vision during locomotion in people with PD. In addition, studying PD as a model, in which the basal ganglia circuits are damaged and walking impairments are evident, may provide insights into the neural control of human locomotion. This book chapter explores how people with PD use visual information while engaged in different real-world walking activities, such as walking on regular pathway and visual cues, avoiding obstacles, and stepping on targets.

Keywords

Vision • Gait • Eye tracking • Obstacle • Cues • Basal ganglia • Parkinson's disease

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8.1 Introduction

Safe locomotion is underpinned by a well-coordinated process that involves the integration of visual, vestibular, and somatosensory information. Based on the fact that vision can identify environmental hazards, especially the distant ones, one might argue that vision is the predominant sensory system used for guiding locomotion. Details regarding environmental surroundings, identification and characteristics of objects and surfaces, and self-motion information are captured by the visual system. Vision provides information about the body relative to the environment, which is referred to as visual exproprioception (i.e., watching your feet while deliberately stepping on desired locations) [1–3]. This is distinguished from visual exteroception, which refers to information about environmental surroundings (i.e., characteristics such as curb height at the sidewalk). One critical role of the central nervous system is to maintain balance during locomotion in different types of terrains. In this concern, visuospatial information makes possible preventative regulation of gait patterns that ensure effective and safe locomotion [4]. To date, visual inputs are important for employing avoidance strategies, for making proactive adjustments to accommodate different ground terrain, and for navigation [3, 5, 6].

Visual information necessary for safe locomotion is directly acquired by eye movements [7]. Hence, many studies have examined visual sampling during locomotion. Especially, advancements in eye-tracking technology have enabled visual sampling to be quantified during real-world activities, such as steady-state walking, obstacle avoidance, and turning. Visual sampling is defined as the combination of fixations and saccades that are required to gather information about the environment while performing a real-world activity [8]. A fixation is the maintaining of the visual gaze on a single location; it is the activity that stabilizes the fovea (fixations) on areas of interest. Saccade refers to a fast jump-like movement of the eyes between two fixated areas [7]. Both gaze parameters have been used to better understand the role of visual information in the control of human locomotion. Visual sampling of the environment is task dependent and relevant to the task goal [6]. Importantly, disruptions of gaze behavior during locomotion are related to age, cognitive function, neurodegenerative diseases, and increased risk of falling [8–11].

Parkinson's disease (PD) is a neurological pathology characterized by progressive degeneration of dopaminergic neurons in the *substantia nigra pars compacta* of the basal ganglia. The decreased amount of dopamine compromises the optimum amount of neuromotor impulses required for the accurate motor control. As a consequence, people with PD demonstrate impaired motor control. Especially, gait and postural control are affected by the PD. Parkinsonian gait is characterized by shortened step length, reduced velocity, reduced upper limb swing, festination, and "freezing" episodes [12–14]. Impairments of postural control in people with PD include stooped posture (i.e., forward head, rounded shoulders, thoracic hyperkyphosis, and increased forward flexion of the trunk) and difficulties compensating for postural instabilities [15, 16]. In addition to motor impairments,

people with PD demonstrate a range of cognitive and visual deficits. For example, attention and executive functions are commonly impaired in PD and are related to increased gait variability and slowness [17, 18]. Visual deficits in PD include reduced contrast sensitivity, visuospatial impairments, hypometric voluntary, and variable reflexive saccades [11, 19–22]. More importantly, motor control and visual mechanisms are also interlinked with attentional resources, which are mediated by cognitive ("top-down") processes [23]. That being said, studying PD as a model may help to further clarify the links between these motor, cognitive, and visual impairments. This book chapter explores how people with PD use visual information while engaged in different real-world walking activities, revisiting the experiments conducted by our research group.

8.2 Defining a Protocol for Assessing Visual Control of Locomotion in Parkinson's Disease

Although people with PD are argued to be more dependent on vision to guide locomotion [24, 25], both clinical practice and research evidence suggest that PD alters responses to visual inputs [26, 27]. For example, visual stimuli such as doorways, narrow roads, cluttered environments, and people walking in opposite direction can trigger "freezing" of gait episodes. In opposition, visual cues such as transverse lines placed on the ground can increase step length and release freezing episodes. Understanding these paradoxical responses has been one of the greatest challenges faced by researchers interested in the visual control of locomotion in PD. In this section, we briefly present what would be an appropriate experimental setup for investigations in this field and what our group has explored so far.

One might suggest that the experimental setup to investigate the visual control of locomotion in PD should include an appropriate walking task. Since vision is important for making on-line adjustments to guarantee safe locomotion in the real world [3, 5, 6, 9, 10], it makes sense to use tasks that would require visual guidance to select specific foot placement and/or avoid potential hazards. Obstacle avoidance, walking on a cued path and stepping on targets are only a few examples of tasks that might be used to this end and are those explored in our experiments. These tasks simulate real-world walking tasks and, therefore, give us valuable data to better understand the role that vision plays in guiding locomotion in people with PD.

Determining where, when, what, and how visual information is acquired by the patients during walking activities is critical to understand the visual control of locomotion in PD. Thus, the experimental setup should also include visual sampling measures. Recording gaze behavior (via mobile eye trackers and/or electro-oculography) would be the most direct approach to addressing this important aspect. The combination of fixations and saccades would be the outcomes to represent the way participants explore and acquire visual information from the environment. Alternatively, one could quantify the visual demand to complete the walking task. Aftab Patla proposed the use of what he named as "voluntary visual

sampling paradigm" to assess the visual demand to complete a walking task [28]. In this approach, participants are asked to walk while wearing liquid crystal glasses. These glasses have two states: (i) in the opaque state, the glasses eliminate any form or motion information (i.e., participants cannot see through the glasses) while maintaining a general nonspecific ambient light level; (ii) in the transparent state, the glasses provide participants with a normal view of the surroundings (i.e., participants can actually see through the glasses). Importantly, the transition from an opaque to the transparent state of the glasses is under the subjects' control. Participants press a handheld switch to make the glasses transparent when they need to sample the environment, and the visual samples are recorded by researchers. In other words, participants are able to voluntarily visually sample the environment at any time, any place, and for however long they deem necessary. The voluntary visual sampling paradigm provides valuable data on when sampling the environment is critical and the amount of visual information that is necessary to complete the walking task. The experiments from our group explored both approaches (gaze measures and voluntary visual sampling) presented in this paragraph.

While focused on understanding the role of specific pieces of visual information (i.e., optic flow, exproprioception, etc.) during locomotion in PD, researchers could opt to manipulate the visual information available during the walking task. For example, our group explored the role that optic flow plays on guiding locomotion in patients with PD by comparing the behavior under conditions of normal vision and suppressed optic flow. We suppressed the optic flow by offering only static visual samples during the walking task, as static flashes of transparent state of liquid crystal glasses (similar to stroboscopic lighting used in dance clubs). In addition, we explored the role of exproprioceptive visual information by comparing the behavior under conditions of normal vision and one in which we removed exproprioception from lower limbs. Participants were asked to walk while wearing a blind attached to the trunk to avoid them to view their lower limbs. In the following sections, we describe three experiments in which we explored the suggested aspects to be included in a protocol to investigate the visual control of locomotion in PD.

8.3 Experiment #1: Optic Flow and Voluntary Visual Sampling During Normal Walking and Obstacle Avoidance in Parkinson's Disease

Previously, Azulay and colleagues [24] suggested that people with PD are more dependent on dynamic visual information, or optic flow, to guide locomotion on a regular pathway. These authors observed that, relative to normal lightning, only people with PD significantly reduced stride length and velocity under stroboscopic lighting. However, at the moment of our experiment #1, these findings had not been confirmed by other groups. Then we decided to conduct a similar study with the increment of exploring the voluntary visual sampling paradigm, which could help us better understand whether people with PD are more dependent on visual

information than healthy adults to guide locomotion. In addition, although previous research had reported foot clearance difficulties during obstacle avoidance by people with PD [14, 29], none had investigated the role of visual information, especially optic flow, on their control of step adjustments while crossing obstacles. Then, our experiment #1 investigated the contributions of optic flow and the characteristics of voluntary visual sampling during normal walking [30] and obstacle avoidance in people with PD [31].

People with mild to moderate PD ($n = 12$) and age- and gender-matched healthy individuals ($n = 12$) were asked to walk at preferred speed along a pathway 8 m long under different conditions. Two task conditions were tested: normal walking and obstacle avoidance; for the obstacle avoidance task, participants stepped over a foam-made obstacle (low or high; ankle height or half-knee height, respectively) positioned in the middle of the pathway. Three visual sampling conditions were tested: dynamic, static, and voluntary visual sampling. Participants wore liquid crystal glasses for visual manipulation. Under the dynamic condition, the glasses were transparent (normal lighting). Under the static condition, the glasses provided static visual samples at 3 Hz (only flashes of transparent state; similar to Azulay's study [24]), suppressing the optic flow. Under the voluntary visual sampling condition, participants pressed a handheld switch to make the glasses transparent when they needed to sample the environment. Outcome measures included spatiotemporal parameters of normal walking and obstacle avoidance, the number of obstacle contacts, and the characteristics of voluntary visual sampling.

During normal walking, interaction between groups and visual conditions was not observed for spatiotemporal measures. We found main effect of visual conditions for all spatiotemporal measures. These findings suggest that experimental vision manipulation altered the locomotor behavior of both people with PD and healthy individuals in a similar way. For example, participants of both groups walked slower in the static condition than in the dynamic and voluntary visual sampling conditions; there were no differences between the dynamic and voluntary visual sampling conditions (Fig. 8.1a). Also, there were no differences between groups for voluntary visual sampling measures, which mean that the two groups required the same amount of visual information to complete the normal walking task (Fig. 8.1b). These findings suggest that the visual control of normal walking in people with PD was similar to that observed in healthy controls. In other words, people with PD were not more dependent on visual information or optic flow than healthy individuals to guide locomotion along a regular pathway.

During obstacle avoidance, there were no significant differences between groups for the total duration of visual samples (Fig. 8.2a). Then, while crossing the obstacle in the voluntary visual sampling condition, people with PD required the same amount of visual information as did the healthy participants. With regard to the success rate of obstacle avoidance, people with PD registered more obstacle contacts than did the control group only under the static visual condition (Fig. 8.2b). This finding suggests that optic flow information (present during both dynamic and voluntary visual sampling conditions) was relevant to the individuals with PD with regard to task success. It seems that optic flow, even for intermittent

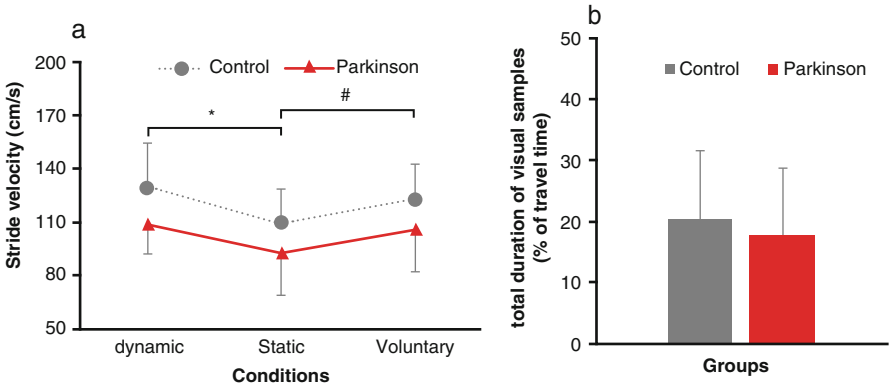

Fig. 8.1 Key findings for the normal walking task of experiment #1. (**a**) Stride velocity values for each group on each visual condition; * indicates a significant difference between dynamic and static conditions for both groups; # indicates a significant difference between static and voluntary visual sampling conditions for both groups. (**b**) Total duration of visual samples by each group during the voluntary visual sampling condition

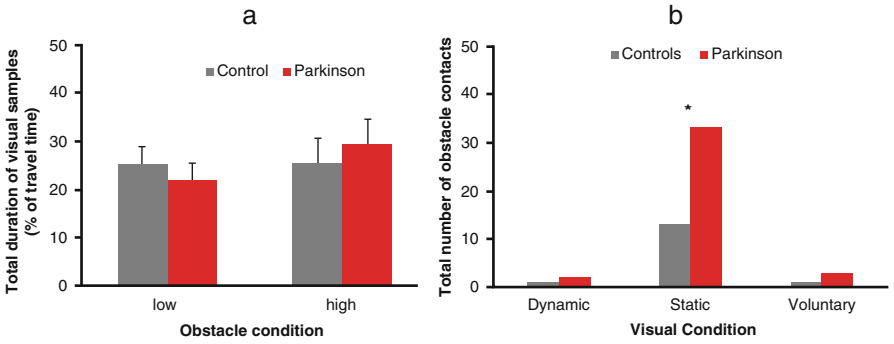

Fig. 8.2 Key findings for the obstacle avoidance task of experiment #1 [31]. (**a**) Total duration of visual samples for each group on each obstacle height during the voluntary visual sampling condition. (**b**) Total number of obstacle contacts for each group on each visual condition; * indicates a significant difference between people with PD and healthy individuals in the static condition

periods (voluntary visual sampling conditions), is necessary for people with PD to adjust movement properly while avoiding the obstacle, probably to compensate the proprioceptive impairment usually observed in people with PD [32]. The availability of only static visual samples may have led patients with PD to have an incorrect perception of limb self-motion (worse than the controls) relative to the obstacle during avoidance. We argue that patients' dependence on visual information seems to be more related to the kind (or quality) rather than the amount of visual information.

8.4 Experiment #2: Which Piece of Visual Information is Key for Gait Benefits Offered by Visual Cues in PD?

Several studies have demonstrated that visual cues such as stripes placed on the ground improve gait in people with PD [24, 33–35]. However, two main gaps in the literature were still to be filled at the moment of this experiment. First, it had not been established the kind of visual information (exproprioceptive or exteroceptive) that is crucial for the benefits in locomotion offered by visual cues in PD. During a cued walking task in which participants are instructed to step on the lines placed on the path, visual exproprioception refers to information of the feet relative to the cues (i.e., watching the foot while stepping on each line); visual exteroception refers to information about the cued pathway (e.g., distance between lines). Second, it had not been established whether people with PD use visual information from cues in a feedforward or on-line mode [1]. Feedforward refers to the use of visual information that was viewed before current step (i.e., looking ahead at one or more steps), while on-line refers to the use of visual information available during current step (i.e., looking at the line while stepping on it). Thus, these two gaps were addressed in the experiment #2 [36].

Participants included people with PD ($n = 19$) and age-matched healthy individuals ($n = 15$). They were asked to walk at preferred speed along a pathway 8 m long under four conditions (Fig. 8.3a), combining two factors (visual cues and exproprioceptive visual information from lower limbs): (i) in the baseline condition, participants walked over a regular black carpet; (ii) in the baseline without

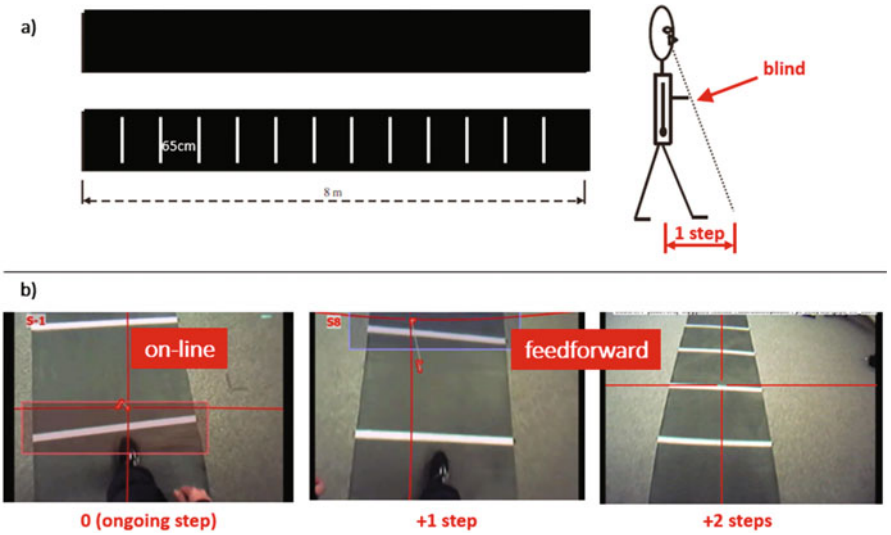

Fig. 8.3 (**a**) Illustration of the pathways (with and without the visual cues) and the blind used to remove exproprioception from lower limbs in the experiment #2. (**b**) Illustration of classification of gaze fixations according to the number of steps participants looked ahead

exproprioception condition, participants walked on the same black carpet while wearing a blind attached to their trunk to remove exproprioceptive information of lower limbs; (iii) in the visual cues condition, participants walked over a black carpet with white horizontal lines (3 cm wide) placed 65 cm apart; (iv) in the visual cues without exproprioception condition, participants walked on the cued pathway while wearing the blind. For the two cued conditions, participants were instructed to step on each line with their heels consecutively as they proceed through the trial. Foot 3-D kinematic data and gaze behavior were recorded, and, most importantly, the two systems were synchronized. For the visual cues condition, we determined how far ahead participants were looking at by calculating the temporal lag between gaze fixation and foot placement (heel contact) on the fixated area. Gaze fixations were classified according to the number of steps participants looked ahead (Fig. 8.3b). For gaze fixations that occurred on the ongoing step, participants were considered to be using the visual information on-line. For gaze fixations that occurred on one or more steps ahead, participants were considered to be using the visual information in a feedforward mode.

In the visual cues condition, both people with PD and healthy participants fixated close to 46 % on visual cues necessary to accomplish the ongoing step, while 54 % of fixations were focused on visual cues one or more steps ahead (Fig. 8.4a). These findings suggest a relatively equal distribution of on-line and feedforward control of gait that does not differ between PD and healthy participants. As expected, individuals with PD walked with shorter steps than healthy controls at baseline condition. When the exproprioception was removed, participants showed the same step length. Interestingly, a similar improvement in step length was evident for people with PD in both cued conditions, with and without exproprioception (Fig. 8.4b). These findings provide evidence that exproprioceptive information is not crucial for gait improvements achieved with visual cues in PD. In other words, people with PD do not have to look at their foot stepping on the cue in order to benefit from visual cues. Thus, environmental information (or exteroceptive information) is the critical piece of visual information to achieve step length benefits with visual cues in people with PD. In addition, we observed that both groups increased step time variability in the two cued conditions relative to the baseline condition (Fig. 8.4c). Increased gait variability is associated with a less automatic control of steps [37, 38], and, therefore, we suggest that participants were planning each step individually in the cued conditions, with a more conscious and hence variable control of gait.

Visual cues seem to work as external drivers that facilitate a compensatory shift to a goal-directed mode of action control. It has been argued that, when patients with PD are instructed to step on the lines, attention is driven to the stepping process [33, 39]. If this is the case, cueing would require increased cortical brain activation, mainly in the prefrontal area. In agreement with this interpretation, Koenraadt et al. [40] observed increased activation in the prefrontal cortex during precision stepping (stepping on consecutive targets) in young adults; these authors suggested that more attention was needed to perform precision stepping in comparison to normal walking. However, it is still to be confirmed in PD. A protocol including measures

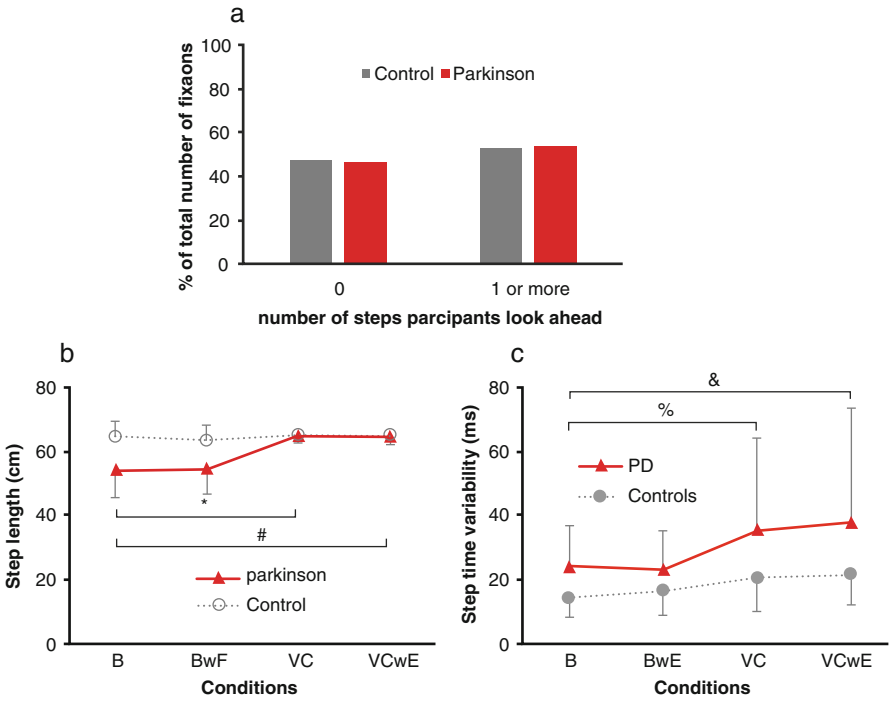

Fig. 8.4 Key findings of experiment #2. (**a**) Number of occurrences as a function of the number of steps participants looked ahead in the visual cues condition. (**b**) Step length values for each group on each experimental condition; B, baseline; BwE, baseline without exproprioception; VC, visual cue; VCwE, visual cue without exproprioception; * indicates significant difference between baseline and visual cue condition for people with PD; # indicates significant difference between baseline and visual cue without exproprioception condition for people with PD; (**c**) Step time variability values for each group on each experimental condition; % indicates significant difference between baseline and visual cue condition for both groups; & indicates significant difference between baseline and visual cue without exproprioception condition for both groups

of cortical brain activation (e.g., electroencephalography or functional near-infrared spectroscopy) during both normal and cued walking is recommended to address this gap in the literature.

8.5 Experiment #3: Temporal Relationship Between Gaze and Stepping Patterns in People with PD (A Precision Stepping Task)

It is well established that people with PD demonstrate a greater risk of falling [41], and tripping over obstacles is one of the major causes of falls in this clinical population [42]. As complex environments require visually guided foot placement on specific locations for safe locomotion, an impaired ability to sample visual

information from the environment might be a contributing factor to tripping and falling. Previous research has shown that inappropriate gaze strategies used by older adults contribute to inaccurate feet placement to meet environmental demands during adaptive walking and increase the risk of falling. For example, Chapman and Hollands [9, 10] observed that in a cluttered environment, older adults prone to falling prioritize the planning of future actions over the accurate execution of ongoing stepping movements. As part of experiment #3 [43], we investigated whether this would also apply to people with PD.

Participants included people with PD ($n = 24$) and age-matched healthy individuals ($n = 18$). They were asked to walk at preferred speed along a pathway 8 m long with foot placement targets positioned in the central area (Fig. 8.5). Two conditions were tested: The "one target" condition required participants to place their right foot onto target 1; the "two target" condition required participants to place their right foot onto target 1 and their left foot onto target 2. Participants were instructed to place each foot accurately in the central area of the targets. Foot 3-D kinematic data and gaze behavior were recorded, with two systems being synchronized. Outcome measures included absolute error (accuracy; measured as the distance between the center of the foot from the center of target 1) of foot placement onto target 1 and the time interval between the gaze transfer away from target 1 and foot contact on the same target. This protocol allowed us to determine the timing relationships between participants' eye and stepping movements during target-directed gait.

When there was a single target in the travel path (one target condition), both groups fixated the target until after foot contact on the target. When challenged with an additional target (two target condition), both groups transferred their gaze from the first target prior to foot contact (Fig. 8.6a). Although it did not reach significance, it is important to note that people with PD transferred their gaze away from the first target 76 ms earlier than healthy individuals during the two target condition (people with PD: 193 ms; control group: 117 ms). That may have influenced foot placement accuracy. Interestingly, only people with PD decreased the accuracy of anterior-posterior foot placement onto the first target when there was more than one target in the pathway (Fig. 8.6b); healthy individuals completed the task with minimal anterior-posterior foot placement error irrespective of the task complexity. Premature gaze transfer was associated with decline in stepping accuracy

Fig. 8.5 Illustration of the two conditions of experiment #3

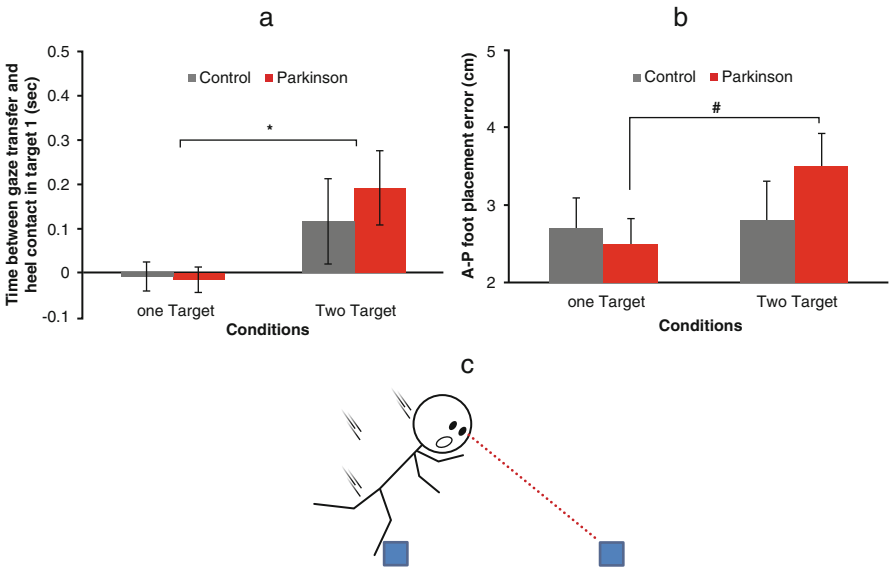

Fig. 8.6 (**a**) Time between gaze transfer and heel contact in the first target for each group on each condition; * indicates a significant difference between conditions (one vs. two targets) for both groups. (**b**) Anterior-posterior foot placement error for each group on each condition; # indicates a significant difference between conditions for people with PD only. (**c**) Illustration of tripping caused by an inappropriate early gaze deviation from the first of two obstacles in the pathway

(anterior-posterior absolute error) in people with PD only. These findings suggest that people with PD are more dependent than healthy individuals on visual information in an on-line manner to guarantee accurate foot placement onto intended stepping locations. Adoption of an early gaze deviation from stepping location, which is associated with loss of accuracy of stepping movements, may contribute to an increased likelihood of trips and falls in people with PD (Fig. 8.6c).

Findings from experiment #3 allow us to make one major suggestion for clinical practice. Improving gaze strategy (i.e., fixating the stepping location until after the foot had landed) should be a new target for interventions in PD. Such approach has potential to minimize foot placement errors and avoid tripping on obstacles during activities of real life, with ultimate impact on fall occurrences.

8.6 Conclusions and Future Directions

Putting all together, we have seen that:

(i) While performing a normal walking task or an obstacle avoidance task, people with PD require the same amount of visual information as do healthy individuals.

(ii) People with PD are more dependent on the quality of visual information available (e.g., optic flow, even for intermittent periods) for successful performance of obstacle avoidance.
(iii) People with PD use visual information from visual cues in both on-line and feedforward fashions.
(iv) Exteroceptive information (e.g., distance between stripes and position of foot placement location) is critical to achieving step length benefits with visual cues in PD.
(v) People with PD are more dependent than healthy individuals on visual information in an on-line manner (i.e., looking at the target location until the moment of foot placement) to guarantee accurate foot placement on intended stepping locations.

Although the three experiments described above and others in the literature provide insights into relevant aspects of visual control of locomotion in people with PD, much work remains to be done in this research area. Since cognitive impairments are common in PD and likely affect visual sampling, future research should focus on the contribution of cognitive resources to visual sampling by patients with PD during real-world walking activities. It is important to keep in mind that simulating real-world walking tasks in the lab may not be enough to observe natural behavior of patients, which limits the interpretation of findings. Therefore, future research should assess patients while performing walking tasks in the home or community environments.

References

1. Patla AE. How is human gait controlled by vision? Ecol Psychol. 1998;10:287–302.
2. Rhea CK, Rietdyk S. Visual exteroceptive information provided during obstacle crossing did not modify the lower limb trajectory. Neurosci Lett. 2007;418:60–5.
3. Marigold DS. Role of peripheral visual cues in online visual guidance of locomotion. Exerc Sport Sci Rev. 2008;36(3):145–51.
4. Patla AE, Prentice SD, Robinson C, Neufeld J. Visual control of locomotion: strategies for changing direction and for going over obstacles. J Exp Psychol Hum Percept Perform. 1991;17:603–34.
5. Patla AE. Understanding the roles of vision in the control of human locomotion. Gait Posture. 1997;5:54–69.
6. Marigold DS, Patla AE. Gaze fixation patterns for negotiating complex ground terrain. Neuroscience. 2007;144:302–13.
7. Hansen DW, Ji Q. In the eye of the beholder: a survey of models for eyes and gaze. IEEE Trans Pattern Anal Mach Intell. 2010;32:478–500.
8. Stuart S, Alcock L, Galna B, Lord S, Rochester L. The measurement of visual sampling during real-world activity in Parkinson's disease and healthy controls: a structured literature review. J Neurosci Methods. 2014;222:175–88.
9. Chapman GP, Hollands MA. Evidence for a link between changes to gaze behaviour and risk of falling in older adults during adaptive locomotion. Gait Posture. 2006;24:288–94.

10. Chapman GP, Hollands MA. Evidence that older adult fallers prioritise the planning of future stepping actions over the accurate execution of ongoing steps during complex locomotor tasks. Gait Posture. 2007;26:59–67.
11. Galna B, Lord S, Daud D, Archibald N, Burn D, Rochester L. Visual sampling during walking in people with Parkinson's disease and the influence of environment and dual-task. Brain Res. 2012;1473:35–43.
12. Morris M, Iansek R, Mcginley J, Matyas T, Huxham F. Three-dimensional gait biomechanics in Parkinson's disease: evidence for a centrally mediated amplitude regulation disorder. Mov Disord. 2005;20:40–50.
13. Yang YR, Lee YY, Cheng SJ, Lin PY, Wang RY. Relationships between gait and dynamic balance in early Parkinson's disease. Gait Posture. 2008;27:611–5.
14. Vitório R, Pieruccini-Faria F, Stella F, Gobbi S, Gobbi LTB. Effects of obstacle height on obstacle crossing in mild Parkinson's disease. Gait Posture. 2010;31:143–6.
15. Bloem BR, Beckley DJ, Van Dijk JG, Zwinderman AH, Remler MP, Roos RC. Influence of dopaminergic medication on automatic postural responses and balance impairment in Parkinson's disease. Mov Disord. 1996;11:509–21.
16. McVey MA, Stylianou AP, Luchies CW, Lyons KE, Pahwa R, Jernigan S, et al. Early biomechanical markers of postural instability in Parkinson's disease. Gait Posture. 2009;30 (4):538–42.
17. Hausdorff JM, Schweiger A, Herman T, Yogev-Seligmann G, Giladi N. Dual-task decrements in gait: contributing factors among healthy older adults. J Gerontol A Biol Sci Med Sci. 2008;63(12):1335–43.
18. Montero-Odasso M, Bergman H, Phillips NA, Wong CH, Sourial N, Chertkow H. Dual-tasking and gait in people with mild cognitive impairment. The effect of working memory. BMC Geriatr. 2009;1:9–41.
19. Davidsdottir S, Cronin-Golomb A, Lee A. Visual and spatial symptoms in Parkinson's disease. Vision Res. 2005;45:1285–96.
20. Chambers JM, Prescott TJ. Response times for visually guided saccades in persons with Parkinson's disease: a meta-analytic review. Neuropsychologia. 2010;48(4):887–99.
21. van Stockum S, MacAskill MR, Anderson TJ. Impairment of voluntary saccades and facilitation of reflexive saccades do not co-occur in Parkinson's disease. J Clin Neurosci. 2012;19 (8):1119–24.
22. Anderson TJ, MacAskill MR. Eye movements in patients with neurodegenerative disorders. Nat Rev Neurol. 2013;9(2):74–85.
23. Botha H, Carr J. Attention and visual dysfunction in Parkinson's disease. Parkinsonism Relat Disord. 2012;18:742–7.
24. Azulay JP, Mesure S, Amblard B, Blin O, Sangla I, Pouget J. Visual control of locomotion in Parkinson's disease. Brain. 1999;122:111–20.
25. Azulay JP, Mesure S, Blin O. Influence of visual cues on gait in Parkinson's disease: contribution to attention or sensory dependence? J Neurol Sci. 2006;248(1-2):192–5.
26. Davidsdottir S, Wagenaar R, Young D, Cronin-Golomb A. Impact of optic flow perception and egocentric coordinates on veering in Parkinson's disease. Brain. 2008;131(Pt 11):2882–93.
27. Cowie D, Limousin P, Peters A, Day BL. Insights into the neural control of locomotion from walking through doorways in Parkinson's disease. Neuropsychologia. 2010;48(9):2750–7.
28. Patla AE, Adkin A, Martin C, Holden R, Prentice S. Characteristics of voluntary visual sampling of the environment for safe locomotion over different terrains. Exp Brain Res. 1996;112(3):513–22.
29. Galna B, Murphy AT, Morris ME. Obstacle crossing in people with Parkinson's disease: foot clearance and spatiotemporal deficits. Hum Mov Sci. 2010;29:843–52.
30. Vitório R, Lirani-Silva E, Barbieri FA, Raile V, Batistela RA, Stella F, et al. The role of vision in Parkinson's disease locomotion control: free walking task. Gait Posture. 2012;35:175–9.
31. Vitório R, Lirani-Silva E, Barbieri FA, Raile V, Stella F, Gobbi LTB. Influence of visual feedback sampling on obstacle crossing behavior in people with Parkinson's disease. Gait Posture. 2013;38:330–4.

32. Konczak J, Corcos DM, Horak F, Poizner H, Shapiro M, Tuite P, et al. Proprioception and motor control in Parkinson's disease. J Mot Behav. 2009;41:543–52.
33. Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease: normalization strategies and underlying mechanisms. Brain. 1996;119:551–68.
34. Lebold CA, Almeida QJ. An evaluation of mechanisms underlying the influence of step cues on gait in Parkinson's disease. J Clin Neurosci. 2011;18:798–802.
35. Spaulding SJ, Barber B, Colby M, Cormack B, Mick T, Jenkins ME. Cueing and gait improvement among people with Parkinson's disease: a meta-analysis. Arch Phys Med Rehabil. 2013;94:562–70.
36. Vitório R, Lirani-Silva E, Pieruccini-Faria F, Moraes R, Gobbi LTB, Almeida QJ. Visual cues and gait improvement in Parkinson's disease: which piece of information is really important? Neuroscience. 2014;277:273–80.
37. Hausdorff JM, Balash J, Giladi N. Effects of cognitive challenge on gait variability in patients with Parkinson's disease. J Geriatr Psychiatry Neurol. 2003;16:53–8.
38. Hausdorff JM. Gait variability: methods, modeling and meaning. J Neuroeng Rehabil. 2005;2–9.
39. Morris ME, Iansek R, Matyas TA, Summers JJ. Ability to modulate walking cadence remains intact in Parkinson's disease. J Neurol Neurosurg Psychiatry. 1994;57(12):1532–4.
40. Koenraadt KL, Roelofsen EG, Duysens J, Keijsers NL. Cortical control of normal gait and precision stepping: an fNIRS study. Neuroimage. 2014;85(Pt 1):415–22.
41. Allen NE, Schwarzel AK, Canning CG. Recurrent falls in Parkinson's disease: a systematic review. Parkinsons Dis. 2013;2013:906274.
42. Ashburn A, Stack E, Ballinger C, Fazakarley L, Fitton C. The circumstances of falls among people with Parkinson's disease and the use of falls diaries to facilitate reporting. Disabil Rehabil. 2008;30:1205–12.
43. Vitório R, Gobbi LTB, Lirani-Silva E, Moraes R, Almeida QJ. Synchrony of gaze and stepping patterns in people with Parkinson's disease. Behav Brain Res. 2016;307:159–64.

Gait Disturbances in Movement Disorders: A Motor-Cognitive Problem

9

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Abstract

Individuals with movement disorders (MD) have gait deficits including impaired automaticity and instability. Despite specific impairments in motor control networks, recent evidence suggests that cognitive deficits are common in individuals with MD and can magnify their gait problems. In this chapter, we present studies showing that locomotor difficulties among individuals with MD are mainly observed in those with cognitive deficits and during complex walking situations in which demands in cognitive and sensorimotor processing increase. Overall, studies demonstrate that gait disturbances and cognitive impairment coexist in MD and together accelerate the loss of mobility in these individuals.

Keywords

Movement disorders • Elderly • Gait • Cognition • Motor control • Falls • Mobility • Motor planning

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9.1 The Relationship Between Gait and Cognition in Movement Disorders

Movement disorders (MD) result from dysfunction of subcortical areas, such as the basal ganglia (BG), as well as cortical areas within the brain. Neurodegenerative processes can compromise the automaticity of well-learned movements such as gait, where increased conscious control (and hence cognitive systems) may be relied upon to perform movements. The relationship between cognitive control and movement performance is often observed when individuals with MD have their attention divided by secondary tasks while walking. As a result of divided attention, walking performance in those with MD can be profoundly impaired compared to healthy individuals. In addition, individuals with MD who have cognitive decline appear to be more susceptible to gait impairments due to their inability to use cognitive resources required to plan and control movements. During complex walking situations (e.g., cluttered environment), the increased cognitive and sensory processing required to plan gait modifications may have a strong impact on the walking performance of individuals with MD. Thus, abnormal gait control while walking in complex situations may result from an overloaded or inefficient cognitive system in MD. In this chapter we review different aspects of gait control associated with cognition in different MD, and in different walking contexts.

9.2 Parkinson's Disease

Parkinson's disease (PD) is the most prevalent MD. The incidence of PD is nearly 1 % in the population and 2 % in people over 60 years of age [1–3]. PD is characterized by an accelerated degenerative process of dopaminergic neurons in the *substantia nigra pars compacta* of the BG. Decreased striatal dopamine affects the functioning of all structures which comprise the BG and consequently impairs the control of well-learned movements, such as gait [4]. In PD, gait impairments are characterized by reductions in step length and velocity, and also increased step-to-step variations in time and distance (i.e., gait variability) [5–7]. These gait impairments may be even greater when individuals with PD have their attention divided by secondary tasks during walking [8–14].

Specifically, velocity and gait variability are the most sensitive gait parameters to dual-task interference in PD. Although the diversity of dual tasks found in the literature makes comparison between studies difficult, gait of individuals with PD is more influenced during the performance of more complex secondary tasks. For example, gait impairments are worse when individuals with PD walk while subtracting by 7's compared to subtracting by 3's [9], or when they are asked to carry a tray with glasses compared to a tray without glasses [12], or when those glasses are full compared to empty glasses [13]. Among gait parameters affected by dual-task interference, gait variability has been shown to be a sensitive measure when dual-task complexity has been increased and may represent the processing

overload created by these tasks. It is important to note, however, that the relationship between gait and cognition may vary depending on the cognitive status and specific motor symptoms (e.g., freezing of gait) of individuals with PD. These aspects will be further discussed in the sections below.

9.3 The Influence of Cognitive Decline on Gait of Individuals with Parkinson's Disease

Previous research has shown that approximately 26 % of non-demented individuals with PD may exhibit a form of mild cognitive impairment, with main deficits being found in the executive/attentional, memory, and visuospatial domains [15]. In addition, a longitudinal study demonstrated that by the 10-year mark from diagnosis, approximately 46 % of individuals with PD had been clinically diagnosed with dementia [16]. These findings support the notion that motor and cognitive dysfunction may coexist in PD even in the early stages of disease and increase in severity in the later stages of disease progression. Interestingly, it has been shown that individuals with predominant postural instability and gait dysfunction (PIGD) have faster rate of decline in both cognitive and motor function than those with predominant tremor symptoms [17]. This selective association between cognitive impairment and PIGD symptoms suggests that the progression of cognitive and mobility deficits may share similar underlying mechanisms in PD.

Early studies investigated the influence of specific cognitive functions on gait in PD. Deficits in attention and executive functions were associated with impaired gait in individuals with PD, especially during dual-task walking [9, 18]. In contrast to research in older adults with mild cognitive impairment [19], performance on memory tests were not associated with changes in gait in PD. These findings support the notion that specific cognitive functions (i.e., attention and executive functions) are associated with gait disturbances in PD.

Executive functions compose a cognitive domain required for our ability to inhibit irrelevant information, to update or maintain relevant information in memory, to plan complex actions, and to general attentional control [20]. These functions allow the adaptation of responses to novel situations and play an important role when managing situations in which automatic responses are not sufficient for optimal performance. Thus, deficits in executive functions may interfere with the ability of individuals with PD to adapt gait when dealing with complex environments (e.g., cluttered spaces) or performing multiple tasks.

With respect to gait, poor performance on tests assessing executive functions has been associated specifically with reduction in speed and increased step-to-step variability when individuals with PD perform dual-task walking [9]. According to Yogev and colleagues [9], individuals with PD may use cognitive resources to alleviate difficulties to maintain the consistency of walking rhythm. Hence, if individuals with PD present impaired cognitive function (e.g., executive functions), it is reasonable to predict that they may be less able to maintain a stable walking

rhythm. Since unstable walking rhythm has been associated with increased risk of falls in older adults [21], this may imply greater risk of falls among individuals with PD with cognitive decline compared to those with normal cognitive status.

In addition to the findings described above, a relationship between cognition and a very debilitating symptom known as freezing of gait has also been identified in individuals with PD. The role of cognitive deficits in the freezing phenomenon is described in greater detail in the following section.

9.4 The Relationship Between Cognition and Freezing of Gait in Parkinson's Disease

Freezing of gait (FOG) is a phenomenon that affects nearly 50 % of all individuals with PD, being more prevalent during later stages of the disease [22, 23]. FOG can be defined as a sudden interruption of walking progression which makes individuals feel as if their feet are "glued" to the ground, irrespective of their intention to move forward [24]. Several perspectives have attempted to explain this phenomenon in PD including reduced cognitive processing capacity [25, 26], impaired bilateral coordination [27], impaired sensory-perceptual processing [28–30], motor planning deficits [31–33], asymmetric degeneration of subcortical areas (e.g., BG and pedunculopontine nucleus) [34, 35], and altered emotional processing [36, 37]. Individuals with PD who experience FOG present typical gait disturbances such as increased step-to-step variability [38], shortened step length [31], and asymmetrical steps [27]. Importantly, when individuals with PD who experience FOG have to adapt their walking pattern to change direction (turning), deal with obstacles (doorways) or situations that impose greater cognitive demand (dual-task), these gait abnormalities are exacerbated and freezing episodes are more likely to occur [28, 39–41]. Although the underlying mechanisms of FOG are unknown, research has consistently demonstrated an association between FOG and deficits in executive functions. More specifically, FOG has been associated with difficulties in attentional set-shifting [42] and conflict resolution [26]. In addition, the notion that deficits in cognitive networks could contribute to FOG has been supported by neuroimaging research.

Brain imaging studies in virtual reality environments have exposed patients to conditions which elicit FOG episodes, for example, locomotion through narrow spaces in addition to attention demanding dual-tasks [25]. It was found that an overload in attentional networks during these FOG provoking situations increased the inhibitory output from BG to motor centers in the brainstem in individuals with PD, such as the mesencephalic locomotor area and the pedunculopontine nucleus, which are areas responsible for the initiation and cessation of locomotion in humans [43]. However, this abnormal inhibitory output from BG to locomotor centers in the brainstem was stronger among freezers compared to non-freezers. Results from imaging studies may help to explain why complex walking situations which demand increased cognitive control and sensorimotor integration for stepping adaptations elicit FOG episodes. Thus, understanding how complex walking

situations influence gait in PD help us better understand the underlying mechanisms of severe gait impairments such as FOG, as well as clarifying the relationship between cognition and gait in PD.

9.5 Cognition and Complex Gait Navigation in Parkinson's Disease

Walking in real-world situations often requires step adaptations to change directions and to avoid contact with obstacles. These gait modifications impose increased demand on cognitive and sensory processing. Reduced striatal dopaminergic activity may limit processing resources magnifying motor disturbances when individuals with PD face complex locomotor situations including obstacle avoidance [44, 45]. Consequently, during these demanding locomotor situations, gait impairments can be exacerbated (slowness, FOG, trips, and the loss of balance) in individuals with PD. Recent studies have shown that approaching obstacles, a situation that demands planning, can overload cognitive processing and exacerbates gait impairments (e.g., increased step-to-step variability) in individuals with PD [28, 29, 32, 33]. Additionally, executive function decline is associated with erroneous stepping planning during obstacle crossing [32]. Planning refers to the preparation/conceptualization of movements. Importantly, gait disturbances associated with increased motor planning complexity were found to be exacerbated in freezers especially when they performed a secondary cognitive task during obstacle approaching and crossing. Another recent study also showed that decreased visual feedback of self-motion overloaded processing resources of individuals with PD increasing the effects of dual-task on gait control [46]. This study showed that individuals with PD tripped more on a visible obstacle when they needed to rely on their impaired proprioceptive system (walking in the dark) and perform a cognitive dual-task compared to healthy individuals. Gait instability (step-to-step variability) during obstacle approach in individuals with PD was higher than in healthy controls only during the condition of reduced visual feedback of self-motion (dark). This result suggests that processing resources to plan gait adaptations may become overloaded because individuals with PD would be using cognition (e.g., attention) to supervise faulty sensorimotor integration to prevent motor and planning errors. Difficulties to automatically process sensory information could force patients to control movements using a more conscious control [47] which can be observed in increased gait variability and dual-task cost while approaching the obstacle. In support of this interpretation, previous research has shown that when individuals with PD are trained to focus attention on sensorimotor information (moving with eyes closed), a significant improvement in gait was found during a complex walking task involving postural threat [48]. Hence, attention might be used by individuals with PD to compensate for sensory-perceptual impairments. Thus, it is important to consider the relationship between impaired sensorimotor integration and the reliance on high-order cognitive processing (e.g., executive functions) in individuals with PD.

Another complex locomotor situation which results in gait difficulties among individuals with PD is turning. Difficulties to turn around the body axis are one of the most common complaints among people with PD, and it may cause extreme gait slowness, loss of balance, and FOG. It has been demonstrated that individuals with FOG had greater gait variability than non-freezers when performing sharper turns (180°) compared wider turns (90°) [40]. In this study, the gait abnormalities found in freezers were suggested to be associated with increased cognitive demand to control steps during sharper turns. Another study demonstrated that freezers had increased brain activity in prefrontal areas compared to healthy age-matched controls only prior to a planned turn compared to an unplanned turn. This study suggested that turning deficits in individuals with FOG may be related to an overload in cognitive processing during movement planning [49]. Therefore, in individuals with PD and especially those with FOG, processing capacity may be overloaded when planning complex gait adjustments. This overload in processing capacity may be attributed to both reduced striatal dopaminergic activity (i.e., reduced BG processing capacity) and impaired cognitive function (executive functions) which may magnify sensorimotor deficits (e.g., slowness, instability) while performing drastic gait and postural adjustments during locomotion.

9.6 Falls and Cognition in Parkinson's Disease

Falls are one of the main causes of hospitalization, decreased mobility, and poor quality of life among individuals with PD [50, 51]. Although the underlying mechanism of falls in PD may be multifactorial, a relationship between cognition and increased risk of falls has been previously demonstrated [52, 53]. Interestingly, a study showed that individuals with PD and elderly fallers have common deficits in executive functions that distinguish them from elderly non-fallers [54]. As previously described, impairments in executive functions may contribute to an inability to adapt gait especially during complex walking situations and may expose individuals with PD to greater risk of falls. In addition to cognitive decline, individuals with PD who have a high incidence of falls also present greater step-to-step variability compared to those with low incidence of falls [55]. Therefore, deficits in executive functions and gait stability (e.g., increased step-to-step variability) are hallmarks of PD fallers [56].

Although the degeneration of the dopaminergic system plays a critical role on gait impairments in PD, recent research has shown that individuals with PD who frequently fall had reduced thalamic cholinergic activity compared to PD non-fallers [57]. Thalamic cholinergic activity is modulated by a brainstem structure called pedunculopontine nucleus, which plays an important role on gait and balance control [43]. Therefore, it might be that impaired cholinergic activity in the pedunculopontine nucleus may also contribute to gait impairments and increased risk of falls among those with PD. This is in line with previous research showing that gait parameters important to gait stability, such as stride time and double support variabilities, are not responsive to dopaminergic medication [58, 59].

In support of the assumption that cholinergic and dopaminergic systems have different but not independent roles in gait control, a recent review [60] identified that falls' incidence in PD may increase if cognitive networks modulated by the cholinergic system are not able to supervise errors in sensorimotor integration, which is primarily modulated by the dopaminergic system. In other words, falls may reflect an inability of individuals with PD to use attentional resources to supervise sensorimotor feedback.

9.7 Huntington's Disease

Huntington's disease (HD) is an MD caused by progressive degeneration of the striatum (i.e., caudate nucleus and putamen). The putamen and the caudate nucleus are important neural structures for sensorimotor [61] and cognitive [62] processing, respectively. Impairments in these subcortical structures affect the ability of human beings to suppress motor outputs and behaviors. In addition to subcortical deficits, cortical areas are also affected by HD. The combination of subcortical and cortical impairments can cause significant cognitive and behavioral deficits, in addition to severe movement disturbances. Individuals with HD exhibit hyperkinetic and involuntary movements (chorea) in later stages of the disease. The pathological mechanism underlying movement impairments in HD also have an important influence on gait.

Gait characteristics in HD include wide base of support, lateral swaying, spontaneous knee flexion, variable cadence, and parkinsonian features [63]. Although individuals with HD present some parkinsonian gait characteristics (e.g., variability and slowness), the abnormal gait pattern observed in HD is not associated with deficits to regulate the amplitude of steps, such as in PD. Individuals with HD exhibit difficulties to maintain a consistent timing or pace between steps. This increased gait variability of step timing in individuals with HD may be indicative of poor stability and reduced gait automaticity.

Due to extensive subcortical and cortical damage, it is challenging to establish causality between striatum damage and cognitive deficits in individuals with HD. Previous research has shown that cognitive deficits found in individuals with HD differ from those observed in Alzheimer's disease, which is a neurological disease known to affect primarily cortical areas [64]. For example, Brandt and colleagues [64] showed that while individuals with Alzheimer's disease showed deficits in memory (word recall) and orientation (date) sections of the Mini-Mental State Exam, those with HD were more impaired in executive functions (serial subtractions). More specifically, Lawrence et al. [65] found that individuals with HD present a specific pattern of deficits in executive functions such as planning and set-shifting. The pattern of deficits in executive functions observed in individuals with HD differed from that found in individuals with PD (subcortical), but it was similar to individuals with frontal lobe lesions (cortical). Thus, even though cognitive deficits in HD may be primarily caused by striatal dysfunction, cortical dysfunction as a result of widespread lesions in the brain may also contribute to

these cognitive deficits. If this is the case, it might be that the ability of individuals with HD to compensate for gait deficits through the cognitive system may be limited especially when cognitive resources are challenged while walking.

A study revealed that a cognitive dual-task (counting backwards in steps of 2 or 3) affected gait in individuals with HD more than a motor task (carrying a tray with glasses) [66]. The cognitive dual-task provoked slower gait speed, decreased cadence, and shorter stride length (all compared to free gait condition), whereas the same dual-task interference was not observed in healthy control individuals. This study showed that individuals with HD depend on attentional resources to control their steps while walking. Given that attentional control over steps should improve gait impairments in HD, directing attention to a metronome while walking should improve the timing control of steps. However, a study demonstrated that individuals with HD did not improve gait under these conditions [67]. According to this study, the lack of gait improvement while external cues were provided to individuals with HD was due to attentional deficits. Thus, attentional deficits may affect the ability of these individuals to synchronize their steps with external cues (e.g., auditory signals produced by a metronome).

Taken together, the severity of cognitive deficits in HD may influence gait and prevent these individuals from utilizing cognitive resources to compensate for gait deficits.

9.8 Progressive Supranuclear Palsy

Progressive supranuclear palsy (PSP) is a MD caused by abnormal accumulation of tau protein-positive filamentous known as neurofibrillary tangles in the glia and neurons [68–70]. This abnormal neuronal formation is found to be more concentrated in areas of the brainstem responsible for gaze and gait control. Since motor symptoms in PSP and PD are very similar, the absence of downgaze is the most important criterion to differentiate PSP from PD. Movement impairments observed in individuals with PSP consist of the absence of downgaze movements, staring look due to eyelid retraction, trunk rigidity, bradykinesia, hypometric movements, and FOG. Deficits in gait and postural control are thought to be related to an increased number of falls among those with PSP [71]. In addition, when compared to PD, individuals with PSP may exhibit shorter step length and increased cadence when asked to walk fast [72]. These walking patterns suggest that gait deficits in PSP may result from difficulties setting an appropriate step length to modulate gait speed at the cortical level. Additionally, cortical areas in the brain that underlie executive functions are also affected by PSP, posing a complex scenario which involves the deterioration of both gait and cognitive functions.

Cognitive deficits in individuals with PSP are mainly characterized by slowness of thinking and impaired executive functions, although memory and visuospatial processing (e.g., difficulty to identify shapes and objects accurately) may also be impaired [73, 74]. Individuals with PSP also have impaired performance on cognitive tests sensitive to frontal lobe dysfunction. More specifically, Robbins and

colleagues [75] showed that individuals with PSP performed worse than age-matched controls on short-term memory, spatial working memory, planning, and set-shifting tests. Although the pattern of deficits found in individuals with PSP resemble those observed in individuals with PD, deficits were found to be more severe in individuals with PSP when compared to those with PD [75]. Since PSP is a rare MD, a small number of studies investigating the relationship between gait and cognition exist in the literature.

Previous research has shown that individuals with PSP who fall more frequently were more affected by a cognitive-motor task (counting backwards) than those with a low incidence of falls [76]. This study concluded that gait impairments and falls may result from a combination of deficits in functioning of the brainstem and prefrontal cortical regions in individuals with PSP. Furthermore, a recent study showed that individuals with PSP with high incidence of falls demonstrated greater impairment in executive and visuospatial functions compared to those with a lower incidence of falls [71]. These findings suggest that impairments in gait and balance in individuals with PSP may be strongly influenced by cognition and may affect the ability of individuals with PSP to navigate safely.

Although the relationship between cognition, gait impairments, and falls in PSP seems obvious, the contribution of downgaze impairments to falls incidence remains unclear and might have been a confounder in previous investigations. Downgaze deficits could have a negative effect on planning of foot elevation when individuals with PSP walk on uneven terrains. Furthermore, difficulties to shift gaze to the lower visual field while walking could cause erroneous step adjustments consequently contributing to trips, loss of balance, and falls. Therefore, studies should investigate not only falls occurrence among individuals with PSP but also how these falls happened. For example, if the majority of falls happened after trips on uneven terrain, it is possible that downgaze impairments have important contribution to falls compared to situations where patients were distracted while walking on even terrains. However, this hypothesis still needs to be confirmed in future studies.

9.9 Final Considerations

In conclusion, individuals with PD, HD, or PSP may present similar gait deficits that could be caused by decreased movement automaticity. Consequently, individuals with these MD may rely on the cognitive system to compensate for gait deficits. The coexistence of both motor and cognitive deficits in most MD is important to note. Furthermore, the severity of cognitive deficits, such as attention, may limit the ability of those with MD to adapt gait in complex situations and utilize sensory information to improve gait. Therefore, rehabilitation programs should combine motor and cognitive therapies to improve gait in MD.

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References

1. Alves G, Forsaa EB, Pedersen KF, Dreetz Gjerstad M, Larsen JP. Epidemiology of Parkinson's disease. J Neurol. 2008;255 Suppl 5:18–32.
2. Rajput AH, Birdi S. Epidemiology of Parkinson's disease. Parkinsonism Relat Disord. 1997;3 (4):175–86.
3. de Lau LM, Breteler MM. Epidemiology of Parkinson's disease. Lancet Neurol. 2006;5 (6):525–35.
4. Poldrack RA, Sabb FW, Foerde K, Tom SM, Asarnow RF, Bookheimer SY, et al. The neural correlates of motor skill automaticity. J Neurosci. 2005;25(22):5356–64.
5. Morris ME, Iansek R, Matyas TA, Summers JJ. The pathogenesis of gait hypokinesia in Parkinson's disease. Brain. 1994;117(Pt 5):1169–81.
6. Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms. Brain. 1996;119(Pt 2):551–68.
7. Rochester L, Baker K, Nieuwboer A, Burn D. Targeting dopa-sensitive and dopa-resistant gait dysfunction in Parkinson's disease: selective responses to internal and external cues. Mov Disord. 2011;26(3):430–5.
8. Hausdorff JM, Balash J, Giladi N. Effects of cognitive challenge on gait variability in patients with Parkinson's disease. J Geriatr Psychiatry Neurol. 2003;16(1):53–8.
9. Yogev G, Giladi N, Peretz C, Springer S, Simon ES, Hausdorff JM. Dual tasking, gait rhythmicity, and Parkinson's disease: which aspects of gait are attention demanding? Eur J Neurosci. 2005;22(5):1248–56.
10. Baker K, Rochester L, Nieuwboer A. The immediate effect of attentional, auditory, and a combined cue strategy on gait during single and dual tasks in Parkinson's disease. Arch Phys Med Rehabil. 2007;88(12):1593–600.
11. Rochester L, Hetherington V, Jones D, Nieuwboer A, Willems AM, Kwakkel G, et al. Attending to the task: interference effects of functional tasks on walking in Parkinson's disease and the roles of cognition, depression, fatigue, and balance. Arch Phys Med Rehabil. 2004;85 (10):1578–85.
12. Rochester L, Hetherington V, Jones D, Nieuwboer A, Willems AM, Kwakkel G, et al. The effect of external rhythmic cues (auditory and visual) on walking during a functional task in homes of people with Parkinson's disease. Arch Phys Med Rehabil. 2005;86(5):999–1006.
13. Bond JM, Morris M. Goal-directed secondary motor tasks: their effects on gait in subjects with Parkinson disease. Arch Phys Med Rehabil. 2000;81(1):110–6.
14. O'Shea S, Morris ME, Iansek R. Dual task interference during gait in people with Parkinson disease: effects of motor versus cognitive secondary tasks. Phys Ther. 2002;82(9):888–97.
15. Aarsland D, Bronnick K, Williams-Gray C, Weintraub D, Marder K, Kulisevsky J, et al. Mild cognitive impairment in Parkinson disease: a multicenter pooled analysis. Neurology. 2010;75 (12):1062–9.
16. Williams-Gray CH, Mason SL, Evans JR, Foltynie T, Brayne C, Robbins TW, et al. The CamPaIGN study of Parkinson's disease: 10-year outlook in an incident population-based cohort. J Neurol Neurosurg Psychiatry. 2013;84(11):1258–64.
17. Burn DJ, Rowan EN, Allan LM, Molloy S, O'Brien JT, McKeith IG. Motor subtype and cognitive decline in Parkinson's disease, Parkinson's disease with dementia, and dementia with Lewy bodies. J Neurol Neurosurg Psychiatry. 2006;77(5):585–9.
18. Lord S, Rochester L, Hetherington V, Allcock LM, Burn D. Executive dysfunction and attention contribute to gait interference in 'off' state Parkinson's Disease. Gait Posture. 2010;31(2):169–74.
19. Montero-Odasso M, Oteng-Amoako A, Speechley M, Gopaul K, Beauchet O, Annweiler C, et al. The motor signature of mild cognitive impairment: results from the gait and brain study. J Gerontol A Biol Sci Med Sci. 2014;69(11):1415–21.

20. Miyake A, Friedman NP, Emerson MJ, Witzki AH, Howerter A, Wager TD. The unity and diversity of executive functions and their contributions to complex "Frontal Lobe" tasks: a latent variable analysis. Cogn Psychol. 2000;41(1):49–100.
21. Springer S, Giladi N, Peretz C, Yogev G, Simon ES, Hausdorff JM. Dual-tasking effects on gait variability: the role of aging, falls, and executive function. Mov Disord. 2006;21 (7):950–7.
22. Giladi N, Kao R, Fahn S. Freezing phenomenon in patients with parkinsonian syndromes. Mov Disord. 1997;12(3):302–5.
23. Giladi N. Freezing of gait. Clin overview Adv Neurol. 2001;87:191–7.
24. Nutt JG, Bloem BR, Giladi N, Hallett M, Horak FB, Nieuwboer A. Freezing of gait: moving forward on a mysterious clinical phenomenon. Lancet Neurol. 2011;10(8):734–44.
25. Shine JM, Matar E, Ward PB, Bolitho SJ, Gilat M, Pearson M, et al. Exploring the cortical and subcortical functional magnetic resonance imaging changes associated with freezing in Parkinson's disease. Brain. 2013;136(Pt 4):1204–15.
26. Vandenbossche J, Deroost N, Soetens E, Spildooren J, Vercruysse S, Nieuwboer A, et al. Freezing of gait in Parkinson disease is associated with impaired conflict resolution. Neurorehabil Neural Repair. 2011;25(8):765–73.
27. Plotnik M, Giladi N, Balash Y, Peretz C, Hausdorff JM. Is freezing of gait in Parkinson's disease related to asymmetric motor function? Ann Neurol. 2005;57(5):656–63.
28. Almeida QJ, Lebold CA. Freezing of gait in Parkinson's disease: a perceptual cause for a motor impairment? J Neurol Neurosurg Psychiatry. 2010;81(5):513–8.
29. Silveira CRA, Ehgoetz Martens KA, Pieruccini-Faria F, Bell-Boucher D, Roy EA, Almeida QJ. Disentangling perceptual judgment and online feedback deficits in Parkinson's freezing of gait. J Neurol. 2015;262(7):1629–36.
30. Ehgoetz Martens KA, Pieruccini-Faria F, Almeida QJ. Could sensory mechanisms be a core factor that underlies freezing of gait in Parkinson's disease? PLoS One. 2013;8(5), e62602.
31. Chee R, Murphy A, Danoudis M, Georgiou-Karistianis N, Iansek R. Gait freezing in Parkinson's disease and the stride length sequence effect interaction. Brain. 2009; 132:2151–60.
32. Pieruccini-Faria F, Jones JA, Almeida QJ. Motor planning in Parkinson's disease patients experiencing freezing of gait: the influence of cognitive load when approaching obstacles. Brain Cogn. 2014;87:76–85. Epub 2014/04/15.
33. Knobl P, Kielstra L, Almeida Q. The relationship between motor planning and freezing of gait in Parkinson's disease. J Neurol Neurosurg Psychiatry. 2012;83(1):98–101.
34. Pieruccini-Faria F, Ehgoetz Martens KA, Silveira CRA, Jones JA, Almeida QJ. Side of basal ganglia degeneration influences freezing of gait in Parkinson's disease. Behav Neurosci. 2015;29(2):214–8.
35. Fling BW, Cohen RG, Mancini M, Nutt JG, Fair DA, Horak FB. Asymmetric pedunculopontine network connectivity in parkinsonian patients with freezing of gait. Brain. 2013;136(Pt 8):2405–18.
36. Lieberman A. Are freezing of gait (FOG) and panic related? J Neurol Sci. 2006;248 (1-2):219–22.
37. Ehgoetz Martens KA, Ellard CG, Almeida QJ. Does anxiety cause freezing of gait in Parkinson's disease? PLoS One. 2014;9(9), e106561.
38. Hausdorff JM, Schaafsma JD, Balash Y, Bartels AL, Gurevich T, Giladi N. Impaired regulation of stride variability in Parkinson's disease subjects with freezing of gait. Exp Brain Res. 2003;149(2):187–94.
39. Nieuwboer A, Dom R, De Weerdt W, Desloovere K, Janssens L, Stijn V. Electromyographic profiles of gait prior to onset of freezing episodes in patients with Parkinson's disease. Brain. 2004;127(Pt 7):1650–60.
40. Bhatt H, Pieruccini-Faria F, Almeida QJ. Dynamics of turning sharpness influences freezing of gait in Parkinson's disease. Parkinsonism Relat Disord. 2013;19(2):181–5.

41. Spildooren J, Vercruysse S, Desloovere K, Vandenberghe W, Kerckhofs E, Nieuwboer A. Freezing of gait in Parkinson's disease: the impact of dual-tasking and turning. Mov Disord. 2010;25(15):2563–70.
42. Naismith SL, Shine JM, Lewis SJ. The specific contributions of set-shifting to freezing of gait in Parkinson's disease. Mov Disord. 2010;25(8):1000–4.
43. Pahapill PA, Lozano AM. The pedunculopontine nucleus and Parkinson's disease. Brain. 2000;123(Pt 9):1767–83.
44. Pieruccini-Faria F, Jones JA, Almeida QJ. Insight into dopamine-dependent planning deficits in Parkinson's disease: a sharing of cognitive & sensory resources. Neuroscience. 2016;318:219–29.
45. Pieruccini-Faria F, Vitorio R, Almeida QJ, Silveira CRA, Caetano MJ, Stella F, et al. Evaluating the acute contributions of dopaminergic replacement to gait with obstacles in Parkinson's disease. J Mot Behav. 2013;45(5):369–80.
46. Pieruccini-Faria F, Ehgoetz Martens KA, Silveira CRA, Jones JA, Almeida QJ. Interactions between cognitive and sensory load while planning and controlling complex gait adaptations in Parkinson's disease. BMC Neurol. 2014;14(1):250.
47. Redgrave P, Rodriguez M, Smith Y, Rodriguez-Oroz MC, Lehericy S, Bergman H, et al. Goal-directed and habitual control in the basal ganglia: implications for Parkinson's disease. Nat Rev Neurosci. 2010;11(11):760–72.
48. Sage MD, Almeida QJ. Symptom and gait changes after sensory attention focused exercise vs aerobic training in Parkinson's disease. Mov Disord. 2009;24(8):1132–8.
49. Maidan I, Bernad-Elazari H, Gazit E, Giladi N, Hausdorff JM, Mirelman A. Changes in oxygenated hemoglobin link freezing of gait to frontal activation in patients with Parkinson disease: an fNIRS study of transient motor-cognitive failures. J Neurol. 2015.
50. Gazibara T, Pekmezovic T, Tepavcevic DK, Tomic A, Stankovic I, Kostic VS, et al. Circumstances of falls and fall-related injuries among patients with Parkinson's disease in an outpatient setting. Geriatr Nurs. 2014;35(5):364–9.
51. Grimbergen YA, Munneke M, Bloem BR. Falls in Parkinson's disease. Curr Opin Neurol. 2004;17(4):405–15.
52. Amboni M, Barone P, Hausdorff JM. Cognitive contributions to gait and falls: evidence and implications. Mov Disord. 2013;28(11):1520–33.
53. Camicioli R, Majumdar SR. Relationship between mild cognitive impairment and falls in older people with and without Parkinson's disease: 1-Year Prospective Cohort Study. Gait Posture. 2010;32(1):87–91.
54. Hausdorff JM, Doniger GM, Springer S, Yogev G, Simon ES, Giladi N. A common cognitive profile in elderly fallers and in patients with Parkinson's disease: the prominence of impaired executive function and attention. Exp Aging Res. 2006;32(4):411–29.
55. Yogev G, Plotnik M, Peretz C, Giladi N, Hausdorff JM. Gait asymmetry in patients with Parkinson's disease and elderly fallers: when does the bilateral coordination of gait require attention? Exp Brain Res. 2007;177(3):336–46.
56. Amboni M, Barone P, Iuppariello L, Lista I, Tranfaglia R, Fasano A, et al. Gait patterns in Parkinsonian patients with or without mild cognitive impairment. Mov Disord. 2012;27 (12):1536–43.
57. Bohnen NI, Muller ML, Koeppe RA, Studenski SA, Kilbourn MA, Frey KA, et al. History of falls in Parkinson disease is associated with reduced cholinergic activity. Neurology. 2009;73 (20):1670–6.
58. Blin O, Ferrandez AM, Pailhous J, Serratrice G. Dopa-sensitive and dopa-resistant gait parameters in Parkinson's disease. J Neurol Sci. 1991;103(1):51–4.
59. Lord S, Baker K, Nieuwboer A, Burn D, Rochester L. Gait variability in Parkinson's disease: an indicator of non-dopaminergic contributors to gait dysfunction? J Neurol. 2011;258 (4):566–72.
60. Sarter M, Albin RL, Kucinski A, Lustig C. Where attention falls: increased risk of falls from the converging impact of cortical cholinergic and midbrain dopamine loss on striatal function. Exp Neurol. 2014;257:120–9.

61. Goble DJ, Coxon JP, Van Impe A, Geurts M, Van Hecke W, Sunaert S, et al. The neural basis of central proprioceptive processing in older versus younger adults: an important sensory role for right putamen. Hum Brain Mapp. 2012;33(4):895–908.
62. Lewis SJ, Dove A, Robbins TW, Barker RA, Owen AM. Striatal contributions to working memory: a functional magnetic resonance imaging study in humans. Eur J Neurosci. 2004;19 (3):755–60. Epub 2004/02/27.
63. Koller WC, Trimble J. The gait abnormality of Huntington's disease. Neurology. 1985;35 (10):1450–4.
64. Brandt J, Folstein SE, Folstein MF. Differential cognitive impairment in Alzheimer's disease and Huntington's disease. Ann Neurol. 1988;23(6):555–61.
65. Lawrence AD, Sahakian BJ, Robbins TW. Cognitive functions and corticostriatal circuits: insights from Huntington's disease. Trends Cogn Sci. 1998;2(10):379–88.
66. Delval A, Krystkowiak P, Delliaux M, Dujardin K, Blatt JL, Destee A, et al. Role of attentional resources on gait performance in Huntington's disease. Mov Disord. 2008;23(5):684–9.
67. Thaut MH, Miltner R, Lange HW, Hurt CP, Hoemberg V. Velocity modulation and rhythmic synchronization of gait in Huntington's disease. Mov Disord. 1999;14(5):808–19.
68. Esterhammer R, Schocke M, Seppi K. Basal ganglia cellular pathology in multiple system atrophy, progressive supranuclear palsy and Parkinson disease. Can quantitative magnetic resonance spectroscopic imaging make the difference? Eur J Neurol. 2010;17(9):1111–2.
69. Piao YS, Hayashi S, Wakabayashi K, Kakita A, Aida I, Yamada M, et al. Cerebellar cortical tau pathology in progressive supranuclear palsy and corticobasal degeneration. Acta Neuropathol. 2002;103(5):469–74.
70. Ghatak NR, Nochlin D, Hadfield MG. Neurofibrillary pathology in progressive supranuclear palsy. Acta Neuropathol. 1980;52(1):73–6.
71. Kim SL, Lee MJ, Lee MS. Cognitive dysfunction associated with falls in progressive supranuclear palsy. Gait Posture. 2014;40(4):605–9.
72. Egerton T, Williams DR, Iansek R. Comparison of gait in progressive supranuclear palsy Parkinson's disease and healthy older adults. BMC Neurol. 2012;12:116.
73. Burrell JR, Hodges JR, Rowe JB. Cognition in corticobasal syndrome and progressive supranuclear palsy: a review. Mov Disord. 2014;29(5):684–93.
74. Grafman J, Litvan I, Stark M. Neuropsychological features of progressive supranuclear palsy. Brain Cogn. 1995;28(3):311–20.
75. Robbins TW, James M, Owen AM, Lange KW, Lees AJ, Leigh PN, et al. Cognitive deficits in progressive supranuclear palsy, Parkinson's disease, and multiple system atrophy in tests sensitive to frontal lobe dysfunction. J Neurol Neurosurg Psychiatry. 1994;57(1):79–88.
76. Lindemann U, Nicolai S, Beische D, Becker C, Srulijes K, Dietzel E, et al. Clinical and dual-tasking aspects in frequent and infrequent fallers with progressive supranuclear palsy. Mov Disord. 2010;25(8):1040–6.

The Influence of Muscle Fatigue on Walking: The Role of Aging and Parkinson's Disease

10

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Abstract

Muscle fatigue causes motor adjustments in functional activities, including locomotion. Fatigue development is mediated by a range of individual factors such as the presence of pathologies, age, physical activity level, and type of activity performed. Specifically regarding gait control, muscle fatigue conditions have been associated with changes in stability and safety. To understand how muscle fatigue affects walking, mainly in complex environments, such as obstacle crossing or stepping down a curb, and whether the motor adjustments are age and Parkinson's disease (PD)-related, our group conducted a series of experiments involving walking tasks and muscle fatigue. In these studies, we investigated the influence of physical activity level, age, PD, and complexity of the environment in the context of muscle fatigue and walking. In this chapter, firstly, we define muscle fatigue. Then, we explain how the physiology of muscle fatigue can influence motor control. Furthermore, we consider fatigue in the context of aging and PD and indicate the effects of muscle fatigue on gait parameters. Finally, we discuss the main findings from our studies, comparing them with the literature and pointing out the practical applications.

Keywords

Muscle fatigue • Gait • Parkinson's disease • Aging • Physical activity level • Motor control • Obstacle avoidance

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10.1 Introduction

Fatigue is a normal physiological reaction to prolonged or intensive activity. Fatigue involves muscle weakness, feelings of tiredness, and reduced energy [1]. Furthermore, fatigue is known to be a safety hazard and may, for example, increase the risk of falling in younger and older individuals and people with Parkinson's disease (PD) [2–7]. Fatigue development is mediated by a range of individual factors such as the presence of pathologies [1], age (older adults are induced to fatigue faster than younger adults) [2], physical activity level (inactive individuals are induced to fatigue faster than active individuals) [3], and the type of activity performed (aerobic or strength exercise) [8]. In healthy younger and older individuals, fatigue is predictable and transient and responds to rest [9]. However, for people with PD, fatigue seems to be unpredictable, poorly responsive to rest, out of proportion to the activities performed, present at rest, and interferes with daily activities [9].

Fatigue can affect an individual's performance in work and home settings or cause disability, and it is recognized as a serious social problem [10, 11]. As consequence of fatigue, productivity is decreased in the workplace and at home, and adjustments during walking occur, causing errors [12]. Fatigue is considered one of the most disabling symptoms in people with PD [13]. Therefore, fatigue can contribute to disabilities in occupational activities and in daily living, for example by limiting the number of stairs that a person can descend or ascend, or causing problems in crossing or circumventing a person or object. Such disabilities can deteriorate the quality of life of an individual. In people with PD, for example, fatigue can limit the ability to maintain hobbies and participate in social activities [14].

The increasing prevalence of fatigue and its negative effects on healthy and neurological individuals has led to an increase in the quantity of research about the effects of fatigue in daily activities, including locomotion. With regard to fall prevention in older people, muscle fatigue has been shown to be an important factor to be considered as it affects balance and functional performance and, therefore, further predisposes older adults to falls than younger individuals [3, 4]. In addition, reports from people with PD have revealed that fatigue is one of the most common symptoms felt by this population. [9, 15]. However, fatigue not only affects older and neurological populations. Up to 56 % of employees in 15 European countries reported fatigue at work [16], and one-third of the US workforce experienced fatigue in the workplace [17]. As a result, governments have increased the regulation of fatigue [18, 19]. Although fatigue can be considered a negative consequence of physical activity, it works as a safety mechanism by preventing injury due to overexertion [20, 21].

The literature provides some evidence that the effects of muscle fatigue on gait are age and PD related, indicating that new muscle synergy occurs in the presence

of muscle fatigue, as observed by changes in spatiotemporal, mechanic, and muscle activation gait parameters [2–8, 22–25]. Therefore, understanding the consequences of fatigue in different age groups and in people with PD could help to avoid falls, slip, trips, and injuries during walking, mainly in complex environmental settings, such as obstacle avoidance and stepping down a curb. Furthermore, fatigue may be a useful model to study the effects of disease as it impairs muscle torque-generating capacity, yet does not include confounding factors such as pain or swelling [22].

To elucidate the theme in this chapter, firstly, we define fatigue. Then, we explain how the physiology of muscle fatigue can influence motor control. Furthermore, we consider fatigue in the context of aging and PD and indicate the effects of muscle fatigue on gait parameters. Finally, we discuss the main findings from our studies and indicate explanations for our results according to the literature, in addition to suggesting practical applications.

10.2 Definition of Fatigue

The multidimensional characteristic of fatigue complicates a clear definition. General fatigue is characterized by difficulty in initiating and sustaining mental and physical tasks in the absence of motor or physical impairment [26, 27]. Fatigue has a great number of causes [28], but in general, could be described as a failure to maintain an exercise at the same intensity [29]. Despite physical causes, fatigue could be associated with a clinical state, such as cancer, PD, and multiple sclerosis, among others [30]. According to Smets and collaborators [31], fatigue is multidimensional, including general fatigue, physical fatigue, reduced motivation, reduced activity, and mental fatigue. However, when we restrict ourselves to muscle fatigue, it is easier to define.

Muscle fatigue is the most commonly related symptom by many workers while performing their jobs [17]. Twenty-five to thirty-three percent of falls in the workplace occur due to muscle fatigue. This fact can be explained by the fact that muscle fatigue decreases motor control and the efficiency to perform daily activities [32]. According to the Ciba Foundation Symposium, held in 1980, muscle fatigue is "the inability of a physiological process to continue functioning at a particular level and/or the inability of the total organism to maintain a predetermined exercise intensity" [33] or as a loss of muscle performance during repeated or continuous activation [34]. To illustrate this definition, let's think about the submaximal repetitive contraction. Muscle fatigue will appear when the force or power output cannot be generated. In this case, a decrease in velocity could be considered as a fatigue situation. However, during isometric conditions, the fatigue situation could

be defined as the incapacity to maintain the force (required or expected). In addition, muscle fatigue can be defined by the concept of endurance, in which muscle fatigue is defined as the time to failure in maintaining a target muscle force or power output [35].

10.3 Physiology of Muscle Fatigue

Muscle fatigue is caused by limitations in the ability of the nervous system to generate a sustained signal and the reduced ability of calcium to stimulate contraction [36]. Consequently, muscle fatigue causes a reduction in the maximal voluntary force until the required or expected effort cannot be achieved. An individual who is performing a resistance exercise, at submaximal effort, is then no longer able to maintain the exercise, as the maximal force-generating capacity of muscles is less than the target submaximal effort.

It has been suggested that both central and peripheral factors are responsible for muscle fatigue [37]. Central fatigue is defined as a progressive reduction in voluntary activation of muscle during exercise [37, 38], which appears to be task dependent and may be mediated by intrinsic motoneuronal, spinal, and supraspinal factors [37]. Central fatigue is related to a reduction in the number of motor units activated or a reduction in the shot frequency of the motor units [36]. Peripheral fatigue is produced by changes at or below the neuromuscular junction [37], which is associated with failure of the surface membrane and T-tubular action potential propagation, the coupling mechanism between action potential and calcium release, or calcium regulation at the level of the contractile elements [36]. During exercise, failure to maintain the initial maximal effort is a combination of central and peripheral fatigue factors [37]. In other words, muscle fatigue affects the intrinsic properties of the muscle and also compromises the function of the central nervous system in driving the motoneurons adequately.

10.4 Muscle Fatigue in Aging

The prevalence of general fatigue in daily activities is inconsistent and varies from 7 to 98 % according to age and neurological disease [39–41]. For younger adults, more than half of individuals report moderate or severe fatigue [42], while in people over 70 years of age, it was found that 98 % of subjects report fatigue symptoms [19]. Fatigue is considered as one of the factors that cause disability, including in people with PD, limiting the ability to maintain hobbies and participate in social activities [7, 9, 43].

Older adults have indicated that muscle fatigue influences functional ability as reflected in walking, cleaning the house, general household chores, taking exercise, and lifting things [19]. During the aging process, deterioration of the neuromotor

and musculoskeletal systems occurs [44], and it is possible to make a correlation between this deterioration and fatigue. Besides this, older people commonly present impairment during walking [45], such as reduced gait velocity, when compared to younger people [44], and the dysfunction of muscle and proprioception [46]. Sarcopenia, which is defined as a loss of muscle mass and function [47], is another effect of aging that contributes to the prevalence of fatigue in older people. However, the effects of the aging process on muscle fatigue remain unclear [48].

There is a contradiction regarding age-related muscle fatigue. While some studies show less muscle fatigue in older people [13, 49], many other studies indicate that there is an increase in fatigue with aging [35, 50, 51]. Two theories have been used to explain this behavior: (i) reduced force generation or muscle weakness due to sarcopenia, which would make older adults more susceptible to muscle fatigue, and (ii) higher percentage of type I fibers in muscles of older adults, showing higher reflex excitability than muscles containing high percentages of type II fibers [38, 49]; this would make older adults relatively more resistant to fatigue induction. Moreover, the inconsistencies may be due to differences in contraction modes, protocols, muscle group, or subject characteristics [35, 52]. Nevertheless, the age of the individuals may be important in fatigue response since the effects of muscle fatigue seem to be aging dependent. Therefore, age is an important consideration in studies on fatigue and its effects on motor performance [38].

10.5 Parkinson's Disease and Fatigue

PD is the second most common neurodegenerative disease, just behind Alzheimer's disease. People with PD commonly relate muscular weakness during the progression of the disease [53], and its appearance could be connected with the disease onset [54]. Several studies have shown that fatigue is a major problem for more than half of patients with PD [9, 55], around 58.1 % of patients [56], demonstrating the high incidence of fatigue in people with PD [57]. However, there is little information about fatigue and PD in the literature.

People with PD present higher fatigue symptoms in several dimensions (general, mental, physical, reduced activity level, and low activation) than neurologically healthy individuals, and this seems not to be influenced by the physical activity level [7]. Kluger and colleagues [9] in a recent review listed important aspects of fatigue related to PD, as follows: (i) distinguishing fatigue from related constructs (i.e., sleepiness); (ii) recognizing that subjective fatigue complaints and objective performance fatigability are dissociable phenomena, (iii) using strict criteria to define clinical significance, (iv) specifying which performance domains are affected by fatigue (i.e., motor, cognitive), and (v) identifying causal factors by function and neuroanatomical location. Some of these items help to justify why fatigue can influence movement control, such as walking.

Fatigue in PD is related to some mitochondrial dysfunctions that PD people present [58]. In addition, the high presence of fatigue in people with PD is linked to striatal and limbic serotonergic dysfunction [59] that, in turn, is associated with gait

abnormalities [60]. Other causes that link people with PD with fatigue are sleep disorders, medications, and depression [61]. Furthermore, people with PD have reduced capacity to generate force and avoid muscle fatigue (they are induced to fatigue faster) compared to control individuals [7]. Although fatigue is more related to PD, the literature presents little information about the influence of the disease on muscle fatigue, especially on walking.

10.6 Muscle Fatigue and Walking

Muscle fatigue seems to induce negative changes in motor control. Loss of control from muscle fatigue can result in deficits in the capacity to regulate the mechanisms of movement such as muscular disorganization, modification of both the peripheral proprioceptive system and central processing of proprioceptive information, or deficits of planning and movement [12, 13]. Hence, muscular fatigue makes the regulation of postural sway more cognitively dependent [62], considering that the central processing factors are an important limitation for movement and postural stability. Another important aspect for loss of balance control might be the centrally adaptive changes in anticipatory postural adjustments [63, 64]. In this case, early onset of anticipatory postural adjustments may reflect an attempt by the central nervous system to compensate the deficits caused by muscle fatigue.

Gait adjustments, especially in complex environments, require adequate muscle strength in lower limb muscles, mainly for safely crossing an obstacle and stepping down a curb [65]. This requirement might not be met when muscles are fatigued. Muscle fatigue also impairs motor control by increasing muscle co-contraction and limiting fast corrective movements [66, 67], and by adversely affecting proprioception, movement coordination, and muscle reaction times [5, 6, 23], which are important components of balance control [4–6]. A new segmental organization in the presence of muscle fatigue is required to maintain motor performance, as observed by changes in time and spatial gait parameters [4, 22–25]. To understand how muscle fatigue affects walking, mainly in complex environments such as obstacle crossing or stepping down a curb, and whether such adjustments are age and PD related, our group have conducted a series of muscle fatigue and walking experiments, which are presented in Table 10.1.

10.7 Remarks and Considerations

This chapter investigates the mechanics and control adjustments on walking under leg muscle fatigue according to aging and PD, mainly considering gait environments (unobstructed walking and obstacle avoidance) and physical activity level (see Table 10.1). In Fig. 10.1, we present a summary of our findings, indicating similarities in the effects of muscle fatigue on walking in younger adults, older adults, and people with PD. Younger adults, older people, and people with PD increased step velocity and step width (with the exception of people with PD) and

Table 10.1 Description of manuscripts that examined the effects of muscle fatigue on walking. yo = years old. ROM—range of motion

Article	Aim	Sample	Fatigue protocol/experimental condition	Main results
Barbieri et al. [68]	To investigate foot placement relative to the curb in the final stride before stepping down, with and without ankle and knee muscle fatigue	10 healthy younger people—27.60 ± 2.79 yo, 69.21 ± 10.76 kg, 1.80 ± 0.08 m	Knee muscles, sit-to-stand (0.5 Hz); ankle muscles—repeated standing calf raise exercise (0.5 Hz)/five trials of stepping down a 10-cm elevation	Participants increased variability of footstep horizontal distance with muscle fatigue. In addition, there was an increase in variability of knee ROM, reduced step length and step duration, and increased step width and step velocity with muscle fatigue. Finally, participants increased ankle and hip ROM and decreased knee ROM
Barbieri et al. [3]	To analyze the effects of muscle fatigue in active and inactive younger adults on the kinematic and kinetic parameters of even and uneven walking	20 younger male adults: active group –24.7 ± 2.85 yo, 73.2 ± 4.4 kg, 1.78 ± 0.10 m; inactive group – 24.74 ± 2.95 yo, 82.7 ± 17.6 kg, 1.78 ± 0.06 m	Sit-to-stand (0.5 Hz)/three trials of unobstructed and obstructed gait	The endurance time in the fatigue protocol was lower for the inactive group. Both groups increased step width and stride speed and reduced stride duration and braking vertical force. The findings were independent of task and physical activity level
Barbieri et al. [69]	To evaluate the effects of muscle fatigue of quadriceps and triceps surae muscles at landing in stepping down in ongoing gait	10 healthy volunteers (5 men and 5 women): 27.60 ± 2.79 yo, 69.21 ± 10.76 kg, 1.81 ± 0.08 m	Knee muscles, sit-to-stand (0.5 Hz); ankle muscles—repeated standing calf raise exercise (0.5 Hz)/five trials of stepping down a 10-cm elevation	Fatigue did not cause a change in the frequency of heel or toe landing. The results indicated that in stepping down fatigue effects are compensated by redistributing work to unfatigued muscle groups. In addition, subjects increased step width, but did not adjust step length (in both muscle fatigue) and step duration (only for ankle muscle fatigue)

(continued)

Table 10.1 (continued)

Article	Aim	Sample	Fatigue protocol/experimental condition	Main results
Barbieri et al. [2]	To analyze the impact of age and leg muscle fatigue on gait parameters while unobstructed level ground walking and in obstacle crossing during walking	120 participants: G20: 24.0 ± 2.88 yo, 77.96 ± 14.74 kg, 1.78 ± 0.04 m G30: 32.5 ± 2.74 yo, 79.26 ± 14.23 kg, 1.74 ± 0.06 m G40: 44.0 ± 2.98 yo, 84.63 ± 15.51 kg, 1.75 ± 0.06 m G50: 54.5 ± 3.25 yo, 82.17 ± 13.40 kg, 1.71 ± 0.07 m G60: 64.0 ± 2.68 yo, 77.99 ± 12.61 kg, 1.71 ± 0.06 m G70: 74.5 ± 4.5 yo, 73.0 ± 12.26 kg, 1.65 ± 0.06 m	Sit-to-stand (0.5 Hz)/three trials of gait with and without obstacle	Leg muscle fatigue caused age-dependent changes in both unobstructed level ground walking and obstacle crossing during walking, such as reduced stride duration, increased stride speed, larger step width, and leading heel clearance. The adjustments in gait with fatigue in both tasks were more pronounced in participants over 40 years old than in younger individuals
Santos et al. [7]	To analyze the effects of lower limb muscle fatigue on gait in people with PD and healthy individuals, grouped according to physical activity level	10 active (67 ± 5.12 yos) and 10 inactive (71.7 ± 5 yo) people with PD and 10 active (67.5 ± 6.5 yo) and 10 inactive (71.4 ± 6.4 yo) neurologically healthy individuals	Sit-to-stand (0.5 Hz)/three trials of unobstructed gait	Physical activity level did not affect the gait changes occurring after lower limb muscle fatigue, either in patients with PD or in healthy individuals. Both groups increased stride length and velocity and decreased stride duration and braking vertical impulse after muscle fatigue. Gait changes were less pronounced in PD (i.e., no change in step width or double support duration)

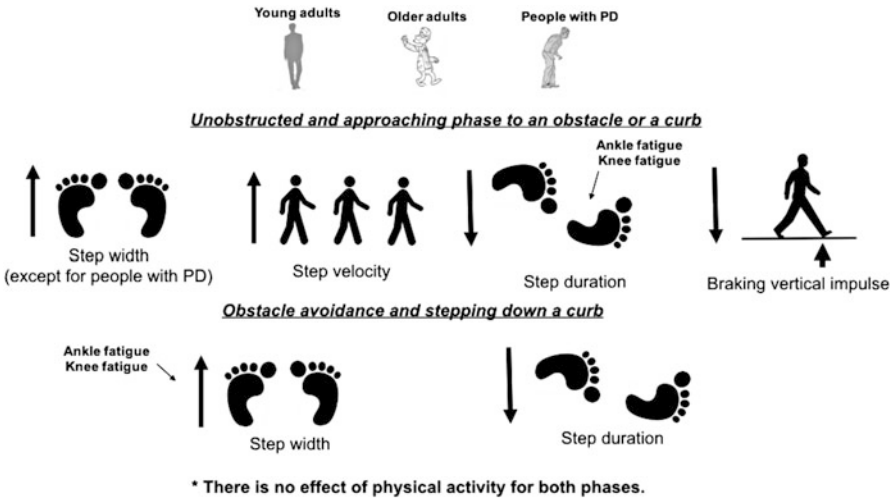

Fig. 10.1 Summary of similar findings of the effects of muscle fatigue on gait in younger adults, older adults, and people with PD

decreased step duration and braking vertical impulse during unobstructed walking or the approach phase to an obstacle or curb (Fig. 10.1). During obstacle avoidance or stepping down a curb, individuals increased the basis of support (step width) and reduced step duration. In addition, in both types of walking, physical activity level had no effects on the adjustments in walking after muscle fatigue in younger adults, older adults, or people with PD. These findings seem to indicate that gait adjustments are necessary to improve stability of gait after muscle fatigue, regarding age and PD. On the other hand, some strategies were more pronounced in older adults compared to (i) younger adults, for example, a reduced trail limb heel clearance and stride duration and increased stride length and gait speed, which appeared to reflect an attempt to maintain balance and safety, probably to counteract adverse fatigue effects, but could present a high risk of falling [2]; (ii) people with PD did not change the basis of support and double support duration after muscle fatigue, while older people adjusted these parameters to improve the control of mass in the mediolateral direction [7].

All individuals who participated in our studies [2, 3, 7, 68, 69] demonstrated reduced muscle strength after muscle fatigue (Fig. 10.2). This is an important aspect to explain the changes in walking. The protocols used in the studies (for knee and ankle fatigue) most likely affected the entire legs and not just the specific muscles. In addition, the movement to muscle fatigue may also have contained eccentric components [70]. Therefore, the fatigue protocol may have affected various muscles of the (whole) leg and not an isolated muscle group [2, 3, 7, 68, 69]. Fatigue of multiple muscles simultaneously impairs production of joint moments and hence absorption of kinetic energy [71, 72] and joint stabilization [22, 24, 73] through eccentric actions. The neuromuscular system may be able to compensate the

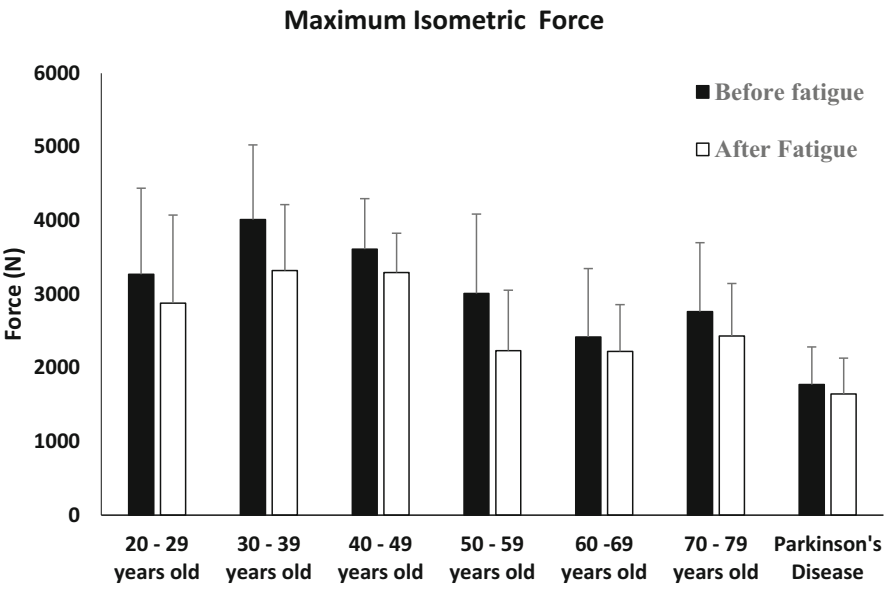

Fig. 10.2 Maximum isometric force of participants according to age and disease [2, 3, 7]

deficits caused by muscle fatigue of a single muscle group by adapting the activity of antagonistic and synergistic muscles, whereas this may be not possible if these muscle groups are also fatigued [74]. Finally, it has been suggested that with more muscles fatigued, there are changes in the supraspinal activity, regulating the activity of other muscles, but also of muscle spindles [75]. The effects of fatigue-related changes on proprioception may have contributed to destabilizing the gait [70].

The decrease in stride duration and increased step width (with the exception of people with PD) after fatigue induction were consistent with changes observed in our studies, independent of age (younger adults and older people), task (unobstructed walking, obstacle avoidance, and stepping down a curb), and level of physical activity (active and inactive individuals). The reduction in step duration after muscle fatigue seems to be the preferred strategy to deal with balance threats [76]. A reduction in this parameter can improve center of mass control and facilitate control of the body center in the mediolateral and anteroposterior directions. In addition, increased step width provides a larger margin of safety in controlling mediolateral movements of the body's center of mass [76–78].

Another common adjustment, which occurs in people of all ages and those with PD, is an increase in step/stride velocity during walking. First, this behavior could be a direct consequence of the reduction in step duration and step length. An increase in step length is a common adjustment due to muscle fatigue, but it is not used by younger adults. Increased step length provides a larger margin of safety in controlling anteroposterior movements of the body's center of mass [77].

During gait, increasing step length may maintain the center of mass on the base of support, making it easier to come to a stop in a single step and avoiding a forward fall [77, 78]. Therefore, changes in stride length and duration seem to be more important in terms of balance control then the result on gait speed, improving gait stability [76, 79]. On the other hand, increases in step velocity could be an attempt by the participants to perform the task as quickly as possible. Increasing speed could be a risky strategy as it decreases the time to acquire information, process, plan, and react during the movement [80, 81] and to integrate sensory information [82]. This is more dangerous when the individuals need to cross an obstacle or step down a curb, which may increase the risk of falling, especially in people with PD, who have impaired sensory integration capacity [83, 84].

Interestingly, people with PD seem to be less able to deal with the effects of muscle fatigue on walking than older adults. After muscle fatigue, both groups (people with PD and older adults) increased stride length and decreased stride duration, but only the control group performed adjustments in step width and double support duration to improve mediolateral balance. The lack of change in individuals with PD may suggest a stiffness strategy (robust system) that represents difficulty in performing adjustments [82]. Furthermore, people with PD present deficits in sensorimotor integration [79], which may be associated with commitments in perception of fatigue [7] and consequently gait adjustments to improve safety. Another alternative explanation for the less pronounced changes in people with PD could be that the central activation of their muscles in the fatigue protocol was insufficient to cause muscle overload and induce metabolic fatigue [85].

Muscle fatigue seems to similarly affect unobstructed walking, obstacle avoidance, and stepping down a curb [2, 3]. The adjustments were similar in the three conditions, indicating that the effect of fatigue on muscle condition is independent of walking type. However, due to major challenges during adaptive walking (obstacle avoidance and stepping down), it is possible the risk of falls is more elevated after muscle fatigue than in the unobstructed condition. Changes in the approach phase indicated that the individuals are adjusting their walk prior to the obstacle or curb (feedforward manner). This might be regarded as an anticipatory strategy facilitating foot placement in relation to the obstacle or curb [2, 3], but an error in this phase can compromise the plan to perform the task successfully.

Physical activity level seems to have no effects on the context of muscle fatigue and walking. Although the active groups demonstrated better muscle condition, which was demonstrated by higher maximum isometric force for active individuals (younger adults: 3346.70 ± 1002.3 N, older adults: 2576.03 ± 623.67 N; people with PD: 1785.56 ± 559.58 N) than inactive individuals (younger adults: 3099.1 ± 1351.9 N, older adults: 2402.67 ± 1026.75 N; people with PD: 1762.04 ± 461.0 N) and higher time to induce muscle fatigue in active individuals (Active: younger adults: 1018.4 ± 697.4 s; older adults: 408.6 ± 234.5 s; people with PD: 107.6 ± 39.5 s; Inactive: younger adults: 416.4 ± 381.7 s; older adults: 122.3 ± 133.2 s; people with PD: 80.8 ± 21.3 s), the active and inactive individuals

performed similar gait adjustments under muscle fatigue. Despite these findings, we consider physical activity as an important aspect to improve gait control, mainly in people with PD as physical activity can counteract the advance of the disease [86].

10.8 Practical Applications and Future Directions

From the findings presented in this chapter, it is possible to make some important observations about muscle fatigue and walking. The studies identified that muscle fatigue negatively affects stability during unobstructed gait as well as during obstacle crossing and stepping down a curb. The compensatory adjustments of gait parameters when fatigued appeared not to depend on physical activity levels in younger adults, older adults, or people with PD. Moreover, these adjustments might be caused by changes in muscle synergy. After muscle fatigue, it is necessary to increase the recruitment of motor units to develop the strength that is needed to generate adequate torque during gait. Therefore, the increased recruitment of motor units helps to explain the changes in step duration, width, and velocity, which were similar adjustments presented by the individuals.

Despite this similar behavior in all individuals, aging and PD seem to exacerbate the effects of muscle fatigue on walking. Modulations in both unobstructed walking and obstacle avoidance start after 40 years of age, with a risky strategy for older individuals over 60 years of age. This can be explained due to the effects of aging on physiology, such as muscle strength decline. The decline in strength combined with muscle fatigue further impairs strength production, increasing the risk of falling during gait [4]. As a consequence, although older people were able to adjust their walking patterns after leg muscle fatigue, these adjustments were less effective to maintain balance than in younger adults. In PD the situation is more complex, since the patients demonstrate rigid systems during gait. People with PD seem to have difficulties performing adjustments when necessary, because they present stiffness [87], which is explained by a co-activation that is common in people with PD [54] and by the lack of synergy, indicating a problem in motor output to control the movement [88]. Thus, the next step in our studies is to understand the effects of fatigue on muscle activation during gait.

In general, our studies provide further insight into the effects of muscle fatigue on different types of walking in diverse groups. This information is important in order to understand balance control in fatigued situations and possibly prevent falls in working and elderly populations. The findings may be used by professionals to improve gait stability under muscle fatigue and deal with fatigue in complex walking settings in older people and people with PD, for example, using a larger step width and step velocity. Physical exercise may be a strategy to improve or maintain muscle conditions and avoid muscle fatigue, consequently, preventing falls. Physical exercise promotes several benefits to physical capacity (e.g., strength, cardiovascular resistance, balance, flexibility, and coordination) and increases stability during gait [86]. However, since we did not find effects of

physical activity, further studies are necessary to investigate what kind of exercise programs are better for improving muscle fatigability to prevent falls [89].

Another alternative strategy to avoid the consequences of muscle fatigue is through recovery. Understanding the effects of fatigue recovery on gait could be important information for the individual to avoid the risk of falling and seems important to maintain performance [70]. A comprehensive and descriptive understanding of fatigue recovery may be fundamental to determining whether aged humans can perform and repeat requisite movements successfully [38, 70]. The period of rest is important to recover the muscle condition and, consequently, gait parameters. However, Barbieri and colleagues [85] showed that 20 min of rest is not sufficient to recover the gait parameters in younger individuals. This represents a negative aspect as there is a long time to recovery from muscle fatigue, which represents a prolonged risk to stability and the high energetic cost can impair security during gait. Thus, future studies on balance and gait control may also need to address fatigue recovery, especially in older adults and people with PD.

Therefore, according to the findings in this chapter, we can conclude that muscle fatigue changed the gait parameters in different environments in younger adults, older adults, and people with PD. The aging process and PD represent an increased risk of falling, which seems to be associated with poor muscle conditions. However, there are still some gaps in the literature on this theme, making further knowledge necessary.

References

1. Kroenke K, Wood DR, Mangelsdorff AD, Meier NJ, Powell JB. Chronic fatigue in primary care. Prevalence, patient characteristics, and outcome. JAMA. 1988;260:929–34.
2. Barbieri FA, Santos PCR, Simieli L, Orcioli-Silva D, Van Diëen JH, Gobbi LTB. Interactions of age and leg muscle fatigue on unobstructed walking and obstacle crossing. Gait Posture. 2014;39:985–90.
3. Barbieri FA, Santos PCR, Vitório R, van Dieën JH, Gobbi LTB. Effect of muscle fatigue and physical activity level in motor control of the gait of young adults. Gait Posture. 2013;38:702–7.
4. Helbostad JL, Leirfall S, Moe-Nilssen RS, Letvold O. Physical fatigue affects gait characteristics in older persons. J Gerontol A Biol Sci Med Sci. 2007;62:1010–5.
5. Parijat P, Lockhart TE. Effects of lower extremity muscle fatigue on the outcomes of slip-induced falls. Ergonomics. 2008;51:1873–84.
6. Parijat P, Lockhart TE. Effects of quadriceps fatigue on the biomechanics of gait and slip propensity. Gait Posture. 2008;28:568–73.
7. Santos PCR, Gobbi LTB, Orcioli-Silva D, Simieli L, Van Diëen JH, Barbieri FA. Effects of leg muscle fatigue on gait in patients with Parkinson's disease and controls with high and low levels of daily physical activity. Gait Posture. 2016;47:86–91.
8. Barbieri FA, Santos PCR, Vitório R, Lirani-silva E, Gobbi LTB, van Diëen JH. Systematic review of the effects of fatigue on spatiotemporal gait parameters. J Back Musculoskelet Rehabil. 2013;26(2):125–31.
9. Kluger BM, Herlofson K, Chou KL, Lou JS, Goetz CG, Lang AE, Weintraub D, Friedman J. Parkinson's disease-related fatigue: a case definition and recommendations for clinical research. Mov Disord. 2016;31(5):625–31.

10. Hancock PA, Desmond PA. Stress, workload, and fatigue. London: Lawrence Erlbaum Associates; 2000.
11. Cohen S, Kessler RC, Gordon L. Measuring stress, a guide for health and social scientists. Oxford: Oxford University Press; 1995.
12. Halvani GH, Zare M, Hobobati H. The fatigue in workers of Iran Central Iron ore company in Yazd. Int J Occup Med Environ Health. 2009;22(1):19–26.
13. Tralongo P, Respini D, Ferrau F. Fatigue and aging. Crit Rev Oncol/Hematol. 2003;8:S57–64.
14. Zesiewicz TA, Patel-Larson A, Hauser RA, Sullivan KL. Social security disability insurance (SSDI) in Parkinson's disease. Disabil Rehabil. 2007;29:1934–6.
15. Garcia-Ruiz PJ, Chaudhuri KR, Martinez-Martin P. Non-motor symptoms of Parkinson's disease: a review from the past. J Neurol Sci. 2014;338(1-2):30–3.
16. Benavides FG, Benach J, Ez-roux AV, Roman C. How do types of employment relate to health indicators? Findings from the second European survey on working conditions. J Epidemiol Community Health. 2000;54(7):494–501.
17. Swaen GM, Van Amelsvoort LG, Bültmann U, Kant IJ. Fatigue as a risk factor for being injured in an occupational accident: results from the Maastricht Cohort Study. Occup Environ Med. 2003;60(1):i88–92.
18. Liao S, Ferrell BA. Fatigue in an older population. J Am Geriatr Soc. 2000;48(4):426–30.
19. Dawson D, Mcculloch K. Managing fatigue: it's about sleep. Sleep Med Rev. 2005;9 (5):365–80.
20. Ratel S, Duche P, Williams CA. Muscle fatigue during high-intensity exercise in children. Sports Med. 2006;36:1031–65.
21. Abbiss CR, Laursen PB. Models to explain fatigue during prolonged endurance cycling. Sports Med. 2005;35:865–98.
22. Murdock GH, Hubley-Kozey CL. Effect of a high intensity quadriceps fatigue protocol on knee joint mechanics and muscle activation during gait in young adults. Eur J Appl Physiol. 2012;112(2):439–49.
23. Lin D, Nussbaum MA, Seol H, Singh NB, Madigan ML, Wojcik LA. Acute effects of localized muscle fatigue on postural control and patterns of recovery during upright stance: influence of fatigue location and age. Eur J Appl Physiol. 2009;106:425–34.
24. Granacher U, Wolf I, Wehrle A, Bridenbaugh S, Kressig RW. Effects of muscle fatigue on gait characteristics under single and dual-task conditions in young and older adults. J Neuroeng Rehabil. 2010;9:7–56.
25. Olson MW. Trunk extensor fatigue influences trunk muscle activities during walking gait. J Electromyogr Kinesiol. 2010;20(1):17–24.
26. Friedman JH, Alves G, Hagell P, Marinus J, Marsh L, Martinez-Martin P, Goetz CG, Poewe W, Rascol O, Sampaio C, Stebbins G, Schrag A. Fatigue rating scales critique and recommendations by the movement disorders society task force on rating scales for Parkinson disease. Mov Disord. 2010;25(7):805–22.
27. Lou JS. Physical and mental fatigue in Parkinson's disease: epidemiology pathophysiology and treatment. Drugs Aging. 2009;26(3):195–208.
28. Egerton T, Brauer SG, Cresswell AG. Fatigue after physical activity in healthy and balance-impaired elderly. J Aging Phys Act. 2009;17(1):89–105.
29. Booth FW, Thomason DB. Molecular and cellular adaptation of muscle in response to exercise: perspectives of various models. Physiol Rev. 1991;71(2):541–85.
30. Toth MJ, Gottlieb SS, Goran MI, Fisher ML, Poehlman ET. Daily energy expenditure in free-living heart failure patients. Am J Physiol. 1997;272(3 Pt 1):E469–75.
31. Smets EM, Garssen B, Bonke B, De Haes JC. The multidimensional fatigue inventory (MFI) psychometric qualities of an instrument to assess fatigue. J Psychosom Res. 1995;39 (3):315–25.
32. Ribeiro F, Oliveira J. Aging effects on joint proprioception: the role of physical activity in proprioception preservation. Eur Rev Aging Phys Act. 2007;4(2):71–6.

33. Edwards RHT. Biochemical basis of fatigue in exercise performance: catastrophe theory of muscular fatigue. In: Knuttgen HG, Vogel JA, Poortmans J, editors. Biochemistry of exercise muscle structure, energy metabolism and the level V. Champaign: Human Kinetics; 1983. p. 3–28.
34. Stackhouse SK, Stevens JE, Lee SC, Pearce KM, Snyder-Mackler L, Binder-Macleod SA. Maximum voluntary activation in nonfatigued and fatigued muscle of young and elderly individuals. Phys Ther. 2001;81:1102–9.
35. Kent-Braun JA. Skeletal muscle fatigue in old age: whose advantage? Exerc Sport Sci Rev. 2009;37(1):3–9.
36. Powers S, Howley E. Exercise physiology: theory and application to fitness and performance. 8th ed. Columbus: McGraw-Hill; 2011.
37. Gandevia SC. Spinal and supraspinal factors in human muscle fatigue. Physiol Rev. 2001;81 (4):1726–71.
38. AllmanBL RCL. Neuromuscular fatigue and aging: central and peripheral factors. Muscle Nerve. 2002;25(6):785–96.
39. Loge JH, Ekeberg O, Kaasa S. Fatigue in the general Norwegian population: normative data and associations. J Psychosom Res. 1998;45(1):53–65.
40. Pawlikowska T, Chalder T, Hirsch SR, Wallace P, Wright DJ, Wessely SC. Population based study of fatigue and psychological distress. BMJ. 1994;308(6931):763–6.
41. Chalder T, Berelowitz G, Pawlikowska T, Watts L, Wessely S, Wright D. Development of a fatigue scale. J Psychosom Res. 1993;37(2):147–53.
42. Amaducci CM, Mota DD, Pimenta CA. Fatigue among nursing undergraduate students. Rev Esc Enferm USP. 2010;44(4):1052–8.
43. Santos PCR, Barbieri FA, Orcioli-Silva D, Simieli L, Gobbi LTB. Effects of physical activity levels on fatigue perception in patients with Parkinson's disease and neurologically healthy individuals. Health. 2014;6:1–6.
44. Kang HG, Dingwell JB. Separating the effects of age and walking speed on gait variability. Gait Posture. 2008;27(4):572–7.
45. Owings TM, Grabiner MD. Step width variability, but not step length variability or step time variability, discriminates gait of healthy young and older adults during treadmill locomotion. J Biomech. 2004;37(6):935–8.
46. Shaffer SW, Harrison AL. Aging of the somatosensory system: a translational perspective. Phys Ther. 2007;87(2):193–207.
47. Curtis L, Litwic A, Cooper C, Dennison E. Determinant of muscle and bone aging. J Cell Physiol. 2015;230(11):2618–25.
48. Yassierli NMA, Iridiastadi H, Wojcik LA. The influence of age on isometric endurance and fatigue is muscle dependent: a study of shoulder abduction and torso extension. Ergonomics. 2007;50(1):26–45.
49. Lanza IR, Russ DW, Kent-Braun JA. Age-related enhancement of fatigue resistance is evident in men during both isometric and dynamic tasks. J Appl Physiol. 1985;2004:967–75.
50. Baudry S, Klass M, Pasquet B, Duchateau J. Age-related fatigability of the ankle dorsiflexor muscles during concentric and eccentric contractions. Eur J Appl Physiol. 2007;100 (5):515–25.
51. McNeil CJ, Rice CL. Fatigability is increased with age during velocity-dependent contractions of the dorsiflexors. J Gerontol A Biol Sci Med Sci. 2007;62:624–9.
52. Katsiaras A, Newman AB, Kriska A, Brach J, Krishnaswami S, Feingold E, Kritchevsky SB, Li R, Harris TB, Schwartz A, Goodpaster BH. Skeletal muscle fatigue, strength, and quality in the elderly: the Health ABC Study. J Appl Physiol. 2005;99(1):210–6.
53. Kakinuma S, Nogaki H, Pramanik B, Morimatsu M. Muscle weakness in Parkinson's disease: isokinetic study of the lower limbs. Eur Neurol. 1998;39(4):218–22.
54. Cano-de-la-cuerda R, Perez-de-heredia M, Miangolarra-page JC, Munoz-hellin E, Fernandez-de-las-penas C. Is there muscular weakness in Parkinson's disease? Am J Phys Med Rehabil. 2010;89(1):70–6.

55. Friedman JH, Brown RG, Comella C, Garber CE, Krupp LB, Lou JS, Marsh L, Nail L, Shulman L, Taylor CB, Working Group on Fatigue in Parkinson's Disease. Fatigue in Parkinson's disease: a review. Mov Disord. 2007;22(3):297–308.
56. Barone P, Antonini A, Colosimo C, Marconi R, Morgante L, Avarello TP, Bottacchi E, Cannas A, Ceravolo G, Ceravolo R, Cicarelli G, Gaglio RM, Giglia RM, Iemolo F, Manfredi M, Meco G, Nicoletti A, Pederzoli M, Petrone A, Pisani A, Pontieri FE, Quatrale R, Ramat S, Scala R, et al. The PRIAMO study: a multicenter assessment of nonmotor symptoms and their impact on quality of life in Parkinson's disease. Mov Disord. 2009;24(11):1641–9.
57. Friedman JH, Friedman H. Fatigue in Parkinson's disease. Neurology. 1993;43(10):2016–8.
58. Parker Jr WD, Swerdlow RH. Mitochondrial dysfunction in idiopathic Parkinson disease. Am J Hum Genet. 1998;62(4):758–62.
59. Pavese N, Metta V, Bose SK, Chaudhuri KR, Brooks DJ. Fatigue in Parkinson's disease is linked to striatal and limbic serotonergic dysfunction. Brain. 2010;133(11):3434–43.
60. Alves G, Wentzel-Larsen T, Larsen JP. Is fatigue an independent and persistent symptom in patients with Parkinson disease? Neurology. 2004;63(10):1908–11.
61. Knie B, Mitra MT, Logishetty K, Chaudhuri KR. Excessive daytime sleepiness in patients with Parkinson's disease. CNS Drugs. 2011;25(3):203–12.
62. Vuillerme N, Forestier N, Nougier V. Attentional demands and postural sway: the effect of the calf muscles fatigue. Med Sci Sports Exerc. 2002;34(12):1907–12.
63. Strang AJ, Berg WP. Fatigue-induced adaptive changes of anticipatory postural adjustments. Exp Brain Res. 2007;178(1):49–61.
64. Strang AJ, Choi HJ, Berg WP. The effect of exhausting aerobic exercise on the timing of anticipatory postural adjustments. Sports Med Phys Fitness. 2008;48(1):9–16.
65. Petrella JK, Kim JS, Tuggle SC, Hall SR, Bamman MM. Age differences in knee extension power, contractile velocity, and fatigability. J Appl Physiol. 2005;98:211–20.
66. Izawa J, Rane T, Donchin O, Shadmehr R. Motor adaptation as a process of reoptimization. J Neurosci. 2008;28(11):2883–91.
67. Dw F, Liaw G, Te M, Osu R, Burdet E, Kawato M. Endpoint stiffness of the arm is directionally tuned to instability in the environment. J Neurosci. 2007;27(29):7705–16.
68. Barbieri FA, Lee YJ, Gobbi LTB, Pijnappels M, van Diëen JH. The effect of muscle fatigue on the last stride before stepping down a curb. Gait Posture. 2013;37(4):542–6.
69. Barbieri FA, Gobbi LTB, Lee YJ, Pijnappels M, van Diëen JH. Effect of triceps surae and quadriceps muscle fatigue on the mechanics of landing in stepping down in ongoing gait. Ergonomics. 2014;57(6):1–9.
70. Barbieri FA, Beretta SS, Pereira VAI, Simieli L, Orcioli-silva D, Santos PCR, van Diëen JH, Gobbi LTB. Recovery of gait after quadriceps muscle fatigue. Gait Posture. 2016;43:270–4.
71. Prilutsky BI, Herzog W, Leonard TR, Allinger TL. Role of the muscle belly and tendon of soleus, gastrocnemius, and plantaris in mechanical energy absorption and generation during cat locomotion. J Biomech. 1996;29(4):417–34.
72. DeVita P, Blankenship-Hunter P, Skelly WA. Effects of a functional knee brace on the biomechanics of running. Med Sci Sports Exerc. 1992;24(7):797–806.
73. Lewek MD, Rudolph KS, Snyder-Mackler L. Quadriceps femoris muscle weakness and activation failure in patients with symptomatic knee osteoarthritis. J Orthop Res. 2004;22 (1):110–5.
74. Boyas S, Remaud A, Bisson EJ, Cadieux S, Morel B, Bilodeau M. Impairment in postural control is greater when ankle plantar flexors and dorsiflexors are fatigued simultaneously than when fatigued separately. Gait Posture. 2011;34:254–9.
75. Pearson K, Gordon J. Spinal reflexes. In: Kandel ER, Schwartz JH, Jessell TM, editors. Principles of neural science. New York: McGraw Hill; 2000. p. 713–36.
76. Hak L, Houdijk H, Steenbrink F, Mert A, van der Wurff P, Beek PJ, van Dieën JH. Speeding up or slowing down? Gait adaptations to preserve gait stability in response to balance perturbations. Gait Posture. 2012;36(2):260–4.

77. Hof AL, Gazendam MG, Sinke WE. The condition for dynamic stability. J Biomech. 2005;38 (1):1–8.
78. Hof AL, van Bockel RM, Schoppen T, Postema K. Control of lateral balance in walking. Experimental findings in normal subjects and above-knee amputees. Gait Posture. 2007;25 (2):250–8.
79. Brujin SM, van Dieën JH, Meijer OG, Beek PJ. Is slow walking more stable? J Biomech. 2009;42(10):1506–12.
80. Gobbi LTB, Silva JJ, Barbieri FA. Do men and women use similar adaptive locomotion to clear static and dynamic obstacles? Hum Mov. 2011;12(1):75–80.
81. Silva JJ, Barbieri FA, Gobbi LTB. Adaptive locomotion for crossing a moving obstacle. Motor Control. 2011;15(3):419–33.
82. Lewis GN, Byblow WD. Altered sensorimotor integration in Parkinson's disease. Brain. 2002;125(Pt 9):2089–99.
83. Vitório R, Gobbi LT, Lirani-Silva E, Moraes R, Almeida QJ. Synchrony of gaze and stepping patterns in people with Parkinson's disease. Behav Brain Res. 2016;307:159–64.
84. Vitório R, Lirani-Silva E, Barbieri FA, Raile V, Stella F, Gobbi LT. Influence of visual feedback sampling on obstacle crossing behavior in people with Parkinson's disease. Gait Posture. 2013;38(2):330–4.
85. Stevens-Lapsley J, Kluger BM, Schenkman M. Quadriceps muscle weakness, activation deficits, and fatigue with Parkinson disease. Neurorehabil Neural Repair. 2012;26(5):533–41.
86. Gobbi LTB, Oliveira-Ferreira MD, Caetano MJ, Lirani-Silva E, Barbieri FA, Stella F, Gobbi S. Exercise programs improve mobility and balance in people with Parkinson's disease. Parkinsonism Relat Disord. 2009;15:S49–52.
87. Carpenter MG, Allum JHJ, Honegger F, Adkin AL, Bloem BR. Postural abnormalities to multidirectional stance perturbations in Parkinson's disease. J Neurol Neurosurg Psychiatry. 2004;75(9):1245–54.
88. Takakusaki K, Tomita N, Yano M. Substrates for normal gait and pathophysiology of gait disturbances with respect to the basal ganglia dysfunction. J Neurol. 2008;255 Suppl 4:19–29.
89. Ijmker T, Houdijk H, Lamoth CJ, Beek PJ, van der Woude LH. Energy cost of balance control during walking decreases with external stabilizer stiffness independent of walking speed. J Biomech. 2013;46(13):2109–14.

Parkinson's Disease and Gait Asymmetry

11

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Abstract

Walking is considered symmetric in neurologically healthy individuals. However, asymmetry begins to occur with aging, and could be indicative of neurodegenerative diseases, such as Parkinson's disease (PD). The aim of this chapter is to discuss gait asymmetry in people with PD. Specifically, we present a general idea about unilateral signs/symptoms of PD and its influence on asymmetry and review the literature about unobstructed gait asymmetry in people with PD. Finally, we show the effects of obstacle crossing and auditory cues on gait asymmetry in people with PD. Previous studies have indicated that people with PD presented greater asymmetry in the temporal parameters compared to neurologically healthy older subjects. For obstacle crossing during walking, both people with PD and neurologically healthy older individuals demonstrated a higher symmetric index for step duration during obstacle

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crossing while walking compared to unobstructed walking. Therefore, obstacle avoidance increases gait asymmetry of neurologically healthy older individuals and people with PD. Auditory cues decreased asymmetry for step length, duration and velocity, and cadence in individuals with PD compared to neurologically healthy older individuals. However, only neurologically healthy older individuals demosntrated greater asymmetry in the trials with auditory cues for step length, duration and velocity, and cadence. Therefore, auditory cues seem to have no effects on gait asymmetry for unobstructed and obstacle walking in individuals with PD, and impair gait asymmetry in neurologically healthy older individuals during obstacle avoidance.

Keywords
Asymmetry • Walking • Parkinson's disease • Sidedness • Aging • Obstacle avoidance • Auditory cues

11.1 Introduction

The term asymmetry can mean both structural and functional left–right dissimilarities [1]. Asymmetry can be considered as differences in performance and motor control between sides. The concepts of symmetry and asymmetry are closely tied to function of the two hemispheres of the human brain. Despite having a preferred limb during tasks, walking is considered symmetric in neurologically healthy individuals [2]. However, asymmetry begins to occur with aging [2], and could be indicative of neurodegenerative diseases [3, 4], such as Parkinson's disease (PD). PD is characterized by a predominantly unilateral appearance of tremor, rigidity, and bradykinesia [5]. However, there is no obvious cause for the preferential unilateral neurodegenerative process [6].

The study of asymmetry in walking in people with PD may help in understanding the pathogenesis of the disease [6]. Clarifying what is responsible for motor asymmetry in PD is certainly important for understanding the cause of PD and its progression [7]. However, the motor control mechanisms of the limbs during walking are not well understood. The few studies that have analyzed asymmetry in gait evaluated tasks which presented little challenge, for example, walking without environmental demands (unobstructed walking). Challenging tasks, such as crossing an obstacle during walking, represent daily activities and can help in understanding the unilateral symptoms of PD. Therefore, in this chapter we discuss gait asymmetry in people with PD. Specifically, we present a general idea about unilateral signs/symptoms of PD and its influence on asymmetry and review the literature with regard to unobstructed gait asymmetry in people with PD. Finally, we show the effects of obstacle crossing and auditory cues on gait asymmetry in people with PD.

11.2 Parkinson's Disease and Unilateral Motor Impairment

PD is characterized by the dysfunction or death of neurons that produce dopamine. PD affects 0.3 % of the general population, with the highest prevalence in individuals over 60 years old [8]. PD causes degeneration mainly in the substantia nigra of the basal ganglia that is located in the midbrain, causing several forms of motor impairment [9]. The reduced level of dopamine inhibits the thalamus cortical and brainstem motor systems, compromising other brain structures [10]. This degeneration affects the planning and execution of motor actions in people with PD, especially repetitive, simultaneous, and sequential movements [11].

The diagnosis of PD is generally detected through the characteristic signs and symptoms of the disease. The principal signs and symptoms of PD used for diagnosis are bradykinesia, akinesia, hypometry, resting tremors, and muscle stiffness [11]. In addition, the unilateral predominance of the disease is another important indicator for PD diagnosis [6, 7], which is related to asymmetry between the sides.

Sidedness is used to differentiate PD from other neurodegenerative diseases [6]. More than half of people with PD present sidedness in motor impairment [12–16], commonly indicated by the Unified Parkinson's Disease Rating Scale [12, 17, 18]. The unilateral predominance of signs and symptoms affects patients in the early stages of the disease and may persist throughout the progression period of the disease—20–30 years [6, 19]. Previous studies have reported that impairment in the lower and upper limbs of people with PD occurs in accordance with the extent of brain damage in regions containing dopamine in each hemisphere [20–22]. Individuals with unilateral PD present significant differences in striatal uptake between sides, in both the caudate and putamen nuclei [23, 24]. In addition, the dominant hemisphere has a widespread distribution in the thalamus circuitry [25–27].

Structural, genetic, environmental, and toxic or metabolic mechanisms could explain this asymmetry in PD signs and symptoms [6]. However, two theories are most accepted to explain the unilateral signs and symptoms [6]. Before detailing the explanations, it is interesting to indicate that motor signs and symptoms emerge after the loss of about 50–70 % of the nigral neurons [9, 10]. The first theory indicates that there are normal inborn differences in the numbers of nigral dopaminergic neurons. Consequently, from birth, the number of nigral neurons on one side may be lower than that on the other side. The degenerative process of the disease affects both sides equally, but the side with the primarily reduced number of neurons reaches the critical point of vulnerability earlier. The second theoretical possibility is that, for some reason, one substantia nigra is more vulnerable than the other, and once the degenerative process starts, accelerated cell death occurs first on that side (see reference [6] for more details).

11.3 Gait Asymmetry in People with Parkinson's Disease

Due to sidedness of motor impairments, people with PD present asymmetrical adjustments in walking. However, the mechanisms that cause gait asymmetry in people with PD are not well known. Gait asymmetries in PD reflect an asymmetrical depletion of dopamine in the substantia nigra [28, 29]. This results in asymmetrical deregulation of the striatum, leading to further asymmetrical dysfunction of multiple circuits involving the basal ganglia and cortical regions [30, 31].

Previous studies have indicated that people with PD presented greater asymmetry in the temporal parameters compared to neurologically healthy older subjects [4, 32–37]. Specifically, swing time and its variability are the most asymmetrical parameters in people with PD, which are related to freezing episodes, falls, and decreased executive function [4, 32–35]. However, step length [38] and ground reaction forces [32] seem to be symmetrical parameters in unobstructed walking for individuals with PD. Table 11.1 shows previous studies that investigated gait asymmetry in people with PD. It is important to indicate that the findings of these studies were from walking without environmental demand. The presence of an obstacle during walking could increase gait asymmetry due to the increased complexity of the task. In the next section we present our findings on the effects of obstacle avoidance on gait asymmetry in people with PD.

To mitigate the effects of the signs and symptoms, especially asymmetry, and to improve the quality of life of people with PD, some interventions are used. The most common is pharmacological treatment. Although pharmacological treatment is effective at improving motor performance in people with PD, it presents some side effects. Therefore, other interventions have also been used, such as the external cues paradigm [39, 40] to reduce asymmetry without side effects. Verbal external [41], visual [42], rhythmic [43, 44], and auditory [45–47] cues are conventionally used in PD treatment. Previous studies have indicated that auditory cues are most effective for people with PD [48, 49], which improve rhythmicity, walking speed, and step length [47, 50–52]. One possible explanation for the improvement in gait from auditory cues is that movement requires less effort and attention, which could facilitate walking [44]. However, the use of external cues, especially auditory cues, has not been sufficiently studied with respect to the effects on asymmetry in people with PD. It is possible, this intervention could decrease walking asymmetries in people with PD. The results of our study about the effects of auditory cues on gait asymmetry in unobstructed and with obstacle walking in people with PD are presented in the following sections.

Table 11.1 Methodological information and results of studies that investigated unobstructed walking symmetry in PD

Author	Purpose	Samples	Environment and condition	Main findings
Plotnik et al. [4]	Examine if freezing of gait is related to motor asymmetry	$n = 24$ (PD FOG+) H&Y = 3 (OFF); 2.7 (ON) $n = 12$ (PD FOG-) H&Y = 2.8 (OFF); 2.7 (ON)	Walk at their normal pace on level ground for a total of 80 m	The group PD FOG+ showed larger asymmetry in ON and OFF state, compared to the group PD FOG- No correlation was found between asymmetry of clinical symptoms and gait asymmetry
Baltadjieva et al. [32]	Characterize gait asymmetry in patients with de novo PD	n =35 (de novo PD) H&Y = 1–2, 5 n =22 (control group)	Walk on level ground (80 m) at self-selected usual walking speed	Increased gait asymmetry is already viewed in de novo PD patients in temporal parameters (swing time), but not in ground reaction forces
Yogev et al. [33]	Assess the contribution of cognitive function to gait symmetry	n =21 (PD group) H&Y = 2–3 n =15 (older fallers) n =11 (control group)	Walk 25 m at comfortable pace under two walking conditions: usual walking and dual tasking (cognitive loading)	For both people with PD and older fallers: Gait asymmetry values were significantly higher during the usual walking condition in people with PD Gait asymmetry significantly increased when they walked and performed a dual task compared to the usual walking condition Gait asymmetry was associated with gait speed and gait variability, but no correlations were found for PD motor symptoms
Nanhoe-Mahabier et al. [38]	Assess spatial step asymmetry during gait in patients with PD with and without freezing of gait	$n = 12$ (PD FOG+) H&Y = 2.4 $n = 15$ (PD FOG-) H&Y = 2.1 $n = 15$ (control group)	Walking 8 m overground at preferred speed and on treadmill at preferred speed	Step time asymmetry was larger in PD FOG- compared to control group during treadmill walking There were no differences in step length variation and asymmetry between PD FOG- and PD FOG+ UPDRS score was significantly associated with spatial step asymmetry

(continued)

Table 11.1 (continued)

Author	Purpose	Samples	Environment and condition	Main findings
Frazzitta et al. [36]	Evaluate the relationship between gait asymmetry and freezing of gait and the effects of treadmill treatment on asymmetry	n =30 (PD FOG+) n =30 (PD FOG-) H&Y = 3	4-week rehabilitation treatment using a treadmill with auditory and visual cues	Significant correlation between gait asymmetry and FOGQ, between improvement in gait asymmetry and improvement in right stride length and improvement in FOGQ
Park et al. [37]	Investigate possible changes in coupling patterns, symmetry, complexity, and variability of lower extremity joints in gait of people with PD	n = 10 (DP group) n = 10 (control older adults) n = 20 (young adults)	Walking on a split-belt treadmill for 5 min at preferred velocity	Aging and PD significantly increased lower limb joint asymmetry People with PD presented greater asymmetry than the control older adults

FOG+ with freezing of gait, *FOG−* without freezing of gait, *FOGQ* freezing of gait questionnaire, *ON* under medication effects, *OFF* without medication effects, *H&Y* Hoehn and Yahr scale, *UPDRS* Unified Parkinson's Disease Rating Scale

11.4 Effects of Obstacle Avoidance on Gait Asymmetry in People with Parkinson's Disease

Unobstructed walking of individuals with PD is asymmetric. However, spatial parameters [38] and ground reaction forces [32] seem to be symmetric in unobstructed walking. Despite important findings about gait asymmetry in previous studies, analysis of asymmetry during complex gait tasks, such as obstacle crossing during gait, which are performed daily by this population [53], is neglected. Obstacle crossing during walking is one of the main factors leading to falling in people with PD [53–55]. Bilateral asymmetry reflects medial–lateral imbalance and instability and, hence, a predisposition to falls [33]. Previous researches has indicated that obstacle crossing during walking increases gait impairment in people with PD, mainly the hypometry and bradykinesia [53, 56–60], decreasing the toe clearance during obstacle crossing and changing the foot position in relation to the obstacle [53, 54, 61, 62]. Therefore, due to the increase in difficulty, complexity, and motor requirements during obstacle crossing for people with PD, asymmetry during this type of gait could be exacerbated.

We investigated the influence of obstacle crossing on gait asymmetry in individuals with PD and neurologically healthy older individuals (control group) (Table 11.2). Participants performed five trials of unobstructed gait and ten trials of obstacle crossing during gait (five trials of crossing with each leg) at a self-selected speed. Both people with PD and neurologically healthy older individuals (control group) demonstrated a higher symmetric index [63–65] for step duration during obstacle crossing while walking compared to unobstructed walking. Obstacle crossing increased step duration of both crossing step limbs compared to step duration in unobstructed walking and increased step duration of the step with the most affected (people with PD)/non-dominant (control group) limb compared to the step with the least affected/dominant limb. Our findings confirmed the idea that obstacle crossing increased gait asymmetry compared to unobstructed walking.

Obstacle crossing requires that individuals perform adjustments during walking, necessitating rapid planning and execution of adaptive movements [66], which seems to exacerbate gait asymmetry. Obstacle avoidance involves complex circuitry, such as the parietal and motor cortex, the lateral cerebellum, and pontine gray nuclei [66], which appears to be more susceptible to sudden interference. For example, obstacle avoidance is characterized by reduced velocity, shortened step length, and larger step width [53, 55, 57, 67, 68], which needs a more conservative and cautious strategy. The combination of consciously controlled walking with deliberate obstacle avoidance may well outweigh the available resources, causing these adjustments and increasing gait asymmetry. Older people already present deficits in the circuitry indicated below, mainly in peripheral structures [69], which could cause gait asymmetry during complex locomotion. These structures are more impaired in PD, affecting the basal ganglia and requiring greater compensatory involvement from other parts of the brain during walking [70]. In addition, individuals with PD present a progressive difference in striatal uptake between the cerebral hemispheres in both the caudate and putamen nuclei and in the

Table 11.2 Means and standard deviations of the walking parameters and symmetric index [63–65] on unobstructed walking and walking with obstacle crossing in individuals with PD and neurologically healthy older individuals

	People with PD			Neurologically healthy older individuals		
	Least/dominant limb	Most/non-dominant limb	Symmetric index (%)	Least/dominant limb	Most/non-dominant limb	Symmetric index (%)
Unobstructed walking						
Step length (cm)	57.96 ± 6.77	59.0 ± 7.10	2.30 ± 2.09	63.57 ± 4.25	62.88 ± 5.84	1.94 ± 2.02
Step duration (s)	0.54 ± 0.06	0.54 ± 0.05	1.23 ± 0.77	0.50 ± 0.05	0.51 ± 0.05	1.07 ± 0.86
Step width (cm)	10.55 ± 3.51	11.00 ± 3.62	6.39 ± 6.84	10.53 ± 2.24	10.86 ± 2.87	3.82 ± 3.10
Step velocity (cm/s)	108.57 ± 18.62	110.69 ± 20.33	2.60 ± 1.96	126.16 ± 1085	123.27 ± 11.97	1.89 ± 1.80
Double support (%)	24.61 ± 2.91	26.10 ± 1.82	4.33 ± 4.74	23.28 ± 2.05	24.44 ± 1.88	2.95 ± 2.69
Obstacle walking						
Step length (cm)	63.42 ± 6.06	64.14 ± 6.73	1.88 ± 1.45	65.23 ± 6.9	65.9 ± 6.43	2.11 ± 1.75
Step duration (s)	0.74 ± 0.09	0.75 ± 0.08	2.30 ± 1.67	0.65 ± 0.07	0.68 ± 0.07	2.52 ± 1.84
Step width (cm)	10.63 ± 1.89	11.21 ± 2.13	5.94 ± 5.80	13.08 ± 4.69	13.10 ± 3.77	7.57 ± 5.85
Step velocity (cm/s)	87.01 ± 13.8	87.14 ± 15.98	2.20 ± 1.55	101.71 ± 11.7	98.96 ± 13.78	2.77 ± 1.60
Double support (%)	15.9 ± 2.40	16.42 ± 2.18	2.96 ± 3.59	15.21 ± 2.43	15.94 ± 1.90	5.10 ± 4.10

cortical–basal ganglia–thalamic circuitry [25–27], which cause gait asymmetry and seem to increase during challenging tasks. Therefore, obstacle avoidance increases gait asymmetry in neurologically healthy older individuals and people with PD.

11.5 Influence of Auditory Cues on Gait Asymmetry during Obstacle Avoidance

Previous studies have indicated that auditory cues are the most effective cues for people with PD [48, 49]. Auditory cues are external stimuli associated with the initiation and ongoing facilitation of a movement [40], which improves rhythmicity, walking speed, and step length [47, 50–52]. Due to auditory cues that influence the neural circuitry regulating gait, Brodie and collaborator [73] indicated that symmetry matched auditory cues compensated the unsteady gait in the majority people with PD, but did not interfere in gait asymmetry. Despite this, the authors suggest that during challenging walking task, such as turning or obstacle crossing, auditory cues could decrease gait asymmetry.

Our group investigated the influence of auditory cue on gait asymmetry during obstacle avoidance in people with PD and neurologically healthy older individuals. Participants performed five trials of unobstructed gait and ten trials of obstacle crossing during gait (five trials of crossing with each leg) with and without auditory cues. First, the individuals performed the trials without auditory cues. For the auditory cue trials, the cue (controlled by a metronome) was customized for each individual according to the cadence determined by three trials performed prior to starting the experiment.

People with PD and neurologically healthy older individuals presented different behaviors for gait asymmetry when auditory cues were provided (Table 11.3). Individuals with PD demonstrated lesser asymmetry for step length, duration and velocity, and cadence than neurological healthy older individuals when auditory cues were offered. For individuals with PD, auditory cues in unobstructed walking decreased step velocity asymmetry. Only neurologically healthy older individuals showed greater asymmetry in the trials with auditory cues for step length, duration and velocity, and cadence. In addition, the control group demonstrated greater asymmetry for stride velocity with auditory cues in obstacle crossing compared to unobstructed walking. Finally, neurologically healthy older individuals increased gait asymmetry (step length, velocity, and cadence) during obstacle crossing with auditory cues compared to without auditory cues.

Auditory cues had no effects on gait asymmetry for unobstructed and obstacle walking in individuals with PD. Only step velocity in unobstructed walking improved gait asymmetry with auditory cues. The findings corroborate with Brodie and collaborators [73] who did not find an influence of auditory cues on gait asymmetry. This finding could be interpreted as asymmetry in morphology, musculature, and joints related to aging than directly through mechanisms related to the diagnosis of PD [73]. However, although auditory cues influence the neural circuitry regulating gait [43, 46], improving automatic movement [71, 72] and

Table 11.3 Means and standard deviations of the walking parameters and symmetric index [63–65] without and with auditory cues in people with PD and neurologically healthy older individuals

Symmetric index (%)	People with PD		Neurologically healthy older individuals	
	Without auditory cues	With auditory cues	Without auditory cues	With auditory cues
Unobstructed walking				
Step length	2.30 ± 2.09	1.60 ± 1.50	1.94 ± 2.02	1.93 ± 1.16
Step duration	1.23 ± 0.77	1.17 ± 0.70	1.07 ± 0.86	1.05 ± 0.67
Step width	6.39 ± 6.84	3.60 ± 3.17	3.82 ± 3.10	3.63 ± 3.50
Step velocity	2.60 ± 1.96	1.50 ± 1.19	1.95 ± 1.72	2.29 ± 1.30
Cadence	1.24 ± 0.77	1.17 ± 0.72	1.06 ± 0.87	1.03 ± 0.67
Double support	4.33 ± 4.74	3.49 ± 2.48	2.50 ± 2.41	2.76 ± 4.35
Obstacle walking				
Step length	1.88 ± 1.45	2.34 ± 1.36	2.28 ± 1.69	5.74 ± 3.91
Step duration	2.30 ± 1.67	2.43 ± 2.97	1.96 ± 0.68	7.44 ± 4.80
Step width	5.94 ± 5.80	7.60 ± 5.24	7.57 ± 5.85	15.36 ± 16.18
Step velocity	2.20 ± 1.55	3.18 ± 2.50	2.48 ± 1.53	10.53 ± 6.86
Cadence	2.30 ± 1.66	2.47 ± 3.01	1.98 ± 0.64	7.47 ± 4.70
Double support	2.96 ± 3.59	3.34 ± 3.99	5.20 ± 4.72	7.62 ± 3.85

decreasing asymmetry, when individuals with PD cross an obstacle, auditory cues did not improve, but did not impair, gait asymmetry. The findings seem to indicate that people with PD do not use auditory cues, or that obstacle avoidance impairs the benefit of auditory stimuli in gait asymmetry. An important aspect to analyze in future studies is if people with PD are able to synchronize their steps (heel contact) with auditory cues. It is possible that the higher demands of this task prevent adequate the use adequately of the auditory information.

More surprising were the results for neurologically healthy older individuals. The combination of obstacle avoidance and auditory cues increased gait asymmetry. Auditory cues seem to disrupt the gait pattern of neurologically healthy older individuals, which increases gait variability [39]. Possibly the combination of auditory cues and obstacle crossing increases the robustness of the task, increasing gait pattern disruption. Auditory cues seem to interfere with well-learned movement patterns in people without basal ganglia deficits, which perturbs the functioning of internal mechanisms [73]. Therefore, the use of auditory cues for neurologically healthy older individuals during obstacle avoidance seems not to be recommended.

11.6 Final Remarks

Neurologically healthy older individuals seem to start presenting asymmetry in gait, probably caused by aging effects. However, gait asymmetry is exacerbated in people with PD due to the effects of the disease that generate unilateral motor

degradation. Several studies have found greater unobstructed gait asymmetry in individuals with PD, especially for temporal gait parameters. However, little is known about gait asymmetry during obstacle avoidance in both people with PD and neurologically healthy older individuals.

Obstacle avoidance seems to increase gait asymmetry in both people with PD and neurologically healthy older individuals. Differently from unobstructed gait, the effects of aging seem to increase gait asymmetry in complex walking, independent of the effects of PD. Older people already present deficits in brain circuitry, which are more impaired in people with PD, increasing gait asymmetry during obstacle avoidance. Older individuals, and particularly people with PD, are especially impaired to switching in motor tasks, requiring subjects to make corrective steps instead of walking straight [66], predominantly during challenging walking tasks. It is possible that the basal ganglia are important for set-dependent adaptation of movement patterns for changes in condition, indicating that individuals have inflexible control [74], which is aggravated by sensory and perceptual deficits such as in people with PD [75].

Auditory cues seem to have no effects on gait asymmetry for unobstructed and obstacle walking in individuals with PD, but they impair gait asymmetry in neurologically healthy older individuals during obstacle avoidance. Possibly the combination of auditory cues and obstacle crossing disrupted the gait pattern in neurologically health older individuals, increasing gait asymmetry. In addition, individuals with PD were not able to use auditory cues to improve gait asymmetry during obstacle avoidance. This task seems to be a complex movement for people with PD that demands more movement coordination and attention, impairing the use of auditory stimulus.

References

1. Hugdahl K. Symmetry and asymmetry in the human brain. Eur Rev. 2005;13:119–33.
2. Sadeghi H, Allard P, Prince F, Labelle H. Symmetry and limb dominance in able-bodied gait: a review. Gait Posture. 2000;12(1):34–45.
3. Wall JC, Turnbull GI. Gait asymmetries in residual hemiplegia. Arch Phys Med Rehabil. 1986;67(8):550–3.
4. Plotnik M, Giladi N, Balash Y, Peretz C, Hausdorff JM. Is freezing of gait in Parkinson's disease related to asymmetric motor function? Ann Neurol. 2005;57(5):656–63.
5. Hughes AJ, Daniel SE, Kilford L, Lees AJ. Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study of 100 cases. J Neurol Neurosurg Psychiatry. 1992;55(3):181–4.
6. Djaldetti R, Ziv I, Melamed E. The mystery of motor asymmetry in Parkinson's disease. Lancet Neurol. 2006;5(9):796–802.
7. Melamed E, Poewe W. Taking sides: is handedness involved in motor asymmetry of Parkinson's disease? Mov Disord. 2012;27(2):171–3.
8. de Lau LM, Breteler MM. Epidemiology of Parkinson's disease. Lancet Neurol. 2006;5(6):525–35.
9. Takakusaki K, Saitoh K, Harada H, Kashiwayanagi M. Role of basal ganglia-brainstem pathways in the control of motor behaviors. Neurosci Res. 2004;50(2):137–51.

10. Obeso JA, Rodriguez-Oroz MC, Rodriguez M, Lanciego JL, Artieda J, Gonzalo N, Olanow CW. Pathophysiology of the basal ganglia in Parkinson's disease. Trends Neurosci. 2000;23 (10 Suppl):S8–19.
11. Mazzoni P, Shabbott B, Cortés JC. Motor Control Abnormalities in Parkinson's Disease. Cold Spring Harb Perspect Med. 2012;2(6):a009282.
12. Uitti RJ, Baba Y, Whaley NR, Wszolek ZK, Putzke JD. Parkinson disease: handedness predicts asymmetry. Neurology. 2005;64(11):1925–30.
13. Yust-Katz S, Tesler D, Treves TA, Melamed E, Djaldetti R. Handedness as a predictor of side of onset of Parkinson's disease. Parkinsonism Relat Disord. 2008;14(8):633–5.
14. Stewart KC, Fernandez HH, Okun MS, Rodriguez RL, Jacobson CE, Hass CJ. Side onset influences motor impairments in Parkinson disease. Parkinsonism Relat Disord. 2009;15 (10):781–3.
15. Haaxma CA, Helmich RC, Borm GF, Kappelle AC, Horstink MW, Bloem BR. Side of symptom onset affects motor dysfunction in Parkinson's disease. Neuroscience. 2010;170 (4):1282–5.
16. van der Hoorn A, Bartels AL, Leenders KL, de Jong BM. Handedness and dominant side of symptoms in Parkinson's disease. Parkinsonism Relat Disord. 2011;17(1):58–60.
17. Elbaz A, McDonnell SK, Maraganore DM, Strain KJ, Schaid DJ, Bower JH, et al. Validity of family history data on PD: evidence for a family information bias. Neurology. 2003;61 (1):11–7.
18. Toth C, Rajput M, Rajput AH. Anomalies of asymmetry of clinical signs in parkinsonism. Mov Disord. 2004;19(2):151–7.
19. Verreyt N, Nys GM, Santens P, Vingerhoets G. Cognitive differences between patients with left-sided and right-sided Parkinson's disease. A review. Neuropsychol Rev. 2011;21 (4):405–24.
20. Kumar A, Mann S, Sossi V, Ruth TJ, Stoessl AJ, Schulzer M, Lee CS. [11c]dtbz-pet correlates of levodopa responses in asymmetric Parkinson's disease. Brain. 2003;126:2648–55.
21. Foster ER, Black KJ, Antenor-Dorsey JA, Perlmutter JS, Hershey T. Motor asymmetry and substantia nigra volume are related to spatial delayed response performance in Parkinson disease. Brain Cogn. 2008;67(1):1–10.
22. Lewis MM, Smith AB, Styner M, Gu H, Poole R, Zhu H, et al. Asymmetrical lateral ventricular enlargement in Parkinson's disease. Eur J Neurol. 2009;16(4):475–81.
23. Knable MB, Jones DW, Coppola R, Hyde TM, Lee KS, Gorey J, Weinberger DR. Lateralized differences in iodine-123-ibzm uptake in the basal ganglia in asymmetric Parkinson's disease. J Nucl Med. 1995;36(7):1216–25.
24. Tatsch K, Schwarz J, Mozley PD, Linke R, Pogarell O, Oertel WH, Kung HF. Relationship between clinical features of Parkinson's disease and presynaptic dopamine transporter binding assessed with [123i]IPT and single-photon emission tomography. Eur J Nucl Med. 1997;24 (4):415–21.
25. Johnson-Frey SH. The neural bases of complex tool use in humans. Trends Cogn Sci. 2004;8 (2):71–8.
26. Binkofski F, Buccino G, Zilles K, Fink GR. Supramodal representation of objects and actions in the human inferior temporal and ventral premotor cortex. Cortex. 2004;40(1):159–61.
27. Potgieser AR, de Jong BM. Different distal-proximal movement balances in right- and left-hand writing may hint at differential premotor cortex involvement. Hum Mov Sci. 2011;30 (6):1072–8.
28. Booij J, Tissingh G, Boer GJ, Speelman JD, Stoof JC, Janssen AGM, Wolters EC, van Royen EA. [123I] FP-CIT SPECT shows a pronounced decline of striatal dopamine transporter labelling in early and advanced Parkinson's disease. J Neurol Neurosurg Psychiatry. 1997;62:133–40.
29. Kaasinen V, Nurmi E, Bergman J, Eskola O, Solin O, Sonninen P, Rinne JO. Personality traits and brain dopaminergic function in Parkinson's disease. Proc Natl Acad Sci U S A. 2001;98 (23):13272–7.

30. Clower DM, Dum RP, Strick PL. Basal ganglia and cerebellar inputs to 'AIP'. Cereb Cortex. 2005;15(7):913–20.
31. Middleton FA, Strick PL. Basal ganglia and cerebellar loops: motor and cognitive circuits. Brain Res Brain Res Rev. 2000;31(2-3):236–50.
32. Baltadjieva R, Giladi N, Gruendlinger L, Peretz C, Hausdorff JM. Marked alterations in the gait timing and rhythmicity of patients with de novo Parkinson's disease. Eur J Neurosci. 2006;24(6):1815–20.
33. Yogev G, Plotnik M, Peretz C, Giladi N, Hausdorff JM. Gait asymmetry in patients with Parkinson's disease and elderly fallers: when does the bilateral coordination of gait require attention? Exp Brain Res. 2007;177(3):336–46.
34. Plotnik M, Hausdorff JM. The role of gait rhythmicity and bilateral coordination of stepping in the pathophysiology of freezing of gait in Parkinson's disease. Mov Disord. 2008;23 (2):444–50.
35. Plotnik M, Giladi N, Hausdorff JM. Bilateral coordination of gait and Parkinson's disease: the effects of dual tasking. J Neurol Neurosurg Psychiatry. 2009;80(3):347–50.
36. Frazzitta G, Pezzoli G, Bertotti G, Maestri R. Asymmetry and freezing of gait in parkinsonian patients. J Neurol. 2013;260(1):71–6.
37. Park K, Roemmich RT, Elrod JM, Hass CJ, Hsiao-Wecksler ET. Effects of aging and Parkinson's disease on joint coupling, symmetry, complexity and variability of lower limb movements during gait. Clin Biomech. 2016;33:92–7.
38. Nanhoe-Mahabier W, Snijders AH, Delval A, Weerdesteyn V, Duysens J, Overeem S, Bloem BR. Walking patterns in Parkinson's disease with and without freezing of gait. Neuroscience. 2011;182:217–24.
39. Baker K, Rochester L, Nieuwboer A. The effect of cues on gait variability-reducing the attentional cost of walking in people with Parkinson's disease. Parkinsonism Relat Disord. 2008;14(4):314–20.
40. Rochester L, Burn DJ, Woods G, Godwin J, Nieuwboer A. Does auditory rhythmical cueing improve gait in people with Parkinson's disease and cognitive impairment? A feasibility study. Mov Disord. 2009;24(6):839–45.
41. Behrman AL, Teitelbaum P, Cauraugh JH. Verbal instructional sets to normalise the temporal and spatial gait variables in Parkinson's disease. J Neurol Neurosurg Psychiatry. 1998;65 (4):580–2.
42. Azulay JP, Mesure S, Amblard B, Blin O, Sangla I, Pouget J. Visual control of locomotion in Parkinson's disease. Brain. 1999;122:111–20.
43. Del Olmo MF, Ariasa P, Furiob MC, Pozoc MA, Cudeiro J. Evaluation of the effect of training using auditory stimulation on rhythmic movement in parkinsonian patients - a combined motor and [18F]-FDG PET study. Parkinsonism Relat Disord. 2006;12(3):155–64.
44. Rochester L, Hetherington V, Jones D, Nieuwboer A, Willems AM, Kwakkel G, Van Wegen E. The effect of external rhythmic cues (auditory and visual) on walking during a functional task in homes of people with Parkinson's disease. Arch Phys Med Rehabil. 2005;86 (5):999–1006.
45. Almeida QJ, Frank JS, Roy EA, Patla AE, Jog MS. Dopaminergic modulation of timing control and variability in the gait of Parkinson's disease. Mov Disord. 2007;22(12):1735–42.
46. Hausdorff JM, Lowenthal J, Herman T, Gruendlinger L, Peretz C, Giladi N. Rhythmic auditory stimulation modulates gait variability in Parkinson's disease. Eur J Neurosci. 2007;26 (8):2369–75.
47. Picelli A, Camin M, Tinazzi M, Vangelista A, Cosentino A, Fiaschi A, Smania N. Three-dimensional motion analysis of the effects of auditory cueing on gait pattern in patients with Parkinson's disease: a preliminary investigation. Neurol Sci. 2010;31(4):423–30.
48. Rochester L, Nieuwboer A, Baker K, Hetherington V, Willems AM, Chavret F, Kwakkel G, Van Wegen E, Lim I, Jones D. The attentional cost of external rhythmical cues and their impact on gait in Parkinson's disease: effect of cue modality and task complexity. J Neural Transm. 2007;114(10):1243–8.

49. Nieuwboer A, Baker K, Willems AM, Jones D, Spildooren J, Lim I, Rochester L. The short-term effects of different cueing modalities on turn speed in people with Parkinson's disease. Neurorehabil Neural Repair. 2009;23(8):831–6.
50. Willems AM, Nieuwboer A, Chavret F, Desloovere K, Dom R, Rochester L, Jones D, Kwakkel G, Van Wegen E. The use of rhythmic auditory cues to influence gait in patients with Parkinson's disease, the differential effect for freezers and non-freezers, an explorative study. Disabil Rehabil. 2006;28(11):721–8.
51. van Iersel MB, Munneke M, Esselink RA, Benraad CE, Olde Rikkert MG. Gait velocity and the timed-up-and-go test were sensitive to changes in mobility in frail elderly patients. J Clin Epidemiol. 2008;61(2):186–91.
52. Hausdorff JM. Gait dynamics in Parkinson's disease: common and distinct behavior among stride length, gait variability, and fractal-like scaling. Chaos. 2009;19(2):026113.
53. Vitorio R, Pieruccini-Faria F, Stella F, Gobbi S, Gobbi LT. Effects of obstacle height on obstacle crossing in mild Parkinson's disease. Gait Posture. 2010;31(1):143–6.
54. Galna B, Peters A, Murphy AT, Morris ME. Obstacle crossing deficits in older adults: a systematic review. Gait Posture. 2009;30(3):270–5.
55. Galna B, Murphy AT, Morris ME. Obstacle crossing in people with Parkinson's disease: foot clearance and spatiotemporal deficits. Hum Mov Sci. 2010;29(5):843–52.
56. Sofuwa O, Nieuwboer A, Desloovere K, Willems AM, Chavret F, Jonkers I. Quantitative gait analysis in Parkinson's disease: comparison with a healthy control group. Arch Phys Med Rehabil. 2005;86(5):1007–13.
57. Pieruccini-Faria F, Menuchi MRTP, Vitório R, Gobbi LTB, Stella F, Gobbi S. Kinematic parameters for gait with obstacles among elderly patients with Parkinson's disease, with and without levodopa: a pilot study. Braz J Phys Ther. 2006;10(2):233–9.
58. Yang YR, Lee YY, Cheng SJ, Lin PY, Wang RY. Relationships between gait and dynamic balance in early Parkinson's disease. Gait Posture. 2008;27(4):611–5.
59. Vitorio R, Teixeira-Arroyo C, Lirani-Silva E, Barbieri FA, Caetano MJ, Gobbi S, et al. Effects of 6-month, multimodal exercise program on clinical and gait parameters of patients with idiopathic Parkinson's disease: a pilot study. ISRN Neurol. 2011;2011:714947.
60. Vitório R, Lirani-Silva E, Barbieri FA, Raile V, Batistela RA, Stella F, Gobbi LT. The role of vision in Parkinson's disease locomotion control: free walking task. Gait Posture. 2012;35 (2):175–9.
61. van Hedel HJ, Waldvogel D, Dietz V. Learning a high-precision locomotor task in patients with Parkinson's disease. Mov Disord. 2006;21(3):406–11.
62. Dietz V, Michel J. Locomotion in Parkinson's disease: neuronal coupling of upper and lower limbs. Brain. 2008;131(Pt 12):3421–31.
63. Herzog W, Nigg BM, Read LJ, Olsson E. Asymmetries in ground reaction force patterns in normal human gait. Med Sci Sports Exerc. 1989;21:110–4.
64. Beretta VS, Gobbi LTB, Lirani-Silva E, Simieli L, Orcioli-Silva D, Barbieri FA. Challenging postural tasks increase asymmetry in patients with Parkinson's disease. PLoS One. 2015;10(9), e0137722.
65. Barbieri FA, Polastri PF, Baptista AM, Lirani-Silva E, Simieli L, Orcioli-Silva D, Beretta VS, Gobbi LTB. Effects of disease severity and medication state on postural control asymmetry during challenging postural tasks in individuals with Parkinson's disease. Hum Mov Sci. 2016;46:96–103.
66. Snijders AH, Weerdesteyn V, Hagen YJ, Duysens J, Giladi N, Bloem BR. Obstacle avoidance to elicit freezing of gait during treadmill walking. Mov Disord. 2010;25(1):57–63.
67. Patla AE, Prentice SD, Robinson C, Neufeld J. Visual control of locomotion: strategies for changing direction and for going over obstacles. J Exp Psychol Hum Percept Perform. 1991;17 (3):603–34.
68. Patla AE. Strategies for dynamic stability during adaptive human locomotion. IEEE Eng Med Biol Mag. 2003;22:48–52.

69. Seidler RD, Bernard JA, Burutolu TB, Fling BW, Gordon MT, Gwin JT, Kwak Y, Lipps DB. Motor control and aging: links to age-related brain structural, functional, and biochemical effects. Neurosci Biobehav Rev. 2010;34(5):721–33.
70. Berardelli A, Rothwell JC, Thompson PD, Hallett M. Pathophysiology of bradykinesia in Parkinson's disease. Brain. 2001;124(Pt 11):2131–46.
71. Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms. Brain. 1996;119(2):551–68.
72. Cunnington R, Iansek R, Bradshaw JL. Movement-related potentials in Parkinson's disease: external cues and attentional strategies. Mov Disord. 1999;14(1):63–8.
73. Brodie MA, Dean RT, Beijer TR, Canning CG, Smith ST, Menant JC, Lord SR. Symmetry matched auditory cues improve gait steadiness in most people with Parkinson's disease but not in healthy older people. J Parkinsons Dis. 2015;5(1):105–16.
74. Cools R, Barker R, Sahakian B, Robbins T. L-dopa medication remediates cognitive inflexibility, but increases impulsivity in patients with Parkinson's disease. Neuropsychologia. 2003;41:1431–41.
75. Almeida QJ, Frank JS, Roy EA, Jenkins ME, Spaulding S, Patla AE, Jog MS. An evaluation of sensorimotor integration during locomotion toward a target in Parkinson's disease. Neuroscience. 2005;134:283–93.

Effects of Physical Activity on Walking in Individuals with Parkinson's Disease

12

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Abstract

Considering our strongest involvement with walking behavior of people with Parkinson's disease (PD), this chapter develops the relationship between physical exercise and walking in this population. Among the cardinal and the secondary signs and symptoms of the disease, we find the gait impairments as one of the most disabling behaviors of people with PD. In this chapter, that issue is treated with respect to different intervention types and procedures and their effects on two walking conditions (over even and uneven surfaces), especially because only under challenging conditions this behavior is more impaired. Specifically, we are detailing many physical exercise interventions with respect to type, duration, and frequency and showing specific effects on gait parameters. According to the topics discussed in this chapter, we concluded that even though physical exercise is gaining evidence to improve gait impairments and other motor and non-motor signs and symptoms of the PD, there is no consensus with respect to the better intervention characteristics to promote effects on walking behavior of people with PD.

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12.1 Introduction

This chapter develops three major aspects: the characteristics of locomotor behavior that are impaired with the disease and the effects of physical exercise interventions on gait parameters in both over even and uneven terrains. Then the aim of this chapter is to present the most recent evidences of the effects of physical exercise interventions on walking behavior of people with Parkinson's disease (PD).

12.2 Locomotion in Parkinson's Disease

Walking impairments showed by people with PD in both even and uneven terrains are associated to tripping and body unbalanced which increase their fall episodes [1, 2], since most falls happen during walking in this population. The amount of fall episodes in people with PD is approximately two- or even threefold bigger than their healthy peers [1, 3] and is the second major cause of their hospitalization [4], mainly due to fractures [5, 6]. In addition to fractures, the consequences of a fall episode are related to physical, psychological, and functional impairments such as fear of falling, decrease in mobility, and loss of independency to perform activities of daily living [7, 8]. Therefore, researchers all over the world have focused on both to reveal disease-related locomotor behavior and to develop interventions to prevent fall episodes.

Related to deficits in locomotor behavior due to the disease, walking independency is impaired in people with PD in two ways: general and specific. In a more general descriptive way, people with PD show reduction in overall mobility [9], decrease in walking velocity and in range of motion (bradykinesia and hypometria), rigidity (stooped posture), and postural instability [10–18]. In a specific descriptive way, spatial and temporal parameters of gait are also impaired such as stride length, double support duration, gait velocity, and cadence [19–24].

These deficits are more pronounced when the locomotor control of people with PD is challenging as walking over uneven terrain or dual-task condition. The major gait modulations under dual-task conditions are decrease in gait speed and in the stride length and the increase in gait variability [25–29]. Walking over uneven terrain requires constant adaptations in gait parameters. Since people with PD also face uneven terrain in their everyday life, a recent and large set of studies had been published on obstacle avoidance describing the walking behavior by means of kinematic parameters [18, 30–34]. The results of these studies revealed that two

major disease features (bradykinesia and hypometria) can be seen in both the approaching and the crossing phases: reduction in stride length and stride velocity, decreased distances between leading foot and the obstacle before and after crossing, and similar toe clearance when people with PD were compared with their aged-matched healthy controls.

Dopaminergic medications are the gold standard treatment for people with PD, and its effects on motor function, mobility, and postural instability have been established. Specifically related to falls, dopamine replacement has minimum effects on postural instability [35–39], while in walking over even terrain, the dopamine therapy increases gait parameters such as stride length and velocity, and there is no effect on stride duration and the time of the swing phase [12, 40]. In a more challenging condition, such as walking over obstacles, Pieruccini-Faria and colleagues [41] revealed that dopaminergic replacement has no effect on spatial parameters, but increased step time during the approach phase, while the toe clearance of the trailing limb was decreased without medication during the crossing phase.

Cognition is also impaired in people with PD that influence their motor symptoms [42]. Some cognitive aspects such as attention affect the walking stability [43], which is related to the increased risk of falls [28, 44]. However, the major focus of this chapter is related to the effects of physical exercise in walking behavior; the reader can find more specific information in Chaps. 5 and 10 of this book.

12.3 Walking and Exercise in Parkinson's Disease

Physical exercise has been used in PD with the purpose to ameliorate the signs and symptoms that are little or nonresponsive to the dopaminergic therapy such as postural instability, freezing of gait, cognitive impairment, and depression [45–47]. Multiple benefits of the physical exercise have been demonstrated [48, 49], and the regular enrolment in physical activity is associated to a better physical function and quality of life [50]. This kind of intervention associated to the medications has potential to increase the dopaminergic levels [51], to change the gray matter [52], and to improve the cerebral connectivity [53]. Recent studies have been suggested that physical exercise can retard the disease progression and induce plastic effects in the patients' brain [54–57].

Interventions with physical exercise have been purposed to ameliorate the walking performance such as hypokinesia, postural instability, and freezing of gait [58–60], to improve safety while walking, and to prevent falls [61], especially when the dopaminergic replacement is not effective for some gait abnormalities and fall episodes [62]. Bloem and collaborators [7] demonstrated that the majority of falls happen when the patients are in the ON state of medication, i.e., when the symptoms are under medication control. Since the effects of the medication decrease with the disease progression and the debilitating side effects improve [63], it is necessary to investigate intervention strategies to improve gait parameters.

Different types of physical exercise interventions have been presented in the literature as promoters of positive gait modifications in people with PD [64–66]. Interventions with locomotion exercises can improve kinematic gait parameters such as walking velocity and cadence and step length and width [62, 67], as well as improvements in clinical measures, mobility, and quality of life [68–70], assessed by the *Unified Parkinson's Disease Rating Scale* (UPDRS), the 6 min walking test, the *Balance Evaluation Systems Test* (*BESTest*), the Timed Up and Go (TUG) test, and the *Parkinson's Disease Questionnaire—39 items* (PDQ-39) [71–75].

In general, the gains provided by the physical exercise are maintained when the sessions are from 2 to 3 times a week with duration from 4 to 12 weeks [48, 76]. Specifically for locomotion exercises, interventions with duration from 2 to 6 months, with frequency from 1 to 3 times a week and from 30 to 70 min per session, have been showed relevant improvements [59].

Several types of interventions with physical exercises have been used to improve walking behavior, as resistance, aerobic, and multimodal exercises [67, 77–80], which will be detailed in the next session of this chapter. Some strategies associated with the intervention with locomotion exercises have been used to optimize the exercise benefits such as Nordic walking, sensory cues, and treadmill walking [60, 63, 81, 82]. Nordic walking is characterized by the use of poles to help the walking skill. This strategy has demonstrated superior results for locomotor behavior specially related to postural stability, stride length, gait pattern, and gait variability [83], as well as to ameliorate functional mobility and quality of life [82, 84] and to maintain the improvements after 5 months of follow-up [84]. The sensory cues are defined as an external stimulation to regulate the movement [85]. Auditory and visual cues have been used to improve the gait parameters in people with PD. Music, counting loud, or metronome beats are applied as auditory cues, while laser pointers, adapted glasses, and ground stripes are used as visual cues [63, 85]. Auditory cues are able to improve cadence, stride length, and velocity, while visual cues have demonstrated relevant effects only in step length. However, so far, it is unknown what type of auditory cues is the most efficient [63, 85]. Locomotor intervention with treadmill has showed relevant and consistent results in some gait parameters such as step length and velocity, but not in walking distance and cadence [60]. When a partial body weight is added with the treadmill, studies have demonstrated significant results in the clinical (UPDRS), balance, and walking impairments [86, 87], in comparison to locomotor intervention with verbal instructions. Nevertheless, when treadmill training is associated with visual cues, better scores for step length, walking velocity, and mobility have been reached and maintained after a follow-up period [88].

Recent technologies have been applied to ameliorate locomotor behavior of the people with PD such as virtual reality, robotic resources, and exergames, which are considered promising types of interventions due to the demonstrated benefits [66, 69]. Virtual reality can promote motor learning through the multisensory perceptions (visual, auditory, and haptic inputs) and movement repetitions to solve problems [89]. Mirelman and colleagues [89] applied treadmill training with a customized virtual reality of complex environment (obstacles) in people

with PD and showed improvements in cognitive, physical, and behavioral aspects. Picelli and collaborators [90] applied robotic resources in combination with treadmill training to rehabilitate walking in patients in mild to moderate stages of the disease and revealed improvements in walking distance and speed. In more advanced stages of the disease, Picelli and collaborators [91] demonstrated the positive effects of the same intervention in postural stability. Exergames (computer games) combining players' physical movements with real-time motion detection have been applied to decrease the motor symptoms in people with PD. Two recent systematic reviews about exergames as a rehabilitation tool were published. Barry, Galna, and Rochester [92] reported that people with PD can play exergames that improve their balance and cognition and reduce the motor severity. Harris and colleagues [93], analyzing two studies that included people with PD, revealed that exergaming can improve static balance and postural control.

Nonconventional therapies such as music and martial arts have demonstrated to be efficient as interventions to improve walking [94–96]. Music-based movement therapy (MbM therapy) and dance are among the interventions related to music [94, 95]. Among the different dance styles, such as waltz and foxtrot, Argentine tango is showing better results in movement control [97]. In general, dance has demonstrated positive results to ameliorate motor impairments, such as balance and walking velocity [95], but Argentine tango showed better results for functional mobility, quality of life, and motor sign severity [97, 98]. Tai Chi is a martial art widely used as a complementary therapy in PD [96]. Li and collaborators [99] applied Tai Chi intervention 2 times a week for 24 weeks with 60 min duration per session and showed an increase in step length, walking velocity, and functional mobility and a decrease in fall episodes that were maintained after a follow-up period of 3 months. Tai Chi intervention also revealed another benefits as the increase in motor function and in balance [96].

As we demonstrated in this section, there is heterogeneity of physical interventions been used to the purpose to ameliorate the walking behavior in people with PD. However, there is no consensus in the literature related to the better physical exercise (type, frequency, intensity, and duration) to be applied for this purpose [66]. On the other hand, there is a positive aspect in this physical exercise diversity, i.e., the patient can choose for the most pleasurable physical exercise since motivation is crucial for adherence and engagement in an intervention program [100, 101], mainly because the great effect of exercise requires regular and sustained participation in the activities [102]. It is important to highlight that it is necessary to conciliate medication treatment and physical exercise intervention to improve postural stability and to reduce fall episodes [65].

In view of the walking impairments over even and uneven terrains showed by people with PD, presented in the beginning of this chapter, it is necessary to understand the effects of physical exercise interventions in each surface. Then, different types of interventions are presented in the next section as well as their effects in walking over even and uneven terrains.

12.4 Physical Exercise in Walking Over Even Terrain

Three different types of interventions with physical exercise have demonstrated major effects in gait parameters over even terrain in people with PD: resistance training [103], aerobic training [79, 82], and multimode training [67, 80, 104].

Resistance training is recommended to people with PD due to their muscular weakness [105–107], which is related to the disease progression [108] and the reduction in physical activity levels [109, 110]. Lower levels of muscular strength, specifically the muscle power, are associated with a decrease in walking speed and fall history [111, 112]. Muscle power training also reduces the bradykinesia symptom [103]. Studies reported that resistance training has consistently improved spatial and temporal gait parameters [103, 113], anticipatory postural adjustments during gait initiation [114], and functional mobility [82, 103, 115]. Related to the characteristics of resistance training, the literature presents interventions with short term, 2–3 months [103, 113, 115, 116], and medium term, 6 months [117], intensity varying from moderate [115, 116] to high [103, 113, 116], duration from 2 to 3 times a week, and the use of uni- and multi-joint machines and elastic bands [118]. In addition, the linear intensity progression in the training load is common. It is recommended that the resistance training must be associated with specific activities that translate the muscle strength and power gains to the patients' daily life activities [119].

Aerobic training was designed for people with PD due to their autonomic dysfunction probably caused by cardiac sympathetic [120], which make them reach the Vo_2max earlier than their healthy peers [121]. This means that their cardiac function must be assessed during interventions, and this also implies that aerobic training is recommended since aerobic resistance is important to their everyday life. Interventions with aerobic training were able to improve the performance in motor actions, postural stability, and walking [79]. Specifically to gait parameters, aerobic training has demonstrated improvements in step and stride length [61, 97, 122, 123], walking velocity, and functional mobility [82, 122, 124–127]. Related to the characteristics of aerobic training, the interventions vary from 6 to 14 weeks' duration, two times a week, and with moderate to high intensities. However, high-intensity aerobic training has moderate effects on walking performance [128]. Treadmill training has been used in aerobic interventions due to both motor learning/control and gait mechanical effects. The constant and repetitive gait imposed by the treadmill promotes a continuous feedback which improves the automatic motor control [129]. Treadmill walking also implies an augmented hip hyperextension and elongated hip and ankle flexors that in consequence increase step length and swing duration and decrease support duration improving propulsion [62, 130, 131].

Multimode training is a type of intervention that integrates multiple components of the functional capacity (strength, flexibility, coordination, etc.), sensorimotor, and cognitive aspects with holistic (generalized interventions) [67, 68, 132–134] and specific purposes (focused interventions) [80, 104]. Related to people with PD, multimode training is based on sensory [135], motor [136, 137], and cognitive [138, 139] deficits. Vitório and colleagues [67] designed a multimode training

oriented to the components of functional capacity, cognitive functions, and mobility. Results related to gait parameters after 6 months of training revealed improvements in step length and stride velocity. The characteristics of the generalized interventions applying multimode training are 12–24 weeks' duration, 2–4 times a week, and intensity with incremental loads by both task complexity and physiologic parameters (1RM, %HRmax). Focused interventions applying multimode training with the purpose of gait improvements tend to use eastern approaches. Tai Chi, with fluent movements and positions, implies mental concentration, postural control, and flexibility [140]. While Tai Chi is a martial art, characterized by rapid movements for self-defense, Qigong with more slow movements with internal attentional focus [141] is more suitable for people with PD. In general, eastern approaches ameliorate mobility, where Qigong practice improves the performance in gait initiation [142], which positively impacts the anticipatory postural adjustments, and Tai Chi has no effect on spatial and temporal gait parameters [140, 142, 143]. Multimode training for walking improvements applied in water revealed positive effects in step length and single and double support time [104]. When a multimode training focused on balance and gait was developed in both indoor and outdoor environment [80], the results revealed improvements in walking velocity and dual-task TUG immediately after 8 weeks of intervention and after 6 months of follow-up, while only dual-task TUG scores were maintained after 12 months of follow-up. The combination focus on balance and gait and the use of outdoor activities challenge not only the components of functional capacity and postural stability but also the performance in real-life situations decreasing attentional demands and improving postural control [29]. Our group just finished a long-duration (8 months, twice a week) intervention comparing three groups of people with PD in stages mild to moderate: multimode training ($n = 88$ attempts), locomotor training ($n = 74$ attempts), and cognitive training ($n = 63$ attempts). Data of the gait parameters were collected in three moments (pretest, post1 after 4 months, and post2 after 8 months), and they are now under processing. A preliminary analysis (unpublished data) demonstrated

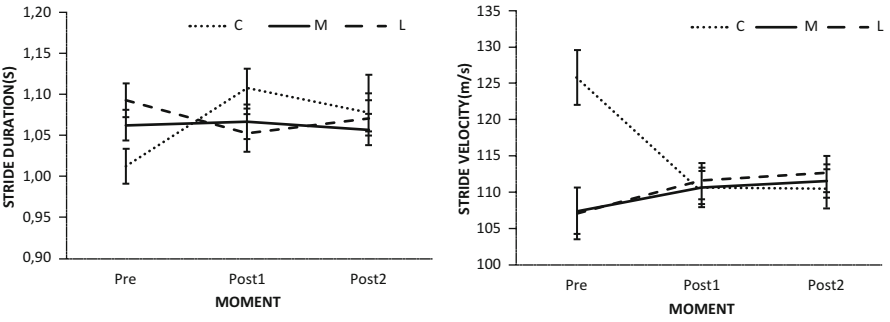

Fig. 12.1 Means and standard errors for stride duration and stride velocity by group (*M* multimode, *L* locomotor, *C* cognitive) and by moment (*pre* pretest, post1 after 4 months, and post2 after 8 months)

interactions between group and moment (Fig. 12.1) for stride duration ($F_{3,\ 222} = 3.54$; $p < 0.012$) and stride velocity ($F_{3,\ 222} = 12.97$; $p < 0.001$).

These results are showing that (1) stride duration is not affected by multimode training, while locomotor training impacts stride duration only in the first 4 months of intervention, and (2) majorly in the first 4 months of intervention, multimode and locomotor trainings have similar effects in increasing stride velocity, while cognitive training decreased the huge values of stride velocity maybe due to the necessity to go outside the home twice a week to participate in the intervention.

12.5 Physical Exercise and Walking Over Uneven Terrain

Adaptive gait is crucial for human locomotion. In everyday life, individuals are constantly crossing over [18] and avoiding obstacles [144] in their travel path. Yet, people walk in different space constraints [145] and in different velocities [146] and perform a secondary task [147]. People with PD show difficulties to perform tasks where it is necessary to adapt their gait pattern, which improve with dopaminergic therapy [41, 148]. Step velocity is one of the spatial and temporal gait parameter that represents the deficits in adaptive gait of people with PD [149]. To increase step velocity is crucial because tasks such as answering a door bell or crossing the street are present in everyday life [150]. This impairment is related to the bradykinesia and to the difficulty in modulating movement according to the temporal demands [151]. Physical exercise can promote changes in gait parameters of people with PD. Short-duration physical exercise (3–6 weeks) is able to ameliorate the gait pattern (stride length) in fast velocity [119, 152–154], independently whether the emphasis is in lower limb range of motion [152] or in treadmill locomotor training [119, 153, 154].

Adaptive gait is also necessary in dual-task situation. Considering that people with PD have attentional deficits that impair dual-task behavior [147], interventions with physical exercises have shown improvements in cognitive parameters, such as executive functions [132] and memory [155]. When locomotor tasks were associated with cognitive demands (verbal fluency, subtractions), Yogev-Seligmann and colleagues [156] observed improvements after 4 weeks in divided attention skills during walking in people with PD, which is important in performing activities of daily living [157]. Using a smartphone applicative, Ginis and collaborators [158] increased the walking velocity during dual task in 13.5 % due to a positive feedback.

Related to crossing over or avoiding obstacles, the literature is rich in describing the adaptive gait of people with PD [18, 34, 41, 144–148]. However, little is known about the effects of physical exercise intervention in these types of adaptive gait. Kim, Jae, and Jeong [159] applied a 12-week, 3 times a week, Tai Chi intervention and observed improvements in dynamical postural stability in both anteroposterior and mediolateral directions to step over a static obstacle. Liao and colleagues [160], using exergames for 6 weeks, 2 times a week, and 45 min per session, focused in

muscle strengthening and balance and revealed that people with PD increased obstacle crossing velocity and the step length.

12.6 Final Remarks

To design an intervention with physical exercise in order to improve gait impairments over even and uneven surfaces in people with PD is still a challenge. Even though physical exercise is gaining evidence to improve gait impairments and other motor and non-motor signs and symptoms of the PD, there is no consensus in the literature with respect to the better intervention characteristics (type, frequency, intensity, and duration of physical exercises) to promote effects on walking behavior of people with PD. However, this diversity facilitates the patients' choice according to their volition and pleasure. Most important now is to encourage people with PD to adopt and maintain an active lifestyle.

References

1. Bloem BR, Hausdorff JM, Visser JE, Giladi N. Falls and freezing of gait in Parkinson's disease: a review of two interconnected, episodic phenomena. Mov Disord. 2004;19 (8):871–84.
2. Stolze H, Klebe S, Zechlin C, Baecker C, Friege L, Deuschl G. Falls in frequent neurological diseases – prevalence, risk factors and aetiology. J Neurol. 2004;251:79–84.
3. Pickering RM, Grimbergen YA, Rigney U, Ashburn A, Mazibrada G, Wood B, Gray P, Kerr G, Bloem BR. A meta-analysis of six prospective studies of falling in Parkinson's disease. Mov Disord. 2007;22(13):1892–900.
4. Temlett JA, Thompson PD. Reasons for admission to hospital for Parkinson's disease. Intern Med J. 2006;36(8):524–6.
5. Johnell O, Melton LJ, Atkinson EJ, O'Fallon WM, Kurland LT. Fracture risk in patients with parkinsonism: a population-based study in Olmsted County, Minnesota. Age Ageing. 1992;21(1):32–8.
6. Williams DR, Watt HC, Lees AJ. Predictors of falls and fractures in bradykinetic rigid syndromes: a retrospective study. J Neurol Neurosurg Psychiatry. 2006;77:468–73.
7. Bloem BR, Grimbergen YA, Cramer M, Willemsen M, Zwinderman AH. Prospective assessment of falls in Parkinson's disease. J Neurol. 2001;248(11):950–8.
8. Adkin AL, Frank JS, Jog MS. Fear of falling and postural control in Parkinson's disease. Mov Disord. 2003;18(5):496–502.
9. Shulman LM, Gruber-Baldini AL, Anderson KE, Vaughan CG, Reich SG, Fishman PS, Weiner WJ. The evolution of disability in Parkinson disease. Mov Disord. 2008;23(6):790–6.
10. Murray MP, Sepic SB, Gardner GM, Downs WJ. Walking patterns of men with parkinsonism. Am J Phys Med Rehabil. 1978;57(6):278–94.
11. Morris ME, Iansek R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease. Brain. 1996;119(2):551–68.
12. Morris ME, Huxham F, McGinley J, Dodd K, Iansek R. The biomechanics and motor control of gait in Parkinson disease. Clin Biomech. 2001;16(6):459–70.
13. Morris M, Iansek R, McGinley J, Matyas T, Huxham F. Three-dimensional gait biomechanics in Parkinson's disease: evidence for a centrally mediated amplitude regulation disorder. Mov Disord. 2005;20(1):40–50.

14. Kimmeskamp S, Hennig EM. Heel to toe motion characteristics in Parkinson patients during free walking. Clin Biomech. 2001;16(9):806–12.
15. Ferrarin M, Carpinella I, Rabuffetti M, Calabrese E, Mazzoleni P, Nemni R. Locomotor disorders in patients at early stages of Parkinson's disease: a quantitative analysis. In Engineering in Medicine and Biology Society, 2006. IEEE Trans Neural Syst Rehabil Eng. 2006;1:1224–7.
16. Yang YR, Lee YY, Cheng SJ, Lin PY, Wang RY. Relationships between gait and dynamic balance in early Parkinson's disease. Gait Posture. 2008;27(4):611–5.
17. Benatru I, Vaugoyeau M, Azulay JP. Postural disorders in Parkinson's disease. Clin Neurophysiol. 2008;38(6):459–65.
18. Vitório R, Lirani-Silva E, Baptista AM, Barbieri FA, dos Santos PCR, Teixeira-Arroyo C, Gobbi LTB. Disease severity affects obstacle crossing in people with Parkinson's disease. Gait Posture. 2014;40(1):266–9.
19. Morris ME, McGinley J, Huxham F, Collier J, Iansek R. Constraints on the kinetic, kinematic and spatiotemporal parameters of gait in Parkinson's disease. Hum Mov Sci. 1999;18 (2):461–83.
20. del Olmo MF, Cudeiro J. Temporal variability of gait in Parkinson disease: effects of a rehabilitation programme based on rhythmic sound cues. Parkinsonism Relat Disord. 2005;11(1):25–33.
21. Sofuwa O, Nieuwboer A, Desloovere K, Willems AM, Chavret F, Jonkers I. Quantitative gait analysis in Parkinson's disease: comparison with a healthy control group. Arch Phys Med Rehabil. 2005;86(5):1007–13.
22. Lewek MD, Poole R, Johnson J, Halawa O, Huang X. Arm swing magnitude and asymmetry during gait in the early stages of Parkinson's disease. Gait Posture. 2010;31(2):256–60.
23. Vitório R, Lirani-Silva E, Barbieri FA, Raile V, Batistela RA, Stella F, Gobbi LTB. The role of vision in Parkinson's disease locomotion control: free walking task. Gait Posture. 2012;35 (2):175–9.
24. Galna B, Lord S, Burn DJ, Rochester L. Progression of gait dysfunction in incident Parkinson's disease: impact of medication and phenotype. Mov Disord. 2015;30(3):359–67.
25. Hausdorff JM, Balash J, Giladi N. Effects of cognitive challenge on gait variability in patients with Parkinson's disease. J Geriatr Psychiatry Neurol. 2003;16(1):53–8.
26. Rochester L, Hetherington V, Jones D, Nieuwboer A, Willems AM, Kwakkel G, Van Wegen E. Attending to the task: interference effects of functional tasks on walking in Parkinson's disease and the roles of cognition, depression, fatigue, and balance. Arch Phys Med Rehabil. 2004;85(10):1578–85.
27. Brown LA, de Bruin N, Doan JB, Suchowersky O, Hu B. Novel challenges to gait in Parkinson's disease: the effect of concurrent music in single-and dual-task contexts. Arch Phys Med Rehabil. 2009;90(9):1578–83.
28. Plotnik M, Dagan Y, Gurevich T, Giladi N, Hausdorff JM. Effects of cognitive function on gait and dual tasking abilities in patients with Parkinson's disease suffering from motor response fluctuations. Exp Brain Res. 2011;208(2):169–79.
29. Kelly VE, Eusterbrock AJ, Shumway-Cook A. A review of dual-task walking deficits in people with Parkinson's disease: motor and cognitive contributions, mechanisms, and clinical implications. Parkinsons Dis. 2012;2012:918719. 14 p.
30. Galna B, Murphy AT, Morris ME. Obstacle crossing in people with Parkinson's disease: foot clearance and spatiotemporal deficits. Hum Mov Sci. 2010;29(5):843–52.
31. Vitório R, Pieruccini-Faria F, Stella F, Gobbi S, Gobbi LTB. Effects of obstacle height on obstacle crossing in mild Parkinson's disease. Gait Posture. 2010;31(1):143–6.
32. Vitório R, Lirani-Silva E, Barbieri FA, Raile V, Stella F, Gobbi LTB. Influence of visual feedback sampling on obstacle crossing behavior in people with Parkinson's disease. Gait Posture. 2013;38(2):330–4.
33. Vitório R, Gobbi LTB, Lirani-Silva E, Moraes R, Almeida QJ. Synchrony of gaze and stepping patterns in people with Parkinson's disease. Behav Brain Res. 2016;307:159–64.

34. Stegemöller EL, Buckley TA, Pitsikoulis C, Barthelemy E, Roemmich R, Hass CJ. Postural instability and gait impairment during obstacle crossing in Parkinson's disease. Arch Phys Med Rehabil. 2012;93(4):703–9.
35. Bloem BR, Beckley DJ, Van Dijk JG, Zwinderman AH, Remler MP, Roos RA. Influence of dopaminergic medication on automatic postural responses and balance impairment in Parkinson's disease. Mov Disord. 1996;11(5):509–21.
36. Horak FB, Frank J, Nutt J. Effects of dopamine on postural control in parkinsonian subjects: scaling, set, and tone. J Neurophysiol. 1996;75(6):2380–96.
37. Schaafsma JD, Giladi N, Balash Y, Bartels AL, Gurevich T, Hausdorff JM. Gait dynamics in Parkinson's disease: relationship to parkinsonian features, falls and response to levodopa. J Neurol Sci. 2003;212(1):47–53.
38. Tunik E, Feldman AG, Poizner H. Dopamine replacement therapy does not restore the ability of parkinsonian patients to make rapid adjustments in motor strategies according to changing sensorimotor contexts. Parkinsonism Relat Disord. 2007;13(7):425–33.
39. Foreman KB, Wisted C, Addison O, Marcus RL, LaStayo PC, Dibble LE. Improved dynamic postural task performance without improvements in postural responses: the blessing and the curse of dopamine replacement. Parkinsons Dis. 2012;2012:692150. 8 p.
40. Blin O, Ferrandez AM, Pailhous J, Serratrice G. Dopa-sensitive and dopa-resistant gait parameters in Parkinson's disease. J Neurol Sci. 1991;103(1):51–4.
41. Pieruccini-Faria F, Vitório R, Almeida QJ, Silveira CRA, Caetano MJD, Stella F, Gobbi S, Gobbi LTB. Evaluating the acute contributions of dopaminergic replacement to gait with obstacles in Parkinson's disease. J Mot Behav. 2013;45(5):369–80.
42. Chaudhuri KR, Healy DG, Schapira AH. Non-motor symptoms of Parkinson's disease: diagnosis and management. Lancet Neurol. 2006;5(3):235–45.
43. Hausdorff JM, Schaafsma JD, Balash Y, Bartels AL, Gurevich T, Giladi N. Impaired regulation of stride variability in Parkinson's disease subjects with freezing of gait. Exp Brain Res. 2003;149(2):187–94.
44. Beauchet O, Annweiler C, Dubost V, Allali G, Kressig RW, Bridenbaugh S, Berrut G, Assal F, Herrmann FR. Stops walking when talking: a predictor of falls in older adults? Eur J Neurol. 2009;16(7):786–95.
45. Lees AJ, Hardy J, Revesz T. Parkinson's disease. Lancet. 2009;373(9680):2055–66.
46. Seppi K, Weintraub D, Coelho M, Perez-Lloret S, Fox SH, Katzenschlager R, Hametner E-M, Poewe W, Rascol O, Goetz CG, Sampaio C. The Movement Disorder Society evidence-based medicine review update: treatments for the non-motor symptoms of Parkinson's disease. Mov Disord. 2011;26 Suppl 3:S42–80.
47. Vorovenci RJ, Biundo R, Antonini A. Therapy-resistant symptoms in Parkinson's disease. J Neural Transm. 2016;123(1):19–30.
48. Goodwin VA, Richards SH, Taylor RS, Taylor AH, Campbell JL. The effectiveness of exercise interventions for people with Parkinson's disease: a systematic review and meta-analysis. Mov Disord. 2008;23(5):631–40.
49. Borrione P, Tranchita E, Sansone P, Parisi A. Effects of physical activity in Parkinson's disease: a new tool for rehabilitation. World J Methodol. 2014;4(3):133–43.
50. Oguh O, Eisenstein A, Kwasny M, Simuni T. Back to the basics: regular exercise matters in Parkinson's disease: results from the National Parkinson Foundation QII registry study. Parkinsonism Relat Disord. 2014;20(11):1221–5.
51. Fisher BE, Li Q, Nacca A, Salem GJ, Song J, Yip J, Hui JS, Jakowec MW, Petzinger GM. Treadmill exercise elevates striatal dopamine D2 receptor binding potential in patients with early Parkinson's disease. Neuroreport. 2013;24(10):509–14.
52. Sehm B, Taubert M, Conde V, Weise D, Classen J, Dukart J, Draganski B, Villringer A, Ragert P. Structural brain plasticity in Parkinson's disease induced by balance training. Neurobiol Aging. 2014;35(1):232–9.
53. Beall EB, Lowe MJ, Alberts JL, Frankemolle AM, Thota AK, Shah C, Phillips MD. The effect of forced-exercise therapy for Parkinson's disease on motor cortex functional connectivity. Brain Connect. 2013;3(2):190–8.

54. Paillard T, Rolland Y, de Souto Barreto P. Protective effects of physical exercise in Alzheimer's disease and Parkinson's disease: a narrative review. J Clin Neurol. 2015;11 (3):212–9.
55. Zou YM, Tan JP, Li N, Yang JS, Yu BC, Yu JM, et al. Do physical exercise and reading reduce the risk of Parkinson's disease? A cross-sectional study on factors associated with Parkinson's disease in elderly Chinese veterans. Neuropsychiatr Dis Treat. 2015;11:695–700.
56. Hirsch MA, Iyer SS, Sanjak M. Exercise-induced neuroplasticity in human Parkinson's disease: what is the evidence telling us? Parkinsonism Relat Disord. 2016;22 Suppl 1: S78–81.
57. Angelucci F, Piermaria J, Gelfo F, Shofany J, Tramontano M, Fiore M, Caltagirone C, Peppea A. The effects of motor rehabilitation training on clinical symptoms and serum BDNF levels in Parkinson's disease subjects. Can J Physiol Pharmacol. 2016;94(4):455–61.
58. Morris ME, Iansek R. Characteristics of motor disturbance in Parkinson's disease and strategies for movement rehabilitation. Hum Mov Sci. 1996;15(5):649–69.
59. Barbieri FA, Simieli L, Orcioli-Silva D, Gobbi LTB. Benefits of physical exercise for patients with Parkinson's disease In: Coelho FGM, Gobbi S, Costa JLR, Gobbi LTB (organizers), editors. Physical exercise in healthy and pathological aging: from theory to practice (in Portuguese). Curitiba: CRV; 2013. p. 325–39.
60. Mehrholz J, Friis R, Kugler J, Twork S, Storch A, Pohl M. Treadmill training for patients with Parkinson's disease. Cochrane Database Syst Rev. 2015;9:CD007830.
61. Protas EJ, Mitchell K, Williams A, Qureshy H, Caroline K, Lai EC. Gait and step training to reduce falls in Parkinson's disease. NeuroRehabilitation. 2005;20(3):183–90.
62. Herman T, Giladi N, Hausdorff JM. Treadmill training for the treatment of gait disturbances in people with Parkinson's disease: a mini-review. J Neural Transm. 2009;116(3):307–18.
63. Spaulding SJ, Barber B, Colby M, Cormack B, Mick T, Jenkins ME. Cueing and gait improvement among people with Parkinson's disease: a meta-analysis. Arch Phys Med Rehabil. 2013;94(3):562–70.
64. Bloem BR, de Vries NM, Ebersbach G. Nonpharmacological treatments for patients with Parkinson's disease. Mov Disord. 2015;30(11):1504–20.
65. Shen X, Wong-Yu IS, Mak MK. Effects of exercise on falls, balance, and gait ability in Parkinson's disease: a meta-analysis. Neurorehabil Neural Repair. 2016;30(6):512–27.
66. Abbruzzese G, Marchese R, Avanzino L, Pelosin E. Rehabilitation for Parkinson's disease: current outlook and future challenges. Parkinsonism Relat Disord. 2016;22 Suppl 1:S60–4.
67. Vitório R, Teixeira-Arroyo C, Lirani-Silva E, Barbieri FA, Caetano MJD, Gobbi S, Stella F, Gobbi LTB, Teixeira-Arroyo C. Effects of a 6-month multimodal exercise program on clinical and gait parameters of patients with idiopathic Parkinson's disease: a pilot study. ISRN Neurol. 2011;2011:714947.
68. Gobbi LTB, Oliveira-Ferreira MD, Caetano MJ, Lirani-Silva E, Barbieri FA, Stella F, Gobbi S. Exercise programs improve mobility and balance in people with Parkinson's disease. Parkinsonism Relat Disord. 2009;15 Suppl 3:S49–52.
69. Rose MH, Løkkegaard A, Sonne-Holm S, Jensen BR. Improved clinical status, quality of life, and walking capacity in Parkinson's disease after body weight-supported high-intensity locomotor training. Arch Phys Med Rehabil. 2013;94(4):687–92.
70. Carvalho A, Barbirato D, Araujo N, Martins JV, Cavalcanti JL, Santos TM, Coutinho ES, Laks J, Deslandes AC. Comparison of strength training, aerobic training, and additional physical therapy as supplementary treatments for Parkinson's disease: pilot study. Clin Interv Aging. 2015;10:183–91.
71. Jenkinson C, Fitzpatrick R, Peto V, Greenhall R, Hyman N. The Parkinson's Disease Questionnaire (PDQ-39): development and validation of a Parkinson's disease summary index score. Age Ageing. 1997;26(5):353–7.
72. Brusse KJ, Zimdars S, Zalewski KR, Steffen TM. Testing functional performance in people with Parkinson disease. Phys Ther. 2005;85(2):134–41.

73. Goetz CG, Poewe W, Rascol O, Sampaio C, Stebbins GT, Counsell C, Giladi N, Holloway RG, Moore CG, Wenning GK, Yahr MD, Seidl L. Movement Disorder Society Task Force report on the Hoehn and Yahr staging scale: status and recommendations. Mov Disord. 2004;19(9):1020–8.
74. Falvo MJ, Earhart GM. Six-minute walk distance in persons with Parkinson disease: a hierarchical regression model. Arch Phys Med Rehabil. 2009;90(6):1004–8.
75. Duncan RP, Leddy AL, Cavanaugh JT, Dibble LE, Ellis TD, Ford MP, Bo Foreman K, Earhart GM. Comparative utility of the BESTest, mini-BESTest, and brief-BESTest for predicting falls in individuals with Parkinson disease: a cohort study. Phys Ther. 2013;93 (4):542–50.
76. Keus SH, Bloem BR, Hendriks EJ, Bredero-Cohen AB, Munneke M; Practice Recommendations Development Group. Evidence-based analysis of physical therapy in Parkinson's disease with recommendations for practice and research. Mov Disord. 2007;22 (4):451–60.
77. Scandalis TA, Bosak A, Berliner JC, Helman LL, Wells MR. Resistance training and gait function in patients with Parkinson's disease. Am J Phys Med Rehabil. 2001;80(1):38–43.
78. Uc EY, Doerschug KC, Magnotta V, Dawson JD, Thomsen TR, Kline JN, Rizzo M, Newman SR, Mehta S, Grabowski TJ, Bruss J, Blanchette DR, Anderson SW, Voss MW, Kramer AF, Darling WG. Phase I/II randomized trial of aerobic exercise in Parkinson disease in a community setting. Neurology. 2014;83(5):413–25.
79. Shu HF, Yang T, Yu SX, Huang HD, Jiang LL, Gu JW, Kuang Y-Q. Aerobic exercise for Parkinson's disease: a systematic review and meta-analysis of randomized controlled trials. PLoS One. 2014;9(7):e100503.
80. Wong-Yu ISK, Mak MKY. Multi-dimensional balance training programme improves balance and gait performance in people with Parkinson's disease: a pragmatic randomized controlled trial with 12-month follow-up. Parkinsonism Relat Disord. 2015;21(6):615–21.
81. Brodie MA, Dean RT, Beijer TR, Canning CG, Smith ST, Menant JC, Lord SR. Symmetry matched auditory cues improve gait steadiness in most people with Parkinson's disease but not in healthy older people. J Parkinsons Dis. 2015;5(1):105–16.
82. Monteiro EP, Franzoni LT, Cubillos DM, de Oliveira Fagundes A, Carvalho AR, Oliveira HB, Pantoja PD, Schuch FB, Rieder CR, Martinez FG, Peyré-Tartaruga LA. Effects of nordic walking training on functional parameters in Parkinson's disease: a randomized controlled clinical trial. Scand J Med Sci Sports. 2016.
83. Reuter I, Mehnert S, Leone P, Kaps M, Oechsner M, Engelhardt M. Effects of a flexibility and relaxation programme, walking, and nordic walking on Parkinson's disease. J Aging Res. 2011;2011:232473.
84. van Eijkeren FJ, Reijmers RS, Kleinveld MJ, Minten A, Bruggen JP, Bloem BR. Nordic walking improves mobility in Parkinson's disease. Mov Disord. 2008;23(15):2239–43.
85. Rocha PA, Porfírio GM, Ferraz HB, Trevisani VF. Effects of external cues on gait parameters of Parkinson's disease patients: a systematic review. Clin Neurol Neurosurg. 2014;124:127–34.
86. Ganesan M, Sathyaprabha TN, Gupta A, Pal PK. Effect of partial weight-supported treadmill gait training on balance in patients with Parkinson disease. PM&R. 2014;6(1):22–33.
87. Ganesan M, Sathyaprabha TN, Pal PK, Gupta A. Partial body weight-supported treadmill training in patients with Parkinson disease: impact on gait and clinical manifestation. Arch Phys Med Rehabil. 2015;96(9):1557–65.
88. Schlick C, Ernst A, Bötzel K, Plate A, Pelykh O, Ilmberger J. Visual cues combined with treadmill training to improve gait performance in Parkinson's disease: a pilot randomized controlled trial. Clin Rehabil. 2016;30(5):463–71.
89. Mirelman A, Maidan I, Herman T, Deutsch JE, Giladi N, Hausdorff JM. Virtual reality for gait training: can it induce motor learning to enhance complex walking and reduce fall risk in patients with Parkinson's disease? J Gerontol A Biol Sci Med Sci. 2011;66A(2):234–40.
90. Picelli A, Melotti C, Origano F, Waldner A, Fiaschi A, Santilli V, Smania N. Robot-assisted gait training in patients with Parkinson disease: a randomized controlled trial. Neurorehabil Neural Repair. 2012;26(4):353–61.

91. Picelli A, Melotti C, Origano F, Waldner A, Gimigliano R, Smania N. Does robotic gait training improve balance in Parkinson's disease? A randomized controlled trial. Parkinsonism Relat Disord. 2012;18(8):990–3.
92. Barry G, Galna B, Rochester L. The role of exergaming in Parkinson's disease rehabilitation: a systematic review of the evidence. J Neuroeng Rehabil. 2014;11(1):33.
93. Harris DM, Rantalainen T, Muthalib M, Johnson L, Teo W-P. Exergaming as a viable therapeutic tool to improve static and dynamic balance among older adults and people with idiopathic Parkinson's disease: a systematic review and meta-analysis. Front Aging Neurosci. 2015;7:167.
94. de Dreu MJ, van der Wilk AS, Poppe E, Kwakkel G, van Wegen EE. Rehabilitation, exercise therapy and music in patients with Parkinson's disease: a meta-analysis of the effects of music-based movement therapy on walking ability, balance and quality of life. Parkinsonism Relat Disord. 2012;18 Suppl 1:S114–9.
95. Sharp K, Hewitt J. Dance as an intervention for people with Parkinson's disease: a systematic review and meta-analysis. Neurosci Biobehav Rev. 2014;47:445–56.
96. Yang Y, Li XY, Gong L, Zhu YL, Hao YL. Tai Chi for improvement of motor function, balance and gait in Parkinson's disease: a systematic review and meta-analysis. PLoS One. 2014;9(7), e102942.
97. Hackney ME, Earhart GM. Effects of dance on movement control in Parkinson's disease: a comparison of Argentine tango and American ballroom. J Rehabil Med. 2009;41(6):475–81.
98. McNeely ME, Mai MM, Duncan RP, Earhart GM. Differential effects of Tango versus dance for PD in Parkinson disease. Front Aging Neurosci. 2015;7:239.
99. Li F, Harmer P, Fitzgerald K, Eckstrom E, Stock R, Galver J, Maddalozzo G, Batya SS. Tai Chi and postural stability in patients with Parkinson's disease. N Engl J Med. 2012;366:511–9.
100. Cano-de-la-Cuerda R, Molero-Sánchez A, Carratalá-Tejada M, Alguacil-Diego IM, Molina-Rueda F, Miangolarra-Page JC, Torricelli D. Theories and control models and motor learning: clinical applications in neurorehabilitation. Neurologia. 2015;30(1):32–41.
101. Ene H, McRae C, Schenkman M. Attitudes toward exercise following participation in an exercise intervention study. J Neurol Phys Ther. 2011;35(1):34–40.
102. Ellis T, Cavanaugh JT, Earhart GM, Ford MP, Foreman KB, Fredman L, Boudreau JK, Dibble LE. Factors associated with exercise behavior in people with Parkinson disease. Phys Ther. 2011;91(12):1838–48.
103. Dibble LE, Hale TF, Marcus RL, Gerber JP, LaStayo PC. High intensity eccentric resistance training decreases bradykinesia and improves quality of life in persons with Parkinson's disease: a preliminary study. Parkinsonism Relat Disord. 2009;15(10):752–7.
104. Ayán C, Cancela JM, Gutiérrez-Santiago A, Prieto I. Effects of two different exercise programs on gait parameters in individuals with Parkinson's disease: a pilot study. Gait Posture. 2014;39(1):648–51.
105. Koller W, Kase S. Muscle strength testing in Parkinson's disease. Eur Neurol. 1986;25 (2):130–3.
106. Kakinuma S, Nogaki H, Pramanik B, Morimatsu M. Muscle weakness in Parkinson's disease: isokinetic study of the lower limbs. Eur Neurol. 1998;39(4):218–22.
107. Cano-de-la-Cuerda R, Pérez-de-Heredia M, Miangolarra-Page JC, Munoz-Hellín E, Fernández-de-las-Penas C. Is there muscular weakness in Parkinson's disease? Am J Phys Med Rehabil. 2010;89(1):70–6.
108. Corcos DM, Chen CM, Quinn NP, McAuley J, Rothwell JC. Strength in Parkinson's disease: relationship to rate of force generation and clinical status. Ann Neurol. 1996;39:79–88.
109. Falvo MJ, Schilling BK, Earhart GM. Parkinson's disease and resistive exercise: rationale, review, and recommendations. Mov Disord. 2008;23(1):1–11.
110. van Nimwegen M, Speelman AD, Hofman-van Rossum EJ, Overeem S, Deeg DJ, Borm GF, van der Horst MHL, Bloem BR, Munneke M. Physical inactivity in Parkinson's disease. J Neurol. 2011;258(12):2214–21.

111. Schilling BK, Karlage RE, LeDoux MS, Pfeiffer RF, Weiss LW, Falvo MJ. Impaired leg extensor strength in individuals with Parkinson disease and relatedness to functional mobility. Parkinsonism Relat Disord. 2009;15(10):776–80.
112. Allen N, Sherrington C, Canning C, Fung V. Reduced muscle power is associated with slower walking velocity and falls in people with Parkinson's disease. Parkinsonism Relat Disord. 2010;16(4):261–4.
113. Dibble LE, Hale TF, Marcus RL, Droge J, Gerber JP, LaStayo PC. High-intensity resistance training amplifies muscle hypertrophy and functional gains in persons with Parkinson's disease. Mov Disord. 2006;21(9):1444–52.
114. Hass CJ, Buckley TA, Pitsikoulis C, Barthelemy EJ. Progressive resistance training improves gait initiation in individuals with Parkinson's disease. Gait Posture. 2012;35:669–73.
115. Schilling BK, Pfeiffer RF, LeDoux MS, Karlage RE, Bloomer RJ, Falvo MJ. Effects of moderate-volume, high-load lower-body resistance training on strength and function in persons with Parkinson's disease: a pilot study. Parkinson Dis. 2010;2010. Article ID 824734.
116. Hirsch MA, Toole T, Maitland CG, Rider RA. The effects of balance training and high-intensity resistance training on persons with idiopathic Parkinson's disease. Arch Phys Med Rehabil. 2003;84(8):1109–17.
117. Allen NE, Canning CG, Sherrington C, Lord SR, Latt MD, Close JCT, O'Rourke SD, Murray SM, Fung VSC. The effects of an exercise program on fall risk factors in people with Parkinson's disease: a randomized controlled trial. Mov Disord. 2010;25(9):1217–25.
118. Chung CLH, Thilarajah S, Tan D. Effectiveness of resistance training on muscle strength and physical function in people with Parkinson's disease: a systematic review and meta-analysis. Clin Rehabil. 2016;30(1):11–23.
119. Shulman LM, Katzel LI, Ivey FM, Sorkin JD, Favors K, Anderson KE, Smith BA, Reich SG, Weiner WJ, Macko RF. Randomized clinical trials of 3 types of physical exercise for patients with Parkinson's disease. JAMA Neurol. 2013;70(2):183–90.
120. Speelman AD, Groothuis JT, van Nimwegen M, van der Scheer ES, Borm GF, Bloem BR, Hopman MTE, Munneke M. Cardiovascular responses during a submaximal exercise test in patients with Parkinson's disease. J Parkinsons Dis. 2012;2(3):241–7.
121. Stanley RK, Protas EJ, Jankovic J. Exercise performance in those having Parkinson's disease and healthy normals. Med Sci Sports Exerc. 1999;31(6):761–6.
122. Sage MD, Almeida QJ. Symptom and gait changes after sensory attention focused exercise vs aerobic training in Parkinson's disease. Mov Disord. 2009;24(8):1132–8.
123. Canning CG, Allen NE, Dean CM, Goh L, Fung VS. Home-based treadmill training for individuals with Parkinson's disease: a randomized controlled pilot trial. Clin Rehabil. 2012;26(9):817–26.
124. Cakit BD, Saracoglu M, Genc H, Erdem HR, Inan L. The effects of incremental speed-dependent treadmill training on postural instability and fear of falling in Parkinson's disease. Clin Rehabil. 2007;21(8):698–705.
125. Fisher BE, Wu AD, Salem GJ, Song J, Lin C-H, Yip J, Cen S, Gordon J, Jakowec M, Petzinger G. The effect of exercise training in improving motor performance and corticomotor excitability in people with early Parkinson's disease. Arch Phys Med Rehabil. 2008;89(7):1221–9.
126. Frazzitta G, Maestri R, Uccellini D, Bertotti G, Abelli P. Rehabilitation treatment of gait in patients with Parkinson's disease with freezing: a comparison between two physical therapy protocols using visual and auditory cues with or without treadmill training. Mov Disord. 2009;24(8):1139–43.
127. Miyai I, Fujimoto Y, Ueda Y, Yamamoto H, Nozaki S, Saito T, Jin Kang MD, et al. Treadmill training with body weight support: its effect on Parkinson's disease. Arch Phys Med Rehabil. 2000;81(7):849–52.
128. Uhrbrand A, Stenager E, Pedersen MS, Dalgas U. Parkinson's disease and intensive exercise therapy – a systematic review and meta-analysis of randomized controlled trials. J Neurol Sci. 2015;353(1):9–19.

129. Trigueiro LCL, Gama GL, Simão CR, de Sousa AVC, Godeiro Júnior CG, Lindquist ARR. Effects of treadmill training with load on gait in Parkinson disease: a randomized controlled clinical trial. Am J Phys Med Rehabil. 2015;94(10):830–7.
130. Morris ME, Matias TA, Iansek R, Summers JJ. Temporal stability of gait in Parkinson's disease. Phys Ther. 1996;76(7):763–89.
131. Pohl M, Rockstroh G, Rückriem S, Mrass G, Mehrholz J. Immediate effects of speed-dependent treadmill training on gait parameters in early Parkinson's disease. Arch Phys Med Rehabil. 2003;84(12):1760–6.
132. Tanaka K, Quadros Jr AC, Santos RF, Stella F, Gobbi LTB, Gobbi S. Benefits of physical exercise on executive functions in older people with Parkinson's disease. Brain Cogn. 2009;69:435–41.
133. Baptista AM, Gobbi LTB, Beretta VS, Vitório R, Teixeira-Arroyo C, Lirani-Silva E, Stella F, Barbieri FA. Long duration exercise program in individuals with Parkinson's disease: effects on functional capacity. Austin Alzheimers J Parkinsons Dis. 2014;1(1):7.
134. Orcioli-Silva D, Barbieri FA, Simieli L, Rinaldi NM, Vitório R, Gobbi LTB. Effects of a multimodal exercise program on the functional capacity of Parkinson's disease patients considering disease severity and gender. Motriz. 2014;20(1):100–6.
135. Patel N, Jankovic J, Hallett M. Sensory aspects of movement disorders. Lancet Neurol. 2014;13(1):100–12.
136. Takakusaki K. Forebrain control of locomotor behaviors. Brain Res Rev. 2008;57(1):192–8.
137. Takakusaki K, Tomita N, Yano M. Substrates for normal gait and pathophysiology of gait disturbances with respect to the basal ganglia dysfunction. J Neurol. 2008;255(4):19–29.
138. Kelly VE, Johnson CO, McGough EL, Shumway-Cook A, Horak FB, Chung KA, Espaye AJ, Revilla FJ, Devoto J, Wood-Siverio C, Factor SA, Cholerton B, Edwards KL, Peterson AL, Quinn JF, Montine TJ, Zabetian CP, Leverenz JB. Association of cognitive domains with postural instability/gait disturbance in Parkinson's disease. Parkinsonism Relat Disord. 2015;21(7):692–7.
139. Varalta V, Picelli A, Fonte C, Amato S, Melotti C, Zatezalo V, Saltuari L, Smania N. Relationship between cognitive performance and motor dysfunction in patients with Parkinson's disease: a pilot cross-sectional study. Biomed Res Int. 2015;2015:365959. 6 p.
140. Yang Y, Qiu WQ, Hao YL, Lv ZY, Jiao SJ, Teng JF. The efficacy of traditional chinese medical exercise for Parkinson's disease: a systematic review and meta-analysis. PLoS One. 2015;10(4):e0122469.
141. Ospina M, Bond K, Karkhaneh M, Tjosvold L, Vandermeer B, Liang Y, et al. Meditation practices for health: state of the research. Evid Rep Technol Assess (Full Rep). 2007;155 (155):1–263.
142. Amano S, Nocera JR, Vallabhajosula S, Juncos JL, Gregor RJ, Waddell DE, Wolf SL, Hass CJ. The effect of Tai Chi exercise on gait initiation and gait performance in persons with Parkinson's disease. Parkinsonism Relat Disord. 2013;19(11):955–60.
143. Hackney ME, Earhart GM. Tai Chi improves balance and mobility in people with Parkinson disease. Gait Posture. 2008;28(3):456–60.
144. Caetano MJD, Lord SR, Schoene D, Pelicioni PHS, Sturnieks DL, Menant JC. Age-related changes in gait adaptability in response to unpredictable obstacles and stepping targets. Gait Posture. 2016;46:35–41.
145. Caetano MJD, Gobbi LTB, Sánchez-Arias MR, Stella F, Gobbi S. Effects of postural threat on walking features of Parkinson's disease patients. Neurosci Lett. 2009;452:136–40.
146. Pereira VAI, Barbieri FA, Vitório R, Simieli L, Lirani-Silva E, Orcioli-Silva D, Gobbi LTB. The motor deficits caused by Parkinson's disease are not able to block adjustments for a safe strategy during obstacle crossing in individuals with moderate disease. Motriz. 2015;21:436–41.
147. Yogev G, Giladi N, Peretz C, Springer S, Simon ES, Hausdorff JM. Dual tasking, gait rhythmicity, and Parkinson's disease: which aspects of gait are attention demanding? Eur J Neurosci. 2005;22:1248–56.

148. Pieruccini-Faria F, Menuchi MRTP, Vitório R, Gobbi LTB, Stella F, Gobbi S. Kinematic parameters for gait with obstacles among elderly patients with Parkinson's disease, with and without Levodopa: a pilot study. Braz J Physiother. 2006;10(2):243–9.
149. Almeida QJ. Timing control in Parkinson's disease. In: Dushanova J, editor. Mechanism in Parkinson's disease – models and treatments. Rijeka: Intech; 2012. p. 39–56.
150. Pelicioni PHS. Impact of Parkinson's disease on kinetics and kinematics features of sit to walk task in fallers and non-fallers (in Portuguese). 2015. http://base.repositorio.unesp.br/bitstream/handle/1149/126354/000841318.pdf.
151. Teasdale N, Phillips J, Stelmach GE. Temporal movement control in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry. 1990;53:862–8.
152. Farley BG, Koshland GF. Training BIG to move faster: the application of the speed-amplitude relation as a rehabilitation strategy for people with Parkinson's disease. Exp Brain Res. 2005;167:462–7.
153. Bello O, Sanchez JA, Lopez-Alonso V, Márquez G, Morenilla L, Castro X, Giraldez M, Santos-García D, Fernandez-del-Olmo M. The effects of treadmill or overground walking training program on gait in Parkinson's disease. Gait Posture. 2013;38:590–5.
154. Harro CC, Schoemaker MJ, Frey OJ, Gamble AC, Harring KB, Karl KL, McDonald JD, Murray CJ, VanDyke JM, Tomassi EM, VanHaitsma RJ. The effects of speed-dependent treadmill training and rhythmic auditory-cued overground walking on gait function and fall risk in individuals with idiopathic Parkinson's disease: a randomized controlled trial. NeuroRehabilitation. 2014;34:557–72.
155. Gobbi LTB, Teixeira-Arroyo C, Lirani-Silva E, Vitório R, Barbieri FA, Pereira MP. Effect of different exercise programs on the psychological and cognitive functions of people with Parkinson's disease. Motriz. 2013;19:597–604.
156. Yogev-Seligmann G, Giladi N, Brozgol M, Hausdorff JM. A training program to improve gait while dual tasking in patients with Parkinson's disease: a pilot study. Arch Phys Med Rehabil. 2012;93:176–81.
157. McKee KE, Hackney ME. The four square step test in individuals with Parkinson's disease: association with executive function and comparison with older adults. NeuroRehabilitation. 2014;35:279–89.
158. Ginis S, Nieuwboer A, Dorfman M, Ferrari A, Gazit E, Canning CG, Rocchi L, Chiari L, Hausdorff JM, Mirelman A. Feasibility and effects of home-based smartphone-delivered automated feedback training for gait in people with Parkinson's disease: a pilot randomized controlled trial. Parkinsonism Relat Disord. 2016;22:28–34.
159. Kim H, Jae HD, Jeong JH. Tai Chi exercise can improve the obstacle negotiating ability of people with Parkinson's disease: a preliminary study. J Phys Ther Sci. 2014;26:1025–30.
160. Liao YY, Yang YR, Cheng SJ, Wu YR, Fuh JL, Wang RY. Virtual reality-based training to improve obstacle-crossing performance and dynamic balance in patients with Parkinson's disease. Neurorehabil Neural Repair. 2015;29:658–67.

Part III

Posture

Older Adults Are Incapable of Changing Sensory-Motor Coupling During Upright Stance with Visual Manipulation and Different Attentional Demands

13

Giovanna Gracioli Genoves, Caroline Sanches, and José Angelo Barela

Abstract

Postural control functioning is characterized by adaptive adjustments, which involves several central nervous system centers. This study investigated the sensory-motor coupling in older and younger adults during concurrent different cognitive tasks while maintaining upright stance with visual manipulation. Fifteen older and 15 younger adults performed 9 trials of upright stance in a moving room. In the first trial, the room remained stationary. In the following 4 trials, the room oscillated but participants were not informed about visual manipulation and in two trials were required to countdown. In the last 4 trials, they were informed about the visual manipulation and were requested to resist to it and repeated the countdown twice again. Results indicated that older swayed with larger magnitude than younger adults. Visual manipulation induced correspondent sway in all participants and information and request to resist reduced visual influence only for young adults when they were not counting. These results suggest that sensory-motor coupling might be changed but requires attentional resources and young adults only achieve it. Differently, older adults are incapable of changing sensory-coupling that might be limited by the lack of attentional resources.

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Elderly • Postural control • Visual • Attention demand • Sensory-motor coupling

13.1 Introduction

Postural control involves an intricate and complex relationship between sensory information and motor activity, with sensory cues coming from multiple sources and the muscular system comprising many muscles. The complexity of the sensory-motor relationship gets even more complex because environmental conditions and the forces acting on the body orientation and equilibrium are constantly changing, requiring a continuous sensory update and muscle activation to accomplish the postural task goal [1].

Aging is associated with changes in many of the performing capabilities, usually leading to a lower adaptability and performance [2, 3], including deterioration of postural control [4, 5]. For instance, several studies have shown that after a certain age, usually around 60 years, in a quiet stance, older adults sway with larger magnitude and/or velocity than younger adults [6–8]. These changes in sway characteristics of older adults are interpreted as a poor functioning of postural control system due to the aging process [2].

Studies have also shown that poor postural control in older adults is associated with changes in sensory information and body sway coupling [5, 8, 9]. For instance, changes in postural control functioning in older adults could be related to how older adults acquire and integrate sensory cues in order to provide precise and reliable information about body sway dynamics and, based upon this information, activate properly the muscles to maintain and/or achieve a desired postural orientation and equilibrium [2].

Postural control performance in older adults is also affected by attentional demands, with larger body sway occurring when upright stance has to be maintained while performing a concomitant cognitive task [10, 11]. Although still debatable, older adults would need to compensate changes in sensory re-weighting and central integrative processes, leading to the need of more cognitive resources to maintain a desired postural orientation. Moreover, the attentional effects on postural control performance is also task-dependent in such a way that more challenging postural control tasks are most affected by concomitant attentional requirements [12, 13]. In this case, the relationship between the acquired information, and the motor response, required to maintaining postural orientation and equilibrium, needs to be far more tied, requiring higher cognitive involvement [8]. Taken together these results, postural control of older adults might require higher attentional demands overloading the cognitive capabilities and, therefore, leading to a poor postural control performance even in daily tasks.

Considering that postural control requires sensory-motor coupling and that higher attentional demands deteriorate postural control performance, one may question if attention is required to perform and/or to improve the relationship between sensory information and body sway. Such question is even more relevant,

considering that when young adults are exposed to sensory manipulation, for instance visual oscillation due to a moving room, correspondent body sway is induced with any discriminative knowledge [7, 14, 15]. More interesting, however, is that when participants are informed that they have been influenced by a "trick" visual manipulation, body sway magnitude and visual influence are reduced [14–16], although with attentional demands needed to reduce visual influence on body sway.

Despite the recent studies, no knowledge is available if older adults would be able to avoid or change sensory-motor coupling as observed for younger adults in situations of visual manipulation. Correspondent body sway has also been induced in older adults due to visual manipulation [5, 8] with older adults being even more influenced by such manipulation [17]. Thus, what would be the effects of requesting them to resist to the visual influence? Considering that older adult postural performance is affected by concomitant attentional requirements and that changes in sensory-motor coupling demand attentional resources, what would be the impact of a concomitant task in older adults sensory-motor coupling? Therefore, the aim of this study was to investigate the sensory-motor coupling in older and younger adults during concurrent different cognitive tasks while maintaining upright stance with visual manipulation.

13.2 Methods

13.2.1 Participants

Fifteen younger (23.00 ± 2.10 years, 1.66 ± 7.60 m, and 64.00 ± 11.80 kg) and 15 older adults ($65.00 \pm 3.6.0$ years, 1.60 ± 6.20 m, and 70.10 ± 9.40 kg) participated in this study, constituting the younger (YA) and the older adults group (OA). The participants did not present any known health impairment that could affect their performance in maintaining upright stance, and all of them signed a written informed consent approved by the Institutional Ethics Committee of State University of São Paulo, Rio Claro.

13.2.2 Procedures

In a single visit to the laboratory, participants were asked to maintain upright and quite stance inside of a moving room, fixating a target (5 cm of diameter, 1 m away) positioned on the front wall. The moving room consisted of three walls and a roof (2.1 m long $\times$ 2.1 m wide $\times$ 2.1 m height), mounted on wheels so that it could be moved back and forth by a servomotor mechanism, while the floor remained motionless. The walls and the roof were white, and black stripes were painted on the walls, creating a pattern of 42 cm wide vertical white and 22 cm wide vertical black stripes. A 20-W fluorescent lamp was attached to the ceiling and used to maintain consistent light throughout data acquisition.

The room's movement was produced by a servomotor system composed of a stepper motor (Ottime, model SM23 SSF11921088), and a motor driver (Ottime, model MBD-278AC) and controlled by specific routines (Motion Planner 4.3). Participants stood on a force platform (Kirstler, model 9286A), with feet parallel and apart at the waist width, and were requested to stand as still as possible with arms hung passively beside their bodies.

Each participant performed nine trials of 60 s each. In the first trial, the room was stationary, and in the remaining trials, the moving room was oscillated back and forward at a peak velocity of 0.6 cm/s, peak-to-peak amplitude of 0.6 cm and frequency of 0.2 Hz. In the first block of moving trials (trials 2, 3, 4, and 5), no information about the room movement was provided and none of the participants discriminate that the room was oscillated (no instruction condition). In the second block of moving trials (trials 6, 7, 8, and 9), participants were informed that the room was oscillating and was inducing corresponding body oscillation, and that they should resist to such room's influence (resist condition). In two trials of both blocks (no instruction and resist conditions), participants were requested to count backwards from 100 in steps of three (100, 97, 94, . . .), performing either a dual- or a tri-task: upright stance with no instruction and counting (dual-task); upright stance, resisting to room's movement and counting (tri-task). The order of the counting trials was defined randomly. Overall, four experimental conditions were performed by the groups: (1) no instruction and no counting; (2) no instruction and counting; (3) resist and no counting; and (4) resist and counting.

The moving room displacement, in the anterior-posterior direction, was obtained using an electrical goniometer (EMG System do Brasil). Data from the force platform and electrical goniometer were synchronized and acquired with an analogic data acquisition unit (ODAU II—OPTOTRAK 3010—Northern Digital, Inc.), at frequency of 100 Hz.

13.2.3 Data Analysis

From the ground reaction forces, center of pressure (CP) was obtained for both anterior-posterior (AP) and medial-lateral (ML) directions. Following, the CP and the moving room position were filtered, second-order zero lag low-pass Butterworth digital filter, with a cut-off frequency of 5 Hz. Because movement of the room occurred in the AP direction, the magnitude of CP sway and the relationship between CP sway and the moving room position were calculated only for this direction.

The magnitude of CP trajectory sway was calculated fitting a first order polynomial to each trial and subtracted from each CP data point of the respective trial to exclude any shifting not related to the movement induced by the moving room. Following, the standard deviation from all values of the corresponding trial was calculated and used to indicate mean sway variability.

The relationship between the movement of the room and CP sway was examined by calculating the following variables: coherence, gain, and phase. Coherence indicates the relationship strength between the room movement and the CP sway.

Coherence values close to one/zero indicate strong/weak dependency between the two signals, and it was calculated at frequency of the driving signal (moving room) at 0.2 Hz. Gain and phase indicate the moving room magnitude influence on the CP sway. Taken together, these variables indicate the coupling structure between body sway and visual information manipulation. These variables were calculated through a transfer function [frequency response function (FRF)], which was computed by dividing the Fourier transforms of CP signals by the Fourier transform of the driving signal (moving room), resulting in a complex-valued transfer function. Gain corresponded to the ratio between the amplitude of CP signals and visual stimulus, at the driving frequency (0.2 Hz). Gain values of one indicate response amplitude similar to the stimulus amplitude and lower/higher values indicate that the response amplitude was lower/higher than the stimulus driving amplitude. Phase depicts the temporal relationship between the oscillation of CP and the visual stimulus. Positive (negative) phase values indicate that sway was ahead (behind) the visual stimulus.

Considering that two trials for each experimental condition were obtained and analyzed, values for each respective condition were averaged and used for further statistical analyses. All the described analyses were computed using custom routines written in Matlab (Math Works, Inc.)

13.2.4 Statistical Analysis

Initially, an analysis of variance (ANOVA), using as factor group (OA and YA) and as dependent variable the mean sway amplitude in the condition that the room remained stationary, was employed. Following, two ANOVAs and one multivariate analysis of variance (MANOVA) were employed, using as factors group (OA and YA) and condition (no information and no counting; no information and counting; resist and no counting; and resist and counting) with repeated measures for the last factor. The dependent variables for each ANOVA were mean sway amplitude and coherence. The dependent variables for the MANOVA were gain and phase. When necessary, univariate analyses and Tukey post hoc tests (HSD Turkey test) were employed. All analyses were performed using SPSS software and the significance level was kept at 0.05.

13.3 Results

Figure 13.1 depicts mean sway amplitude for both groups in the condition that the room remained stationary. ANOVA revealed that older adults swayed with larger magnitude compared to young adults, $F(1,28) = 14.00$, $p < 0.005$.

The manipulation of visual information, due to the moving room, induced corresponding CP sway in both younger and older adults. Figure 13.2 depicts exemplar time series of moving room position and CP sway of an older (a) and a younger adult (b), in the no instruction and no counting condition. Inspection of this

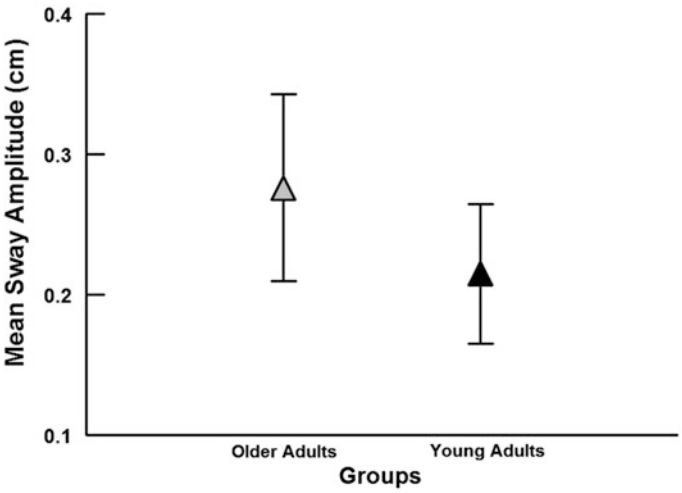

Fig. 13.1 Mean (± SD) values of mean sway amplitude for both older and young adults, in the condition that the room remained stationary

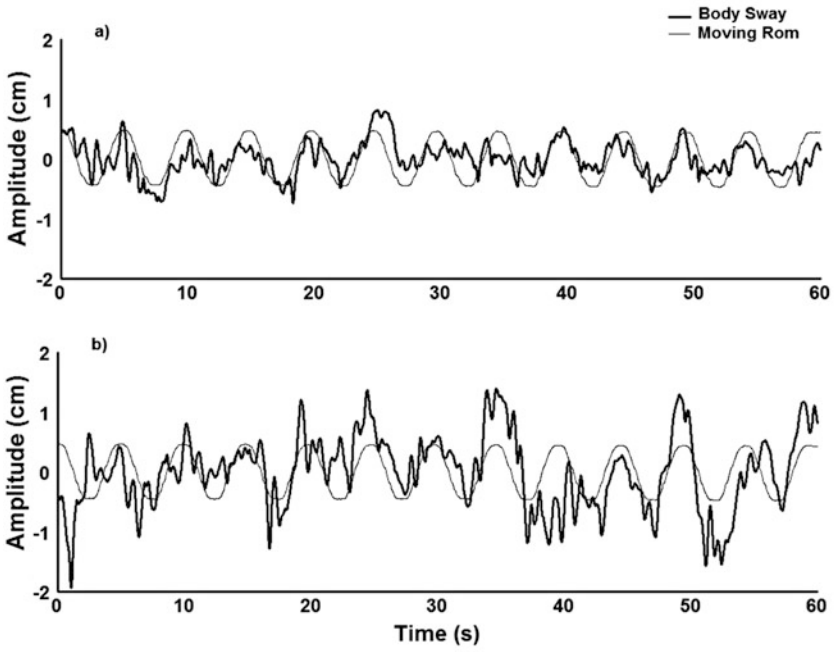

Fig. 13.2 Exemplar time series of the moving room and body sway, in the anterior-posterior direction, for a young (**a**) and an older adult (**b**), in the no instruction and no counting trial

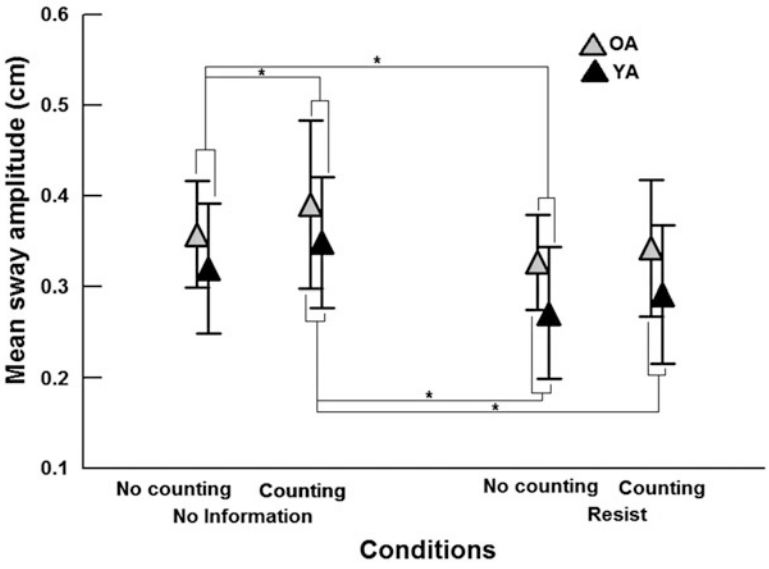

Fig. 13.3 Mean (± SD) values of mean sway amplitude for both older (OA) and young adults (YA) in all four experimental conditions (no instruction and no counting, no instruction and counting, resist and no counting, and resist and counting)

figure shows that both participants followed the visual manipulation, displaying corresponding CP sway as the moving room was oscillated back and forward throughout the trial.

Figure 13.3 depicts mean sway amplitude for both groups in all experimental conditions that the room oscillated. ANOVA revealed group ($F_{1,28} = 4.89$, $p < 0.05$) and condition ($F_{3,84} = 11.00$, $p < 0.001$) effects, but no group by condition interaction ($F_{3,84} = 0.22$, $p > 0.05$). The magnitude of CP sway was larger for the older when compared to younger adults. Post hoc tests indicated that in the no instruction condition, counting increased CP sway magnitude compared to no counting. In the resist condition, CP sway magnitude decreased in the no counting compared to both no counting and counting at the no instruction condition. Finally, CP sway magnitude in the counting was smaller in the resist compared to the no instruction condition.

Figure 13.4 depicts coherence values for both groups in all conditions that the room oscillated. ANOVA revealed no group ($F_{1,28} = 0.64$, $p > 0.05$) and no condition ($F_{3,84} = 2.06$, $p > 0.05$) effect, but revealed group and condition interaction ($F_{3,84} = 4.26$, $p < 0.05$). Post hoc tests revealed that for younger adults' coherence values decreased in the resist and no counting condition when compared with no instruction and counting condition. For the older adults, no difference was observed among all conditions.

Figure 13.5 depicts gain and phase values for both groups in all experimental conditions that the room oscillated. MANOVA revealed no group effect (Wilks'

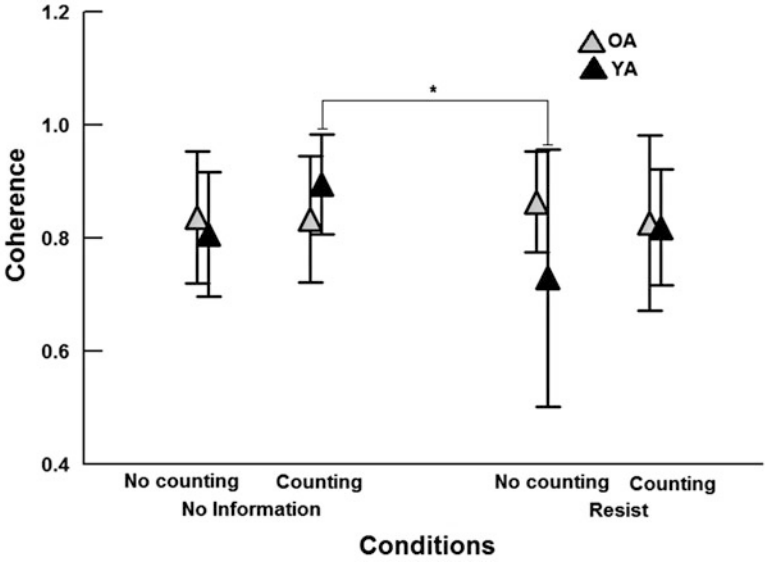

Fig. 13.4 Mean (± SD) values for coherence for both older (OA) and young adults (YA) in all four experimental conditions (no instruction and no counting, no instruction and counting, resist and no counting, and resist and counting)

Lambda = 0.88, $F_{2,27} = 1.70$, $p > 0.05$), condition effect (Wilks' Lambda = 0.37, $F_{6,23} = 6.33$, $p < 0.001$), and no group and condition interaction (Wilks' Lambda = 0.77, $F_{6,23} = 1.11$, $p > 0.05$). Univariate analyses revealed that condition effect occurred only for gain ($F_{3,84} = 9.48$, $p < 0.001$). Post hoc tests indicated that in the resist and no counting condition, gain values dropped compared to both no counting and counting in the no information conditions. Finally, gain values were lower in the resist and counting condition compared to the no information and counting condition.

13.4 Discussion

The purpose of this study was to investigate the sensory-motor coupling in older and younger adults during concurrent different cognitive tasks while maintaining upright stance with visual manipulation. Our results showed that older adults swayed with larger magnitude compared to younger adults, in both with and without visual manipulation, indicating that postural control performance deteriorates with aging. Despite postural control different performance, visual manipulation induces correspondent postural sway in both older and younger adults. Information about visual manipulation and the request to avoid visual sway induction, reduced sway magnitude and visual influence for both older and younger adults. Finally, a concomitant task of counting produced larger sway in

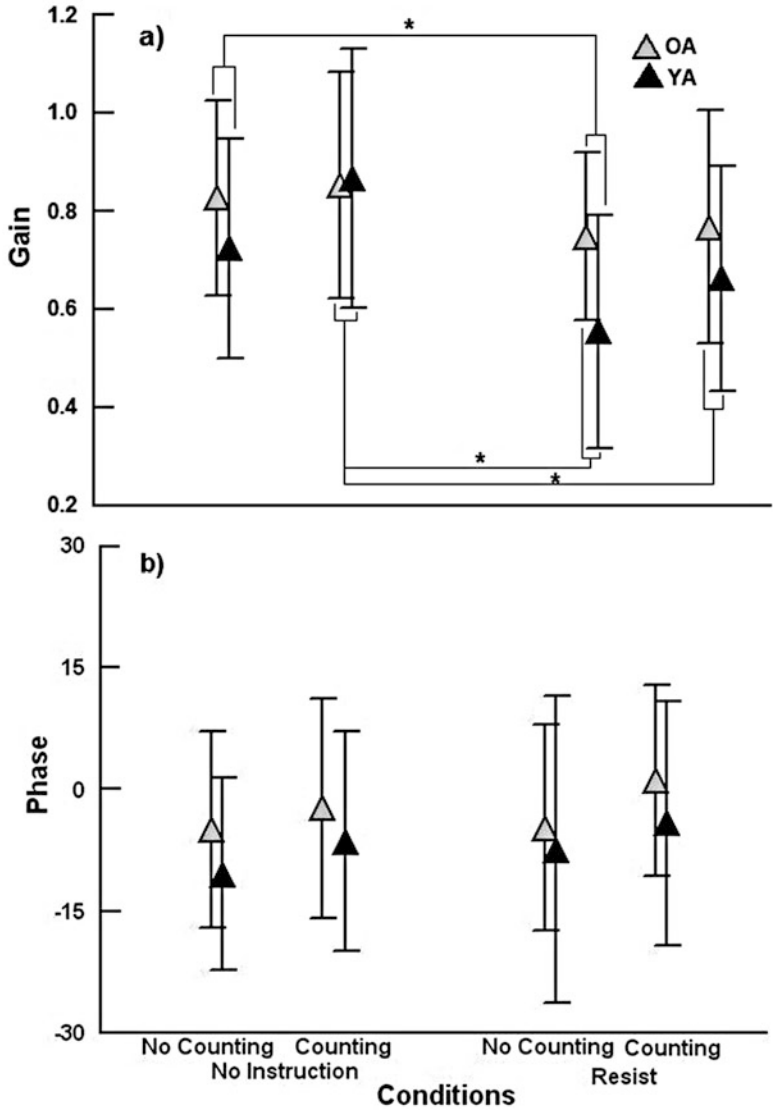

Fig. 13.5 Mean (± SD) values for gain (**a**) and phase (**b**) for both older (OA) and young adults (YA) in all four experimental conditions (no instruction and no counting, no instruction and counting, resist and no counting, and resist and counting)

both older and younger adults, when they were not informed about visual manipulation. Yet, younger participants tried to avoid oscillating with the room, but when they have to count, such efforts to avoid visual oscillation were not observed. Taken together these results, we can suggest that the coupling between visual information and body sway in maintaining upright stance does not require attentional resources

in both older and younger adults. However, older adults are less likely to be able to change this relationship than younger adults because of the attentional requirements need in order to do so.

Postural control worse performance of older adults in upright stance has been previously observed [5, 6, 8, 9] and our present results corroborate previous findings, with older adults displaying larger sway magnitude. Actually, older adults show larger sway magnitude when visual scenario is maintained stationary and when visual manipulation induces postural sway. Larger visual influence on postural magnitude had also been previously shown [17]. Larger sway magnitude in older adults might be due to reduced quality of sensory cues coming from the periphery, providing less accurate sensory information. Yet, changes in central processing might also play an important role in deteriorating postural control performance in older adults. Either explanation or even a combination of both seems to be plausible, based upon ours and other's results [5, 6, 8, 9].

Our present results, in addition, indicate that larger sway magnitude observed in older adults, compared to younger adults, is not because older adults are more susceptible to visual manipulation. Gain values did not differ between older and younger adults, indicating that sway induction due to visual manipulation was similar between the groups. These results are in accordance with those observed in similar conditions of the moving room [5, 9]. Differences in visual influences were only observed in more challenging conditions, when more complex visual patterns were created [5, 9], which were not the case in this study.

Information about visual manipulation and a request to participants resist to it produce a reduction of body sway magnitude (mean sway magnitude—Fig. 13.3) and the influence of visual manipulation (gain—Fig. 13.5) in both older and younger adults. Such behavior had been previously observed for younger adults (e.g., [14, 18, 19]) but had not been observed in older adults. Reduction of sway magnitude and visual influence in older adults are surprising, since older adults are less adaptive in producing changes in visual information and postural sway coupling [20] and more influenced by visual manipulation in more complex conditions [5]. These results indicate that despite not being able to fully ignore visual influence even knowing that visual manipulation is occurring (correspondent sway was still observed), both older and younger adults are able to at least decrease visual influence. Interestingly, younger adults swayed less coherently with the room, trying to avoid visual influence, and such change was not observed for the older adults (coherence—Fig. 13.4).

Sensory influence such as sway induction, even when the information provided is not reliable or accurate, is a remarkable characteristic of postural control system. Such functional characteristic might be due to the necessary cognitive efforts to change the intrinsic sensory and motor relationship [15]. Concomitant cognitive task of counting leaded both older and younger adults be influenced by visual manipulation overcoming the efforts of resisting to it. Coherence and gain values for both older and younger adults were similar comparing no instruction and no counting to resist and counting conditions (Figs. 13.4 and 13.5). Therefore, any change in sensory and motor coupling, at least for postural control upright stance,

seems to require a large amount of cognitive efforts. Younger adults are capable of dispose of a little of this resource, with unfortunately not being the case of older adults. Such suggestion needs to be further investigated and uncovered and might provide important insights to the suggestion that postural control requires attentional resources [21].

13.5 Final Remarks

Sensory-motor coupling in postural control, in regular basis, requires little attentional resources in both older and younger adults. This is a cleaver functioning signature of our central nervous system because attentional resources might be allocated, when necessary, to other needs. In situations of sensory conflicting or unreliable information, younger adults can change the sensory-motor coupling, at least partially, however, demanding cognitive involvement and attentional resources. On other hand, older adults do not show such capability that might be due to lack of attentional resources required to implement such change.

References

1. Horak FB, Macpherson JM. Postural orientation and equilibrium. In: Rowell LB, Shepherd JT, editors. Handbook of physiology. Exercise: regulation and integration of multiple systems. New York: Oxford Univ Press; 1996. p. 255–92.
2. Barela JA, Lopes AG, Razuk M, Barela AMF. Mudanças sensorias e motoras no controle postural decorrentes do processo de envelhecimento. In: Afonso MR, Cavalli AS, Peres AL, editors. Trabalhando com a terceira idade: estudos e pesquisas Pelotas: Editora e Gráfica Universitária; 2011. p. 211–33.
3. Spirduso WW, Francis KL, MacRae PG. Physical dimensions of aging. 2nd ed. Champaign: Human Kinetics; 2005. 384 p.
4. Shumway-Cook A, Woollacott M, Kerns KA, Baldwin M. The effects of two types of cognitive tasks on postural stability in older adults with and without a history of falls. J Gerontol A Biol Sci Med Sci. 1997;52(4):M232–40.
5. Toledo DR, Barela JA. Age-related differences in postural control: effects of the complexity of visual manipulation and sensorimotor contribution to postural performance. Exp Brain Res. 2014;232(2):493–502.
6. Berger L, Chuzel M, Buisson G, Rougier P. Undisturbed upright stance control in the elderly: Part 1. Age-related changes in undisturbed upright stance control. J Mot Behav. 2005;37 (5):348–58.
7. Freitas Junior PB, Barela JA. Alterações no funcionamento do sistema de controle postural em idosos. Uso da informação visual. Rev Port Cien Desp. 2006;6(1):94–105.
8. Prioli AC, Cardozo AS, de Freitas Junior PB, Barela JA. Task demand effects on postural control in older adults. Hum Mov Sci. 2006;25(3):435–46.
9. Toledo DR, Barela JA. Sensory and motor differences between young and older adults: somatosensory contribution to postural control. Rev Bras Fisioter. 2010;14(3):267–75.
10. Marsh AP, Geel SE. The effect of age on the attentional demands of postural control. Gait Posture. 2000;12(2):105–13.

11. Boisgontier MP, Beets IA, Duysens J, Nieuwboer A, Krampe RT, Swinnen SP. Age-related differences in attentional cost associated with postural dual tasks: increased recruitment of generic cognitive resources in older adults. Neurosci Biobehav Rev. 2013;37(8):1824–37.
12. Brown LA, Shumway-Cook A, Woollacott MH. Attentional demands and postural recovery: the effects of aging. J Gerontol A Biol Sci Med Sci. 1999;54(4):M165–71.
13. Redfern MS, Jennings JR, Martin C, Furman JM. Attention influences sensory integration for postural control in older adults. Gait Posture. 2001;14(3):211–6.
14. Barela JA, Weigelt M, Polastri PF, Godoi D, Aguiar SA, Jeka JJ. Explicit and implicit knowledge of environment states induce adaptation in postural control. Neurosci Lett. 2014;566:6–10.
15. Genoves GG, Barela AMF, Sanches C, Barela JA. Attentional effects on sensorimotor coupling in postural control of young adults. Exp Brain Res. 2016;234(12):3641–7.
16. Aguiar SA, Say KG, Lopes AG, Barela JA. Dual task interferes with sensorimotor coupling in postural control. Psychol Neurosci. 2014;7(4):593–9.
17. Wade MG, Lindquist R, Taylor JR, Treat-Jacobson D. Optical flow, spatial orientation, and the control of posture in the elderly. J Gerontol B Psychol Sci Soc Sci. 1995;50(1):p51–4.
18. Freitas Junior PB, Barela JA. Postural control as a function of self- and object-motion perception. Neurosci Lett. 2004;369(1):64–8.
19. Barela AM, Barela JA, Rinaldi NM, de Toledo DR. Influence of imposed optic flow characteristics and intention on postural responses. Motor Control. 2009;13(2):119–29.
20. Barela JA, Genoves GG, Alleoni B, Barela AMF. Visual reweighting in postural control is less adaptative in older adults. Health. 2013;5(12A):74–9.
21. Woollacott M, Shumway-Cook A. Attention and the control of posture and gait: a review of an emerging area of research. Gait Posture. 2002;16(1):1–14.

Contribution of Fingertip Light Touch on Postural Stabilization of Older Adults

14

Ana Maria Forti Barela, Sarah Caporicci, and José Angelo Barela

Abstract

In this study, we investigated the use of somatosensory information by older adults, with and without visual information. Ten older (67.3 ± 4.8 years) and 10 younger adults (24.1 ± 2.5 years) remained in the semi-tandem quiet stance on a force plate in the following experimental conditions, each 30 s long: no vision and no touch (NV-NT), no vision and touch (NV-T), vision and no touch (V-NT), and vision and touch (V-T). On the touch condition, participants touched lightly (<1 N) with their right index finger a rigid instrumented touch bar positioned laterally at their hip level. Mean amplitude of center of pressure (CoP) in the medial–lateral (ML) and anterior–posterior (AP) directions and mean absolute vertical and horizontal (AP and ML) forces applied on the touch bar were calculated. All participants swayed more in the NV-NT condition and presented no difference between V-NT and NV-T. In the NT conditions, older adults swayed more compared to younger adults, but no difference was observed between groups for the AP direction when touch was available. For the ML direction, there was no group difference when older adults used touch compared to younger adults with no touch. Older and younger adults were able to use light touch to reduce body sway; however, older adults presented higher variability to apply vertical and horizontal forces. Somatosensory information contributes to body sway as well as visual information, and older adults can benefit from that

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information, although they need slightly higher levels of applied forces during fingertip touch.

Keywords

Postural control • Motor control • Somatosensory information • Visual information • Elderly • Body sway • Force plate • Touch bar • Semi-tandem stance • Center of pressure

14.1 Introduction

Sensory cues that provides information about the relative body segment orientation and the whole body to the environment is necessary to maintain a stable postural orientation such as upright stance. In order to provide useful information about body orientation, sensory cues coming from different sources (e.g., visual, vestibular, and somatosensory systems) [1] need to be fused in the central nervous system. Based upon this information, appropriate muscle activation is produced in order to achieve or maintain a desirable posture. Therefore, postural control requires sensory cues integration and an intricate relationship between the sensory and motor systems [2].

Several studies have shown that older adults sway with significant larger magnitude during upright stance compared to younger adults [3–6], and such findings suggest poor performance of postural control system due to aging process. Postural control decline in older adults has been explained by peripheral sensory impairment [7–10]. Recently, Toledo and Barela [11] showed that less accurate sensory cue indicating changes in the ankle joint was associated with larger sway in older adults during upright stance.

An alternative strategy to compensate the less accurate sensory cues in older adults would be to enhance information by providing alternative sensory cue sources. For instance, several studies have demonstrated that information from fingertip contact to a rigid surface provides sensory cues, which can be used as an additional source of somatosensory information that could be used to attenuate body sway in upright stance. The use of additional somatosensory information, due to the fingertip contact to a surface, has been observed in newly walking infants [12, 13], children [14], younger adults [15–17], and in individuals with vestibular dysfunction [18], peripheral neuropathy [19], anterior cruciate ligament injury [20], and stroke [21]. Such contact to a surface provides cues that can be used to obtain information about body sway dynamics (position and velocity), leading to anticipatory muscle activity related to postural control and, as a consequence, body sway attenuation occurs [16, 17, 22].

The use of light touch on body sway magnitude of older adults has been investigated by a few studies [23–25] and, despite employing different procedures, these studies have revealed that older adults were able to reduce body sway by lightly touching a stationary surface. However, this finding should be viewed

cautiously because the relationship between changes in applied fingertip forces and body oscillation was not examined [23, 25] and applied force levels have been used above the commonly used levels (<1 N) [25, 26].

Therefore, in this study we investigated the use of somatosensory information coming from light touch, with and without vision in older adults. Our expected results were that older adults could take advantage of using light touch information provided by contacting a stationary surface, but the use of light touch would be slightly different compared to younger adults.

14.2 Materials and Methods

14.2.1 Participants

Ten older (7 females and 3 males) and 10 younger (7 females and 3 males) adults participated in this study. All of them were healthy with no known musculoskeletal injuries or neurological disorders that might impair their ability to maintain balance and had normal or corrected to normal vision at the time of testing. Their mean (± standard deviation, SD) age, height, and mass were, respectively, 67 ± 5 years and 24 ± 3 years, 1.57 ± 0.07 m and 1.67 ± 0.07 m, 71 ± 12 kg and 68 ± 12 kg. Prior to any experimental procedure, participants signed an informed consent form, approved by the Institutional Review Ethics Board.

14.2.2 Apparatus, Task, and Procedures

Participants stood on a force plate (Kistler, Model 9286A), that measures vertical and horizontal ground reaction forces and is used to calculate the center of pressure (CoP). A six-axis sensor (Nano 17 Titanium, ATI, Inc.), that measures forces and moments in the vertical and horizontal directions, was used as a touch contact surface, and it was mounted on a tripod that could be adjusted in height and position, allowing a comfortable arm position as participants touched the contact surface with their right index fingertip. The signals from force plate and the touch contact surface were calibrated in units of force (N). CoP was calculated in centimeter and for the fingertip contact force threshold of 1 N was set and if force levels were up, this value would trigger an auditory alarm.

Participants were asked to maintain a still upright semi-tandem (hallux of right foot touching the medial border of calcaneus of the left foot) stance on the force platform, barefoot, in two visual (vision and no vision) and two fingertip contact (light contact and no contact) conditions. In the vision condition, participants were instructed to look straight ahead towards a target (3 cm diameter) placed 1 m away at eye level. In the no vision condition, participants were required to close their eyes and remained with eyes closed throughout the trial. In the light contact condition, participants were required to place their right index finger on the force sensor (metal surface with 2 cm in diameter) and keep the contact still throughout the trial. If the

auditory alarm would be triggered, they needed to reduce the applied force level while keeping contact with the plate. In the no contact condition, participants were required to maintain both arms hanging passively beside their bodies. Feet position was traced with a marker on top of the force platform to ensure identical foot positions during all trials.

Before each trial, participants were told to look straight ahead and take as much time as necessary to assume a comfortable position with their fingertip either on or off the touch device and with their eyes open or closed, according to the experimental condition, remaining as still as possible. The experimental conditions were: vision and no touch (V-NT), no vision and no touch (NV-NT), vision and light touch, and no vision and light touch. Data from three 30-s trials under each of these four experimental conditions were recorded. These conditions were divided into three periods of randomized trials, totalizing 12 trials, with one minute of rest between each period.

Force data from both force plate and touch device were collected at a frequency of 100 Hz using a personal computer equipped with a data acquisition board (NI-BNC2090, National Instruments, Inc.). Data acquisition was controlled by a dedicated routine written using LabView (National Instruments, Inc.).

14.2.3 Data Analysis

The first and last 2.5 s of data were excluded from analysis to minimize anticipation effects associated with the beginning and end of each trial, remaining 25 s of data for each trial. Acquired force data were then filtered using a fourth-order zero lag low-pass Butterworth digital filter, with a cut-off frequency of 10 Hz. Data from the force plate was used to calculate the CoP for both medial–lateral (ML) and anterior–posterior (AP) directions. Following, the mean sway amplitude (MSA) and force levels were calculated. MSA of CoP was calculated for both ML and AP, by subtracting the average position of CoP from each data point within a trial and, thus, obtaining the average variation around the mean position. Mean contact fingertip contacting force was obtained for each of the force components: vertical, ML, and AP.

Since three trials were acquired for each condition, the MSA and force values acquired in each condition were averaged and used for further analysis. All the calculations and analyses were performed by using custom routines written using MATLAB (MathWorks Inc.).

14.2.4 Statistical Analysis

A multivariate analysis of variance (MANOVA) was performed with groups (younger and older adults), visual (vision and no vision), and fingertip contact conditions (no touch and light contact) as factors, with the last two factors treated as repeated measures. This MANOVA had as dependent variables the MSA values in

the ML and AP directions. Another MANOVA was also performed with groups (younger and older adults) and visual conditions (vision and no vision) as factors, with the last one factor treated as repeated measures. This MANOVA had as dependent variables the mean values of applied forces on the touch device in the three directions (vertical, ML, and AP). When applicable, univariate analyses and the Tukey post hoc tests were employed. An alpha level of 0.05 was adopted for all statistical tests, which were performed using SPSS software.

14.3 Results

All adults were able to use light contact during the upright stance with and without vision. Figure 14.1 depicts an exemplar of times series of CoP in the ML and AP directions in the NV-NT and NV-T conditions of an older and a younger adult maintaining upright stance throughout a trial.

Figure 14.2 depicts mean values of MSA, for both ML (Fig. 14.2a) and AP (Fig. 14.2b) of CoP for both age groups in the experimental conditions. MANOVA revealed group (Wilks' Lambda = 0.271, $F_{2,17} = 22.88$, $p < 0.001$), vision (Wilks' Lambda = 0.068, $F_{2,17} = 117.39$, $p < 0.001$), and touch (Wilks' Lambda = 0.042, $F_{2,17} = 192.88$, $p < 0.001$) main effects, and interaction between group and touch (Wilks' Lambda = 0.585, $F_{2,17} = 6.02$, $p < 0.05$), and vision and touch (V-T)

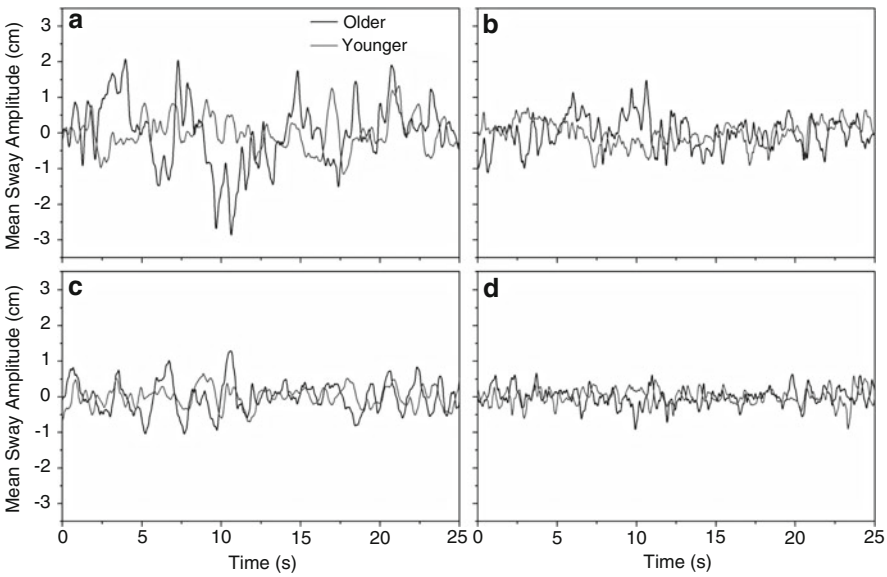

Fig. 14.1 Times series of center of pressure (CoP) in the ML (**a**, **c**) and AP (**b**, **d**) directions in the no vision and no touch (NV-NT; **a**, **b**) and no vision and touch (NV-T; **c**, **d**) conditions of an older and a younger adult maintaining upright stance throughout a trial

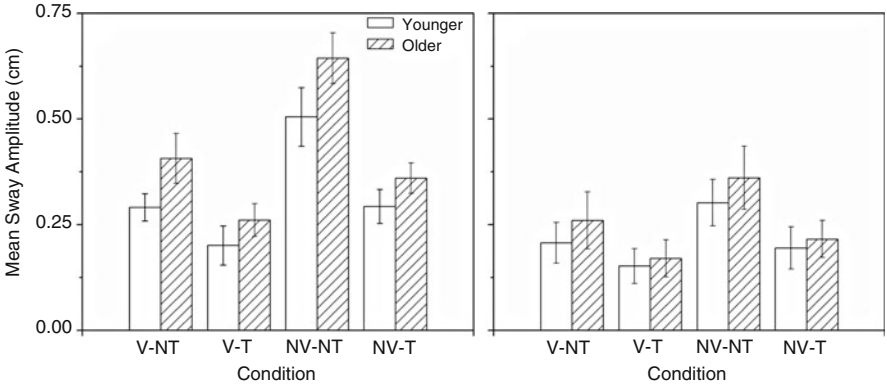

Fig. 14.2 Mean (± SD) of the magnitudes of mean sway amplitude (MSA) of the CoP of younger and older adults during vision and no touch (V-NT), vision and touch (V-T), NV-NT, and NV-T conditions

(Wilks' Lambda = 0.142, $F_{2,17} = 51.22$, $p < 0.001$). Following, the univariate analyses for each direction (ML and AP) will be presented separately.

Univariate analyses for the CoP in the ML direction indicated group ($F_{1,18} = 40.53$, $p < 0.001$), vision ($F_{1,18} = 244.88$, $p < 0.001$), and touch effect ($F_{1,18} = 394.76$, $p < 0.001$) and interaction between group and touch ($F_{1,18} = 12.07$, $p < 0.005$), and V-T ($F_{1,18} = 57.22$, $p < 0.001$). With no vision, older and younger adults showed larger sway compared to with vision condition. Post hoc indicated that participants showed the largest sway in the NV-NT condition and the smallest sway in the V-T condition. With V-NT condition and with no vision and touch (NV-T) condition, body sway was similar. Finally, post hoc indicated that for both younger and older adults, touch reduced sway, but the reduction was lager in older compared to younger adults.

Univariate analyses for the CoP in the AP direction indicated vision ($F_{1,18} = 99.90$, $p < 0.001$), and touch effect ($F_{1,18} = 238.76$, $p < 0.001$) and group and touch ($F_{1,18} = 8.07$, $p < 0.05$) and V-T interaction ($F_{1,18} = 10.60$, $p < 0.005$). Post hoc revealed that without touch, body sway was larger in older compared to younger adults; however, with touch no difference was observed between older and younger adults. Finally, body sway was larger with no vision but only in the condition without touch. When touch was available, body sway was similar with vision and no vision conditions.

Figure 14.3 depicts mean absolute applied forces for both age groups with and without vision conditions. MANOVA revealed only group effect (Wilks' Lambda = 0.492, $F_{3,16} = 5.164$, $p < 0.05$). Univariate analyses showed group effect for the vertical ($F_{1,18} = 4.65$, $p < 0.05$), ML ($F_{1,18} = 15.44$, $p < 0.005$), and AP applied forces ($F_{1,18} = 10.57$, $p = 0.005$). Older adults, pooling vision conditions, applied higher force levels compared to younger adults.

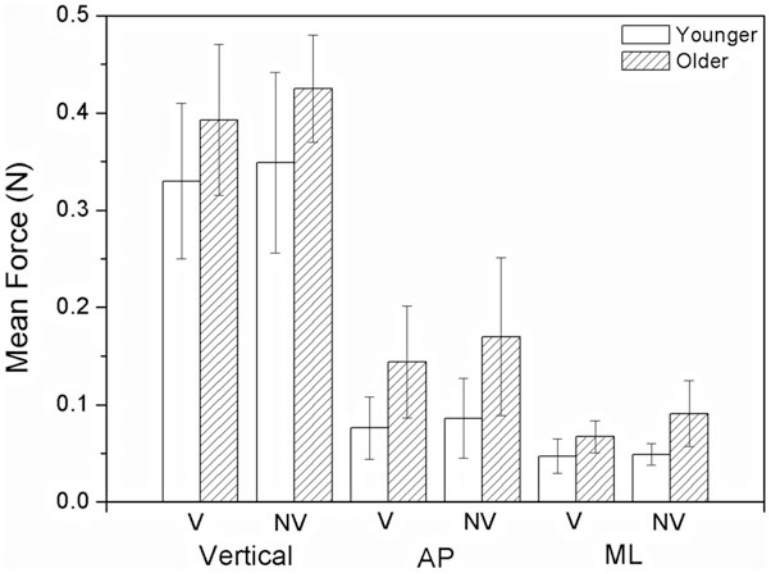

Fig. 14.3 Mean (± SD) of the mean absolute applied forces in the vertical (V), ML, and AP directions of younger and older adults during the light touch conditions with vision (V) and no vision (NV)

14.4 Discussion

The purpose of this study was to investigate the use of somatosensory information, due to lightly touching a stationary surface, with and without vision in older adults. Our results showed that older adults use light touch on a surface to reduce postural control in order to maintain an upright quiet stance. Moreover, in using the enhanced somatosensory cues from the fingertip, the available furnished information is sufficient to reduce some of the sway differences that occur when body sway is compared to younger adults. Finally, older adults applied more force, although within the limit imposed by the required task, compared to younger adults, suggesting that older adults might have some of the peripheral capabilities in acquiring sensory cues slightly altered.

The use of light touch to reduce body sway observed in this study might not constitute a new finding. A few studies have showed that touch to a surface leads to body sway reduction in older adults [23–25]. However, several methodological issues, observed in those previous studies, would prevent us to thoughtfully assume the use of light touch by older adults. Based upon our results, we can suggest that older adults are capable to benefit from touching on a surface even with applied forces below 1 N. Holden and collaborators [15] estimated that mechanical contribution to body sway magnitude due to touching a rigid surface with the index finger in the tandem stance position was well below the observed reduction. Therefore,

such difference was suggested to occur because of the sensory information from the changes of applied forces in the fingertip. Based upon our results, we can assume that older adults can use light touch as an important source of sensory cues to enhance postural control functioning and to reduce postural sway.

An important issue related to changes in postural control is related to which possible changes in the functioning of postural control would be related to worsening postural performance. Recently, Toledo and Barela [11, 27] have shown that in maintaining upright stance, older adults would show larger sway due to reduction of passive motion detection around the ankle. Similarly, studies have suggested that changes in postural control of older adults would be most likely due to peripheral changes [23] while the central processing of sensory cues seems to be intact in older adults [28]. Our results support both suggestions and observations in the way that if sensory cues provided enough information about body dynamics, older adults could maintain upright stance in a similar way as younger adults. Light touch leaded older adults to reduce (ML direction) or even equal (AP direction) sway magnitude to younger adult levels. Therefore, older adults have the capability of maintaining upright stance similar to younger adults, but to do so, they had to enhance sensory cues, such as by lightly contacting a rigid surface.

Despite being able to use light touch, older adults still swayed with larger amplitude in the ML direction compared to younger adults. Why does such difference still persist even with enhanced sensory cues coming from the fingertip? Prioli and collaborators [3, 6] had demonstrated and suggested that the postural control performance is task-dependent in such a way that more demanding postural tasks would require a much closer relationship between sensory information and motor activity in order to achieve a desired performance. Our results also provided evidence for such suggestion. While older adults showed larger sway magnitude in the AP direction with no touch, when touch was made available, no difference was observed compared to the sway magnitude of younger adults. Differently, in the ML direction, even with touch, older adults still swayed with larger magnitude compared to younger adults, indicating that the enhancement of sensory cues due to light touch was not sufficient to overcome the task requirement. Conversely, in the AP direction the cues enhancement was sufficient and younger-like performance was observed.

Finally, a new and important finding was that older adults needed to apply a slightly higher magnitude of force to the surface compared to younger adults. Although studies employing the light touch procedures have limited applied forces up to 1 N, typical adults and children [14] stay below this level, applying force levels around 0.4 N in the vertical direction. Our results also showed levels around the previous observed values for both younger and older adults, although older adults applied a slightly higher force level. Higher levels of applied vertical force have also been observed for Down syndrome [29] and dyslexic children [30]. In the case of dyslexic children, it was suggested that because those children most likely show difficulties in acquiring sensory cues, a strategy adopted by them would be to apply slightly higher levels of force to enhance even further the sensory cues made available by touching the surface. More important, however, is that in doing so,

dyslexic children improved their postural performance to non-dyslexic children levels [31]. Such a phenomenon may also happen with other sensory modalities (e.g., vision), but touch can be easily measured and might become an important strategy to be used in order to understand the use of sensory cues by older adults. Therefore, older adults might suffer from a decline in tactile sensitivity and, because of such decline, it is necessary for a higher level of applied force in order to overcome the peripheral deterioration of sensory cues. Indeed, weaker and delayed proprioceptive afferent inflow to the cortex was observed in older adults and it was associated with deterioration of behavioral response [32, 33].

In sum, older adults can take advantage of lightly touching a rigid surface to improve the functioning of postural control in tasks like maintaining upright stance. Moreover, because light touch might provide an extra source of sensory cues, sensory information related to body dynamics is enhanced and in doing so, older adults might overcome some detrimental changes of peripheral sensory structures as suggested and observed for older adults. But in order to do so, older adults might need to adopt slightly different interaction with the surrounding environment, in our case, touching a surface with higher levels of applied force.

References

1. Nashner LM. Analysis of stance posture in humans. In: Towe AL, Luschei ES, editors. Motor coordination: handbook of behavioral neurology. New York: Plenum Press; 1981. p. 527–65.
2. Horak FB, Macpherson JM. Postural orientation and equilibrium. In: Rowell LB, Shepard JT, editors. Handbook of physiology. New York: Oxford University Press; 1996. p. 255–92.
3. Prioli AC, Cardozo AS, de Freitas Júnior PB, Barela JA. Task demand effects on postural control in older adults. Hum Mov Sci. 2006;25:435–46.
4. Blaszczyk J, Lowe D, Hansen PD. Ranges of postural stability and their changes in the eldery. Gait Posture. 1994;2:11–7.
5. Collins JJ, De Luca CJ, Burrows A, Lipsitz LA. Age-related changes in open-loop and closed-loop postural control mechanisms. Exp Brain Res. 1995;104(3):480–92.
6. Prioli AC, Freitas Junior PB, Barela JA. Physical activity and postural control in the elderly: coupling between visual information and body sway. Gerontology. 2005;51(3):145–8.
7. Lord SR, Ward JA. Age-associated differences in sensori-motor function and balance in community dwelling women. Age Ageing. 1994;23:452–60.
8. Maki BE, McIlroy WE. Postural control in the older adult. Gait Balance Disord. 1996;12 (4):635–58.
9. Sturnieks DL, St George R, Lord SR. Balance disorders in the elderly. Neurophysiol Clin. 2008;38(6):467–78.
10. Woollacott M, Shumway-Cook A. Changes in posture control across the life span: a systems approach. Phys Ther. 1990;70(12):799–807.
11. Toledo DR, Barela JA. Age-related differences in postural control: effects of the complexity of visual manipulation and sensorimotor contribution to postural performance. Exp Brain Res. 2014;232(2):493–502.
12. Barela JA, Jeka JJ, Clark EJ. The use of somatosensory information during the acquisition of independent upright stance. Infant Behav Dev. 1999;22:87–102.
13. Metcalfe JS, McDowell K, Chang TY, Chen LC, Jeka JJ, Clark JE. Development of somatosensory-motor integration: an event-related analysis of infant posture in the first year of independent walking. Dev Psychobiol. 2005;46(1):19–35.

14. Barela JA, Jeka JJ, Clark JE. Postural control in children: coupling to dynamic somatosensory information. Exp Brain Res. 2003;150(4):434–42.
15. Holden M, Ventura J, Lackner JR. Stabilization of posture by precision contact of the index finger. J Vestib Res. 1994;4(4):285–301.
16. Jeka JJ, Lackner JR. Fingertip contact influences human postural control. Exp Brain Res. 1994;100(3):495–502.
17. Jeka JJ, Lackner JR. The role of haptic cues from rough and slippery surfaces in human postural control. Exp Brain Res. 1995;103(2):267–76.
18. Lackner JR, DiZio P, Jeka J, Horak F, Krebs D, Rabin E. Precision contact of the fingertip reduces postural sway of individuals with bilateral vestibular loss. Exp Brain Res. 1999;126 (4):459–66.
19. Dickstein R, Shupert CL, Horak FB. Fingertip touch improves postural stability in patients with peripheral neuropathy. Gait Posture. 2001;14(3):238–47.
20. Bonfim TR, Grossi DB, Paccola CA, Barela JA. Additional sensory information reduces body sway of individuals with anterior cruciate ligament injury. Neurosci Lett. 2008;441(3):257–60.
21. Cunha BP, Alouche SR, Araujo IM, Freitas SM. Individuals with post-stroke hemiparesis are able to use additional sensory information to reduce postural sway. Neurosci Lett. 2012;513 (1):6–11.
22. Jeka JJ, Schoner G, Dijkstra T, Ribeiro P, Lackner JR. Coupling of fingertip somatosensory information to head and body sway. Exp Brain Res. 1997;113(3):475–83.
23. Baccini M, Rinaldi LA, Federighi G, Vannucchi L, Paci M, Masotti G. Effectiveness of fingertip light contact in reducing postural sway in older people. Age Ageing. 2007;36 (1):30–5.
24. Tremblay F, Mireault AC, Dessureault L, Manning H, Sveistrup H. Postural stabilization from fingertip contact: I. Variations in sway attenuation, perceived stability and contact forces with aging. Exp Brain Res. 2004;157(3):275–85.
25. Reginella RL, Redfern MS, Furman JM. Postural sway with earth-fixed and body-referenced finger contact in young and older adults. J Vestib Res. 1999;9(2):103–9.
26. Tremblay F, Mireault A-C, Dessureault L, Manning H, Sveistrup H. Postural stabilization from fingertip contact II. Relationships between age, tactile sensibility and magnitude of contact forces. Exp Brain Res. 2005;164:155–64.
27. Toledo DR, Barela JA. Sensory and motor differences between young and older adults: somatosensory contribution to postural control. Rev Bras Fisioter. 2010;14(3):267–75.
28. Allison LK, Kiemel T, Jeka JJ. Multisensory reweighting of vision and touch is intact in healthy and fall-prone older adults. Exp Brain Res. 2006;175(2):342–52.
29. Gomes MM, Barela JA. Postural control in Down syndrome: the use of somatosensory and visual information to attenuate body sway. Motor Control. 2007;11(3):224–34.
30. Viana AR, Razuk M, de Freitas PB, Barela JA. Sensorimotor integration in dyslexic children under different sensory stimulations. PLoS One. 2013;8(8):e72719.
31. Barela JA, de Freitas PB, Viana AR, Razuk M. Dyslexia and the integration of sensory cues into motor action. Psychology. 2014;5:1870–8.
32. Toledo DR, Manzano GM, Barela JA, Kohn AF. Cortical correlates of response time slowing in older adults: ERP and ERD/ERS analyses during passive ankle movement. Clin Neurophysiol. 2016;127(1):655–63.
33. Toledo DR, Barela JA, Manzano GM, Kohn AF. Age-related differences in EEG beta activity during an assessment of ankle proprioception. Neurosci Lett. 2016;622:1–5.

Effects of Vision on Postural Control in Neurologically Healthy Individuals

15

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Abstract

The purpose of this chapter was to characterize how the postural system of healthy individuals is capable of adaption when facing distinct levels of challenge, all planned to vary the visual context involved. A series of postural studies showed a variety of circumstances in which the postural control system faced both stronger, mechanical, and subtle, sensorial perturbations. Effects of vision were tested in tasks such as moving line-of-gaze in space (during saccadic and smooth pursuit eye movements), grasping an object (under different optic flow conditions), three-ball juggling (under narrow and wide stances), performing a ballet skill (with eyes opened and blindfolded), and driving a car (under simulated conditions); additionally, effects of aging on posture are discussed. In summary, authors suggest that considering the multiple levels of the visual system functioning (eyes, head, trunk, and whole body movements) along with lifespan postural changes is essential to advance our knowledge of the process of acquisition and use of visual information to stabilize posture.

Keywords

Posture • Vision • Eye movement • Head movement • Saccade • Smooth pursuit • Grasping • Juggling • Ballet • Driving

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15.1 Introduction

The maintenance of balance and body orientation in static and dynamic situations is a core element for the performance of daily life, work, exercise, and sport activities [1]. Postural control requires maintaining position of body segments with respect to both other segments and environment (orientation) and controlling internal forces to compensate external forces acting on the body (balance) [2]. To achieve these behavioral goals, the postural control system needs sensory information regarding relative position of body parts and forces acting on it to, then, trigger appropriate muscular contractions in order to maintain or change a given postural position. The sensory bases of this perception–action linkage are complex as information arriving from visual, vestibular, and somatosensory channels must be integrated by the superior levels of central nervous system [3]. On the other hand, any motor action related to the maintenance or achievement of a given body position requires that the effector system works with a multi-segment body, applying specific strategies and muscle activities to constrain many organism degrees of freedom and have them acting as a functional unit [2].

Analyzing the so-called quiet stance, when individuals standing upright are looking straight ahead, is a standard method in posturography that offers the opportunity of better understanding the perceptual–motor complexity of posture [4]. However, exploring situations in which the postural system is challenged by subtle stimuli or dynamical actions may reveal a more comprehensive perspective on postural adaptability and allow novel manipulations of visual information. Thus, the purpose of the present chapter was to characterize how the postural system of healthy individuals is capable of adaption when facing distinct levels of challenge, all planned to vary the visual context involved. A series of postural studies is summarized where the effects of vision were tested in tasks such as moving their line-of-gaze in space (during saccadic and smooth pursuit eye movements), grasping an object (under different optic flow conditions), three-ball juggling (under narrow and wide stances), performing a ballet skill (with eyes opened and blindfolded), and driving a car (under simulated conditions), as shown in the following sections. In addition, a section on a particular type of postural adaptation due to the aging process is presented to describe the effects of progressive perceptual, motor, and cognitive challenges faced by older adults, which is followed by final considerations.

15.2 Static Posture and Eye Movements

Specific characteristics define different gaze behaviors such as fixation, saccades, and smooth pursuit eye movements. In fixation, the eyes remain stationary focusing at a given point in the environment so as to stabilize the visual image on the fovea region allowing the extraction and processing of visual information in detail. Saccades are fast eye movements used to bring a new part of the visual field to the fovea region [5]. Due to this high velocity, the processing of visual information

during the saccade is extremely poor, a phenomenon known as saccadic suppression [6]. Smooth pursuit is the visual tracking of a moving target in which a continuous movement rotates the eyes to compensate for the motion of the visual object, minimizing blur and therefore occurring within a nonstationary frame of reference [7].

The influence of each one of these behaviors on the control of static posture has been the object of innumerous investigations [8, 9]. Besides their great importance on daily life activities, the fact that eye movements do not generate great biomechanical constraints on posture and large center of mass displacements has made the study of their effects on balance even more attractive. In this section, we explore these effects as well as their interaction with the complexity of the postural and visual tasks.

15.2.1 Effects of Distance to Fixed Object

Lee and Lishman [10] argued that the magnitude of the optic flow resulting from movements of the head is a function of the linear distance between visual object and observer. Consequently, bigger changes in optic flow will be produced while fixating an object nearby compared to a distant one when the observer produces a head movement of the same amplitude. Evidence for this interpretation has been provided by the authors by showing that body sway decreases when subjects are fixating nearby targets (0.4 m) compared to distant ones (3 m). Since greater changes in optic flow will be produced for head displacements when the observer is fixating nearby targets, body sway becomes more "detectable" and would be minimized more precisely to stabilize the fixed object in the visual field. On the other hand, for distant targets greater magnitude of body oscillation can be allowed without having a large effect on the stabilization of the visual image [10].

Stoffregen et al. [11] extended this finding demonstrating that when the subject is fixating a distant object but the object nearby is still present in the visual field, body sway is still greater than when the nearby object is being fixated. The authors argue that even though visual information on the nearby object is still available in the optic array, body sway occurred as a function of the task at hand. This means that oscillation was still greater because the subject was fixating a distant target, regardless on the available visual information that might be "automatically" detected by the postural control system but was not part of the suprapostural task [11].

This study has been replicated in further investigations, which moved on to show how these effects interact with more complex postures [12]. Rodrigues et al. [12] explored the effects of object distance on body sway during the maintenance of the kiba-dachi posture, a karate stance which challenges balance specially in the anterior–posterior direction since it requires the person to maintain their legs very distant from each other (larger than shoulder width) with knees flexed and trunk aligned with the feet. Results revealed decreased body sway while fixating a nearby object as compared to a distant one, showing that the effect of distance is also present during the control of such complex posture [12].

15.2.2 Effects of Saccades on Posture

Although a few studies suggested that eye movements might actually increase body sway, this finding was only present in very specific contexts, such as adding cognitive tasks while performing eye movements [13] and exploring these effects in patients with spontaneous nystagmus as opposed to planned voluntary saccades [8, 14]. Overall, the literature provides solid evidence that saccades decrease body sway in neurologically healthy individuals [15]. This is exemplified by experiments showing reduced body oscillation during saccades performed both horizontally and vertically [16] and at near and far distances between observer and visual object [17]. Several studies confirm this pattern of results [18, 19]. Interestingly, eye movements performed with eyes closed do not reduce body sway [19].

Stoffregen et al. [9] argue that the reason why body oscillation is reduced during eye movements is to achieve the goal of stabilizing the visual scene, allowing eye movements to be performed more accurately. Two mechanisms are thought to be responsible for visually detecting body sway and stabilizing posture, which are referred to as afferent and efferent motion perception, or ocular and extraocular mechanisms, respectively [20]. The first, afferent motion perception is a feedback based mechanism which uses information contained in the optic flow to minimize retinal slip and therefore stabilize the distance between the eye and the visual environment. The second, efferent motion perception is based on either the copy of the motor command, named efference copy—which is a feedforward mechanism that predicts the sensory consequences of the movement produced—or the extraocular muscle afferents, named re-afferences, that are consecutive to eye movements [20].

Rodrigues et al. [18] investigated not only the effects of saccades on postural stabilization but also their interaction with the complexity of the postural and eye-movements tasks. While performing horizontal saccades at different frequencies (0.5 and 1.1 Hz), young adults controlled their balance by standing either with feet apart at shoulder width or pressed together reducing stability especially in medial–lateral axis. Results indicated in general reduced body sway during saccades compared to fixation and in narrow compared to wide stance, as expected, but also that specifically in the anterior–posterior axis body sway was greater for narrow compared to wide stance only in the condition of high frequency (1.1 Hz) saccades. This shows that the reduction in body sway which takes place during eye movements was actually stronger with the combination of the most complex postural (narrow stance) and eye-movements (1.1Hz saccades) tasks. Therefore, the reduced time for the planning of the next saccade which occurs for the high frequency saccades compared to the low frequency ones might have required greater stabilization of posture in the non-stable narrow stance condition in order to accurately fulfill the demands of the eye-movements task being performed. The authors argue that the fact that the frequency of saccades could affect posture differently according to the stance condition is in line with an adaptive resource-sharing model, proposed by Mitra and Fraizer [21–23], which

postulates that postural and suprapostural tasks compete for the same limited resources which will be dedicated to either one of these tasks according to the demands of each one [18].

15.2.3 Effects of Smooth Pursuit on Posture

Although saccades clearly stabilize posture, contradictory results have been presented on whether performing smooth pursuit eye movements decreases or increases body oscillation. Visual tracking of a moving target has been shown to cause larger body sway, especially when the target appeared in a dark background [8, 24]. The lack of spatial reference of a dark background together with additional manipulations on stance conditions in these studies—using complex tasks such as heel-to-toe position and standing on a foam cushion—makes these results able to be interpreted only in very specific contexts.

Recent evidence has been provided, however, indicating that smooth pursuit eye movements performed in a regular spatial frame of reference—i.e., not in darkness—which is the most common situation observed in daily life activities, actually decrease body sway, stabilizing posture similarly to saccades [25]. In this investigation, Rodrigues et al. [25] varied the velocity of saccades and smooth pursuit eye movements and demonstrated that both types of eye movements decreased body oscillation compared to fixation, regardless of their velocities.

There are important differences between saccades and smooth pursuit eye movements. These include saccadic suppression—which drastically reduces the capacity for processing of visual information during saccades but does not exist for smooth pursuit, the nonstationary spatial frame of reference that occurs during smooth pursuit but not during saccades, and perhaps more importantly, the fact that while smooth pursuit are movements entirely based on feedback to keep the target at the fovea region continuously, saccades need to be planned beforehand due to their high velocity, leaving no space for corrections during their execution. Nevertheless, even with these different aspects, both eye-movements tasks seem to reduce body sway, leaving authors to conclude that the mechanisms involved in the execution of saccades and smooth pursuit eye movements share similarities in postural control [25], possibly the fact that greater postural stabilization is required to perform both tasks accurately [9].

15.3 Posture, Optic Flow, and Arm Movements

15.3.1 Effects of Grasping Movements Under Different Optic Flow Conditions

Many environmental aspects such as the optical flow and one's own movement can affect posture stabilization. However, little is known about how these two features can be combined as participants maintain upright stance during grasping actions.

Table 15.1 Rodrigues et al.'s [25] experimental conditions combining manipulations of moving room, ball, and catching action

Condition	Room	Ball	Grasping
1	Stationary	Stationary	Absent
2	Stationary	Stationary	Present
3	Stationary	Approaching	Present
4	Approaching	Stationary	Absent
5	Moving back	Stationary	Absent
6	Approaching	Stationary	Present
7	Moving back	Stationary	Present
8	Approaching	Approaching	Present
9	Moving back	Approaching	Present

Rodrigues et al. [25] examined the effects of discrete movements of a moving room (approaching to or moving back from participants) combined with grasping an object on postural control stability. Twelve participants stood upright inside a moving room, moved discretely approaching or moving away from the participants, as they had to remain still or grasping a stationary or an approaching ball, totalizing nine experimental conditions (Table 15.1).

A pulley system was connected to a servomechanism in order to move simultaneously the room and a shaft, which had a ball (6 cm of diameter) fixed in its end. Forward and backward movements of the room were associated with clockwise and anticlockwise rotations of the pulley system, moving the shaft in speed and amplitude proportional to the movement of the room in the same or opposite directions. Participants were instructed to stay in the starting position as stable as possible and keep it through the end of the experimental trials with no grasping (conditions 1, 4, and 5), grasping the ball after the light being turned on with no movement of the room and of the ball (condition 2), and grasping the ball after the light being turned on simultaneously to the room and shaft movements initiation (conditions 3, 6, 7, 8, and 9). The discrete movement of the room had approximate duration of two seconds, with peak velocity of 2.6 cm/s and amplitude of 2.5 cm [26]; the shaft approached the participant, being moved approximately during two seconds, with peak velocity of 74.8 cm/s, and amplitude of 53 cm. During all conditions, participants were required to stand upright for 18 s, maintaining fixation with their eyes on the ball. Each trial was divided into four time windows: prior to room movement initiation (four seconds), during room movement (two seconds), after room movement—first and second periods (four seconds each), totalizing 14 s; the remaining time was excluded from analysis. The variables body displacement and correlation coefficient were obtained during the room movement; the variable mean sway amplitude was used to infer stability prior to and after movement of the room.

Rodrigues et al. [25] found that grasping leads to increased effect of moving room on induced body sway, as body displacement is larger during grasping. However, body sway is more related to room movement when the room is moved back compared to the approaching movement. Despite inducing larger body sway during the movement of the moving room, grasping disruption of postural

equilibrium is even larger when there is no moving room visual flow. These results also showed that grasping a stationary ball in a moving scenario is worse, leading to larger body sway induced by the moving room and such effect is even larger in the moving back movement of the room. Such postural equilibrium disruption is still observed after the grasping has ended (following the four seconds).

It was surprising that grasping during room discrete movement leads to a larger sway; literature had pointed out only correspondent body sway in children [27], adults [26], and even in elderly [28]. This new finding can be explained by the fact that when adults have to gather information about the ball position, they are more influenced by the peripheral flow. In the context of a moving room with no arm movement involved, it is known that flow with large lamellar (peripheral) structure generates stronger sway response than more radially (centrally) structured flow [29]. In line with this reasoning is that although vision of the hand in Rodrigues et al.'s [25] experiment is peripherally available during its initial movement, the use of central vision during the later stages of grasping is advantageous for controlling movement amplitude as the limb decelerates [30].

The correlation coefficient in the moving back condition, which indicates the relationship between moving room and body sway, was dependent on direction, being stronger in the condition of backward optical flow, with the room moving away from the participant. It is well known that infant rhesus monkeys [31] and infant humans [32] show defensive reactions to approaching (looming) patterns of optic flow; these responses were in clear contrast to those related to objects approaching on a non-collision path or a shrinking (as opposed to a looming) pattern [33]. In the moving room paradigm, the room approaches the observers who respond to the optical flow (looming) as if it arose from their own postural sway [34], attempting to keep a constant relation with the room's walls. As in Rodrigues et al.'s [25] experiment, a grasping action was required, and the postural adjustment in response to the approaching movement of the room would result in increase of hand–ball distance; instead, a sway stabilization took place to allow a proper grasping movement. Differently, the room moving back from participants seemed to have facilitated grasping as they both were in the same direction.

In short, Rodrigues et al. [25] showed that grasping leads to increased effect of moving room on induced body sway. In this context, posture was more affected when the room was moved back compared to the approaching movement. Based upon these results, authors suggest that postural control takes advantage of the structured optical flow available due to room movement, even when posture might be destabilized due to one's own movement, to maintain postural equilibrium.

15.3.2 Effects of Juggling Movements Under Different Bases of Support Conditions

Cascade ball juggling is a complex perceptual–motor skill, which requires an efficient postural stabilization. Experience in the juggling task may play a role in the linkage between manual and postural tasks. To clarify how the postural system

works in this context, Rodrigues et al. [35] investigated effects of experience (expert and intermediate groups) and feet distance (wide and narrow stances) on body sway of jugglers during three-ball cascade juggling. Participants stood barefoot on the force plate (some participants wore a gaze tracking system), with feet maintained in wide and narrow conditions and performed three 40-s trials of the three-ball juggling task.

The main finding of Rodrigues et al. [35] was that only intermediate jugglers showed smaller oscillation in the narrow as compared to wide stance. Expert jugglers showed a superior postural stabilization in both stances, with more consistent patterns of center of pressure displacements characterized by measures of mean velocity and amplitude in both AP and ML axes, corroborating the notion according to which juggling skill level is associated with decreased body sway. Previous studies have shown that expertise similarly affect other motor skills, such as rifle shooting, manual rhythmic movements, circus activities, and gymnastics [36–40]. In a study with jugglers, Leroy et al. [41] found that although experts and intermediate participants showed similar body sway amplitude (measured through sacrum lateral displacement), the skilled group was able to perform anticipatory postural adjustments differently from novice jugglers.

Interestingly, gaze behavior seems to participate in controlling more complex postural tasks. The increased body sway of intermediate jugglers could be explained by the larger area of gaze displacement observed throughout each trial. A more stable, spatially reduced gaze pattern, named gaze-through strategy [42], may improve movement planning via the attentional system [43, 44]. During a gaze fixation, balls move in the retinal periphery, which most likely involves a stage operating in gaze-centered coordinates which is more accurate [42, 45]. With a more stable gaze, movement planning is improved because the entire visual background is stable since allocentric and gaze-centered representations become aligned [42, 46]. Although visual information acquisition through fixation patterns (number of fixations, mean duration of fixations and its variability) was not affected by group or condition in Rodrigues et al.'s study [35], the significant base of support by group interaction found for the area of gaze displacement seems to corroborate the experts' gaze-through strategy, suggesting an attentional linkage between postural (center of pressure) and visual (gaze) stability. Although acknowledging the small sample size of gaze data, authors interpreted these results as a confirmation that experts were less dependent on foveal vision and more capable of decoupling the control of posture and bimanual rhythmic arm movements [36, 46].

Rodrigues et al. [35] found a surprising significant effect of base of support; greater postural instability was expected during more challenging tasks, but the opposite was observed. Particularly, intermediate jugglers increased the sway laterally more than experts to facilitate their arms movement pattern, as shown by the significant main effect of group on the ML sway amplitude. Additionally, intermediate jugglers showed significantly smaller oscillation in the narrow stance, indicating that experts were less affected by the basis of support reduction than intermediate jugglers. Expert jugglers showed differences in body oscillation between narrow and wide stances, which were not significant while the

intermediate group was clearly affected by the smaller basis of support and had to reduce more drastically their body sway to keep balance and the juggling task ongoing. Authors interpreted their results as corroborating previous studies which showed effects of the experience and motor specialization on the improvement of postural control [41, 47–49].

In sum, Rodrigues et al. [35] concluded that experts' body sway was characterized by lower velocity and smaller amplitude as compared to intermediate group. Interestingly, authors found that the more challenging (narrow) basis of support caused significant attenuation in body sway only for the intermediate group, suggesting that expertise in cascade juggling was associated with refined postural control.

15.4 Eye–Head Stabilization in Complex Contexts

The role of visual information on the control of movement has been investigated in a variety of complex contexts such as everyday life and sport activities [50–53]. The eyes move around the environment to align fovea region to a specific portion of visual scene for processing of visual information in detail [51]. Acquisition of useful visual information in dynamical postures involves controlled coordination between body, head, and eye movements. Eye and head stabilization during the execution of motor actions is an important mechanism to keep stationary the image on the retina in order to ensure a clearer visual processing during the execution of motor actions [52, 53].

Two kinds of compensatory mechanisms participate in the stabilization of eyes and head during dynamical postures, making the eyes to move in line with head rotations. In the vestibulo-ocular reflex, head rotation velocity is detected by semicircular channels (vestibular system) that provide sensory signals to the oculomotor system to move the eyes in the opposite direction. The second mechanism, the optokinetic reflex, is engaged in adjusting the actual velocity of the retinal image with the velocity of eyes rotation in the same direction [51]. In this section, it will be discussed the role of eye and head stabilization in several everyday life and sport activities that directly affect visual information acquisition and the maintenance of dynamical stability.

15.4.1 Stabilizing Posture During Dynamical Skills: Ballet and Table Tennis

Visual information during the execution of a ballet pirouette is the crucial sensory input used to maintain postural stability. However, visual processing is impaired during the rotation of the trunk, head, and eyes that can affect dynamical posture. Denardi et al. [53] investigated the role of visual information on postural control of expert ballet dancers with analyses of trunk, head, and eyes movements during the execution of a pirouette with (opened eyes) and without (blindfolded eyes) vision available. Gaze stability for as long as possible during performance is an important strategy to minimize rotational flow on the retina generated by the movement of

whole body [52–54]. In Denardi et al.'s [53] experiment, gaze stability strategy was checked with analyses of the quiet eye period. The concept of quiet eye proposed by Vickers [54, 55] suggested that a longer final fixation before the initiation of movement is used to extract the maximum of the critical information from environment to define details of motor performance. Denardi et al. [53] observed a longer quiet eye period in the beginning of the pirouette turns; however, there was absence of correlation between quiet eye duration and postural stability. In contrast with previous studies [52, 54, 56], a longer period of quiet eye did not cause changes on pirouette performance. However, the authors assumed that visual information acquired during gaze stability period was sufficient for expert ballet dancers to achieve dynamical postural requirements of the task [53]. In addition, a clear sequence of trunk, head, and eyes rotations in the execution of the pirouette with opened eyes was observed. In the initiation of movement, trunk rotation starts to turn following with the rotation of the head and finishing with the delayed eyes rotation. At the end, eyes rotation starts following to the head and trunk rotations sequentially. Interestingly, in the experimental conditions without visual information (blindfolded eyes) the rotations of the head and trunk were coupled [53]. These findings showed evidence that when visual information was available, head movements were delayed to maintain the gaze stability as long as possible in order to ensure a useful extraction of information during quiet eye period. Eye and head stabilization during quiet eye period facilitated visual information acquisition to perform the task [53].

Rodrigues, Vickers, and Williams [52] investigated the coordination of head, eye, and arm movements of skilled and less skilled table tennis players; although they did not focus directly on postural control, head contribution to visual stability may help in understanding the role played by vision in posture. Their experimental task consisted in a forehand drive shot to one of the two cued target areas, which was illuminated in order to generate distinct temporal conditions: pre-cue, before serve; early cue, during the initial portion of ball flight; and late-cue, during the final portion of ball flight. The duration of quiet eye was reduced according to the increase in complexity of the task (from pre- to early cue and from early to late-cue conditions). Eye–head stabilization was defined as the period in which head and eye were aligned. During the most difficult condition (late-cue), eye–head stabilization was less frequent suggesting that task complexity disrupted optimal conditions for information acquisition via eye–head stability, which occurred along with a reduction in the quiet eye duration. These changes in eye–head stabilization and quiet eye period during more complex task induced a lower accuracy performance for all players (51, 52).

15.4.2 Eye and Head Stabilization During Driving

Driving a car is a particularly complex type of locomotion supported by visual information available in the surrounding environment. Eye and head movements have been studied during driving situations to determine drivers' visual search

strategies [50, 51, 57]. Land and Lee [58] investigated the steering control on a road with steeper bends, recording steering wheel angle and drivers' gaze direction. Authors found that before the car entering on the bend, drivers spent much of time fixating the tangent point—defined as the most salient point inside of the bend—and also observed a clear correlation between gaze and steering wheel angle, with gaze movements preceding the rotation of the steering wheel by about 0.8 s. The authors suggested that gaze angle directed at the tangent point can provide the signal needed for the arms to control the steering wheel [58]. In another study, Land and Tatler [57] examined the eye and head movements of a Formula 3 racing driver in a winding road. The racing driver fixated most of the time close, but not exactly, at the tangent point before starting to turn the steering wheel [57]. Interestingly, head movements from central line of the lane to the tangent point begun 1.5 s before starting cornering, and the correlation between driver's head direction and the rotation of the vehicle was stronger ($r = 0.96$) than correlation between gaze and head direction ($r = 0.3$). Authors suggested that even though the racing driver did not displace his gaze exactly to the tangent point location, driver's head direction provided sufficient information for the steering wheel control [57].

Carizio [50] investigated the effects of cognitive workload (use of cell phone) on gaze and head stability during simulated driving. The use of a cell phone in speakerphone mode or holding the device in the driver's hand can interfere in the attentional mechanism of drivers and tend to disrupt perceptual and motor performance. Drivers had eyes and head movements recorded while driving in a driver simulator under three experimental conditions: control (no talking, no cell phone), speakerphone mode (talking, with cell phone in speakerphone mode), and holding in hand (talking, with cell phone held in the hand). Results revealed that drivers performed a higher number of fixations per time unit and a higher spread of fixation locations in the horizontal and vertical axes during both cell phone conditions as compared to the control condition. Similar to gaze behavior, variance of head position and orientation also increased during both speakerphone mode and holding in hand conditions of mobile phone use. These findings suggest that increased cognitive workload/task complexity provoked a decrease in eye and head stabilization [50], corroborating previous studies on eye–head stabilization in sports [52].

In natural contexts as those referred above [50, 52, 53, 57], eye and head movements seem to work together to extract useful visual information from a relevant location at a proper time. Particularly, head control plays a critical role in maintaining gaze stability in such complex contexts which is in line with previous evidence of linkage between eye movements and postural control discussed above [50, 52, 53]. The activity of eyes, head, trunk, and whole body emphasizes the organism collective effort towards an optimal acquisition of useful visual information; the observed coupling between gaze and motor behavior evidenced that visual information is essential for planning motor actions and maintaining dynamical posture in complex contexts [50–54].

15.5 Effects of Aging on Eye Movements and Postural Control

In addition to increased complexity of sensory stimuli and motor actions involved in natural situations, the aging process represents a progressive challenge and requires important adaptation of older adults. Aging is known to affect both the motor and sensory systems which are intimately related in the control of static posture [59]. Older adults show poorer performance compared to young adults in both postural control [59–61] and eye-movements tasks [62–64]. Specifically, postural control has been shown to be affected by aging in both simple [65] and complex postural tasks [66]. For this reason, the risk of falls in older adults has received great attention from researchers and authorities representing an important public health issue [67]. Mechanisms contributing to poorer postural control in older adults include motor deficits such as muscle weakness [68], sensory deficits in visual [69], vestibular [70], and somatosensory systems [71], and limitations in attentional capabilities [72]. Importantly, besides causing specific deficits in both motor and sensory systems contributing to postural control, aging has been shown to affect also sensorimotor integration in the control of posture [26, 66].

Deficits caused by aging specifically in the visual system such as a reduced visual acuity [73] and a poorer contrast sensibility [74] provoke an impaired visual perception to older adults. Furthermore, the effect of aging on performance of eye-movements tasks has also been well documented. Older adults perform saccades less accurately [63], with slower reaction times [75] and with reduced amplitude, velocity, and frequency [76] compared to young adults. The gain [77] and accuracy [64] of smooth pursuit eye movements are also affected by aging. A comparison between the performance of eye movements in real-world and laboratory situations has shown that in real world performance of saccades is greatly deteriorated by aging while performance on tracking movements can remain unaffected by aging probably due to the use of additional sensory cues compensating for aging deficits [76]. Interestingly, the effects of aging on eye-movements performance have been shown also for common tasks such as reading [78] and have been even proposed as an indicator on driver's ability in older adult drivers [79] and as a tool for detecting mild cognitive impairments [80].

Similar to results shown previously from young adults [18], Aguiar et al. [59] also explored the effects of saccades at different frequencies and different stance conditions in postural control but in a population of older adults. Discussions around the adaptive resource-sharing model would be enriched with this investigation since the sensory and motor deficits presented by older adults would potentially alter these limited resources available for postural and suprapostural tasks and therefore how the postural control system would give priority to one task over another. Results showed decrease in body sway during saccades compared to fixation, which is the same result encountered in young adults. Interestingly, no difference was found between narrow and wide stance conditions according to the frequency of saccades, which was present for young adults. Authors concluded that older adults present a more rigid postural control strategy, which does not allow larger body sway during a more complex postural task [59]. However, Laufer et al.

[60] investigated postural stability of young and older adults during upright stance on restricted basis of supported 85 cm above ground level with both opened and closed eyes. Postural threat did not affect young adults; however, older adults showed a greater mean power frequency of displacement of center of pressure [60]. The authors concluded that older adults adopted an inadequate postural response during postural threat that increased body sway. The increasing of complexity of the postural task may be a challenge to older adults since the postural control system is affected by aging. In Aguiar et al.'s experiment [59], even with a more complex postural task (restricted basis of support) older adults maintained their body sway unaltered with different frequencies of horizontal saccadic eye movements. According to the authors, older adults performed a more rigid postural strategy to prioritize the postural performance during a threatening situation, which did not allow the influence of saccadic eye movements on body sway. Interestingly, Paquette and Fung [62] found that older adults performed more corrective saccadic movements and with greater latency between stimulus onset and saccadic eye-movement onset with a moving surface compared to young adults. The authors suggested that older adults have visual and postural performance impaired during threatening stability situations. In general, saccadic eye movements reduce body sway of young and older adults; however, during more challenging postural tasks the relationship between eye movements and postural control seems to be altered with aging.

15.6 Final Considerations

Understanding the effects of vision on postural control in healthy individuals requires an extended notion of visual system which includes postural elements. For instance, the notion that "eyes-in-the-head-on-the-body-resting-on-the-ground" [81] are continuously searching for relevant information in the optic flow favors the simultaneous consideration of gaze and postural data to discuss balance control. Postural adjustments seem to support optimal gaze behavior during simple and complex actions. Although optic flow results from translational components of head movement in space and eye movements add rotational components to the flow on the retina [82, 83], a process of minimization of rotational consequences to the flow, called gaze stabilization [84], seems advantageous to optimize translational information acquisition with respect to the perceiver. As human visual input depends on the dynamics of all body parts, studies on posture should explore this collective postural effort (resulting from movements of eyes, head, trunk, and whole body) to acquire the relevant information available in the optic flow, needed for successful action.

Following this suggestion, the series of studies presented in this chapter showed a variety of circumstances in which the postural control system faced both stronger, mechanical perturbations and subtle, sensory challenges. During quiet stance, posture is more stabilized when the fixed object is nearby as compared to a distant one, the principle which holds for complex stances, such as karate kiba-dachi; besides distinct control mechanisms, saccadic and smooth pursuit eye movements directed to predictable and continuous stimulus also tend to reduce body sway.

In contexts with more complex postural requirements, adaptations in the perception–action linkage are multivariate. Grasping action leads to increased effect of optic flow (moving room) on induced body sway; posture was more affected when the room was moved back compared to the approaching movement, suggesting that postural control takes advantage of the structured optical flow available, even when posture might be destabilized due to one's own movement, to maintain postural balance. In cascade juggling, expertise reduces body sway of participants; the more challenging (narrow) basis of support caused significant attenuation in body sway only for the intermediate group, suggesting that experts have a refined postural control. During a pirouette in ballet or a forehand drive in table tennis, eye and head stabilization was strategically adopted in order to facilitate the extraction of useful information to control the motor behavior; increasing of task complexity (removing visual information in ballet or delaying the visual cue in table tennis) caused impairment on eye–head stabilization which disrupted performance. In simulated driving, an increase in cognitive load generated by the use of cell phone resulted in higher relative number of fixations and spatial spread of fixation locations, as well as increased variance of head position and orientation, suggesting a worsening of postural stability and visual information acquisition.

Particularly, characterizing the effects of vision on postural control in healthy older adults adds even more complexity to the debate. The natural perceptual, motor, and cognitive deficits due to the aging process push the limits of adaptation. In general, the older adults show a more rigid postural control strategy, with increased probabilities of instability and risk of falls. In summary, considering the multiple levels of the visual system functioning (with eyes, head, trunk, and whole body movements) along with lifespan postural changes is essential to advance our knowledge of the process of acquisition and use of visual information to stabilize posture.

References

1. Zatsiorsky VM. Kinematics of human motion. Champaign: Human Kinetics; 1998.
2. Horak FB, MacPherson JM. Postural orientation and equilibrium. In: Rowell LB, Sherpherd JT, editors. Handbook of physiology: a critical, comprehensive presentation of physiological knowledge and concepts. New York: Oxford University Press; 1996.
3. Nashner LM. Analysis of stance posture in humans. In: Towe AL, Luschei ES, editors. Motor coordination. New York: Plenum Press; 1981. p. 527–65.
4. Duarte M, Freitas SMSF. Revision of posturography based on force plate for balance evaluation. Rev Bras Fisioter. 2010;14:183–92.
5. Carpenter RHS. Movements of the eyes. London: Pion Limited; 1988.
6. Volkmann FC, Schick AM, Riggs LA. Time course of visual inhibition during voluntary saccades. J Opt Soc Am. 1968;58:562–9.
7. Krauzlis R. Recasting the smooth pursuit eye movement system. J Neurophysiol. 2004;91:591–603.
8. Glasauer S, Schneider E, Jahn K, Strupp M, Brandt T. How the eyes move the body. Neurology. 2005;65:1291–3.

9. Stoffregen T, Bardy B, Bonnet C, Hove P, Oullier O. Postural sway and the frequency of horizontal eye movements. Motor Control. 2007;11:86–102.
10. Lee DN, Lishman JR. Visual proprioceptive control of stance. J Hum Mov Stud. 1975;1:87–95.
11. Stoffregen TA, Pagulayan RJ, Bardy BG, Hettinger LJ. Modulating postural control to facilitate visual performance. Hum Mov Sci. 2000;19:203–20.
12. Rodrigues ST, Jardim JG, Siqueira NS, Paula F, Aguiar SA, et al. Estabilização postural do olhar: Efeitos da distância do objeto [Postural stabilization of looking: effects of distance to target]. Braz J Mot Behav. 2011;6:7–15.
13. Hunter MC, Hoffman MA. Postural control: visual and cognitive manipulations. Gait Posture. 2001;13:41–8.
14. Jahn K, Strupp M, Krafczyc S, Schüler O, Glasauer S, Brandt T. Suppression of eye movements improves balance. Brain. 2002;125:2005–11.
15. Bonnet CT, Baudry S. Active vision task and postural control in healthy, young adults: synergy and probably not duality. Gait Posture. 2016;48:57–63.
16. Rougier P, Garin M. Performing saccadic eye movements or blinking improves postural control. Motor Control. 2007;11:213–23.
17. Rey F, Lê T, Bertin R, Kapoula Z. Saccades horizontal or vertical at near or at far do not deteriorate postural control. Auris Nasus Larynx. 2008;35:185–91.
18. Rodrigues S, Aguiar S, Polastri P, Godoi D, Moraes R, Barela J. Effects of saccadic eye movements on postural control stabilization. Motriz. 2013;19:614–9.
19. Stoffregen T, Bardy B, Bonnet C, Pagulayan R. Postural stabilization of visually guided eye movements. Ecol Psychol. 2006;18:191–222.
20. Guerraz M, Bronstein AM. Ocular versus extraocular control of posture and equilibrium. Neurophysiol Clin Neurophysiol. 2008;38:391–8.
21. Mitra S. Adaptive utilization of optical variables during postural and suprapostural dual-task performance: comment on Stoffregen, Smart, Bardy, and Pagulayan. J Exp Psychol Hum Percept Perform. 2004;30:28–38.
22. Mitra S, Fraizer E. Effects of explicit sway minimization postural-suprapostural dual-task performance. Hum Mov Sci. 2004;23:1–20.
23. Mitra S. Postural costs of suprapostural task load. Hum Mov Sci. 2003;22:253–70.
24. Laurens J, Awai L, Bockish C, Hegemann S, van Hedel H, Dietz V, et al. Visual contribution to postural stability: Interaction between target fixation or tracking and static or dynamic large-field stimulus. Gait Posture. 2010;31:37–41.
25. Rodrigues S, Bertoloni G, Moraes R, Barela A, Savelsbergh G, Barela J. Controlling posture during grasping with different optical flow conditions. Hauppauge: Nova Science Publishers; 2015. p. 1–18.
26. Prioli AC, Freitas Júnior PB, Barela JA. Physical activity and postural control in the elderly: coupling between visual information and body sway. Gerontology. 2005;51:145–8.
27. Perotti Júnior A, Barela J, Polastri P, Tani G. Influência de diferentes informações comportamentais na dinâmica intrínseca entre informação visual e oscilação corporal [Influence od different behavioral informations in intrinsic dynamics between visual information and body sway]. Brazilian J Mot Behav. 2007;2:40–50.
28. Wade M, Lindquist R, Taylor J, Treat-Jacobson D. Optical flow, spatial orientation, and the control of posture in the elderly. J Gerontol Ser B Psychol Sci Soc Sci. 1995;50:51–4.
29. Stoffregen T. Flow structure versus retinal location in the optical control of stance. J Exp Psychol Hum Percept Perform. 1985;11:554–65.
30. Lawrence G, Khan M, Buckolz E, Oldham A. The contribution of peripheral and central vision in the control of movement amplitude. Hum Mov Sci. 2006;25:326–38.
31. Schiff W, Caviness J, Gibson J. Persistent fear responses in rhesus to the optical stimulus of "looming". Science. 1962;136:982–3.
32. Bower T, Broughton J, Moore M. Infant responses to approaching objects: An indicator of response to distal variables. Percept Psychophys. 1971;9:193–6.

33. Bruce V, Green P. Visual perception: physiology, psychology and ecology. 2nd ed. Hove: Lawrence Erlbaum Associates Ltd; 1990.
34. Stoffregen T, Riccio G. Responses to optical looming in the retinal center and periphery. Ecol Psychol. 1990;2:251–74.
35. Rodrigues ST, Polastri PF, Gotardi GC, Aguiar SA, Mesaros MR, Pestana MB, et al. Postural control during cascade ball juggling: effects of expertise and base of support. Percept Mot Skills. 2016;123(1):279–94.
36. Amado AC, Palmer CJ, Hamill J, van Emmerik REA. Coupling of postural and manual tasks in expert performers. Hum Mov Sci. 2016;46:251–60.
37. Era P, Konttinen N, Mehto P, Saarela P, Lyytinen H. Postural stability and skilled performance – a study on toplevel and naive rifle shooters. J Biomech. 1996;29:301–6.
38. Garcia RS, Hayes SJ, Williams AM, Bennett SJ. Multisensory perception and action in 3-ball cascade juggling. J Mot Behav. 2013;45:29–36.
39. Sahli S, Ghroubia S, Rebaic H, Chaâbaned M, Yahiaa A, Pérennoue D, et al. The effect of circus activity training on postural control of 5-6-year-old children. Sci Sports. 2013;28:11–6.
40. Gautier G, Thouvarecq R, Larue J. Influence of experience on postural control: effect of expertise in gymnastics. J Mot Behav. 2008;40:400–8.
41. Leroy D, Thouvarecq R, Gautier G. Postural organization during cascade juggling: influence of expertise. Gait Posture. 2008;28:265–70.
42. Dessing JC, Rey FP, Beek PJ. Gaze fixation improves the stability of expert juggling. Exp Brain Res. 2012;216:635–44.
43. Williams AM, Davids K. Visual search strategy, selective attention, and expertise in soccer. Res Q Exerc Sport. 1998;69:111–28.
44. Shulman GL, Remington RW, Mclean JP. Moving attention through visual space. J Exp Psychol Hum Percept Perform. 1979;5:522–6.
45. Dessing JC, Crawford JD, Medendorp WP. Spatial updating across saccades during manual interception. J Vis. 2011;11:1–18.
46. Huys R, Beek PJ. The coupling between point-of-gaze and ball movements in three-ball cascade juggling: the effects of expertise, pattern and tempo. J Sports Sci. 2002;20:171–86.
47. Marin L, Bardy BG, Bootsma RJ. Level of gymnastic skill as an intrinsic constraint on postural coordination. J Sports Sci. 1999;17:615–26.
48. Perrin P, Deviterne D, Hugel F, Perrot C. Judo, better than dance, develops sensorimotor adaptabilities involved in balance control. Gait Posture. 2002;15:187–94.
49. Yoshitomi SK, Tanaka C, Duarte M, Lima F, Morya E, Hazime F. Respostas posturais à perturbação externa inesperada em judocas de diferentes níveis de habilidade [Postural responses to unexpected external perturbations in judo athletes of different skill levels]. Rev Bras Med do Esporte. 2006;12:159–63.
50. Carizio BG. Efeito distrator do telefone celular sobre a condução simulada de automóveis: situações de uso manual e viva-voz (Dissertação) [Distractor effect of cell phone on the simulated car driving: In hand and speakerphone mode situations (Dissertation)]. Bauru: Faculdade de Arquitetura, Artes e Comunicação [Faculty of Architecture, Arts and Comunication], Universidade Estadual Paulista [São Paulo State University]; 2016.
51. Land MF. Eye movements and the control of actions in everyday life. Prog Retin Eye Res. 2006;25:296–324.
52. Rodrigues ST, Vickers JN, Williams M. Head, eye and arm coordination in table tennis. J Sports Sci. 2002;20:187–200.
53. Denardi R, Ferracioli M, Rodrigues S. Informação visual e controle postural durante a execução da pirouette no ballet [Visual information and postural control during a pirouette in ballet]. Rev Port Ciências do Desporto. 2008;8:241–50.
54. Vickers JN. Visual control when aiming at a far target. J Exp Psychol Hum Percept Perform. 1996;22:342–54.
55. Vickers JN. Origins and current issues in Quiet Eye research. Curr Issues Sport Sci. 2016;1:101.

56. Vickers JN, Adolphe RM. Gaze behaviour during a ball tracking and aiming skill. Int J Sport Vis. 1997;4:18–27.
57. Land MF, Tatler BW. Steering with the head: the visual strategy of a racing driver. Curr Biol. 2001;11:1215–20.
58. Land MF, Lee DN. Where we look when we steer. Nature. 1994;369:742–4.
59. Aguiar SA, Polastri PF, Godoi D, Moraes R, Barela JA, Rodrigues ST. Effects of saccadic eye movements on postural control in older adults. Psychol Neurosci. 2015;8:19–27.
60. Laufer Y, Barak Y, Chemel I. Age-related differences in the effect of a perceived threat to stability on postural control. J Gerontol A Biol Sci Med Sci. 2006;61:500–4.
61. Prioli AC, Freitas Junior PB, Barela JA. Physical activity and postural control in the elderly: coupling between visual information and body sway. Gerontology [Internet]. 2005;51:145–8. Recuperado de: http://www.karger.com/doi/10.1159/000083984.
62. Paquette C, Fung J. Old age affects gaze and postural coordination. Gait Posture [Internet]. 2011;33:227–32. Recuperado de: http://dx.doi.org/10.1016/j.gaitpost.2010.11.010.
63. Warren DE, Thurtell MJ, Carroll JN, Wall M. Perimetric evaluation of saccadic latency, saccadic accuracy, and visual threshold for peripheral visual stimuli in young compared with older adults. Invest Ophthalmol Vis Sci. 2013;54:5778–87.
64. Bozhkova VP, Surovicheva NS, Nikolaev DP, Nikolaev IP, Bolshakov AS. Smooth pursuit in elderly adults studied with apparent motion. Perception. 2015;44:1040–53.
65. Cattagni T, Scaglioni G, Laroche D, Gremeaux V, Martin A. The involvement of ankle muscles in maintaining balance in the upright posture is higher in elderly fallers. Exp Gerontol. 2016;77:38–45.
66. Prioli AC, Cardozo AS, de Freitas Junior PB, Barela JA. Task demand effects on postural control in older adults. Hum Mov Sci. 2006;25:435–46.
67. Ambrose AF, Cruz L, Paul G. Falls and fractures: a systematic approach to screening and prevention. Maturitas. 2015;82:85–93.
68. Orr R. Contribution of muscle weakness to postural instability in the elderly. A systematic review. Eur J Phys Rehabil Med. 2010;46:183–220.
69. Bennett PJ, Sekuler R, Sekuler AB. The effects of aging on motion detection and direction identification. Vision Res. 2007;47:799–809.
70. Fife TD, Baloh RW. Disequilibrium of unknown cause in older people. Ann Neurol. 1993;34:694–702.
71. Toledo DR, Barela JA. Sensory and motor differences between young and older adults: somatosensory contribution to postural control. Rev Bras Fisioter. 2010;14:267–75.
72. Shumway-Cook A, Woollacott M, Kerns KA, Baldwin M. The effects of two types of cognitive tasks on postural stability in older adults with and without a history of falls. J Gerontol. 1997;52:232–40.
73. Owsley C. Aging and vision. Vision Res. 2011;51:1610–22.
74. West SK, Munoz B, Rubin GS, Schein OD, Bandeen-Roche K, Zeger S, et al. Function and visual impairment in a population-based study of older adults the SEE project. Invest Ophthalmol Vis Sci [Internet]. 1997;38:72–82. Recuperado de: http://www.ncbi.nlm.nih.gov/pubmed/9008632.
75. Moschner C, Baloh RW. Age-related changes in visual tracking. J Gerontol. 1994;49:235–8.
76. Dowiasch S, Marx S, Einhauser W, Bremmer F. Effects of aging on eye movements in the real world. Front Hum Neurosci. 2015;9.
77. Ross RG, Olincy A, Harris JG, Radant A, Adler LE, Compagnon N, et al. The effects of age on a smooth pursuit tracking task in adults with schizophrenia and normal subjects. Biol Psychiatry. 1999;46:383–91.
78. Wang J, Li L, Li S, Xie F, Chang M, Paterson KB, et al. Adult age differences in eye movements during reading: the evidence from Chinese. J Gerontol B Psychol Sci Soc Sci. 2016;0:1–10.
79. Schmitt KU, Seeger R, Fischer H, Lanz C, Muser M, Walz F, et al. Saccadic eye movement performance as an indicator of driving ability in elderly drivers. Swiss Med Wkly. 2015;145.

80. Seligman SC, Giovannetti T. The potential utility of eye movements in the detection and characterization of everyday functional difficulties in mild cognitive impairment. Neuropsychol Rev. 2015;25:199–215.
81. Gibson J. The ecological approach to visual perception. Boston: Houghton-Mifflin Company; 1979.
82. Cutting JE. Wayfinding from multiple sources of local information in retinal flow. J Exp Psychol Hum Percept Perform. 1996;22:1299–313.
83. Kim N, Turvey MT, Growney R. Wayfinding and the sampling of optical flow by eye movements. J Exp Psychol Hum Percept Perform. 1996;22:1314–9.
84. Daniel BM, Lee DN. Development of looking with head and eyes. J Exp Child Psychol. 1990;50:200–16.

Balance Control in Older Adults

16

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Abstract

To avoid falls during everyday movements, we need to maintain balance, i.e., control the position of our body's center of mass relative to our base of support. The balance control system comprises sensory subsystems, their afferent nerves, an extensive brain network, and the motor system. Physiological aging of each of these subsystems may limit our ability to control balance in standing and walking. Methods based on questionnaires, functional tests of posture and movement, and advanced methods that quantify the ability to control or regain balance or the performance of specific subsystems of the balance control system are available to test balance control in older adults.

Keywords

Falls • Gait • Posture • Standing • Aging • Brain • Muscle • Vestibular system • Visual system • Proprioception

16.1 Introduction

The basic activities that humans perform seemingly automatically, like standing and walking, require active control of balance to prevent falls. Unfortunately, aging has detrimental effects on balance control and this can lead to falls, which cause a serious threat to the individuals involved and a burden on society. At least 25 % of individuals aged 65 years and over falls at least once per year and half of these individuals falls even more frequently [1]. About 5 % of these falls results in serious injuries such as head injuries and hip or wrist fractures. According to the World

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Health Organization, annual fall rates resulting in emergency department visits range between 6 and 9 per 10,000 people aged over 60, of which 50 % leads to hospital admission. The number of injuries caused by falls is projected to be 100 % higher in the year 2030 due to the aging of the population. Consequently, the direct medical costs related to falls will increase over the next 15 years with 170 % [2]. In addition to physical injuries and the related loss of independence, falls often cause fear of falling and avoidance of physical activity, with negative effects for health, independence, and quality of life.

Most falls occur during everyday movements like standing, walking, and transitioning, for example, from sitting on a chair to standing upright [3]. Falls are often caused by the physical status of the individual but also by environmental factors. Fit and healthy older adults, for example, commonly fall during activities outdoors due to major balance perturbations (e.g., tripping over irregular pavement), while more fragile older adults more often fall indoors due to minor perturbations, sometimes even without clear external cause [4].

What does it take to adequately control balance during everyday movement tasks and what are the effects of aging on balance control and how can we assess balance? In this chapter we try to answer these questions by giving a description of the balance control system and its function in standing and walking. In addition, we address the age-related anatomical and physiological processes that affect this system and the consequences thereof for balance in standing and walking. Moreover, we provide an overview of commonly used clinical as well as more novel and advanced methods for assessment of balance (impairments).

16.2 The Balance Control System

Balance control can be defined as the control of the body center of mass (COM) relative to the base of support (BOS). In a static situation, such as in upright stance on one or two legs, the COM has to be kept over the BOS. When, in a dynamic situation, the COM is positioned outside the BOS, it has to be brought back over the BOS, or the BOS must be enlarged or moved to attain the same situation (Fig. 16.1). To change the position of the COM, two strategies can be used [5]. Muscles around the ankle joint can create a joint torque to accelerate the body around the ankles in the desired direction. Alternatively, movements of proximal body parts can cause a change in total amount of rotation (angular momentum) of the body, which accelerates the body COM in the right direction. An example of the latter is the swaying of the arms to maintain balance on the edge of a cliff. Finally, also the BOS can be moved or enlarged by stepping or by grabbing for hand support.

Given our upright posture, gravity exerts a continuous perturbing effect, which tends to move the COM out of the BOS. Because stiffness of lower limb joints and of the muscle spanning these joints is insufficient to counteract this effect, even unperturbed upright stance requires active balance control [6]. This is reflected in the continuous movement of the COM during standing, called postural sway (Fig. 16.2). To activate muscles appropriately to move the COM in the desired

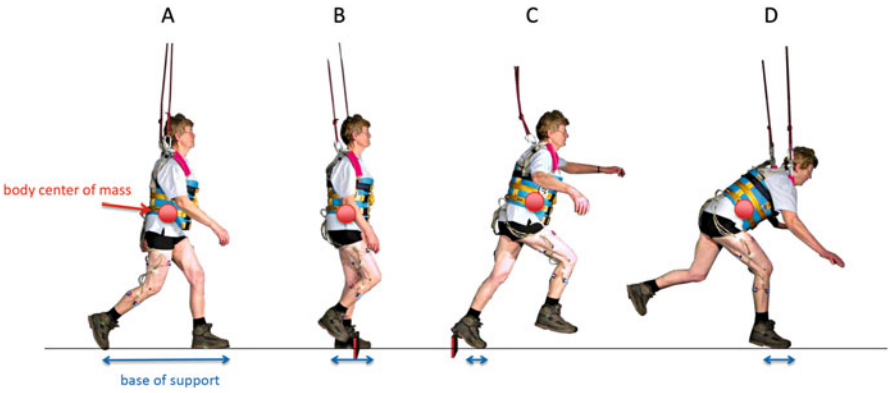

Fig. 16.1 Illustration of the essentials of balance control by means of the situations before and after a balance perturbation during gait (tripping). To control balance the body COM (the location of the point around which the body mass is distributed) has to be maintained above the BOS, formed by the feet (and sometimes by other body parts in contact with the world, such as the hand on a cane). (**a**) The BOS in fore-aft direction is formed by both feet and the COM is located above it. (**b**) The BOS is formed by the left foot only and the COM has a forward velocity. The swing leg, which would normally be placed forward to displace the BOS, is blocked by an obstacle, due to which balance is perturbed, i.e., the COM travels beyond the BOS. (**c**) During balance recovery the forward rotation can be decelerated by generating fast and large muscle force and moments in the stance leg, especially around the left ankle. (**d**) The blocked leg is placed over the obstacle with a large and fast step to displace the BOS far enough forward to regain balance

direction and over the desired range, the nervous system requires information on the current position and velocity of the COM. This information is also indispensable to correctly adjust the BOS if needed. Sensory information thus plays an important role in balance control. The sensory modalities involved, the central nervous system (CNS), and the muscles thus form a feedback system (Fig. 16.3). The sensory system measures the current status; the CNS decides on actions needed to maintain or regain balance and based on this activates muscles; the muscles perform the required mechanical actions and the result is measured again by the sensory system. In addition to feedback, anticipation plays an important role in balance control; sensory information is used to predict future perturbations and anticipatory action can be taken to attenuate or even avoid a perturbation. The balance control system is summarized in Table 16.1 and more extensively described in the subsequent sections.

16.2.1 The Sensory System

The vestibular, the visual, and the proprioceptive and exteroceptive somatosensory systems all contribute to balance control (Fig. 16.3). Adequate functioning of these systems and their connections with the CNS is crucial.

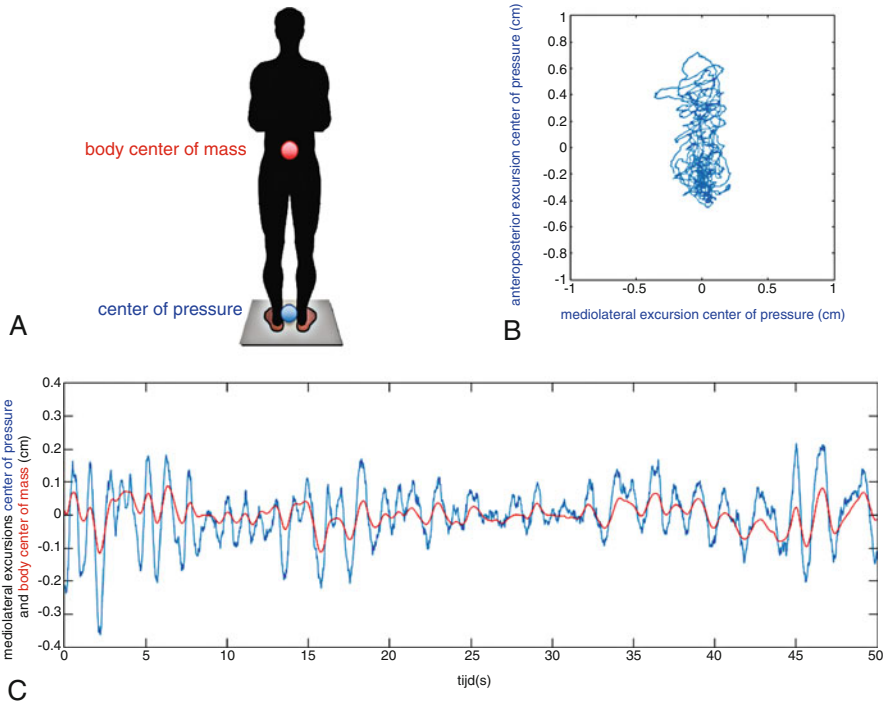

Fig. 16.2 Balance control in standing. (**a**) In upright stance the COM is located approximately in the center of the BOS; small movements of the COM and of the point of application of the ground reaction force are present continuously. (**b**) The fore-aft and sideward movements of the COM and of the center of pressure, the point of application of the ground reaction force, within the BOS illustrate the seemingly random movements within a relatively small part of the BOS. (**c**) The sideward movements of the COM (*red*) and BOS (*blue*) as a function of time illustrate that the movements of these points are related but not equal. For an explanation of their mutual relation, see, for example, [68]. Although the position of the COM has to be controlled to maintain balance, the point of application of the ground reaction force is easier to measure, and consequently it is often used to assess balance control

The vestibular system is localized in the head, behind the ears, and senses linear and angular accelerations of the head. Because gravity is basically a linear acceleration, it also provides information on the orientation of the head relative to the vertical. Afferent connections run mainly to the cerebellum, either directly or via the vestibular nuclei, which also connect to the thalamus and to motor neurons of eye muscles and skeletal muscles, providing the means for very fast feedback. The importance of the vestibular system for maintenance of vertical postures can be demonstrated by artificial stimulation. A weak current stimulation between the mastoid processes during standing causes an illusion of sway toward the side of the cathode and elicits sway in the opposite direction [7]. A substantial percentage of adults over 70 years of age show impaired vestibular function. Anatomically, a loss of sensors in the vestibular organs is apparent and this coincides with lower amplitudes of responses elicited by vestibular stimulation. Although it is well

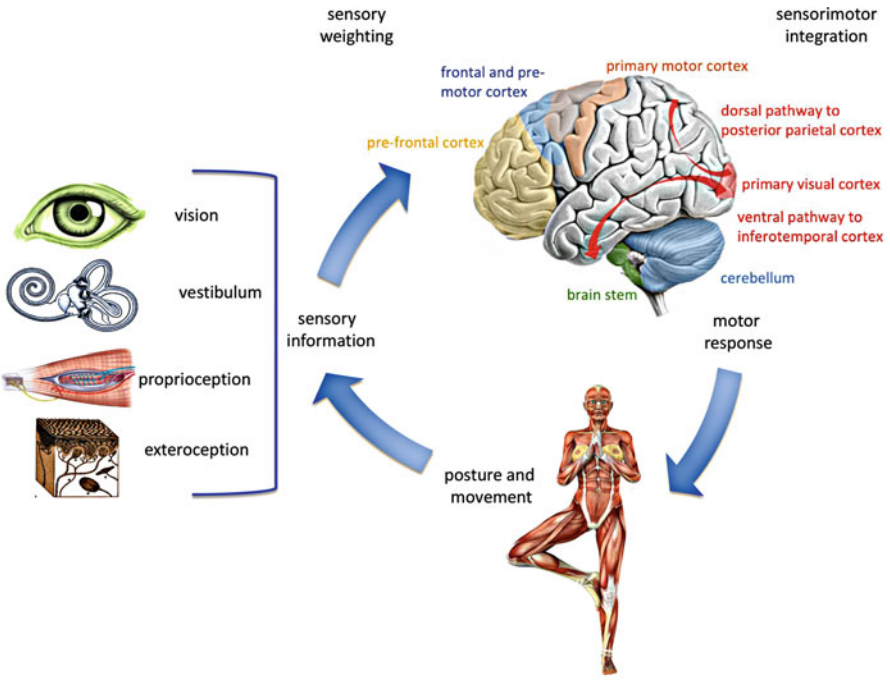

Fig. 16.3 The most important elements of the balance control feedback loop. The sensor system, consisting of the visual, vestibular, and proprioceptive and exteroceptive system provides information to the central nervous system. The most relevant brain areas for balance control are indicated, except for the thalamus which is not visible due to its central location. The central nervous system weights and integrates information from the different sensory modalities and generates motor responses to activate the muscles to adapt posture or movements to maintain balance

established that vestibular disorders increase fall risk, it is not yet clear whether reduced vestibular function in healthy, older adults also has this effect [8].

The visual system converts light patterns on the retina into an image of the environment, including an estimate of the vertical [9]. Consequently, an environment that provides inconsistent or incorrect clues on the orientation of the vertical, like a building with diagonal window frames, is often experienced as unpleasant. Self-movement causes changes of the light pattern on the retina, which thus provides information on the linear and angular movements of the head. The optical nerve connects the retina to the thalamus and the midbrain, and it is assumed that the latter connections are particularly important for balance control. The importance of visual information for balance control becomes immediately apparent from the increase in postural sway when one closes the eyes [10] or when a large part of the environment within the field of view is moving [11]. Visual information is also essential for anticipation to potential balance threats. From the fiftieth decade onwards, changes of the eyes lead to a reduction in visual acuity, contrast

Table 16.1 Balance control system: most important subsystems, functions, and effects of aging

Balance control system	Subsystems	Function in balance control	Effects of aging
Sensory system	Vestibular system	Senses linear and angular acceleration of the head and the gravitational acceleration	Loss of sensors; decreased amplitude of vestibular reflexes
	Visual system	Senses linear/angular acceleration of the head, vertical and horizontal references, and potential balance threats in the environment	Loss of sensors; reduced acuity, contrast sensitivity, depth perception, adaptation to low lighting and varying distance to objects in environment
	Proprioceptive somatosensory system (muscle spindles, tendon organs, joint and skin receptors)	Senses posture and movement of body segments relative to each other	Loss of number and sensitivity of sensors; reduced joint position sense
	Exteroceptive somatosensory system (receptors in foot soles and hands)	Senses tactile information and forces exerted on environment via feet and hands	Loss of sensors; reduced sensitivity foot soles
Central nervous system	Spinal cord and brainstem	Spinal reflexes and automated balance responses	Loss of the white matter (myelin sheets and myelinized nerve fibers); slowing down of transmission of sensory information
	Sensory nuclei, (pre-) motor, frontal and prefrontal cortex	Anticipation to self-generated and external perturbations; inhibition of planned activities	Loss of the white (myelin sheets and myelinized nerve fibers) and gray matter (nerve cells); impaired sensory integration and inhibition
	Cerebellum	Sensorimotor integration	Loss of the gray matter (nerve cells); impaired coordination
Motor system	Motor units	Force regulation	Loss of number of motor units; reduced precision
	Muscles	Force production	Loss (mainly type II) muscle fibers; reduced muscle strength and power
	Tendons	Force transmission from muscle to skeleton	Reduced stiffness; slower force transmission

sensitivity, and depth perception and to a reduced ability to adjust to poor lightning and varying distance to objects in the environment. Especially the reductions in depth perception and contrast sensitivity are correlated to increased fall risk in daily life [8], as is the use of multifocal glasses, which hampers perception of objects in close proximity of the feet [12].

The proprioceptive somatosensory system is distributed over the whole body and comprises muscle spindles, Golgi tendon organs, and joint and skin receptors [13]. This system provides information on the orientation and movement of body segments relative to each other, for which the muscle spindles are the main source of information. The muscle spindles encode changes in muscle length and the speed thereof and are at the spinal level directly connected to the motor neurons of the homonymous muscles and via interneurons to nearby heteronymous muscles. In addition, ascending pathways relay this information to the cerebellum and thalamus. Muscle spindles can be stimulated with mechanical vibrations between 50 and 100 Hz. Studies using this type of stimulation have shown that muscle spindles in leg, trunk, and neck muscles are important for balance control [14], and specifically the quality of proprioceptive information from ankle muscles appears important as this is strongly correlated to the ability to stand on one leg [15]. Compared to standing, balance control during walking is much less affected by proprioception [14], but vibration of the gluteus medius muscle during gait does have some effect on balance control in the frontal plane [16]. With aging, the number of muscle spindles as well as the sensitivity of remaining spindles decrease. This is reflected in a reduced acuity in perception of the posture and movements of joints. Reductions in acuity of posture and movement perception of the knee, ankle, and big toe have been shown to be related to postural sway, to a reduced ability to stand on one leg, and to the risk of falling in daily life [17].

The exteroceptive somatosensory system comprises a range of receptors in the soles of the feet that can provide information on the forces acting on the feet based on the resulting strain of the skin. The importance of these receptors for balance control is illustrated, among other factors, by the increase in postural sway that can be elicited by electrical stimulation of the foot soles [18]. Tactile information from other body parts, such as the hands through contact with an external object, even if that does not offer mechanical support, also contributes to balance control [19]. With aging, the number and the sensitivity of skin receptors in the foot soles decreases. Consequently, the threshold to perceive changes in pressure on the foot sole increases and the acuity for two-point discrimination decreases. These sensory losses are correlated to postural sway and fall risk [17].

Afferent nerve connections from the sensory system to the CNS lose nerve fibers and remaining nerve fibers lose their myelin sheaths. This leads to a reduction in nerve conduction and hence slowing down of balance feedback responses [17].

16.2.2 The Central Nervous System

At the lowest level of the CNS, in the spinal cord, proprioceptive and exteroceptive signals are integrated to evoke motor responses in case of balance perturbations. The spinal reflexes are mainly organized locally, i.e., they mainly involve muscles around the perturbed joint and they are aimed at correcting posture or movement of that joint. These reflexes are not always functional from a balance control perspective and they may even be counterproductive [20].

Balance control requires integrated responses of muscles around several joints simultaneously. The brain stem can generate these automated responses and animal experiments have shown that these remain intact even when all connections with higher brain centers have been disrupted. Yet higher centers of the brain are involved in more complex balance responses, if only because they are based on integration of information from several sensory modalities, as described above. This is important for two reasons. First, estimates of balance can be based on weighted information from different modalities, because their reliability is not perfect and does vary over time. Using a weighted sum of the estimates from single sources provides a robust estimate and the flexibility of weighting allows adaptation to, for example, environmental conditions. Second, integration of information is necessary because, for example, the meaning of signals from the vestibular or visual systems depends on the orientation of the head relative to the rest of the body, for which only proprioceptive information is available. The cerebellum appears to play a key role in adjustment of the motor activity aimed at maintaining and restoring balance on the basis of sensory information regarding the state of the body and the environment [21]. Patients with cerebellar disorders therefore show severe balance problems and they are characterized by compensatory behavior, such as a wide stance to enlarge the BOS.

Predictable balance perturbations can be avoided or attenuated by anticipatory actions. For example, when a planned movement like picking up an object will perturb balance, anticipatory muscle activity or movement of the body acting in the direction opposite to the perturbation is initiated [22]. In such anticipatory actions, the cortex and specifically the premotor cortex play an important role [23]. Similar anticipatory actions can be observed when an external but predictable balance perturbation occurs. When an obstacle that may perturb balance is seen during walking, it may be avoided by adjusting limb trajectory [24]. Such tasks involve visual processing via multiple cortical pathways [25, 26] (Fig. 16.3). Also memory plays a role, since, for example, visual information on obstacle dimensions is no longer available when stepping over an obstacle with the trailing leg. Prefrontal areas are in all likelihood involved in the planning of such adjustments in motor behavior and in inhibiting the initially planned activity. However, since reactions to suddenly appearing obstacles are markedly faster than voluntary reactions, it seems likely that subcortical areas of the brain initiate the responses, while higher centers further modify them [27].

The role of the cortex in responding to balance perturbations is debated, but there is evidence to suggest that premotor and primary motor cortices play a role in the

preselection of postural responses based on information available prior to the perturbation and in adjusting responses later in time [28]. Also balance control in unperturbed gait is related to cortical activity in the left premotor area [29]. All in all, a very extensive network appears active in balance control, with key roles for the cerebellum, premotor and motor areas, and the prefrontal cortex. From this perspective it comes as no surprise that cortical lesions after stroke and Alzheimer disease, or even simultaneously performing a demanding cognitive task, can seriously impair balance control and increase the risk of falls.

With aging, the brain loses neurons and neural connections. These losses vary across brain areas, with most prominent and least prominent losses in prefrontal areas and the cerebellum [30]. These losses affect the integration of sensory information for balance control. Consequently, older adults are more sensitive to sensory perturbations, such as those caused by artificial stimulation, but also real-life perturbations, such as those caused by movement of objects that occupy a large part of the visual field. Also older adults need more time to adjust when the reliability of one of the sources of sensory information changes [8, 31]. Possibly this is caused by a reduced ability to inhibit incoming information due to degeneration of the prefrontal cortex, but fear of losing balance may be an alternative cause [32]. These age-related brain changes are related to reduced balance control, limitations of mobility, and an increased fall risk [33].

16.2.3 The Motor System

Muscles and tendons comprise the most important components of the motor part of the balance control system. To correct large balance perturbations, fast and forceful contractions of muscles in legs [34], trunk [35], and even arms [36] are required, while balance control in unperturbed stance requires only moderate but accurate force production. The calf muscles and the hip abductor muscles play key roles in control of balance during bipedal stance in, respectively, the sagittal and frontal planes [37]. The latter muscles are also key in the control of unipedal stance [38], and both of these muscle groups appear to play an important role in balance control during gait [39, 40]. Muscle strength depends on the number of parallel muscle fibers and consequently on the cross-sectional area of the muscle. The rate at which these forces are transferred to the skeleton and hence at which movements can be controlled largely depends on tendon stiffness. Finally, the accuracy of muscle for production depends mainly on the number of motor units per muscle.

The physiological cross-sectional area of muscles decreases between 20 and 80 years of age, due to a loss of predominantly the fast type II muscle fibers. Muscle strength is maintained more or less up to 50 years of age but gradually declines after that by about 50 % at age 80. The strength of hip abductors is correlated to the time that older adults are able to stand on one leg [38] and the strength of the knee extensor muscles is correlated to the stability of gait [41]. In addition, strength and cross-sectional area of trunk muscles were reported to be negatively correlated to postural sway and fall risk [42]. These associations suggest that muscle strength can

be a limiting factor in dealing with minor balance perturbations that occur during standing and walking.

The rate at which muscles can increase force and the power that muscles can produce, i.e., the product of muscle force and shortening velocity, also decrease with age [43]. This is due to changes in the contractile properties of muscle fibers but also to a decreased stiffness of muscle tendons [44]. The age-related loss of muscle power is about twice as large as the loss in muscle strength, and consequently older adults are limited particularly in performing fast dynamic movement tasks [8]. Balance recovery responses are a clear example of this. Older adults are far less often successful in regaining their balance after tripping over an obstacle than young adults, and this holds especially for older adults with weak and slow muscles [45].

The age-related loss of the number of motor units per muscle, which is most pronounced in distal muscles, and the increase in variability of motor unit firing reduce the precision with which older adults can produce muscle force [46]. It has been shown that older adults with a history of falls are less precise in producing muscle forces than their healthier counterparts [47].

16.2.4 Conclusion

Age-related physiological changes of all the sensory subsystems and their afferent nerves, of parts of the CNS, and of muscles and tendons can all limit the ability to control balance. The functional decline of parts of the balance control system and its effect on balance control can be compensated by other parts of the system and can vary widely between individuals.

16.3 Balance Control in Standing

In standing, the BOS is formed by one or both feet. When standing upright, the COM will be at about 1 m above the support surface, which implies that body sway over a small angle can cause a substantial gravitational torque. Active control of balance is required to counteract these gravitational torques. In the subsequent sections, we will describe how balance is controlled in unperturbed, quiet standing and how balance is corrected after external perturbations. In addition, we describe how balance control in standing is usually assessed and how it changes with aging, with an emphasis on the factors that limit balance control in these situations.

16.3.1 Unperturbed Stance

In standing upright, we sway continuously and the COM moves apparently randomly over the BOS (Fig. 16.2). The continuous COM movement is a consequence of the disturbing effects of gravity and of muscle activity. The magnitude of

postural sway is often used as a measure of the quality of balance control. In two-legged stance, the COM moves over a small and central part of the BOS. Balance control is more challenged when the BOS is reduced, by placing the feet close together, in tandem stance, or by standing on one leg. If instructed to do so, we can usually decrease the amount of postural sway, which implies that we do not normally attempt to minimize it, possibly because this costs energy [48], but there is also evidence to suggest that we produce a certain amount of postural sway to gather sensory information on balance [49].

Older adults generally show a larger postural sway than young adults, especially in the mediolateral direction [50], and their sway amplitude increases more when they close their eyes [51]. Consequently, many older adults are incapable of standing in tandem stance with their eyes closed. However, the increase in sway is also larger in older adults than in young adults when proprioception is perturbed [31]. More generally, it thus seems that older adults are less able to compensate for perturbations of one of the sensory modalities that contribute to postural control. Surprisingly, sometimes, postural sway in upright stance is smaller in old than in young adults, possibly because young adults can afford not to control their balance very tightly, as standing does not challenge them. This explanation is supported by the finding that young adults decreased their sway more than old adults when asked to lean maximally forward or backward [52].

The quality of balance control in standing can be limited by age-related sensory impairments, as described in Sect. 16.2 of this chapter, but also by changes in the CNS. Experiments in which subjects perform cognitive dual tasks, for example, counting down from 100 by 7, show that either balance or the cognitive task is more affected by simultaneous performance than in young adults. This is possibly related to the fact that the cortex is more involved in balance control [33]. Cortical involvement appears to be determined by the relative difficulty of the task; hence the higher cortical activity in older adults is probably compensatory [53]. However, impaired brain function can limit balance control. This is shown by the effect of orthostatic hypotension on balance, where insufficient regulation of blood pressure, which is common among older adults, causes insufficient oxygen supply to the brain. The speed at which blood pressure is regulated after transitioning from sit to stance in older adults is associated with the time that balance can be maintained in tandem stance with eyes closed [54]. Also an association between cognitive abilities and balance control in stance has been found [55]. Finally, the motor system can limit balance control. The time that older adults can maintain balance in various standing postures is associated with muscle strength [56], and lower precision of muscle force production is associated with larger postural sway [57].

16.3.2 External Perturbation of Standing Balance

If a mechanical perturbation causes or threatens the body COM to move outside the BOS, reactive balance control is needed. Reactive balance control is usually tested by sudden and unexpected translations or rotations of a platform that the person

stands on. Generally, older adults show larger postural sway after such perturbations than young adults [58, 59], and they produce higher joint torques to regain balance [58]. Also older adults and especially adults with balance problems more often make a step, using displacement of the BOS to regain balance [60, 61].

Balance perturbations cause early spinal reflexes and later responses generated by higher centers of the CNS. The early responses can be delayed in older adults by around 5 ms relative to young adults, probably as a result of lower nerve conduction velocity. Delays of about 20 ms are found for the later responses and this is attributed to slower information processing in the brain [33]. The magnitude of postural sway after perturbations is associated with thresholds of sensors in foot soles [59], as well as with delays of early and late responses [33]. In addition, the magnitude of sway and the necessity to make a step after perturbations are associated with muscle strength of ankle and trunk muscles [59, 62], in line with the relation between trunk muscle strength and fall risk as described above. The effect of muscle strength on perturbation responses is striking, since older adults were shown to produce higher joint torques than young adults and because these torques were well below maximum levels predicted based on muscle strength [58]. Possibly, the rate at which muscle force and hence joint torques can be developed is the true limiting factor, as this is in turn associated with muscle strength.

16.3.3 Conclusion

The degree to which older adults are able to constrain postural sway to a small part of the BOS and the ability to remain standing when the BOS is reduced are limited by age-related changes of the sensory system, the CNS, and the motor system. The ability to regain standing balance without stepping appears to be limited mainly by the rate at which muscle activation can be increased and at which the muscles can produce force, which are determined by changes in the peripheral and central nervous system, as well as the muscles themselves.

16.4 Balance Control in Gait

In this section, we will describe how balance is controlled, how perturbations are avoided, and how balance is regained during gait. Methods to assess balance control will be discussed, and an overview is given of the changes that occur with aging as well as of the factors that limit balance control during gait in healthy older adults.

16.4.1 Unperturbed Gait

During gait, the body COM has a forward velocity and consequently the BOS has to be displaced to prevent falling. The properties of the human body greatly simplify

the control in the fore-aft direction when walking at constant velocity. The inertia of the legs and the velocity of the body more or less make the leg swing forward just enough to obtain the correct new BOS to maintain balance and gait velocity. Obviously this is a simplification of reality and active control is necessary to deal with variations in the environment and control of balance in the mediolateral direction is even more challenging [63].

Several measures to characterize balance control during gait have been developed and many of these are based on the notion that the movements in unperturbed gait are expected to be regular (i.e., subsequent steps should resemble each other) and that the movement should be stable (i.e., small perturbations of gait are quickly corrected). Many different parameters quantify the (ir-)regularity or (in-)stability of gait [64]; it is however beyond the scope of this chapter to describe these in detail, suffices to state that larger irregularity (or variability) and lower stability are related to age [65], fall history [66], and fall risk [67].

As in standing, the position of the COM relative to the edges of the BOS provides a measure of the margin of safety in balance control. For gait, the velocity of the COM has to be taken into account [68]. The so-obtained dynamic margin of safety ("margin of stability") is frequently used to assess mediolateral balance. Older adults walk with wider steps than young adults and, although this is seen as a compensation to increase the BOS, this is associated with an increase in the amplitude and velocity of sideward movement of the body COM, and when environmental conditions dictate a smaller step width, this leads to a reduced safety margin [69].

Although it is well established that balance control in gait is dependent on sensory information and affected by larger parts of the CNS (see Sect. 16.2), little is known about the interactions and the limiting factors in these systems regarding balance control in gait. It is known that muscle strength is related to gait stability [41] and regularity [70]. Also it has been found that reduced mediolateral margins of safety are related to degenerative changes in the brain, indicating that individuals with impaired connectivity between subcortical and prefrontal brain areas do not adequately compensate for their impaired balance control by regulating step width [71].

16.4.2 Avoidance of Balance Perturbation in Gait

In daily-life gait, we encounter all sorts of potential balance perturbations that we should try to avoid, or when we fail to do so, we must try to regain balance in order to prevent falling. Tripping is reported to be the most common cause of falls among healthy older adults both indoors and outdoors [72]. In this paragraph and the next, we will concentrate on avoiding balance perturbations and recovering from balance perturbations, with tripping as a point in case.

When an obstacle blocks the planned path of the swing leg, it is avoided by either elevating the swing leg higher and lengthening its step or by shortening the step by placing the leg in front of the obstacle to cross it with the other leg first. In young

adults, the choice between these strategies appears to be made such that the deviation relative to the initially planned step is minimal [73]. Older adults however appear to prefer lengthening their step [74]. This preference may reduce the probability of loosing balance, as it avoids that the leg is placed underneath or even behind the body COM [75]. However, it may also be a consequence of older adults not being able to react quick enough to perform the short-step strategy [76].

Obviously, visual information is crucial for adequate obstacle avoidance. The length of the ultimate step before the obstacle and the path of the leg over the obstacle are adjusted to position, height, and other properties of the obstacle [77]. Young adults gather the information needed to do so in the few meters before reaching the obstacle. The duration of visual fixation of the obstacle depends on obstacle height, suggesting that detailed information is gathered [78]. This implies that visual acuity and contrast sensitivity at a few meters distance, but also short-term memory, are important for successful avoidance and may explain why wearing multifocal glasses increases fall risk (Sect. 16.2).

When older adults cross an obstacle, they do so with smaller toe clearance than young adults [79]. They are also less successful in avoiding obstacles when these are first seen when they are already close. Interestingly, this effect is much smaller than one would expect based on age effects in reaction time tests [76]. The importance of cognitive processes for obstacle avoidance is evident from the fact that cognitive impairments [80] and dual task performance [81] reduce the probability of success at this task.

16.4.3 Perturbations of Balance in Gait

If an obstacle cannot be avoided, a collision of the swing leg with the obstacle will cause a trip; the forward placement of the leg is hampered and, if one does not react adequately, a forward rotation of the body, further accelerated by gravity, ends in a fall. Balance recovery requires that one decelerates the forward rotation and places one leg over the obstacle to obtain an adequate BOS and to continue walking (Fig. 16.1). Two types of recovery reactions have been described [82]: if the leg is blocked early in the swing phase, it is quickly elevated over the obstacle and placed behind the obstacle, and, if the leg is blocked late in the swing phase, it is placed on the floor in front of the obstacle and subsequently the other leg steps over the obstacle first. In both cases, the stance leg during the trip has an important contribution to deceleration of the forward rotation by a fast and forceful forward directed push-off. At about 80 ms after impact with obstacle, functional changes in muscle activity are observed in both legs, probably triggered by proprioceptive information from the skin or distal joints and muscles of the blocked leg [83]. Changes in movements of the head occur too late for visual and vestibular inputs to contribute to the generation of these early responses.

Older adults are clearly less successful in performing balance recovery reactions after tripping than young adults. They decelerate the forward rotation less and place their recovery leg less far over the obstacle [84] and fall more often than young

adults [72]. The reaction time after tripping as measured from changes in muscle activity does not appear to be the limiting factor as it is only marginally different between young and older adults and not different between older adults who fall and do not fall after these perturbations [34]. In simulations of the recovery reaction, in which people are released from a forward-leaning position, the velocity of the forward movement of the recovery leg appears to be the limiting factor for successful recovery. A lower rate of the increase in neural drive to the leg muscles appears to underlie this lower movement speed [85]. Successful recovery after tripping is actually also determined by the degree to which one is able to decelerate the forward movement of the trunk [86]. Muscles in the stance leg and trunk are responsible for decelerating the forward rotation of the body before a leg is placed over the obstacle [87, 88]. This requires fast adaptations of leg muscle activity and a maximal activity of the calf muscles. The joint torques produced by these muscles are adapted more slowly in old than in young adults, and this is at least in part due to a slower increase in neural drive to these muscles [84]. Older adults with a lower leg muscle strength and a lower maximum rate of joint torque development around knee and ankle joints fall more often after being tripped than their stronger and faster peers [45].

16.4.4 Conclusion

The quality of balance control and the ability to avoid obstacles during gait both decline with age. It is as of yet not clear what the limiting factors are. Muscle strength is associated with the quality of balance control during gait, but this may not reflect causality. Cognitive impairment appears to play a role, although its importance remains to be determined. The ability to regain balance after a perturbation of gait also declines with age, and this is caused by a reduced ability to generate fast changes in muscle force, probably due to changes in the CNS as well as in the muscles and muscle tendons themselves.

16.5 Assessment of Balance Control

A variety of methods to assess balance in older adults has been proposed, ranging from questionnaires to extensive test batteries. These methods are often used to establish (causes of) balance problems, to target interventions, and to assess the effects of interventions. The most common clinical tests assess static balance in standing or dynamic balance during functional movements such as stepping and walking. In general, these clinical tests measure the success or failure to maintain balance during a certain task or the quality of the performance of a balance task. In contrast, physiological tests assess the potential impairments underlying balance problems [89]. In this section, we discuss subjective methods, and static, dynamic, and functional balance tests, and we present tests aimed at establishing the causes of balance problems. The information described below is summarized in Table 16.2.

Table 16.2 Overview of tests of the balance control system

Type of measurement	Test	Way of testing	Score
Subjective tests	Fall history	Have you fallen in the last 12 months? If so, how often?	Dichotomous score (yes/no) or number of falls
	Falls Efficacy Scale International (FES-I) [91]	16-item questionnaire on fear of falling during several activities	Ordinal score 16–64; high score = high fear
	Activities-specific Balance Confidence (ABC) scale [94]	16-item questionnaire on confidence in performance of several activities without losing balance or falling	Ordinal score per item 0–100; high score = high confidence
Static balance tests	(Semi)tandem stance	Maintain for 10 s, can be combined with closing eyes, unstable support surface, dual task; stopwatch	Dichotomous score (yes/no) or duration; shorter duration = worse balance
	Instrumented (semi)tandem stance	Maintain for 10 s, can be combined with closing eyes, unstable support surface, dual task; force plate, camera system, or inertial measurement unit	Quantitative score (displacement/speed/ acceleration body center of mass or center of pressure); faster, larger sway = worse balance
Dynamic balance tests	Limits of stability (LOS) test [98]	Self-induced maximal movement in eight directions without stepping; force plate (e.g., NeuroCom; Biodex Balance System)	Quantitative score; smaller/ slower displacement = worse balance
	MEdioLateral Balance Assessment (MELBA) test [99]	Self-induced mediolateral weight shifts; force plate or camera system	Quantitative score; lower frequency/ velocity = worse balance
	Balance perturbations [e.g., 102, 103, 106]	Platform translation/rotation or upper body push/pull in standing or walking; instrumented platform or treadmill	Dichotomous score (yes/no stepping response) or quantitative score; stronger perturbation of standing or gait = worse balance

Functional balance tests	Performance-Oriented Mobility Assessment (POMA or Tinetti) [107]	Balance evaluation (13 balance tasks, scored 0–16) and mobility evaluation (6 gait tasks, scored 0–12)	Ordinal score 0–28; lower score = worse balance/mobility; score <19 high fall risk
	Berg Balance Scale (BBS) [108]	14 items, scored with 5-point scale	Ordinal score 0–56; lower score = worse balance; score <43 high fall risk
	Timed Up and Go test (TUG) [109]	Time to get up from chair, walk 3 m, turn around, walk back and sit down; stopwatch	Continuous score (total time); longer time = worse balance/mobility
	Short Physical Performance Battery (SPPB) [110]	5 items (gait, 3 balance tests, repeated sit-to-stand)	Ordinal score 0–12; lower score = worse balance/mobility
Subsystem test	Physiological Profile Assessment (PPA) [116] www.neura.edu.au/fbrg	15 items (3 vision, 3 proprioception, 3 muscle strength, 2 reaction time, 4 postural sway) or short version with 5 items (1 per domain)	1 score for fall risk based on z-scores reference group
	Balance Evaluation Systems Test (BESTest) [117] http://www.bestest.us	27 items (5 biomechanical, 3 balance, 5 transitions, 5 reactive, 3 sensory, 7 gait) or short version with 14 items	Ordinal score 0–108 (short version 0–28); lower score = worse balance/mobility
	Sensory organization test (SOT) [121]	Postural sway in six conditions: eyes open/closed, rigid/unstable support surface, fixed/moving visual surround (e.g., NeuroCom)	Quantitative score based on sway per condition and weighted average total score

16.5.1 Subjective Assessment of Balance Problems

Assessment of balance control usually starts with asking question, for example, on fall history. Having fallen in the preceding year is a strong predictor of future falls [e.g., 67] and hence an important indication of balance problems. In addition, individuals may report to experience balance problems or fear of falling. Such indicators of balance problems can be assessed systematically with questionnaires on fear of falling or balance confidence [90]. Fear of falling, defined as the concern that one may fall during daily activities, can be measured with the Falls Efficacy Scale International (FES-I), a 16-item questionnaire [91], or its shortened 7-item version [92]. A high fear of falling is associated with worse balance performance in standing and in gait [90]. As an alternative to these questionnaires, the icon-FES uses images of activities and asks the individual to indicate whether these would elicit fear of falling [93]. The Activities-specific Balance Confidence (ABC) scale is a commonly used 16-item questionnaire [94] to measure balance confidence, the inverse of fear of falling, which as such has a more positive connotation. It reflects one's confidence in the ability to maintain balance during daily activities.

16.5.2 Static Balance Tests

Widely used methods for static balance testing are based on assessment of the ability to maintain balance over a constrained BOS, for example, with the feet close together or in tandem stance for a certain period of time. The 10-s (semi-)tandem stance is probably the most commonly used. Inability to perform this test is indicative of impaired balance control [95]. To make such tests more challenging and to specifically address the role of the proprioceptive system, the visual system, or cognitive processing, the same tests can be performed with eyes closed, on a compliant surface, or with a cognitive dual task.

Static balance assessments can be enhanced by measuring postural sway with a camera system or by inertial sensors measuring the movement of body segments, from which the movements of the COM can be estimated. This so-called posturography allows measurement of the amplitude, velocity, or acceleration of movements of the COM in fore-aft and sideward directions. This postural sway is also often estimated from movements of the point of application of the ground reaction force (or center of pressure) (Fig. 16.2), measured with a force plate or cheaper alternatives such as the Wii Balance Board [96]. At low movement speed, the center of pressure gives a good approximation of the movements of the body COM, but at higher speeds the difference between the amplitude of the COM and the center of pressure increases. The quality of balance control as measured this way decreases with age (see Sect. 16.3), but the predictive value thereof for fall risk is not clear yet [97]. The interpretation of these measures is thus still open to discussion.

16.5.3 Dynamic Balance Tests

Dynamic posturography tests the responses to self-imposed or externally imposed balance perturbations. A commonly used clinical approach to measure balance under self-induced perturbations is the limits of stability (LOS) test, which measures the maximum range of motion that one can make by leaning the body in a specified direction without stepping [98]. A recent method, called MEdioLateral Balance Assessment (MELBA), tests the ability to follow a moving target by making sideward movements of the COM at incremental speed. This test has been shown to be reliable, sensitive to differences between young and older adults, and indicative of balance problems during gait on a treadmill and in daily life [99]. In addition, the test results are correlated to strength of the hip abductor muscles [100]. Another promising, new, and simple balance test assesses the distance that one can walk over a narrow beam [101].

Platform translations and rotations or devices generating pushes or pulls are used to measure the ability to resist perturbations or to regain balance after perturbations [e.g., 102, 103]. The necessity to make a step, especially after sideward or backward perturbations of standing balance, is a strong predictor of future falls [104, 105]. A drawback of these tests is that standardized perturbations do not necessarily exceed the capacity of the individual, causing ceiling effects. More advanced instruments that standardize the perturbation intensity, but also allow to gradually increase it, are needed to bypass this problem [e.g., 62, 106]. Such instruments are usually expensive and their added value in clinical context is still being studied.

16.5.4 Functional Balance Tests

The most commonly used functional, clinical balance tests are the Tinetti Balance Test, or Performance-Oriented Mobility Assessment (POMA) [107], the Berg Balance Scale (BBS) [108], the Timed Up and Go test (TUG) [109], and the Short Physical Performance Battery (SPPB) [110]. These tests measure the ability to maintain balance in tasks such as getting up from a chair, standing, and walking. These tests are simple and cheap and have been shown to have good reliability within and between observers [89]. However, other aspects of physical capacity also determine the outcomes, and balance problems are identified only when these are already quite severe [95]. Relatively fit, community-dwelling older adults are often able to use compensatory strategies masking impairments of their balance control system during such tests [95], leading to ceiling effects and making these tests less suitable for early detection of balance problems [89].

The outcomes of clinical, functional balance tests are often interpreted in relation to fall risk. However, their predictive value for falls is rather limited. The TUG is, for example, often used to assess fall risk but in fact does not differentiate older fallers from non-fallers [111]. Also for the other tests, there is no or only very limited evidence for a predictive value with respect to fall risk [89]. These tools are indicative of increased risk only in populations that they were tested for. In

addition, the outcomes are not very sensitive to change, for example, changes due to interventions to prevent falls [112].

Currently, instrumented versions of functional balance tests are developed and used. Sensors are employed during the tests to obtain more objective and precise results and a higher sensitivity to subtle balance impairments. Accelerometers and gyroscopes provide detailed information on timing and execution of movements in functional tasks such as walking [66] and getting up from a chair [113]. The outcomes are promising indicators of balance problems and fall risk, but also for these measures, solid evidence is lacking [114]. Accelerometers and gyroscopes can also be used in daily life. Recent studies have shown that information on the stability of walking [67] and standing up from a chair [115] acquired from acceleration data obtained in daily life have added value for the prediction of fall risk, but the clinical value thereof still needs to be demonstrated.

16.5.5 Testing the Balance Control System

Tests of the balance control system aim at establishing the specific nature and/or causes of balance problems, to allow tailored interventions. The Physiological Profile Assessment (PPA) is an extensive test battery that addresses visual, vestibular, and proprioceptive functions as well as leg muscle strength, reaction time, and static and dynamic posturography [116]. The items were selected based on their importance for balance control and the PPA has good predictive value for falls [116]. The Balance Evaluation Systems Test (BESTest) comprises a number of items from previously developed functional tests (such as the TUG and LOS) and aims to assess specific domains of balance control, such as stability in reaching and walking, anticipatory and reactive control, and also some determinants of balance control such as muscle strength [117]. Although not specifically developed to this end, the BESTest has good predictive value for fall risk [118].

Sensory manipulations are often used to establish specific causes of balance problems. A commonly used test is the sensory organization test (SOT), which comprises a movable force plate and a moveable environment to impose visual perturbations. Using six specific conditions with eyes open or closed, the differences in postural sway between conditions provide information on the relative contributions of the visual, proprioceptive, and vestibular system to balance control. In addition, the results provide information on the ability to compensate for unreliable sensory information from the modalities involved [119].

More advanced tests to assess the contributions of different subsystems to balance control are under development, for example, for standing balance [120] or for balance in treadmill walking [121]. These methods combine sensory and mechanical perturbations to provide a detailed identification of the balance control system. The ability to maintain balance during gait in complex environments, which challenges cognitive function can be tested with tools such as the “interactive walkway,” which uses Kinect sensors and projections of obstacles or targets on the floor [122].

16.5.6 Conclusion

No single test is sufficient to fully characterize static or dynamic balance control and to predict falls [123]. Dependent on the clinical question at hand, a combination of tests will be needed. It is striking that anticipatory control, such as when avoiding obstacles, receives limited attention in clinical balance testing, while this might be an important determinant of fall risk. Furthermore, clinical tests, by necessity, standardize and simplify the complexity of balance-threatening conditions that one may be exposed to in daily life. Although dynamic tests can provide information on reactive control of balance, these are usually applied in standing postures and not during dynamic activities such as walking. It is still unclear to what extent such simplifications threaten the validity of the outcomes. From this perspective, the development of balance assessment based on ambulatory measurements in daily life seems a promising approach to obtain a more complete picture.

References

1. Lord SR, Sherrington C, Menz HB. Falls in older people: risk factors and strategies for prevention. 2nd ed. Cambridge: Cambridge University Press; 2001.
2. WHO. WHO global report on falls prevention in older age. Geneva: WHO Press; 2007. P. 1–7, 47.
3. Robinovitch SN, Feldman F, Yang Y, Schonnop R, Leung PM, Sarraf T, et al. Video capture of the circumstances of falls in elderly people residing in long-term care: an observational study. Lancet. 2013;381(9860):47–54.
4. Kelsey JL, Berry SD, Procter-Gray E, Quach L, Nguyen US, Li W, et al. Indoor and outdoor falls in older adults are different: the maintenance of balance, independent living, intellect, and Zest in the Elderly of Boston Study. J Am Geriatr Soc. 2010;58(11):2135–41.
5. Hof AL. The equations of motion for a standing human reveal three mechanisms for balance. J Biomech. 2007;40:451–7.
6. Loram ID, Lakie M. Direct measurement of human ankle stiffness during quiet standing: the intrinsic mechanical stiffness is insufficient for stability. J Physiol. 2002;545:1041–53.
7. Iles JF, Baderin R, Tanner R, Simon A. Human standing and walking: comparison of the effects of stimulation of the vestibular system. Exp Brain Res. 2007;178(2):151–66.
8. Sturnieks DL, St George R, Lord SR. Balance disorders in the elderly. Neurophysiol Clin. 2008;38(6):467–78.
9. St George RJ, Fitzpatrick RC. The sense of self-motion, orientation and balance explored by vestibular stimulation. J Physiol. 2011;589:807–13.
10. Paulus WM, Straube A, Brandt T. Visual stabilization of posture. Physiological stimulus characteristics and clinical aspects. Brain. 1984;107(Pt 4):1143–63.
11. Redfern M, Yardley L, Bronstein A. Visual influences on balance. J Anxiety Disord. 2001;15:81–94.
12. Lord SR, Dayhew J, Howland A. Multifocal glasses impair edge-contrast sensitivity and depth perception and increase the risk of falls in older people. J Am Geriatr Soc. 2002;50 (11):1760–6.
13. Proske U, Gandevia SC. The proprioceptive senses: their roles in signaling body shape, body position and movement, and muscle force. Physiol Rev. 2012;92(4):1651–97.
14. Courtine G, De Nunzio AM, Schmid M, Beretta MV, Schieppati M. Stance- and locomotion-dependent processing of vibration-induced proprioceptive inflow from multiple muscles in humans. J Neurophysiol. 2007;97(1):772–9.

15. Son J, Ashton-Miller JA, Richardson JK. Frontal plane ankle proprioceptive thresholds and unipedal balance. Muscle Nerve. 2009;39(2):150–7.
16. Roden-Reynolds DC, Walker MH, Wasserman CR, Dean JC. Hip proprioceptive feedback influences the control of mediolateral stability during human walking. J Neurophysiol. 2015;114(4):2220–9.
17. Shaffer SW, Harrison AL. Aging of the somatosensory system: a translational perspective. Phys Ther. 2007;87(2):193–207.
18. Cofre Lizama LE, Pijnappels M, Verschueren S, Reeves NP, van Dieën JH. Can explicit visual feedback of postural sway efface the effects of sensory manipulations on mediolateral balance performance? J Neurophysiol. 2016;115:907–14.
19. Jeka JJ. Light touch contact as a balance aid. Phys Ther. 1997;77(5):476–87.
20. Welch TD, Ting LH. Mechanisms of motor adaptation in reactive balance control. PLoS One. 2014;9(5), e96440.
21. Manzoni D. The cerebellum may implement the appropriate coupling of sensory inputs and motor responses: evidence from vestibular physiology. Cerebellum. 2005;4(3):178–88.
22. van Dieen J, de Looze M. Directionality of anticipatory activation of trunk muscles in a lifting task depends on load knowledge. Exp Brain Res. 1999;128(3):397–404.
23. Jacobs JV, Lou JS, Kraakevik JA, Horak FB. The supplementary motor area contributes to the timing of the anticipatory postural adjustment during step initiation in participants with and without Parkinson's disease. Neuroscience. 2009;164(2):877–85.
24. Moraes R, Lewis MA, Patla AE. Strategies and determinants for selection of alternate foot placement during human locomotion: influence of spatial and temporal constraints. Exp Brain Res. 2004;159(1):1–13.
25. Hesse C, Lane AR, Aimola L, Schenk T. Pathways involved in human conscious vision contribute to obstacle-avoidance behaviour. Eur J Neurosci. 2012;36(3):2383–90.
26. Patla AE, Goodale MA. Obstacle avoidance during locomotion is unaffected in a patient with visual form agnosia. Neuroreport. 1996;8:165–8.
27. Weerdesteyn V, Nienhuis B, Hampsink B, Duysens J. Gait adjustments in response to an obstacle are faster than voluntary reactions. Hum Mov Sci. 2004;23(3–4):351–63.
28. Jacobs JV, Horak FB. Cortical control of postural responses. J Neural Transm (Vienna). 2007;114(10):1339–48.
29. Bruijn SM, van Dieën JH, Daffertshofer A. Beta activity in the premotor cortex is increased during stabilized as compared to normal walking. Front Hum Neurosci. 2015;9:593.
30. Sullivan EV, Deshmukh A, Desmond JE, Lim KO, Pfefferbaum A. Cerebellar volume decline in normal aging, alcoholism, and Korsakoff's syndrome: relation to ataxia. Neuropsychology. 2000;14(3):341–52.
31. Wiesmeier IK, Dalin D, Maurer C. Elderly use proprioception rather than visual and vestibular cues for postural motor control. Front Aging Neurosci. 2015;7:97.
32. Eikema DJ, Hatzitaki V, Tzovaras D, Papaxanthis C. Age-dependent modulation of sensory reweighting for controlling posture in a dynamic virtual environment. Age (Dordr). 2012;34(6):1381–92.
33. Papegaaij S, Taube W, Baudry S, Otten E, Hortobagyi T. Aging causes a reorganization of cortical and spinal control of posture. Front Aging Neurosci. 2014;6:28.
34. Pijnappels M, Bobbert MF, van Dieën JH. Control of support limb muscles in recovery after tripping in young and older subjects. Exp Brain Res. 2005;160(3):326–33.
35. van der Burg JC, Pijnappels M, van Dieën JH. Out-of-plane trunk movements and trunk muscle activity after a trip during walking. Exp Brain Res. 2005;165(3):407–12.
36. Pijnappels M, Kingma I, Wezenberg D, Reurink G, van Dieen JH. Armed against falls: the contribution of arm movements to balance recovery after tripping. Exp Brain Res. 2010;201(4):689–99.
37. Winter DA, Prince F, Frank JS, Powell C, Zabjek KF. Unified theory regarding A/P and M/L balance in quiet stance. J Neurophysiol. 1996;75(6):2334–43.

38. Allet L, Kim H, Ashton-Miller J, De Mott T, Richardson JK. Frontal plane hip and ankle sensorimotor function, not age, predicts unipedal stance time. Muscle Nerve. 2012;45 (4):578–85.
39. Kim M, Collins SH. Once-per-step control of ankle-foot prosthesis push-off work reduces effort associated with balance during walking. J Neuroeng Rehabil. 2015;12:43.
40. Rankin BL, Buffo SK, Dean JC. A neuromechanical strategy for mediolateral foot placement in walking humans. J Neurophysiol. 2014;112(2):374–83.
41. Toebes MJ, Hoozemans MJM, Dekker J, van Dieen JH. Associations between measures of gait stability, leg strength and fear of falling. Gait Posture. 2015;41:76–80.
42. Granacher U, Gollhofer A, Hortobagyi T, Kressig RW, Muehlbauer T. The importance of trunk muscle strength for balance, functional performance, and fall prevention in seniors: a systematic review. Sports Med. 2013;43(7):627–41.
43. Thelen DG, Schultz AB, Alexander NB, Ashton-Miller JA. Effects of age on rapid ankle torque development. J Gerontol A Biol Sci Med Sci. 1996;51(5):M226–32.
44. Reeves ND, Narici MV, Maganaris CN. Myotendinous plasticity to ageing and resistance exercise in humans. Exp Physiol. 2006;91(3):483–98.
45. Pijnappels M, van der Burg PJ, Reeves ND, van Dieen JH. Identification of elderly fallers by muscle strength measures. Eur J Appl Physiol. 2008;102(5):585–92.
46. Enoka RM, Christou EA, Hunter SK, Kornatz KW, Semmler JG, Taylor AM, et al. Mechanisms that contribute to differences in motor performance between young and old adults. J Electromyogr Kinesiol. 2003;13(1):1–12.
47. Carville SF, Perry MC, Rutherford OM, Smith IC, Newham DJ. Steadiness of quadriceps contractions in young and older adults with and without a history of falling. Eur J Appl Physiol. 2007;100(5):527–33.
48. Houdijk H, Brown S, van Dieen JH. The relation between postural sway magnitude and metabolic energy cost during upright standing on a compliant surface. J Appl Physiol. 2015;119:696–703.
49. Carpenter MG, Murnaghan CD, Inglis JT. Shifting the balance: evidence of an exploratory role for postural sway. Neuroscience. 2010;171(1):196–204.
50. Pasma JH, Bijlsma AY, van der Bij MD, Arendzen JH, Meskers CG, Maier AB. Age-related differences in quality of standing balance using a composite score. Gerontology. 2014;60 (4):306–14.
51. Choy NL, Brauer S, Nitz J. Changes in postural stability in women aged 20 to 80 years. J Gerontol A Biol Sci Med Sci. 2003;58(6):525–30.
52. van Wegen EEH, van Emmerik REA, Riccio GE. Postural orientation: age-related changes in variability and time-to-boundary. Hum Mov Sci. 2002;21(1):61–84.
53. Papegaaij S, Taube W, van Keeken HG, Otten E, Baudry S, Hortobagyi T. Postural challenge affects motor cortical activity in young and old adults. Exp Gerontol. 2016;73:78–85.
54. Pasma JH, Bijlsma AYJ, Klip JM, Stijntjes M, Blauw GJ, Muller M, et al. Blood pressure associates with standing balance in elderly outpatients. PLoS One. 2014;9, e106808.
55. Stijntjes M, Pasma JH, van Vuuren M, Blauw GJ, Meskers CG, Maier AB. Low cognitive status is associated with a lower ability to maintain standing balance in elderly outpatients. Gerontology. 2015;61(2):124–30.
56. Bijlsma AY, Pasma JH, Lambers D, Stijntjes M, Blauw GJ, Meskers CG, et al. Muscle strength rather than muscle mass is associated with standing balance in elderly outpatients. J Am Med Dir Assoc. 2013;14:493–8.
57. Kouzaki M, Shinohara M. Steadiness in plantar flexor muscles and its relation to postural sway in young and elderly adults. Muscle Nerve. 2010;42(1):78–87.
58. Gu M-J, Schultz AB, Shepard NT, Alexander NB. Postural control in young and elderly adults when stance is perturbed: dynamics. J Biomech. 1996;29(3):319–29.
59. Wu G. The relation between age-related changes in neuromusculoskeletal system and dynamic postural responses to balance disturbance. J Gerontol A Biol Sci Med Sci. 1998;53(4):M320–6.

60. Schulz BW, Ashton-Miller JA, Alexander NB. Compensatory stepping in response to waist pulls in balance-impaired and unimpaired women. Gait Posture. 2005;22(3):198–209.
61. McIlroy WE, Maki BE. Changes in early automatic postural responses associated with the prior-planning and execution of a compensatory step. Brain Res. 1993;631(2):203–11.
62. Rijken NH, van Engelen BG, de Rooy JW, Geurts AC, Weerdesteyn V. Trunk muscle involvement is most critical for the loss of balance control in patients with facioscapulohumeral muscular dystrophy. Clin Biomech (Bristol, Avon). 2014;29 (8):855–60.
63. Kuo AD. Stabilization of lateral motion in passive dynamic walking. Int J Rob Res. 1999;18 (9):917–30.
64. Bruijn SM, Meijer OG, Beek PJ, van Dieen JH. Assessing the stability of human locomotion: a review of current measures. J R Soc Interface. 2013;10(83):20120999.
65. Hamacher D, Singh NB, Van Dieen JH, Heller MO, Taylor WR. Kinematic measures for assessing gait stability in elderly individuals: a systematic review. J R Soc Interface. 2011;8 (65):1682–98.
66. Toebes MJ, Hoozemans MJ, Furrer R, Dekker J, van Dieen JH. Local dynamic stability and variability of gait are associated with fall history in elderly subjects. Gait Posture. 2012;36 (3):527–31.
67. van Schooten KS, Pijnappels M, Rispens SM, Elders P, Lips P, van Dieen JH. Ambulatory fall-risk assessment: amount and quality of daily-life gait predict falls in older adults. J Gerontol A Biol Sci Med Sci. 2015;70:608–15.
68. Hof AL, Gazendam MGJ, Sinke WE. The condition for dynamic stability. J Biomech. 2005;38(1):1–8.
69. Arvin M, Mazaheri M, Pijinappels M, Hoozemans MJM, Burger BJ, Verschueren SM, et al. Effects of narrow base gait on mediolateral balance control in young and older adults. J Biomech. 2016;43:1264–7.
70. Kang HG, Dingwell JB. Separating the effects of age and walking speed on gait variability. Gait Posture. 2008;27(4):572–7.
71. Bruijn SM, Van Impe A, Duysens J, Swinnen SP. White matter microstructural organization and gait stability in older adults. Front Aging Neurosci. 2014;6:104.
72. van Dieën JH, Pijnappels M, Bobbert MF. Age-related intrinsic limitations in preventing a trip and regaining balance after a trip. Saf Sci. 2005;43(7):437–53.
73. Patla AE, Prentice SD, Rietdyk S, Allard F, Martin C. What guides the selection of alternate foot placement during locomotion in humans. Exp Brain Res. 1999;128(4):441–50.
74. Weerdesteyn V, Nienhuis B, Mulder T, Duysens J. Older women strongly prefer stride lengthening to shortening in avoiding obstacles. Exp Brain Res. 2005;161(1):39–46.
75. Moraes R, Allard F, Patla AE. Validating determinants for an alternate foot placement selection algorithm during human locomotion in cluttered terrain. J Neurophysiol. 2007;98:1928–40.
76. Chen HC, Ashton-Miller JA, Alexander NB, Schultz AB. Effects of age and available response time on ability to step over an obstacle. J Gerontol. 1994;49(5):M227–33.
77. Patla AE, Rietdyk S, Martin C, Prentice S. Locomotor patterns of the leading and the trailing limbs as solid and fragile obstacles are stepped over: some insights into the role of vision during locomotion. J Mot Behav. 1996;28:35–47.
78. Patla AE, Vickers JN. Where and when do we look as we approach and step over an obstacle in the travel path? Neuroreport. 1997;8:3661–5.
79. McFadyen BJ, Prince F. Avoidance and accommodation of surface height changes by healthy, community-dwelling, young, and elderly men. J Gerontol. 2002;57:B166–74.
80. Persad CC, Giordani B, Chen HC, Ashton-Miller JA, Alexander NB, Wilson CS, et al. Neuropsychological predictors of complex obstacle avoidance in healthy older adults. J Gerontol B Psychol Sci Soc Sci. 1995;50(5):272–7.
81. Chen HC, Schultz AB, Ashton-Miller JA, Giordani B, Alexander NB, Guire KE. Stepping over obstacles: dividing attention impairs performance of old more than young adults. J Gerontol A Biol Sci Med Sci. 1996;51(3):M116–22.

82. Eng JJ, Winter DA, Patla AE. Strategies for recovery from a trip in early and late swing during human walking. Exp Brain Res. 1994;102:339–49.
83. Pijnappels M, Bobbert MF, van Dieen JH. How early reactions in the support limb contribute to balance recovery after tripping. J Biomech. 2005;38(3):627–34.
84. Pijnappels M, Bobbert MF, van Dieen JH. Push-off reactions in recovery after tripping discriminate young subjects, older non-fallers and older fallers. Gait Posture. 2005;21 (4):388–94.
85. Thelen DG, Muriuki M, James J, Schultz AB, Ashton-Miller JA, Alexander NB. Muscle activities used by young and old adults when stepping to regain balance during a forward fall. J Electromyogr Kinesiol. 2000;10(2):93–101.
86. Pavol MJ, Owings TM, Foley KT, Grabiner MD. Mechanisms leading to a fall from an induced trip in healthy older adults. J Gerontol A Biol Sci Med Sci. 2001;56(7):M428–37.
87. Pijnappels M, Bobbert MF, van Dieen JH. Contribution of the support limb in control of angular momentum after tripping. J Biomech. 2004;37(12):1811–8.
88. van der Burg JC, Pijnappels M, van Dieen JH. The influence of artificially increased trunk stiffness on the balance recovery after a trip. Gait Posture. 2007;26(2):272–8.
89. Mancini M, Horak FB. The relevance of clinical balance assessment tools to differentiate balance deficits. Eur J Phys Rehabil Med. 2010;46(2):239–48.
90. Schepens S, Sen A, Painter JA, Murphy SL. Relationship between fall-related efficacy and activity engagement in community-dwelling older adults: a meta-analytic review. Am J Occup Ther. 2012;66(2):137–48.
91. Yardley L, Beyer N, Hauer K, Kempen G, Piot-Ziegler C, Todd C. Development and initial validation of the Falls Efficacy Scale-International (FES-I). Age Ageing. 2005;34(6):614–9.
92. Kempen GI, Yardley L, van Haastregt JC, Zijlstra GA, Beyer N, Hauer K, et al. The short FES-I: a shortened version of the Falls Efficacy Scale-international to assess fear of falling. Age Ageing. 2008;37(1):45–50.
93. Delbaere K, Smith ST, Lord SR. Development and initial validation of the Iconographical Falls Efficacy Scale. J Gerontol A Biol Sci Med Sci. 2011;66(6):674–80.
94. Powell LE, Myers AM. The Activities-specific Balance Confidence (ABC) scale. J Gerontol A Biol Sci Med Sci. 1995;50A(1):M28–34.
95. Pasma JH, Engelhart D, Schouten AC, van der Kooij H, Maier AB, Meskers CG. Impaired standing balance: the clinical need for closing the loop. Neuroscience. 2014;267:157–65.
96. Huurnink A, Fransz DP, Kingma I, van Dieen JH. Comparison of a laboratory grade force platform with a Nintendo Wii Balance Board on measurement of postural control in single-leg stance balance tasks. J Biomech. 2013;46(7):1392–5.
97. Pajala S, Era P, Koskenvuo M, Kaprio J, Tormakangas T, Rantanen T. Force platform balance measures as predictors of indoor and outdoor falls in community-dwelling women aged 63–76 years. J Gerontol A Biol Sci Med Sci. 2008;63(2):171–8.
98. Blaszczyk JW, Lowe DL, Hansen PD. Ranges of postural stability and their changes in the elderly. Gait Posture. 1994;2(1):11–7.
99. Cofre Lizama LE, Pijnappels M, Rispens SM, Reeves NP, Verschueren SM, van Dieen JH. Mediolateral balance and gait stability in older adults. Gait Posture. 2015;42:79–84.
100. Arvin M, van Dieën JH, Faber GS, Pijnappels M, Hoozemans MJM, Verschueren SM. Hip abductor neuromuscular capacity: a limiting factor in mediolateral balance control in older adults? Clin Biomech. 2016;37:27–33.
101. Sawers A, Ting LH. Beam walking can detect differences in walking balance proficiency across a range of sensorimotor abilities. Gait Posture. 2015;41(2):619–23.
102. Weerdesteyn V, Laing AC, Robinovitch SN. The body configuration at step contact critically determines the successfulness of balance recovery in response to large backward perturbations. Gait Posture. 2012;35(3):462–6.
103. Toebes MJ, Hoozemans MJM, Dekker J, van Dieen JH. Effects of unilateral leg muscle fatigue on balance control in perturbed and unperturbed gait in healthy elderly. Gait Posture. 2014;40:215–9.

104. Hilliard MJ, Martinez KM, Janssen I, Edwards B, Mille M-L, Zhang Y, et al. Lateral balance factors predict future falls in community-living older adults. Arch Phys Med Rehabil. 2008;89(9):1708–13.
105. Sturnieks DL, Menant J, Delbaere K, Vanrenterghem J, Rogers MW, Fitzpatrick RC, et al. Force-controlled balance perturbations associated with falls in older people: a prospective cohort study. PLoS One. 2013;8(8), e70981.
106. Rabago CA, Dingwell JB, Wilken JM. Reliability and minimum detectable change of temporal-spatial, kinematic, and dynamic stability measures during perturbed gait. PLoS One. 2015;10(11):e0142083.
107. Tinetti ME. Performance-oriented assessment of mobility problems in elderly patients. J Am Geriatr Soc. 1986;34(2):119–26.
108. Berg K, Wood-Dauphinee S, Williams JI, Gayton D. Measuring balance in the elderly: preliminary development of an instrument. Physiother Can. 1989;41(6):304–11.
109. Podsiadlo D, Richardson S. The timed "Up & Go": a test of basic functional mobility for frail elderly persons. J Am Geriatr Soc. 1991;39(2):142–8.
110. Guralnik JM, Simonsick EM, Ferrucci L, Glynn RJ, Berkman LF, Blazer DG, et al. A short physical performance battery assessing lower extremity function: association with self-reported disability and prediction of mortality and nursing home admission. J Gerontol. 1994;49(2):M85–94.
111. Barry E, Galvin R, Keogh C, Horgan F, Fahey T. Is the Timed Up and Go test a useful predictor of risk of falls in community dwelling older adults: a systematic review and meta-analysis. BMC Geriatr. 2014;14:14.
112. Freiberger E, de Vreede P, Schoene D, Rydwik E, Mueller V, Frändin K, et al. Performance-based physical function in older community-dwelling persons: a systematic review of instruments. Age Ageing. 2012;41(6):712–21.
113. Regterschot GR, Zhang W, Baldus H, Stevens M, Zijlstra W. Sensor-based monitoring of sit-to-stand performance is indicative of objective and self-reported aspects of functional status in older adults. Gait Posture. 2015;41(4):935–40.
114. Shany T, Wang K, Liu Y, Lovell NH, Redmond SJ. Review: are we stumbling in our quest to find the best predictor? Over-optimism in sensor-based models for predicting falls in older adults. Healthc Technol Lett. 2015;2(4):79–88.
115. Iluz T, Weiss A, Gazit E, Tankus A, Brozgol M, Dorfman M, et al. Can a body-fixed sensor reduce heisenberg's uncertainty when it comes to the evaluation of mobility? Effects of aging and fall risk on transitions in daily living. J Gerontol A Biol Sci Med Sci. 2016;71(11):1459–65.
116. Lord SR, Menz HB, Tiedemann A. A physiological profile approach to falls risk assessment and prevention. Phys Ther. 2003;83(3):237–52.
117. Horak FB, Wrisley DM, Frank J. The Balance Evaluation Systems Test (BESTest) to differentiate balance deficits. Phys Ther. 2009;89(5):484–98.
118. Yingyongyudha A, Saengsirisuwan V, Panichaporn W, Boonsinsukh R. The Mini-Balance Evaluation Systems Test (Mini-BESTest) demonstrates higher accuracy in identifying older adult participants with history of falls than do the BESTest, berg balance scale, or timed up and go test. J Geriatr Phys Ther. 2016;39:64–70.
119. Peterka RJ. Sensorimotor integration in human postural control. J Neurophysiol. 2002;88 (3):1097–118.
120. Pasma JH, Engelhart D, Maier AB, Schouten AC, van der Kooij H, Meskers CG. Changes in sensory reweighting of proprioceptive information during standing balance with age and disease. J Neurophysiol. 2015;114(6):3220–33.
121. Chien JH, Eikema DJ, Mukherjee M, Stergiou N. Locomotor sensory organization test: a novel paradigm for the assessment of sensory contributions in gait. Ann Biomed Eng. 2014;42(12):2512–23.
122. Geerse DJ, Coolen BH, Roerdink M. Kinematic validation of a multi-kinect v2 instrumented 10-meter walkway for quantitative gait assessments. PLoS One. 2015;10(10):e0139913.
123. Kingma H, Gauchard GC, de Waele C, van Nechel C, Bisdorff A, Yelnik A, et al. Stocktaking on the development of posturography for clinical use. J Vestib Res. 2011;21(3):117–25.

Falls and Postural Stability in Older Individuals: Implications for Activities of Daily Living

17

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Abstract

The risk of falling is associated with aging-related motor and sensory declines. In fact, a higher incidence of falls has been observed in individuals of over 60 years who are known to have balance problems. The current knowledge regarding the mechanisms involved in the recovery of postural orientation and body equilibrium shows that the main changes occur as individuals become older and in those who are sedentary or are affected by some diseases. Extensive research and preventive efforts regarding the causes and consequences of falling have been conducted to better understand this issue. Fortunately, older individuals who practice regular physical activity present a healthy lifestyle and a lower risk and incidence of falling which may be associated with functioning of the postural control system. Recently, public health policies have been adopted by many countries in order to improve the quality of life of older individuals and reduce the high number of fallers among the elderly.

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17.1 Introduction

The aging process is characterized by individual constraints such as organic, physiological, and psychosocial, which lead to adaptation of even simple daily tasks. Brazil has one of the largest aging populations in the world, increasing from 14 million in 2000 (8.6 % of the population) to 20 million in 2010, and currently comprises 13 % of the population [1]. Health professionals who work with this population are concerned about the high incidence and risk of falls in older individuals and seek strategies to prevent the causes of this phenomenon [2].

A fall can be defined as the "unintentional displacement of the body to below the starting position, with correction of the disability in a timely manner, caused by multifactorial circumstances that compromise stability" [3]. The risk of falls has been associated with intrinsic and/or extrinsic characteristics of older adults. The intrinsic factors are related to physiological alterations during aging, such as decreased visual acuity; the abusive use of drugs/medication; chronic diseases such as hypertension, diabetes, arthritis, osteoarthritis, and osteoporosis, causing deficits in muscle strength, balance, and/or gait; and the psychological status, such as fear of falling, which affects, for instance, performance of the gait [4]. The extrinsic factors are environmental and social circumstances in which the older people are included, such as inadequate lighting, slippery surfaces, carpets without anti-slip coatings or with folds, obstacles when walking, no handrail support, high or narrow steps, and public pathways with holes or irregularities, among others [5].

Worldwide, about 424,000 individuals die each year as a result of falls, and a further 37.3 million fallers require medical attention [6]. Falling is the primary cause of accidental death in people over 65 years and is considered the most serious and frequent home accident, being responsible for 24 % of deaths in the older people. About 30 % of the older people fall each year, and this rate increases up to 40 % among individuals over 80 years and among older women up to 75 years. According to the Brazilian Health Ministry [7], the Public Health System has significant annual expenses related to fracture treatments in older individuals, principally related to hip fractures, which are the leading cause of morbidity and mortality in this population.

Fear or concern about recurrent falls is also associated with reduced mobility, loss of balance confidence, and activity restriction in older people. The evaluation is complex since it may involve physical, behavioral, and functional constraints [8]. For these reasons, it is important to identify possible causes and risk factors and propose several interventions in order to prevent falls in the older individuals.

Studies have shown that peripheral structure impairments, such as muscle weakness, loss of bone mineral density, sensorial constraints, nerve transmission

delays, and a slower reaction time are associated with older fallers [9, 10]. For instance, older individuals with impaired nerve transmission and sensory constraints take more time to initiate the postural response after an environmental disturbance. These delays may lead to a higher center of mass excursion which requires greater production of torque at the ankle and/or hip joints. As older individuals appear to present age-related muscle weakness, balance may not occur.

In addition to these peripheral constraints, recent studies have indicated that balance disorders in older individuals may also occur due to altered central mechanisms. In particular, the older people demonstrate a loss of central neurons and synaptic connections, which reduces processing speed and the ability to perform multiple activities simultaneously, predominantly those involving postural control [11, 12]. Some differences in brain structure (loss of global brain mass, a decline in volume of gray and white matter), biochemistry (differences in brain neurochemistry), and function (changes in brain recruitment patterns) have been observed [13] when comparing older adults with younger ones. Recent evidence has demonstrated that brain activation alters as age increases [14], mainly in areas such as the cerebellum, precuneus, occipital cortex, and basal ganglia, which seem to be associated with ankle muscle activation. These brain areas are increasingly activated in older but not in younger adults during ankle dorsiflexion movements. Since these cortico-cerebellar areas contribute to higher-order cognitive processes [15] and postural and balance control [16], these neural changes have been interpreted as compensatory responses to age-related deficits in sensorimotor control of the distal lower limbs [14].

Therefore, functional constraints in both the central and peripheral nervous systems seem to lead to inappropriate age-related postural responses to a constantly changing sensory environment, which could be associated with an increased risk of falling among this population. For instance, many fall events occur during the performance of activities of daily living (ADLs) which associate two or more tasks simultaneously. A dual task is defined as the simultaneous performance of a primary task, usually postural, in association with another task, denominated secondary, which could be a cognitive task (speaking, counting down, or speaking different names), a simple motor task (holding a glass of water or carrying a tray), a complex motor task (changing pocket coins), or cognitive/motor task (using a telephone while walking) [14, 15]. In general, the performance of dual tasks in an adequate manner involves dividing attention and making more effort to process postural control and sensory information [11, 12]. Older individuals, when performing a dual task, present changes in the maintenance of postural control due to competition between the secondary task and effectiveness of motor responses and sensory input, increasing the body sway to maintain balance [12]. One reason for these results might be the fact that both postural control and cognitive/motor tasks occur at the cortical level, so to perform these activities simultaneously implies competition for cortical processing, causing a reduction in the automatism of the older people to perform simultaneous activities [11].

Currently, many physical exercises/activities have been recommended to maintain appropriate levels of multiple operating systems, and these benefits are also extended to postural control. However, the underlying mechanisms involved in these sensorimotor changes are still poorly understood. The aim of this chapter is to demonstrate the changes in postural control over the years and the mechanisms involved in the recovery of body stability. First, the aspects involved in the functioning of the postural control system will be discussed, after which, a multifactorial approach to reduce falls and the related injuries will be addressed.

17.2 Aging and Postural Control

Historically, postural control was considered a summation of parallel and hierarchical reflex pathways [9, 17, 18]. Currently, it is understood as the complex interaction between multiple neural systems underlying two functional goals: postural orientation, the relative positioning of the body segments with respect to each other and to the environment, and postural equilibrium, the state in which all forces acting on the body are balanced [17].

Considering that postural control involves not only balance but also the ability to assume and maintain a desired orientation, every movement involves postural control [17]. Therefore, the accurate functioning of the postural control system allows us to properly interact with the environment. However, a decline in postural control performance is well documented in the older population [19, 20]. Furthermore, these age-related changes in the postural control system have been related to falling individuals [21, 22]. Piirtola and Era [22] suggested that parameters of mean speed and the root mean square of center of pressure (CP) could be used as predictors of the number of falls among the older people during upright stance. It has been found that older individuals with a history of falls present a larger sway area of CP than older individuals with no history of falls [21]. Therefore, understanding the mechanisms underlying these alterations in the postural control system is critical to fall prevention.

The postural control system involves different control mechanisms and behavioral strategies. Regarding the control mechanisms, the compensatory (feedback) and anticipatory (feedforward) postural adjustments can be mentioned. Compensatory postural adjustments operate on the principle of negative feedback [23]; thus afferent feedback is used to control the position of the body when the initial setting is disturbed [24]. On the other hand, anticipatory postural control is based on predictive control [23], since disturbances are anticipated using higher-order processing rather than simple negative feedback [24]. Thus, this mechanism involves muscle activation prior to the disturbance. In general, feedback postural control is available when subjects are faced with unpredictable externally generated disturbances, and feedforward postural control operates when subjects deal with disturbances generated by their own movements (self-motion) [25].

In the older people, the feedforward postural control mechanism is not used effectively [26, 27], and, for this reason, it seems that older individuals rely more on feedback control [26]. For Dault and coworkers [26], the inability of the older individuals to use feedforward control could be related to motor and sensory modifications associated with aging. This is a problem for the postural control system because both feedback and feedforward control mechanisms must operate in combination to achieve optimal motor control functioning [28]. If muscle activation begins prior to the disturbance, it is possible to minimize the perturbation to vertical posture, and, consequently, less feedback postural control is required. On the other hand, if these anticipatory adjustments are not effective, the disturbance will be greater, and compensatory postural adjustments may not be enough to recover balance.

Behavioral strategies are required to recover balance after disturbances, and these have been widely described [9, 17, 29]. The ankle strategy is the most commonly used and is the most useful for slow and/or small perturbations [17]. In this strategy, the ankle muscles alone act to recover the balance [30]. Thus, after a perturbation, the vertical alignment is altered and the ankle muscles activated in order to produce a torque at the ankle joint. When the ankle muscles are not enough to restore balance, the hip strategy is used [30]. The hip strategy is useful for rapid or large-amplitude disturbances [17] and consists of flexing the hip, moving the center of mass posteriorly, or extending the hip to move the center of mass anteriorly [30]. In addition, there is the stepping strategy, which is usually used when the ankle and hip strategies are inadequate to maintain the center of mass within the limits of stability [9]. The stepping strategy is useful for very large and/or fast perturbations [9, 17] but may also be observed in small perturbations in subjects who have not previously experienced the body position or received instruction on keeping the feet in place [17]. Age-related alterations have also been documented in these behavioral strategies [9, 19, 20]. Some studies have shown that older individuals switch strategies from ankle to hip to recover postural stability even after a small (feedback control) but unpredictable perturbation [10, 31, 32]. Taking into account that postural control functioning is not such efficient in older adults, disturbances of balance are generally more difficult to recover by ankle strategy, and so older people adopt a more conservative hip strategy.

Integration of sensory inputs is crucial to provide accurate information for postural control system functioning. For instance, sensory inputs are used to adjust self-initiated disturbances of stability (feedforward) and also to detect externally triggered disturbances (feedback) [23, 33]. As the body oscillates, a wide variety of receptors are activated and provide different types of inputs to the central nervous system (CNS) to generate proper muscular contractions which are dependent on the goals of the desired task in a specific environmental condition [33].

Generally, information from three sensory systems is available to the postural control system: visual, vestibular, and somatosensory [34, 35]. The visual system provides information about the position and movement of an object in space and the position and movement of the limbs in the environment and the whole body through

the eyes. The aging process reduces acuity, contrast sensitivity, depth perception, and rapid adaptation to changing depth [20, 33], which could make the older people more susceptible to falling. The somatosensory system provides information on the position of the body in space relative to the supporting surface and the position information and relative speed between body segments. The sensors of this system include muscle proprioceptors (Golgi tendon organ and muscle spindles) and joint and skin mechanoreceptors. The reduction in density and sensitivity of dermal mechanoreceptors, the stiffness of tissue, and degeneration of peripheral nerves may be related to decreased skin sensitivity over the years [20, 33]. The vestibular system provides information about the orientation and movement of the head, and the speed of information processing may reduce with age, also resulting in an increased risk of falls.

Falls in older individuals have been associated with multiple underlying causes, such as the sensory reweighting process [9]. Under normal conditions, the sensory systems provide redundant information to the CNS regarding maintenance of body position [35]. However, in constantly changing environments, some sensory cues may become more reliable than others in order to achieve the movement task. When this happens, visual, vestibular, and somatosensory inputs must be dynamically reweighted in order to optimize the control of postural stability [17]. Upweights and downweights of different sensory modalities appear to be linked in a nonlinear process [36, 37]. Some evidence suggests that the older individuals are not able to reweight sensory information appropriately [12, 20, 38]; however, it is still unclear whether such differences are due to central [38, 39] or peripheral mechanisms [40].

Recently, Toledo and Barela [40] found evidence that sensory reweighting in older adults might involve peripheral deterioration. In this study, older adults were submitted to several assessments in order to evaluate central and peripheral mechanisms. Peripheral mechanisms were evaluated by sensory (visual acuity, visual contrast sensitivity, tactile foot sensitivity, and detection of passive ankle motion) and motor (isometric ankle torque, muscular activity latency after stance perturbation) assessments, and central mechanisms were evaluated by postural control assessment (body sway during upright stance and induced by movement of a visual scene). The results revealed that less accurate sensitivity to passive ankle joint motion is related to larger body sway in sensory conflicting situations (induced by movement of a visual scene). Thus, there is evidence that if peripheral sensation is sufficiently intact, there is no difference in the sensory reweighting process between young and older adults (fallers and non-fallers) [41] suggesting that age-related changes in the reweighting process seem more likely to be due to peripheral mechanisms.

Despite these varied aspects, older adults engaged in physical exercise programs have demonstrated more effective performances of the postural control system in many daily activities. Besides the functional fitness improvements from regular physical exercise practice (strength, flexibility, coordination, balance, and resistance cardiovascular), active older individuals are constantly encouraged to interact with changing environments during exercise practicing which requires integration between useful sensory information and appropriate muscle contractions which

could benefit the postural control functioning. Based on these results, many professionals have considered the multifactorial aspects to develop exercise programs and prevent falls.

17.3 Physical Activity and Postural Control of Older Adults

The participation of older individuals in specific or multimodal exercise programs is an important element in the prevention and rehabilitation of multiple aspects associated with falls, such as muscle weakness, deterioration in postural control, and committed instability during locomotion [e.g., 42, 43]. Although prevention of falls has been the goal of several exercise programs, with different contributions to the health style of the older people [e.g., 44–46], understanding of the aspects which might actually change one or more factors associated with the risk of falling is still being investigated. Meta-analyses and reviews about this issue have pointed out, in many cases, incomplete methodologies, without strict definition of the groups or training methods (frequency, intensity, duration) applied in physical activity programs for the older adults which compromise the knowledge of consistent results on the effects of exercise in reducing falls in this population [45, 47, 48].

Despite this lack of information, overall results have shown that the practice of different types of physical exercise has beneficial effects on the health and quality of life of older individuals, mainly due to maintenance of their functional and neural capacity, independent mobility, and ability to carry out ADLs, which has been associated with a lower risk and fear of falling [13, 43, 49, 50]. Table 17.1 summarizes the main findings regarding the effects of exercise/physical activity training on the postural and functional parameters which are associated with the risk of falling in older individuals.

Strength/resistance, balance, and aerobic training are the most popular activities practiced by older adults and the most commonly prescribed among health professionals to prevent the risk of falling [47, 51]. The American Geriatrics Society and British Geriatrics Society Clinical Practice Guidelines for the prevention of falls in older persons recommend additional training for gait and coordination that should be performed 1–3 times a week, with different exercise intensities, and a duration of at least 12 months to achieve the necessary preventive benefits to reduce falls. Pau and collaborators [52] investigated the effects of exercise intensity (light or vigorous) on sitting and standing, static balance, and gait tasks in older adults. The results indicated that vigorous intensity physical activity may reduce body sway in static and dynamic situations and improve gait parameters in the older adults during the performance of ADLs. Light physical activities appear to be also beneficial but only in tasks involving static balance. These findings might be used to plan and to implement more specific and effective fall prevention programs for older people.

Particularly, the decline in postural control and increase in the incidence and risk of falling due to the aging process [21, 22] have led older adults to practice exercises in order to improve or maintain adequate levels of functioning of this

Table 17.1 Summarized exercise/physical activity training effects on postural and functional parameters

Author	Main objectives	Mean age	Intervention/evaluation	Main assessments	Main results
Perrin et al. [42]	Examine the influence of continuous PSA practice on postural control in the elderly	AA: 73.9 years IA: 71.9 years AI:67.9 years II: 73.7 years	Period during which the subjects were engaged in PSA	Posturographic tests (static, dynamic with a single and fast upward tilt, and dynamic with slow sinusoidal oscillations) and electromyography responses	The postural control performance decreased in the order AA > IA > AI > II
Hess et al. [56]	Evaluate the effect of 10-week, high-intensity strength training program	EG: 80.9 years CG: 82.5 years	EG: 10 weeks (3 days/week) I: high-intensity strength training program (quadriceps, hamstrings, tibialis anterior, gastrocnemius muscles)	Pre- and post-training evaluation Berg Balance Scale, TUG, Balance Confidence Scale	EG: improvements on Berg Balance Scale ($p = 0.030$), TUG ($p = 0.045$), Balance Confidence Scale ($p = 0.038$)
Colcombe et al. [50]	Examine whether aerobic fitness training of older adults can increase brain volume in regions associated with age-related declines	EG: 65.5 years CG:66.9 years YA: 18–30 years	EG: 6 months (3 days/week) I: aerobic exercises (cardiorespiratory fitness prescribed from HR) CG: 6 months (3 days/week) I: aerobic exercises (whole-body stretching and toning)	EG and CG: VO2 and MRI images YA: MRI images	EG: increases in brain volume (gray and white matter regions)
Bruin et al. [46]	Evaluate the additional effect of functional exercises on balance and lower extremity function in strength training of older adults	EG: 86.3 years CG: 86.5 years	EG: 12 weeks (2 days/week) I:strength and functional exercises (45 min + 30 min) CG: 12 weeks (2 days/week) I: strength exercises (45 min)	Tinetti Scale, physical performance tests (timed walking measure, chair stand test, tandem stand), balance test, maximal isometric knee extensor muscle force	EG: improvement on Tinetti test ($p = 0.026$), chair stand test ($p = 0.012$), balance test ($p = 0.009$)

Martinez-Amat et al. [54]	Evaluate the effect of a 12-week-specific proprioceptive training program on postural stability, gait, balance, and fall prevention	EG: 79.35 years CG: 77.0 years	EG: 12 weeks (2 days/week) CG: maintain activity levels and medication during the 12-week intervention period	Pre- and post-training evaluation, Tinetti Scale (postural stability, gait, balance, and risk of falling), Berg Balance Test	EG: improvements on ML body sway with EO ($p < 0.05$) and AP body sway with EC ($p < 0.01$) Romberg quotient about surface ($p < 0.05$) and speed ($p < 0.01$) Gait (Tinetti test): improved 14.66 % Balance (Berg test scores): 11.47 %
Lee et al. [44]	Evaluate effects of a multifactorial fall prevention program on fall incidence and physical function in community-dwelling older adults	EG: 75.4 years CG: 76.0 years	EG: 3-month multifactorial intervention program (8-week, 1 day/week + daily home exercise) I: health education, home hazards evaluation/modification, exercise training (strengthening, balance, cardiorespiratory endurance, and flexibility) CG: 3-month multifactorial intervention program I: health education brochures, referrals, and recommendations without direct exercise intervention	Pre- and post-training evaluation Fall incidence (1 fall during the 12-month follow-up), PPA risk index (vision, muscular strength, reaction time, postural sway, and proprioception), TUG test, GDS, FES, IPAQ	Cumulative 1-year fall incidence EG: 25.2 % CG: 27.6 % EG: PPA improvements on fall risk index, reaction time, postural sway with EO, TUG test, and GDS, especially for marked fall risk older individuals

(continued)

Table 17.1 (continued)

Author	Main objectives	Mean age	Intervention/evaluation	Main assessments	Main results
Pau et al. [52]	Assess the effects of vigorous and light physical activity on static balance, gait, and sit-to-stand (STS) tasks in a cohort of healthy older adults	VPA: 69.8 years LPA: 70.3 years	VPA: 12 weeks (3 days/week) I: 45 min with %HRR in the range 60–84 % (aerobic and anaerobic exercises) LPA: 12 weeks (3 days/week) I: %HRR below 40 % (static and dynamic exercises for posture and balance, joint and spine mobility)	Pre- and post-training evaluation Bipedal stances (EO and EC). Unipedal stance (left and right), Gait: spatiotemporal parameters, Sit-to-stand test	VPA: improvements on gait parameters ($p < 0.001$) and STS time ($p < 0.001$), reduced body sway with EC ($p < 0.01$) LPA: decreased swing phase duration ($p = 0.042$) and reduced body sway with EC ($p < 0.05$)
Gianoudis et al. [51]	Evaluate the effectiveness and feasibility of a multimodal exercise program in older adults	EG: 67.7 years CG: 67.2 years	EG: 12 months (3 days/week) – Osteo-cise exercise program I: HV-PRT, weight-bearing impact and challenging balance/mobility activities CG: to continue usual care and to take charge of own musculoskeletal health	Femoral neck and lumbar spine BMD, muscle strength, functional muscle power (Timed Stair Climb test), dynamic balance (Four Square Step and Sit-to-Stand tests), TUG, falls rate	EG: gains in femoral neck and lumbar spine BMD ($p < 0.05$), muscle strength ($p < 0.05$), functional muscle power ($p < 0.05$), and dynamic balance ($p < 0.01$)

PSA practice of physical and sporting activities, *AA* active-active group, *IA* inactive-active group, *AI* active-inactive group, *II* inactive-inactive group, *EG* experimental group, *CG* control group, *BBS* berg balance scale, *TUG* timed Up and Go, *I* intervention, *HR* peak heart rate, VO_2 maximal oxygen uptake, *MRI* magnetic resonance imaging, *min* minutes, *ML* medial lateral, *AP* anterior posterior, *EO* eyes open, *EC* eyes closed, *PPA* Physiological Profile Assessment, *IPAQ* International Physical Activity Questionnaire, *GDS* Geriatric Depression Scale, *FES* Fall Efficacy Scale-International, *VPA* vigorous physical activity, *LPA* light physical activity, *HRR* Heart Rate Reserve, *PRT* progressive resistance training, *HV* high velocity, *BMD* bone mineral density

system [51, 53]. These exercises are based on generating situations involving balance and postural orientation, increased mobility through the environment, shifts of movement direction, postural disturbances through sudden events, and sensory system manipulation [45, 52]. These situations lead the postural control system to produce effective and adaptable motor responses due to the disturbances suffered in these contexts. Prioli and collaborators [38] showed that sedentary older individuals presented larger body sway in response to changing visual stimulus compared to active older and young adults. In addition, active older adults are able to respond more quickly and accurately to situations of sensory conflict than their sedentary peers. These results suggest that exercise practice might improve the perceptual-motor skills of the older adults decreasing the risk of falls associated with the deterioration of the postural control system.

Many studies have compared the effectiveness of different types of exercise on the postural control performance of healthy older people. Stretching and multisensory training [54]; strength and flexibility exercises [47]; functional exercises [46]; tai chi, Pilates, and Yoga [53]; and, recently, cognitive training and training with virtual reality [49, 55] are some of these exercises. For instance, Hess et al. [56] showed that high-intensity strength training improved the postural stability levels and balance confidence of 80-year-old individuals after 10 weeks of training. Overall, all these studies show that the practice of more than one type of exercise appears to be effective for decreasing postural instability, improving functional tasks and ADL performance and, thus, preventing the risk of falls.

Therefore, involvement in physical exercise programs may be crucial to the maintenance and improvement of postural control performance over the years. The period in which this practice occurred in the life of the older individual is also worth noting, since older individuals who have always practiced physical and sporting activities present better postural control performance, followed by older individuals who have recently begun this practice and those who practiced physical/sport activity only at earlier ages [42]. Older individuals who have never practiced regular physical/sports activity present the worst performance of this system, demonstrating greater postural instability compared to the other groups [42].

Recent studies have shown that the benefits of this practice extend beyond the neuromuscular and sensory alterations observed in the peripheral nervous system [13]. Colcombe and collaborators [50] observed an increase in the brain volume of healthy subjects, aged 60–79 years, after only 6 months of regular aerobic training. There is evidence that exercise may increase the performance of prefrontal brain regions, enabling better control of movement and compensation in the functioning of brain areas directly affected by the aging process, which causes balance disorders, lower coordination, and slower movements, mainly during gait [13, 49, 50].

Unfortunately, there is no consensus in the literature about which exercise is better for the maintenance and/or improvement of the postural control system and, consequently, fall prevention. This is most likely because there are no specific exercises to prevent falls; however, it seems that regular physical exercise, regardless of the type, enables the older people to act on the environment and receive

sensory information in order to produce adequate muscular activation. Active older adults might be able to better calibrate their postural responses to environmental contexts.

Currently, many countries offer public policies to reduce the risk of falls in the older individuals due to the high costs of public health hospitalizations [57], reduced functional capacity postfracture, and increased dependence after falls. The main program for the prevention of falls recommended by the World Health Organization [58] comprises educational health, exercise practice, dual-task training, and identification of the risks of falling (individual, environmental, and social). There are many successful programs to prevent falls around the world, such as the Thematic Networks, the Prevention of Falls Network for Dissemination (ProFouND) in Europe, the National Institute for Health and Care Excellence (NICE) in the United Kingdom, the Canadian Falls Prevention Curriculum © supported by the Population Health Fund of the Public Health Agency of Canada, and the National Health Policy for the Elderly in Brazil. The programs propose taking part in a minimum of 50 h of these activities (equivalent to 2 h per week for 6 months, twice a week) for 12 weeks or more [59, 60]. Many programs have helped to reduce the incidence of falls and public costs from hospitalizations and morbidities in the elderly, ensuring the maintenance of functional capacity and quality of life for seniors.

17.4 Final Considerations

Postural control is essential for the majority of daily motor activities. Degradation of this system over the years may be related to the increased risk of falls in the older population. Neural activation and sensorimotor coupling seem to be altered in the older people which lead to poor postural responses to the environment. Exercise practice results in benefits for postural control in neurologically healthy older individuals, improving balance and reducing the risk of falls. Although the mechanisms underlying the improvements in the postural control system are not entirely known, many countries have developed fall prevention programs in order to reduce the incidence of falls and public costs from hospitalizations and morbidities in the older population.

References

1. Instituto Brasileiro de Geografia e Estatística. Censodemográfico 2013. Brasília, DF; 2013.
2. American Geriatrics Society and British Geriatrics Society. Summary of the Updated American Geriatrics Society/British Geriatrics Society clinical practice guideline for prevention of falls in older persons. J Am Geriatr Soc. 2011;59(1):148–57.
3. Silva RB, Eslick GD, Duque G. Exercise for falls and fracture prevention in long term care facilities: a systematic review and meta-analysis. J Am Med Dir Assoc. 2013;14(9):685–9.
4. Ganz SB, Peterson MG, Russo PW, Guccione A. Functional recovery after hip fracture in the subacute setting. HSS J. 2007;3(1):50–7.

5. Gregg EW, Pereira MA, Caspersen CJ. Physical activity, falls, and fractures among older adults: a review of the epidemiologic evidence. J Am Geriatr Soc. 2000;48(8):883–93.
6. Ishigaki EY, Ramos LG, Carvalho ES, Lunardi AC. Effectiveness of muscle strengthening and description of protocols for preventing falls in the elderly: a systematic review. Braz J Phys Ther. 2014;18(2):111–8.
7. Brasil. Health Ministry/Ministério da Saúde. Secretaria de Atenção à Saúde. Departamento de Atenção Básica. Política nacional de atenção básica / Ministério da Saúde, Secretaria de Atenção à Saúde, Departamento de Atenção à Saúde. Brasília: Ministério da Saúde; 2010.
8. Camargos FFO, Dias RC, Dias JMD, Freire MTF. Cross-cultural adaptation and evaluation of the psychometric properties of the Falls Efficacy Scale - International Among Elderly Brazilians (FES-I-BRAZIL). Braz J Phys Ther. 2010;14(3):237–43.
9. Horak FB, Shupert CL, Mirka A. Components of postural dyscontrol in the elderly: a review. Neurobiol Aging. 1989;10:727–38.
10. Mackey DC, Robinovitch SN. Mechanisms underlying age-related differences in ability to recover balance with the ankle strategy. Gait Posture. 2006;23:59–68.
11. Rankin JK, Woollacott MH, Shumway-Cook A, Brown LA. Cognitive influence on postural stability: a neuromuscular analysis in young and older adults. J Gerontol A Biol Sci Med Sci. 2000;55(3):M112–9.
12. Dault MC, Frank JS. Does practice modify the relationship between postural control and the execution of a secondary task in young and older individuals? Gerontology. 2004;50:157–64.
13. Seidler RD, Bernard JA, Burutolu TB, Fling BW, Gordon MT, Gwin JT, et al. Motor control and aging: links to age-related brain structural, functional and biochemical effects. Neurosci Biobehav Rev. 2010;34:721–33.
14. Linortner P, Jehna M, Johansen-Berg H, Matthews PM, Schmidt R, Fazekas F, et al. Aging associated changes in the motor control of ankle movements in the brain. Neurobiol Aging. 2014;35(10):2222–29.
15. Cavanna AE, Trimble MR. The precuneus: a review of its functional anatomy and behavioural correlates. Brain. 2006;129:564–83.
16. Ioffe ME, Chernikova LA, Ustinova KI. Role of cerebellum in learning postural tasks. Cerebellum. 2007;6(1):87–94.
17. Horak FB, MacPherson JM. Postural orientation and equilibrium. In: Rowell LB, Sherpherd JT, editors. Handbook of physiology, critical, comprehensive presentation of physiological knowledge and concepts. New York: Oxford American Physiological Society; 1996. p. 255–92.
18. Reed ES. Changing theories of postural development. In: Woolacott MH, Shumway-Cook A, editors. Development of posture and gait: across the life span. Columbia: University of South Carolina Press; 1989. p. 3–24.
19. Alexander NB. Postural control in older adults. J Am Geriatr Soc. 1994;42(1):93–108.
20. Woollacott MH, Shumway-Cook A, Nashner LM. Aging and posture changes in sensory organization and muscular coordination. Int J Aging Hum Dev. 1986;23:97–114.
21. Melzer I, Kurz I, Oddson LIE. A retrospective analysis of balance control parameters in elderly fallers and non-fallers. Clin Biomech. 2010;25:984–8.
22. Piirtola M, Era P. Force platform measurements as predictors of falls among older people: a review. Gerontology. 2006;52:1–16.
23. Houk JC, Lehman S. Control systems: feedback, feedforward, and adaptive strategies. In: Adelman G, editor. Encyclopedia of neuroscience. Boston: Birk Kauser; 1987. p. 275–7.
24. Gatev P, Thomas S, Kepple T, Hallett M. Feedforward ankle strategy of balance during quiet stance in adults. J Physiol. 1999;514(3):915–28.
25. Dufossé M, Hugon M, Massion J. Postural forearm changes induced by predictable in time or voluntary triggered unloading in man. Exp Brain Res. 1985;60:330–4.
26. Dault MC, Haart M, Geurts AC, Arts IM, Nienhuis B. Effects of visual center of pressure feedback on postural control in young and elderly healthy adults and in stroke patients. Hum Mov Sci. 2003;22:221–36.

27. Woollacott MH, Manchester DL. Anticipatory postural adjustments in older adults: are changes in response characteristics due to changes in strategy? J Gerontol. 1993;48(2): M64–70.
28. Seidler RD, Noll DC, Thiers G. Feedforward and feedback process in motor control. Neuroimage. 2004;22:1775–83.
29. Horak FB, Nashner LM. Central programming of postural movements: adaptation to altered support-surface configurations. J Neurophysiol. 1986;55(6):1369–81.
30. Winter DA. Human balance and postural control during standing and walking. Gait Posture. 1995;3(4):193–214.
31. Barrett RS, Lichtwark GA. Effect of altering neural, muscular and tendinous factors associated with aging on balance recovery using the ankle strategy: a simulation study. J Theor Biol. 2008;254:546–54.
32. Godoi D, Barela JA. Mecanismos de ajustes posturais feedback e feedforward em idosos. Rev Bras Ciênc Esporte. 2002;23(3):9–22.
33. Horak FB. Postural orientation and equilibrium: what do we need to know about neural control of balance to prevent falls? Age Ageing. 2006;35 Suppl 2:ii7–11.
34. Ghez C. Posture. In: Kandel ER, Schwartz JH, Jessel TM, editors. Principles of neural science. 3rd ed. Norwalk: Appleton & Lange; 1991. p. 596–607.
35. Nashner LM. Analysis of stance posture in humans. In: Towe AL, Luschei ES, editors. Motor coordination (Handbook of behavioral neurobiology. Vol. 5). New York: Plenum Press; 1981. p. 527–65.
36. Oie KS, Kiemel T, Jeka JJ. Multisensory fusion: simultaneous re-weighting of vision and touch for the control of human posture. Cognit Brain Res. 2002;14:164–76.
37. Polastri PF, Barela JA, Kiemel T, Jeka JJ. Dynamics of inter-modality re-weighting during human postural control. Exp Brain Res. 2012;223(1):99–108.
38. Prioli AC, Freitas Júnior PB, Barela JA. Physical activity and postural control in the elderly: coupling between visual information and body sway. Gerontology. 2005;51:145–8.
39. Teasdale N, Simoneau M. Attentional demands for postural control: the effects of aging and sensory reintegration. Gait Posture. 2001;14:203–10.
40. Toledo D, Barela JA. Age-related differences in postural control: effects of the complexity of visual manipulation and sensorimotor contribution to postural performance. Exp Brain Res. 2014;232:493–502.
41. Allison LK, Kiemel T, Jeka JJ. Multisensory reweighting of vision and touch is intact in healthy and fall-prone older adults. Exp Brain Res. 2006;175:342–52.
42. Perrin PP, Gauchard GC, Perrot C, Jeandel C. Effects of physical and sporting activities on balance control in elderly people. Br J Sports Med. 1999;33:121–6.
43. Ricci NA, Gonçalves D de FF, Coimbra IB, Coimbra AMV. Fatores associados ao histórico de quedas de idosos assistidos pelo programa de saúde da família. Saude e Soc. 2010;19 (4):898–909.
44. Lee HC, Chang KC, Tsauo JY, Hung JW, Huang YC, Lin SI. Effects of a multifactorial fall prevention program on fall incidence and physical function in community-dwelling older adults with risk of falls. Arch Phys Med Rehabil. 2013;94(4):606–15.
45. Fernandez-Arguelles EL, Rodriguez-Mansilla J, Antunez LE, Garrido-Ardila EM, Munhoz RP. Effects of dancing on the risk of falling related factors of healthy older adults: a systematic review. Arch Gerontol Geriatr. 2015;60(1):1–8.
46. Bruin ED, Murer K. Effect of additional functional exercises on balance in elderly people. Clin Rehabil. 2007;21:112–21.
47. Gschwind YJ, Kressig RW, Lacroix A, Muehlbauer T, Pfenninger B, Granacher U. A best practice fall prevention exercise program to improve balance, strength/ power, and psychosocial health in older adults: study protocol for a randomized controlled trial. BMC Geriatr. 2013;105:1–13.
48. Balzer K, Bremer M, Schramm S, Luhmann D, Raspe H. Fall prevention for the elderly. GMS Health Technol Assess. 2012;8:1–18.

49. Bherer L. Cognitive plasticity in older adults: effects of cognitive training and physical exercise. Ann N Y Acad Sci. 2015;1337:1–6.
50. Colcombe SJ, Erickson KI, Scalf PE, Kim JS, Prakash R, Mcauley E, et al. Aerobic exercise training increases brain volume in aging humans. J Gerontol. 2006;61A(11):1166–70.
51. Gianoudis J, Bailey CA, Ebeling PR, Nowson CA, Sanders KM, Hill K, et al. Effects of a targeted multimodal exercise program incorporating high-speed power training on falls and fracture risk factors in older adults: a community-based randomized controlled trial. J Bone Miner Res. 2014;29(1):182–91.
52. Pau M, Leban B, Collu G, Migliaccio GM. Effect of light and vigorous physical activity on balance and gait of older adults. Arch Gerontol Geriatr. 2014;59(3):568–73.
53. Baker AL, Bird ML, Talesvski J. Effect of pilates exercise for improving balance in older adults. Arch Phys Med Rehabil. 2015;96(4):715–23.
54. Martinez-Amat A, Hita-Contreras F, Lomas-Vega R, Caballero-Martinez I, Alvarez PJ, Martinez-Lópes E. Effects of 12-week proprioception training program on postural stability, gait and balance in older adults: a controlled clinical trial. J Strength Cond Res. 2013;27 (8):2180–88.
55. Duke G, Boersma D, Loza-Diaz G, Hassan S, Geisinger D, Suriyaarachchi P, Sharma A, Demontiero O. Effects of balance training using a virtual-reality system in older fallers. Clin Interv Aging. 2013;8:257–63.
56. Hess JA, Woollacott M. Effect of high-intensity strength-training on functional measures of balance ability in balance-impaired older adults. J Manipulative Physiol Ther. 2005;28:582–90.
57. Lockhart TE. An integrated approach towards identifying age-related mechanisms of slip initiated falls. J Electromyogr Kinesiol. 2008;18:205–17.
58. World Health Organization. WHO global report on falls prevention in older age. France; 2007.
59. National Institute For Health And Care Excellence. Falls: assessment and prevention of falls in older people. NICE clinical guideline 161 guidance. United Kingdom; 2013. nice.org.uk/cg161.
60. Sherrington C, Tiedemann A, Fairhall N, Close JC, Lord SR. Exercise to prevent falls in older adults: an updated meta-analysis and best practice recommendations. Public Health Bull. 2011;22(3-4):78–83.

Exercise to Maximise Postural Control and Reduce the Risk of Falls in Older Age

18

Anne Tiedemann and Catherine Sherrington

Abstract

The risk of experiencing a fall increases with advanced age, with at least one third of community-dwelling people aged 65 years and older falling annually. Falls can have a significant impact on an older person and commonly result in disability, loss of mobility, reduced quality of life and fear of falling. More serious fall-related consequences can include hip fracture, permanent disability, institutionalisation and death. Falling or loss of postural control results from a mismatch between an individual's physical ability and the demands of the environment and/or of the activity being undertaken. The physiological factors that underpin postural control decline with age, leading to a greater risk of falling. Some of these impairments, such as reduced muscle strength, can be improved with exercise, whereas others, such as poor vision, require other interventions. Targeted exercise is crucial for promoting functional independence and reducing the risk of falling in older age. Extensive research evidence strongly supports exercise as an effective single intervention to improve postural control and prevent falls in community-dwelling older people. Programmes that include exercises that challenge balance are proven to be more effective in preventing falls than programmes that do not challenge balance. Exercise should also be progressively challenging and ongoing and of sufficient dose to maximise its benefits in reducing falls. Uptake and long-term adherence to effective exercise programmes is more likely if older people's preferences and individual needs are considered. This chapter outlines the key elements of effective fall prevention exercise programmes for community-dwelling older people.

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Accidental falls • Aged • Exercise • Injury prevention • Postural control • Balance

18.1 Introduction

A fall is defined as "an unexpected event in which the participant comes to rest on the ground, floor, or lower level, as a result of a loss of balance" [1]. Falls affect a significant number of older people worldwide, with over one third of community-dwelling people aged 65 years and older falling one or more times every year. With the worldwide proportion of people aged 65 years and older expected to increase substantially in the years to come and the high resource burden resulting from many falls, this is a public health issue that demands attention.

Falls can have serious long-term consequences for the older person and their families. Falls may result in loss of mobility, ongoing disability, reduced quality of life, social isolation and fear of falling [2]. Most falls result in only minor injuries; however, more serious consequences can include hip fracture, permanent disability, institutionalisation and death.

Globally falls are the second leading cause of unintentional injury death, after road traffic injuries [3], and account for approximately 18 % of emergency hospital admissions in the 65+-year age group [4]. Fall rates in frailer older people, such as those residing in aged care facilities, are reported to be between 40 % and 56 % each year. Furthermore, people who have been hospitalised due to a fall-related injury are more likely to be discharged to a residential aged care facility or to require ongoing assistance at home from community services compared to older people hospitalised for non-fall-related conditions.

In addition to the substantial psychosocial and opportunity costs associated with falls, there is also a high economic cost. Falls are the leading cause of injury-related hospitalisation in people aged 65 years and over and account for 75 % of these hospitalisations. Furthermore, patients admitted to hospital as a result of a fall have a longer average length of stay than those admitted for other reasons. In 2013, the direct medical costs of fall-related injury among older adults in the USA were over $34 billion [5]. These substantial health-care costs will rise in significance if fall prevention is not given more priority in the area of preventive health care.

18.2 Postural Instability and Falls

Postural control is defined as the maintenance of the body's centre of gravity within its base of support during stance or voluntary movements and in response to postural perturbations [6]. Input to the central nervous system is received from the visual, vestibular and somatosensory systems to allow for adjustments to be made to body position to maintain an upright bodily posture.

A fall occurs when an individual's physiology cannot cope with an activity being undertaken and/or the environment in which the activity is being undertaken. Instability and falls in older people can result from impairment in any component of the postural control system, and poor balance is widely acknowledged as being a significant contributor to the increased incidence of falls in people aged 65 and older. If older people encounter a hazard that causes them to trip or slip, they are less likely to recover stability in time to prevent a fall because of age-related impairments in various physiological systems, such as vision, peripheral sensation, muscle strength, reaction time and vestibular sense. Furthermore, when one of the components of the postural control system is deficient, there is a greater reliance on the remaining components to help maintain balance, therefore increasing the demands on the sensorimotor systems and consequently increasing the likelihood of a fall.

It is widely accepted that the risk of falling increases with the number of risk factors present. Falls are not random events and can be predicted by assessing certain risk factors [7, 8]. Some of these risk factors (e.g. reduced muscle strength and impaired balance and gait) can be improved with exercise [9], whereas others (e.g. poor vision, psychoactive medication use) require other intervention approaches.

18.3 Interventions to Prevent Falls

While falls are quite common in older age, a large body of well-conducted research shows that falls are not an inevitable consequence of ageing. Several interventions have been identified as effective strategies to reduce the risk and rate of falls among community-dwelling older people [10].

Clinical trials strongly support exercise as an effective single intervention to prevent falls in community dwellers [10, 11]. Exercise is effective in reducing the risk of falling for both general community dwellers [12] and people at a high risk of falls [13]. The recent Cochrane review of interventions to prevent falls in community-dwelling older people concluded that exercise can reduce the risk and rate of falls in older people by between 15 and 32 %, depending on the type of programme and measures used to assess effectiveness [10].

Multifaceted interventions can also prevent falls [10], and many of these include exercise. Several other single interventions have been demonstrated to prevent falls in randomised controlled trials: home safety modifications in people who have previously fallen [14]; reducing intake of psychoactive medications [15]; enhanced podiatry [16]; cataract surgery [17]; the use of single lens rather than bi-, tri- or multifocal glasses for outdoor mobility [18]; and the insertion of cardiac pacemakers for the small proportion of people who experience blackouts and are diagnosed with the cardio-inhibitory form of carotid sinus hypersensitivity [19]. Impaired muscle strength and poor postural control are known to significantly increase fall risk, making exercise an obvious choice as a fall prevention intervention since exercise is known to impact on these risk factors.

18.4 Effective Fall Prevention Exercise

A systematic review and meta-analysis of fall prevention exercise programmes by our group included 54 randomised controlled trials [11]. The systematic review and meta-analysis aimed to determine the specific fall prevention exercise programme characteristics that were most strongly associated with fall prevention benefits. The results indicated that the optimal exercise programme for preventing falls is one that contains the following three elements: exercises that provides a high challenge to balance, exercise of a high dose and no walking programme. This combination of factors resulted in a significant 38 % reduction in the rate of falls (adjusted pooled rate ratio = 0.62, 95 % confidence interval 0.54–0.73). In comparison, programmes that included a high challenge to balance and a high dose but also included a walking programme resulted in a lower 21 % reduction in the rate of falls (adjusted pooled rate ratio = 0.79, 95 % confidence interval 0.70–0.89). Effective challenge to balance is provided with exercises that are conducted while standing in which participants aim to (a) stand with their feet closer together or on one leg, (b) minimise the use of their hands to assist balance and (c) practise controlled movements of the body's centre of mass.

The apparent detrimental effect on fall rates of the inclusion of walking programmes may be due to (a) increased exposure to hazards that increase fall risk when walking, (b) walking taking time away from high-level balance training or (c) confounding of the results as walking programmes were more likely to be prescribed in high-risk populations (e.g. in residential care) and the beneficial effects of exercise in this population are less marked. While current evidence suggests that walking is not an effective fall prevention strategy in some studies, there are other benefits of walking programmes for older people [20, 21]. We suggest walking training be included in a fall prevention programme as long as balance training is the main programme focus, and walking is additional to it. However, high-risk individuals should not be prescribed brisk walking programmes due to the increased risk of falls with this activity [22]. The Otago Exercise Programme [23] (described below) can be effective in preventing falls and includes the prescription of a walking programme if the exercise provider considers the individual participant to be safely able to undertake such a programme. We suggest that this approach be used for participants in all fall prevention exercise programmes.

When selecting the initial level of balance exercise difficulty, there should be consideration of the capabilities of the individual and procedures to ensure safety. Once the older person has mastered a balance exercise in a stable and confident manner without the need for upper limb support, the exercise should be progressed to increase the challenge to balance. Methods to increase the intensity and effectiveness of balance-challenging exercises over time include (a) using progressively more difficult postures with a gradual reduction in the base of support (e.g. two-legged stand, semi-tandem stand, tandem stand, one-legged stand), (b) using dynamic movements that perturb the centre of gravity (e.g. tandem walk, circle turns, leaning and reaching activities, stepping over obstacles),

(c) specific resistance training for postural muscle groups (e.g. heel stands, toe stands, hip abduction with added weights to increase intensity, unsupported sit to stand practice) and/or (d) reducing sensory input (e.g. standing with eyes closed, standing/walking on an unstable surface such as foam mats) [24]. Further challenge can be provided by the use of dual tasks, such as combining a memory task with a gait training exercise or a hand-eye coordination activity with a balance task. Some examples of exercises which challenge balance and are appropriate for older people to perform are included in Table 18.1. Information is also included about methods for progression of exercise intensity over time.

The systematic review and meta-analysis conducted by Sherrington and colleagues [11] also identified exercise dose as a factor that impacted on fall prevention programme effectiveness, where a higher dose of exercise (e.g. a dose

Table 18.1 Balance-challenging exercises suitable for prescription to older people and methods to progress exercise intensity

Baseline exercise	Progression
Graded reaching in standing	Use narrower foot placement Reach further and in different directions Reach down to a stool or the floor Reach for heavier objects Stand on a softer surface, e.g. foam mat Step while reaching
Stepping in different directions	Take longer or faster steps Step over obstacle Pivot on non-stepping foot
Walking practice	Decrease base of support, e.g. tandem walk Increase step length and speed Walk on different surfaces Walk in different directions Walk around and over obstacles Heel-and-toe walking
Sit to stand	Don't use hands to push off Lower chair height Use a softer chair Add weight (vest or belt)
Heel raises	Decrease hand support Hold raise for longer Perform on one leg at a time Add weight (vest or belt)
Step-ups—forward and lateral	Decrease hand support Increase step height Add weight (vest or belt)
Half squats sliding down a wall	Decrease hand support Hold the squat for longer Move a short distance away from the wall Add weight (vest or belt) Perform on one leg at a time

Adapted from Tiedemann et al. [25]

of more than 50 h of exercise, typically 2 × 1 h sessions per week for 6 months) was associated with a significantly greater fall prevention effect. It is likely that exercise needs to be ongoing to have a lasting effect on fall rates. Therefore, programmes should offer ongoing exercise or encourage people to undertake ongoing exercise at the end of a short-term formal programme as recommended by the American College of Sports Medicine.

18.5 Exercise-Based Fall Prevention Programmes

Programmes that include exercises that challenge balance are more effective in preventing falls than programmes that do not challenge balance. Undertaking specific balance-challenging exercise on a regular basis for a sustained period of time is essential to significantly reduce fall risk. Some examples of effective fall prevention exercise programmes are detailed below.

18.5.1 Otago Exercise Programme of Home-Based Strength and Balance Exercise

The Otago Exercise Programme of home-based strength and balance exercise was developed by researchers in New Zealand in 2001 [26]. It was specifically designed to prevent falls and consists of a set of leg muscle strengthening and balance training exercises progressing in difficulty and a walking plan. In four well-designed randomised controlled trials, it has been shown to be effective in reducing the number of falls and fall-related injuries by 35 % in community-dwelling older adults and had the greatest impact in those aged 80 and older [23].

The Otago Exercise Programme is designed to be delivered during five home visits by a trained instructor, commonly a nurse or physiotherapist. The exercises are individually tailored to the participants' current ability and can be prescribed with the use of ankle cuff weights for the strengthening exercises where appropriate. The exercises take about 30 min to complete and are recommended to be undertaken three times a week. In addition, participants are advised to go for a walk at least twice a week.

Otago Exercise Programme participants receive a booklet with instructions for each exercise prescribed and ankle cuff weights (starting at 1 kg). To promote adherence participants are also encouraged to complete an exercise diary to record the days they undertake the programme. The instructor also provides support and guidance via telephone contact to them each month between home visits. Follow-up home visits are recommended every 6 months.

18.5.2 The Lifestyle-Integrated Functional Exercise (LiFE) Programme

The Lifestyle-integrated Functional Exercise (LiFE) programme was developed by researchers in Australia in 2012 [27] and uses a unique approach of integrating exercise into everyday activities to prevent falls in older people. In a randomised controlled trial involving 317 people aged 70 years and older, the LiFE programme reduced the rate of falls after 12 months by 31 % compared with the control group [27].

The LiFE programme was developed to provide an alternative to typical structured exercise programmes, giving choice for older people who may find such exercise programmes unappealing or unrealistic. It involves embedding balance and lower limb strength training into daily routines such as climbing stairs, stepping over objects and moving from sitting to standing. Rather than prescribing a set of exercises to be carried out at set times several times a week, the LiFE programme activities are recommended to be carried out whenever the opportunity arises during the day.

The LiFE programme is delivered by a trained instructor during five home visits, with two additional booster visits and two follow-up phone calls over a 6-month period. Participants also receive a printed workbook that outlines the activities and how to progress their difficulty over time.

The participant and instructor plan how to embed the programme activities into the participant's daily routine. Each programme is unique as it is tailored to what the participant routinely does and how they decide to embed the activities into their usual daily routine. For example, standing on one leg to challenge balance while waiting for the kettle to boil or squatting down to pick things up from floor level rather than bending in order to strengthen thigh muscles.

18.5.3 Tai Chi

Tai Chi is an ancient form of exercise with its origins in China that is usually performed in a group. There are many different forms of Tai Chi, but all are linked by three main underlying concepts: (a) movement control, where Tai Chi movements are slow, smooth and continuous; (b) weight transference, where Tai Chi movements focus on transferring weight with each step; and (c) integration of mind and body, where Tai Chi movements are practised with a calm and relaxed mind to aid in the relaxation of muscles and joints.

A 2012 Cochrane review of interventions to prevent falls in community-dwelling older people [10] found that Tai Chi was effective in reducing the risk and rate of falls in older people by around 30 %. A sub-group analysis also determined that Tai Chi appears to be more effective in general community-dwelling older people rather than those who are at high risk of falling (i.e. those who have previously fallen).

18.5.4 Other Group-Based Exercise Programmes

Several other group-based fall prevention exercise programmes have been shown to be effective in the research context. Barnett and colleagues conducted a randomised controlled trial among 163 people aged 65 years who were identified as being at risk of falling [28]. The intervention consisted of weekly hour-long group-based exercise classes designed to improve balance, coordination, aerobic capacity and muscle strength. The content of the classes focussed on improving balance during functional activities such as transfers, reaching and stepping. Participants were also encouraged to carry out the exercises at home. The exercises were progressed in complexity and speed over the 12-month study period. At the end of the 12-month period, the intervention group had a 40 % lower rate of falls compared with the no exercise control group.

Similarly, Lord and colleagues [29] conducted a randomised controlled trial among 551 people aged 62 years and older living in retirement villages in Australia. They aimed to determine the effect of participation in an exercise programme on physical functioning and falls. The exercise programme ran for 12 months and consisted of a twice-weekly group-based programme that focussed on reducing fall risk by improving strength, balance, coordination, speed and gait. The exercises were also designed to improve the participants' ability to carry out activities of daily living such as stair climbing, postural transfers and turning and reaching. The hour-long exercise groups were led by trained instructors, and most of the exercises were undertaken while weight bearing, with a major emphasis on social interaction and enjoyment. At the end of the 12-month period, the intervention group had a 22 % lower rate of falls compared with the control group.

Exercises that target strengthening of the lower limb muscle groups [23, 27, 28] and muscles of the ankles and feet [16] have been included in successful fall prevention programmes. As with any effective exercise programme, it is crucial to progress the intensity of the resistance used over time to ensure muscles are overloaded and gains in strength are achieved.

Balance involves anticipatory and ongoing postural adjustments and is thus a coordination task. Activities such as aerobics, tennis and lawn bowls have not been evaluated for their effectiveness in preventing falls, but as they require coordination practice, they are likely to be beneficial in maintaining balance abilities for middle-aged people and abler older people. For older people with poorer postural control, these activities may increase risk of falling so individually prescribed exercises which safely challenge balance may be more appropriate.

There is emerging evidence that yoga-based exercise may be beneficial for preventing falls. A recent systematic review and meta-analysis by the chapter authors [30] identified the effect of yoga on balance and mobility in people aged 60 years and over. Six randomised controlled trials were included, and the results showed that yoga interventions resulted in small improvements in balance and medium improvements in mobility (standardised mean differences of 0.40 and 0.50, respectively). Further research is required to determine whether yoga-related improvements in balance and mobility translate to prevention of falls in older people.

18.6 Implementation Considerations

People aged 85 years and over and those with chronic disease or functional limitations are at a substantially increased risk of falls. Exercise programmes for these groups must be prescribed carefully to ensure they do not cause falls. There is evidence that falls can be prevented in people at increased risk of falls through well-designed exercise programmes. Further research is needed to determine the optimal approach for preventing falls in people with specific medical conditions such as Parkinson's disease and stroke.

Our recently published trial of an individualised home-based fall prevention exercise programme delivered to people recently discharged from hospital [31] found that the intervention significantly improved performance-based mobility but also significantly increased the rate of falls. This result suggests that unsupervised home exercise alone cannot be recommended as a fall prevention intervention for this high-risk group of older people. Further research is required to determine the optimal fall prevention programme for effectively preventing falls in older people recently discharged from hospital.

Exercise guidelines recommend an extended cool-down period after physical activity for older people, to reduce the chance of hypotension, syncope or arrhythmias during the postexercise recovery period. Certain medical conditions require additional precautions for exercise to be undertaken safely and effectively. For example, people with osteoarthritis may require analgesia [32], people with asthma [33] and heart disease/angina [34] may require the use of medication, and people with diabetes may require additional carbohydrate prior to or during exercise [35]. Dehydration is more likely to occur in older people taking diuretics, so fluid intake is recommended before, during and after exercise.

Despite the evidence promoting its benefits, a major limitation of exercise as a public health intervention is low rates of participation, with reported participation rates as low as 6 % for balance training exercise among older people [36]. Consideration of factors that maximise uptake and participation in exercise programmes by older people is important, such as moderate-duration activity, programme accessibility and convenience, emphasis on social aspects, strong leadership and individually tailored exercise. Furthermore, it has been demonstrated that fall prevention programmes that are labelled with a positive health message, such as "healthy ageing," are likely to be more acceptable to older people than those which describe themselves explicitly as "fall prevention" programmes.

18.7 Conclusion

In summary, the research evidence shows that in order to improve postural control and prevent falls in older age, exercise programmes need to target three key elements: gradual reduction in the base of support (move feet closer together in standing or stand on one leg), gradual reduction in the use of upper limb

support to assist balance (reduce the amount of holding on to stable objects) and practice of controlled movements of the body's centre of mass (move to limits of stability).

A range of exercise programmes which target balance and provide ongoing exercise are effective in preventing falls. These include the Otago Exercise Programme of home-based balance and strength training [23], the LiFE programme of embedded balance and strength training into habitual daily routines [27], group-based Tai Chi [37, 38] and other group-based balance and strengthening exercises [28, 29]. As exercises that focussed on muscles of the ankles and feet were considered to be important components of a successful podiatry fall prevention trial [16], such exercises could also be considered for inclusion in general exercise programmes and especially for the high proportion of older people with foot problems. Programme design should meet the needs and abilities of the target population to ensure it provides exercise that is challenging yet safe.

References

1. Lamb SE, Jorstad-Stein EC, Hauer K, Becker C. Prevention of Falls Network Europe and Outcomes Consensus Group. Development of a common outcome data set for fall injury prevention trials: the Prevention of Falls Network Europe consensus. J Am Geriatr Soc. 2005;53:1618–22.
2. Lord SR, Sherrington C, Menz HB, Close JCT. Falls in older people: risk factors and strategies for prevention. 2nd ed. Cambridge: Cambridge University Press; 2006.
3. World Health Organization. WHO global report on falls prevention in older age. 2007.
4. Bell A, Talbot-Stern J, Hennessy A. Characteristics and outcomes of older patients presenting to the emergency department after a fall: a retrospective analysis. Med J Aust. 2000;173 (4):179–82.
5. Stevens JA, Corso PS, Finkelstein EA, Miller TR. The costs of fatal and non-fatal falls among older adults. Inj Prev. 2006;12(5):290–5.
6. Winter DA. ABC (Anatomy, biomechanics and control) of balance during standing and walking. Ontario: Waterloo Biomechanics; 1995.
7. Ganz DA, Bao Y, Shekelle PG, Rubenstein LZ. Will my patient fall? JAMA. 2007;297:77–86.
8. Tiedemann A, Shimada H, Sherrington C, Murray S, Lord SR. The comparative ability of eight functional mobility tests for predicting falls in community-dwelling older people. Age Ageing. 2008;37:1–6.
9. Howe TE, Rochester L, Jackson A, Banks PMH, Blair VA. Exercise for improving balance in older people. Cochrane Database Syst Rev. 2007;(4):CD004963. doi:10.1002/14651858.CD004963.pub2.
10. Gillespie LD, Robertson MC, Gillespie WJ, Sherrington C, Gates S, Clemson LM, et al. Interventions for preventing falls in older people living in the community. Cochrane Database Syst Rev. 2012;(9):CD007146. doi:10.1002/14651858.CD007146.pub3.
11. Sherrington C, Tiedemann A, Fairhall N, Close JCT, Lord SR. Exercise to prevent falls in older adults: an updated meta-analysis and best practice recommendations. N S W Public Health Bull. 2011;22:78–83.
12. Woo J, Hong A, Lau E, Lynn H. A randomised controlled trial of Tai Chi and resistance exercise on bone health, muscle strength and balance in community-living elderly people. Age Ageing. 2007;36(3):262–8.

13. Skelton D, Dinan S, Campbell M, Rutherford O. Tailored group exercise (Falls Management Exercise – FaME) reduces falls in community-dwelling older frequent fallers (an RCT). Age Ageing. 2005;34(6):636–9.
14. Clemson L, Mackenzie L, Ballinger C, Close JCT, Cumming RG. Environmental interventions to prevent falls in community-dwelling older people: a meta-analysis of randomized trials. J Aging Health. 2008;20(8):954–71.
15. Campbell AJ, Robertson MC, Gardner MM, Norton RN, Buchner DM. Falls prevention over 2 years: a randomized controlled trial in women 80 years and older. Age Ageing. 1999;28 (6):513–8.
16. Spink MJ, Menz HB, Fotoohabadi MR, Wee E, Landorf KB, Hill KD, et al. Effectiveness of a multifaceted podiatry intervention to prevent falls in community dwelling older people with disabling foot pain: randomised controlled trial. Br Med J. 2011;342:d3411.
17. Harwood RH, Foss AJ, Osborn F, Gregson RM, Zaman A, Masud T. Falls and health status in elderly women following first eye cataract surgery: a randomised controlled trial. Br J Ophthalmol. 2005;89(1):53–9.
18. Haran MJ, Cameron ID, Ivers RQ, Simpson JM, Lee BB, Tanzer M, et al. Effect on falls of providing single lens distance vision glasses to multifocal glasses wearers: VISIBLE randomised controlled trial. BMJ. 2010;340:c2265.
19. Kenny RAM, Richardson DA, Steen N, Bexton RS, Shaw FE, Bond J. Carotid sinus syndrome: a modifiable risk factor for nonaccidental falls in older adults (SAFE PACE). J Am Coll Cardiol. 2001;38(5):1491–6.
20. Nelson ME, Rejeski WJ, Blair SN, Duncan PW, Judge JO, King AC, et al. Physical activity and public health in older adults: recommendation from the American College of Sports Medicine and the American Heart Association. Med Sci Sports Exerc. 2007;39:1435–45.
21. Murphy MH, Nevill AM, Murtagh EM, Holder RL. The effect of walking on fitness, fatness and resting blood pressure: a meta-analysis of randomised, controlled trials. Prev Med. 2007;44:377–85.
22. Ebrahim S, Thompson PW, Baskaran V, Evans K. Randomized placebo-controlled trial of brisk walking in the prevention of postmenopausal osteoporosis. Age Ageing. 1997;26:253–60.
23. Robertson MC, Campbell AJ, Gardner MM, Devlin N. Preventing injuries in older people by preventing falls: a meta-analysis of individual-level data. J Am Geriatr Soc. 2002;50:905–11.
24. Chodzko-Zajko WJ, Proctor DN, Fiatarone Singh MA, Minson CT, Nigg CR, Salem GJ, et al. ACSM position stand on exercise and physical activity for older adults. Med Sci Sports Exerc. 2009;41:1510–30.
25. Tiedemann A, Sherrington C, Close JCT, Lord SR. Exercise and Sports Science Australia Position Statement on exercise and falls prevention in older people. J Sci Med Sport. 2011;14 (6):489–95.
26. Gardner MM, Buchner DM, Robertson MC, Campbell AJ. Practical implementation of an exercise-based falls prevention programme. Age Ageing. 2001;30:77–83.
27. Clemson L, Singh MAF, Bundy A, Cumming RG, Manollaras K, O'Loughlin P, et al. Integration of balance and strength training into daily life activity to reduce rate of falls in older people (the LiFE study): randomised parallel trial. BMJ. 2012;345:e4547.
28. Barnett A, Smith B, Lord SR, Williams M, Bauman A. Community-based group exercise improves balance and reduces falls in at-risk older people: a randomised controlled trial. Age Ageing. 2003;32(4):407–14.
29. Lord SR, Castell S, Corcoran J, Dayhew J, Matters B, Shan A, et al. The effect of group exercise on physical functioning and falls in frail older people living in retirement villages: a randomized, controlled trial. J Am Geriatr Soc. 2003;51(12):1685–92.
30. Youkhana S, Dean CM, Wolff M, Sherrington C, Tiedemann A. Yoga-based exercise improves balance and mobility in people aged 60 and over: a systematic review and meta-analysis. Age Ageing. 2016;45:21–9.

31. Sherrington C, Lord SR, Vogler CM, Close JCT, Howard K, Dean CM, et al. A post-hospital home exercise program improved mobility but increased falls in older people: a randomised controlled trial. PLoS One. 2014;9(9), e104412.
32. American Geriatrics Society Panel on Exercise and Osteoarthritis. Exercise prescription for older adults with osteoarthritis pain: consensus practice recommendations. J Am Geriatr Soc. 2001;49:808–23.
33. National Asthma Council of Australia. Asthma management handbook. Melbourne; 2006.
34. Briffa T, Maiorana A, Allan R, et al. On behalf of the Executive Working Group and National Forum Participants: National Heart Foundation of Australia physical activity recommendations for people with cardiovascular disease. National Heart Foundation of Australia: Sydney, Australia; 2006.
35. Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C, White RD. Physical activity/ exercise and type 2 diabetes: a consensus statement from the American Diabetes Association. Diabetes Care. 2006;29:1433–8.
36. Merom D, Pye V, Macniven R, van der Ploeg H, Milat A, Sherrington C, et al. Prevalence and correlates of participation in fall prevention exercise/ physical activity by older adults. Prev Med. 2012;55(6):613–7.
37. Li F, Harmer P, Fisher KJ, McAuley E, Chaumeton N, Eckstrom E, et al. Tai Chi and fall reductions in older adults: a randomized controlled trial. J Gerontol A Biol Sci Med Sci. 2005;60:187–94.
38. Voukelatos A, Cumming RG, Lord SR, Rissel C. A randomized, controlled trial of tai chi for the prevention of falls: the Central Sydney tai chi trial. J Am Geriatr Soc. 2007;55:1185–91.

Interlateral Asymmetries of Body Balance Control Resulting from Cerebral Stroke

19

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Abstract

This review is dedicated to synthesize recent findings on interlateral asymmetries of body balance control provoked by uni-hemispheric cerebral stroke, a neural lesion more frequently associated with older individuals. I introduce this theme by exposing results on the main deficits of balance control resulting from stroke-related cerebral lesions. In the main sections, the reviewed findings suggest that reduced balance stability due to stroke results from the impaired capacity to exert muscular forces in a timely and coordinated manner with the paretic leg, associated with low compensatory action of the nonparetic leg, to prevent increased body oscillation. The discussed findings lead to the conclusion that interlateral asymmetry is magnified, with balance control being more critically impaired, when the right cerebral hemisphere is lesioned. Evidence is presented for propositions that right hemisphere specialization could be based on processing of sensory information, and for the alternative proposition that hemispheric specialization is founded on preeminence for impedance control.

Keywords

Posture asymmetry • Weight-bearing asymmetry • Cerebrovascular accident • Right hemisphere specialization • Stance stability • Quiet balance • Perturbed balance • Hemiparesis • Impedance control

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19.1 Introduction

Upright postural control in healthy individuals is characterized by predominantly interlateral symmetric behavior, with weight-bearing and adjustments for maintenance of body equilibrium distributed similarly between the right and left legs. Investigation of unipedal stance has shown that the shared responsibilities of balance control between the two legs are based on symmetric capabilities to deal with requirements of quiet stance and recovery of body equilibrium following a perturbation. Evidence for interlateral symmetric balance control has been presented from research on experts in activities imposing a high demand on body equilibrium. Mertz and Docherty [1], for example, evaluated dancers when landing with either leg on a force plate from dance-specific jumps, requiring anticipatory and corrective movements with the supporting leg. Analysis of ground reaction forces and postural stability indicated symmetric performance between the two legs in the performance of those movements. Further investigation has also found similar performance between the legs for balance control in quiet stance in field hockey athletes [2]. Soccer players represent a particularly interesting group for evaluation of interlateral asymmetry of balance control, given that very fast mobilization of the preferred leg for kicking movements might be expected to induce a specialization of balance control of the supporting leg. Results by Teixeira et al. [3], however, were contradictory to this expectation. Analysis of unipedal balance, both in quiet posture and in tasks requiring fast oscillation of the opposite leg in different directions, showed symmetric profiles between center of pressure (CoP) excursion under each foot. An additional finding of interest in these results was reduced CoP excursion values for either leg in comparison with non-soccer players. This result suggests that the equilibrium gains by training of soccer skills are shared in similar proportions between the two legs, promoting interlateral symmetric performance (see also [4]). The reported performance symmetry between the legs in balance control in healthy young individuals is broken in different diseases more frequently observed in older individuals, which is particularly evident in unilateral cerebral damage by stroke. In these cases, movements of the hemibody contralateral to the lesioned cerebral hemisphere are more impaired (hemiparesis) than movements of the ipsilateral hemibody, leading to functional asymmetry between the two legs for regulation of body equilibrium. In the next sections, I review the literature about interlateral asymmetry between the paretic and nonparetic legs in upright balance control as a result of unilateral cerebral stroke, and evidence for associated hemispheric specialization.

19.2 Impairment of Postural Control by Cerebral Stroke

The capacity to maintain quiet upright balance, through high-frequency small-scale adjustments, and to produce fast large-scale postural reactions with minimal attentional requirements has led to the notion that postural control is essentially automatic. On the other hand, recent investigation on healthy individuals using different

techniques to scan cerebral activation in conditions imposing challenges to balance control has indicated the participation of a highly distributed network involving different cortical areas in the generation of postural responses. More specifically, the circuitry dedicated to the control of body balance has been found to include different sites of the prefrontal cortex [5–7], supplementary motor area [8–11], visual association cortex [12], and the primary motor cortex [13–15]. These cortical structures seem to be linked with subcortical structures [8, 12] for appropriate selection, coordination, and scalability of muscular responses to maintain quiet upright balance or to recover postural equilibrium following an unanticipated perturbation. These results suggest that reflex circuitries associated with postural control are continuously adjusted by descending signals to incorporate higher order output into muscular responses to regulate body balance.

Results showing impairment of balance control due to lesions of different sites of the cerebral cortex by stroke have provided further information on the role played by higher order neural structures in the regulation of upright stance. In the analysis of quiet stance control, cerebral stroke has been shown to induce increased amplitude of CoP sway in both the anteroposterior and mediolateral directions [16–19]. Dickstein and Abulaffio [20] compared body sway at the pelvis and legs of both body sides between individuals with stroke and healthy controls. Results showed that stroke led to larger body oscillation in both the anteroposterior and mediolateral directions, with larger oscillation of the paretic than the nonparetic lower limb. Additionally, they found low temporal synchronization between sway of each leg and the respective pelvis side as well as between the two legs. Further investigation has shown that stroke leads to decreased temporal synchronization between CoP oscillations under each foot in quiet stance as compared to healthy individuals [21]. The latter results also showed that reduced inter-leg synchronization of postural adjustments in individuals with stroke was associated with increased postural sway in the mediolateral direction and increased weight-bearing asymmetry. Further investigation on this point revealed that stroke-related low inter-leg synchronization for maintenance of stable upright posture is correlated with magnitude of motor impairment and frequency of falls [22]. More recently, evidence has been presented that deficits of temporal synchronization of CoP displacements under the feet are associated with spasticity provoked by stroke [23]. These results suggest that one of the consequences of a cerebral lesion by stroke is impairment of intra-leg and between-legs coordination to apply forces to the ground when keeping quiet upright stance. Those coordination deficits may be thought to reduce the magnitude of the force vectors exerted on the ground by synergistic muscle groups, leading to increased body sway.

Although analysis of quiet posture control has indicated important deficits induced by cerebral stroke, the most threatening situation for maintenance of upright posture is represented by unexpected perturbations to stance. In this situation, keeping upright balance requires the production of short-latency and vigorous postural reactions through feet-in-place or stepping responses to recover body equilibrium. The effect of stroke on reactive postural responses has been studied in contexts of self- (involving a combination of anticipatory and reactive

components) or externally produced (requiring pure reactive responses) perturbations. In the context of self-produced perturbations, Garland performed a series of experiments on postural responses to fast voluntary shoulder flexion of the nonparetic participant's arm in individuals with stroke. Results showed that the stroke participants had increased latency and reduced magnitude of muscular activation of the distal posterior leg muscles [24]. Even though stroke participants showed balance-related anticipatory control, they performed worse than age-matched controls. Failure to produce timely and appropriately scaled muscular activation for balance control in coordination with upper limb movements is possibly associated with increased postural instability following a cerebral stroke. The ensuing experiments in this series revealed more evident impairment of muscular activation of the paretic leg in comparison with healthy controls [25], and compensation for weaker activation of the paretic leg muscles by increasing the anticipatory activation of posterior muscles of the nonparetic leg [26]. The differential compensatory activation of the muscles of the nonparetic leg suggests lateralized feedforward adjustments taking into consideration the longer activation onset delays and reduced power of muscular responses of the paretic leg to predictable self-produced balance perturbations.

Evaluation of the effect of cerebral lesion by stroke in the production of pure reactive postural responses to external perturbations has been frequently assessed by moving the basis of support unanticipatedly to the participant. In this situation, the relative position of the body center of mass over the basis of support is suddenly displaced, requiring fast selection and scaling of the appropriate muscle groups to recover upright postural stability. Results of studies employing this experimental strategy to perturb stance have shown that muscular responses of the paretic leg are delayed [27–30] and weaker [28] as compared to healthy individuals. Those deficits of muscular activation of the paretic leg have been shown to be paralleled by impaired dynamic postural control [28], and it seems to be particularly critical in individuals with stroke having a history of falls [30]. The link between deficient muscular activation by stroke and threatening to maintenance of upright balance has been corroborated by a recent investigation showing that sudden slip-like perturbations to upright stance led to falls in 71 % of the participants with stroke in comparison with no falls in age-matched healthy controls [31]. An alternative experimental strategy to produce unanticipated body balance perturbations consists of applying a continuous horizontal load to the participants' trunk, allowing them to assume a stable upright position, and then releasing the load at a time unbeknownst to them. In this situation, the unanticipated release of the external load leads to a fast body sway in the direction opposite to load application. In the epoch immediately following load release, fast and large-scale muscular responses are required to attend the constraint of keeping the feet in place to recover balance stability. Comparison of stroke and age-matched healthy individuals in this task revealed that deficits of activation of posterior muscles of the paretic leg in the participants with stroke were paralleled by reduced velocity of CoP displacement and longer delays to revert the direction of CoP sway [32]. Kinematic analysis showed that participants with stroke responded to the perturbation with increased ankle and hip

angular displacements in comparison to healthy controls. Those effects were particularly evident in individuals with right cerebral hemisphere lesions (results are detailed in a forthcoming section). The reviewed findings on postural responses to perturbed posture suggest that delayed and weaker muscular activation to an unanticipated perturbation in individuals with cerebral lesions by stroke induces impoverished recovery of stance stability, increasing the probability that they suffer falls in analogous daily living situations.

In addition to deficits in the generation of muscular activation to control upright posture following a cerebral lesion, research has suggested that sensory processing also contributes to atypical postural control in individuals who suffered a stroke. In different studies, results have indicated that stroke leads to increased dependence on visual information to control balance in quiet standing. Marigold and Eng [33] found that under full vision cerebral stroke induced increased amplitude of CoP sway in both anteroposterior and mediolateral directions. The difference of postural stability between the stroke and control groups was magnified in the condition of eyes closed, with stroke leading to a proportionally larger increment of CoP sway amplitude in the mediolateral direction in comparison with healthy controls (see also [34, 35]). Further results showing increased postural sway only in individuals with impaired ankle proprioception due to stroke [36] suggest that increased visual dependence is related to poor proprioceptive signals relevant for postural control (cf. [37, 38]). In this case, less reliable proprioceptive information from the ankles as a result of cerebral lesion might be compensated by weighting up vision in sensorimotor integration. That interpretation is supported by results showing that postural stability is impaired in individuals with stroke particularly in experimental conditions involving multiple sensory information manipulation [39, 40].

Misperception of verticality seems to be another sensory-related malfunction of the nervous system due to stroke leading to poor postural control in quiet stance. Bonan [41, 42] found that misperception of verticality in individuals with stroke was correlated with poor scores in body balance tests. More recently, individuals with stroke were evaluated for verticality perception and postural stability in sitting and standing postures for conditions combining stable–unstable surfaces and availability or not of vision [43]. Analysis showed significant correlations between subjective perception of verticality and posturography parameters in all experimental conditions. These results suggest that verticality perception is an important factor for quiet balance control potentially disrupted by stroke.

19.3 Interlateral Asymmetries of Body Balance

Uni-hemispheric cerebral stroke frequently leads to interlateral asymmetries in body balance control. Postural asymmetries are more easily observable in the mediolateral direction, due to an increased proportion of body weight-bearing on one leg than on the other (weight-bearing asymmetry, WBA). However, asymmetric regulation of anteroposterior balance sway between the legs has been shown to be one of the main deleterious consequences of stroke to body balance stabilization.

In the control of quiet standing by individuals with stroke, a greater proportion of body weight is borne more frequently on the nonparetic leg, but in some cases most of the body weight is borne on the paretic leg [44]. While bearing most of the body weight on the nonparetic leg seems to be due to its higher functional capacity to regulate balance control, weight-bearing predominantly on the paretic leg is supposed to result from rehabilitation training with focus on improvement of control of the paretic body side. Even though WBA is usually reduced during the first weeks following stroke, that postural asymmetry persists in some cases [17, 45], being considered as an indicator of incomplete recovery of hemiparesis [46, 47]. Attenuation of WBA has been a concern in rehabilitation programs [48], but similar distribution of body weight between the legs seems to be maintained by means of increased attentional control of body balance [17, 34]. Investigation of the effect of WBA on postural control in healthy individuals has been made by reproducing the body weight distribution between the legs in a similar way as individuals with stroke do. Using this experimental strategy, Rougier and Genthon [49] observed that asymmetric distribution of body weight between the legs in quiet standing leads to increased contribution of the overloaded leg to generate the resulting CoP displacements in the anteroposterior direction. Additionally, it has been shown that WBA modulates the threshold of stepping in response to multidirectional stance perturbations, decreasing the threshold for displacement of the support basis toward the loaded side while increasing the threshold when the support basis is moved toward the unloaded side [50].

Comparison of stroke and age-matched healthy individuals in asymmetric quiet standing has shown different points on interest. A similar effect of WBA between individuals with stroke and controls is forward CoP displacement under the less loaded than under the more loaded leg [51]. Then, forward CoP positioning of the less loaded leg seems to be due to mechanical factors rather than to a neurological deficit associated with limb paresis. The differential results between groups were the following: (a) the less loaded paretic leg failed to produce a longitudinal pattern of CoP displacements as observed in controls, which was paralleled by diminished postural stability; (b) the atypical CoP trajectories under the paretic foot were compensated by increased CoP displacements under the nonparetic foot; and (c) lower balance stability was found when stroke participants failed to compensate for the control deficits of the paretic leg with increased contribution of the nonparetic leg. These results suggest that WBA is not the main cause per se of balance instability in individuals with stroke, while the impoverished control of torques applied on the ground at the ankle of the paretic leg or low compensatory adjustments with the nonparetic leg may be conceived to be the major sources of poor balance stability in these individuals. Further investigation of the effect of WBA on quiet standing control was conducted by comparing the contribution of each leg to balance control between individuals with stroke having different asymmetry profiles: symmetric body weight distribution between the legs versus asymmetric loading of the paretic or the nonparetic leg [44]. Results revealed that asymmetric weight-bearing toward either leg induced increased amplitude of CoP oscillation in the mediolateral direction in comparison to individuals featured by

symmetric weight-bearing distribution between the legs. Additionally, it was found lower contribution of the more loaded leg to body balance in individuals with greater weight-bearing on the paretic than on the nonparetic leg. Reduced contribution to balance control of the loaded paretic leg was associated with greater reliance on visual information in comparison with participants keeping a symmetric weight distribution between the legs. This result suggests low use of lower limbs' proprioception to regulate body balance in those individuals.

Evaluation of dynamic balance in conditions of perturbed stance has extended the conclusions on the effect of WBA on postural responses drawn from quiet standing. van Asseldonk et al. [52] assessed the relative contribution of the paretic and nonparetic ankles in balance responses to continuous random movements of the support basis in individuals with stroke. Analysis was made for the magnitude and timing of the corrective torques on the ground in response to increased body sway on the moving platform. Results showed that healthy controls in condition of WBA presented a linear relation between the contribution of each leg to support body weight and its participation in balance control. In individuals with stroke, conversely, the contribution of the paretic leg to weight-bearing (40–45 %) was much larger than its contribution to balance control (10–20 %). Marigold et al. [29] compared muscular responses between stroke and age-matched healthy participants to unanticipated discrete forward or backward basis of support translation while standing. Evaluation was made in the following stance conditions: increased weight-bearing load, decreased weight-bearing load, and self-selected weight-bearing. Results indicated that in forward platform displacement control participants modulated the motion of the ankle dorsiflexors to different magnitudes of perturbation, with greater magnitude of muscular responses as weight-bearing load increased. Individuals with stroke, on the other hand, failed to modulate the magnitude of muscular responses as a function of WBA in both the paretic and nonparetic legs. When responding to backward platform translations, onset latency for activation of the muscle gastrocnemius medialis was shorter as weight-bearing load increased in controls. In individuals with stroke, conversely, no differences in onset latency of muscular activation were observed as a function of the weight-bearing condition. The impaired capacity to modulate the delay and magnitude of muscular responses was paralleled by cases of falls in individuals with stroke, in comparison to no occurrence of falls in the controls.

Comparison of muscular activation between the paretic and nonparetic limbs in response to balance perturbation has offered further information on postural asymmetries as a result of cerebral stroke. Di Fabio et al. [27] evaluated interlateral asymmetry of postural responses to unanticipated rotation of the support basis, inducing dorsi- or plantar-flexion at the ankle, during a series of anteroposterior platform translations. Results showed longer onset latency for muscle activation in the paretic leg, resulting in asynchronous muscular activation between the lower limbs. In an ensuing experiment, Di Fabio [53] assessed activation of agonist and antagonist muscles in response to rotational and translational displacements of the support basis. A typical activation sequence in this situation was an initial long-latency response in the agonist muscles followed by the activation of the antagonist

muscles. Results showed that the onset of the long-latency-antagonist response was delayed in the paretic distal muscles by 25–40 ms. Further research on perturbed posture has shown that responses of the paretic leg are not only delayed but also weaker [28], requiring compensatory activation of the nonparetic leg to sustain upright stance.

In the search for a deeper understanding of the asymmetries of muscular activation between the paretic and nonparetic legs associated with balance control, Pollock et al. [54] assessed the recruitment and firing rate of motor units of the muscle gastrocnemius medialis during continuous loading pulling the participant's body forward while standing. Body balance perturbation was introduced by applying progressive anteriorly directed loads at the pelvis every 25–40 s until 5 % body mass had been sustained. Results for the pattern of muscular activation showed that EMG magnitude increased with loading (reflecting modulation of motor unit recruitment), but not the motor unit firing rate. That behavior was similar between the legs. Analysis of individual leg contribution to balance control, on the other hand, revealed that CoP maximum anteroposterior displacement and velocity reached higher values under the nonparetic than the paretic leg. Pollock et al.'s results suggest that asymmetric contribution of the paretic and nonparetic legs to body balance control is not due to differential patterns of muscular activation between the legs.

19.4 Interhemispheric Cerebral Asymmetry in Balance Control

In the previous section, the reviewed literature indicates that interlateral performance asymmetries between the legs in individuals with stroke result from the poor capacity of the paretic leg to contribute to body balance stabilization in both quiet and perturbed standing. In the present section, I review evidence that balance control is more deeply impaired in lesions of the right (RHL) than left (LHL) cerebral hemisphere. The notion of right hemisphere preeminence in balance control has emerged from results showing that lesions of the right cerebral hemisphere leads to poorer body vertical orientation [55], and increased body sway [56, 57] in quiet standing. In clinical assessments, RHL has been shown to lead to lower scores [58] in addition to delayed recovery of independent stance [59]. Analysis of dynamic voluntary postural control has indicated that RHL induces poorer CoP displacement [60, 61] and weight-bearing transfer between the legs [62]. These findings are consistent in showing a specialization of the right cerebral hemisphere in upright balance control. One of the most accepted hypotheses for the interhemispheric functional asymmetry in balance control is based on the notion that the right cerebral hemisphere is specialized for spatial attention and/or representation [58], supposedly playing the major role in the elaboration of an internal model of verticality guiding body orientation in space [55, 63]. Recent research has offered some support for this hypothesis by showing a persistent reduction in mediolateral postural asymmetry in individuals with RHL following several sessions of prism adaptation by wearing a pair of glasses producing rightward optical deviation of the

visual field [64]. Improvement of postural control in this case seems to be due to a more accurate calibration of the extra personal space guiding vertical body orientation. Even though processing of the spatial orientation of the body can be conceptualized to be an important factor for quiet balance control, evidence has been presented that impaired stance stability in RHL becomes evident only in the condition of no vision [65]. That finding suggests that right hemisphere specialization in balance control may have origins different from processing of visuo-spatial information.

An alternative proposition based on hemispheric specialization for sensory information processing has been made by Goble et al. [66]. They found that activation of different cortical (parietal, frontal, and insular) and subcortical (basal ganglia) structures by feet muscle spindle stimulation is mainly located in the right cerebral hemisphere. Moreover, they observed that activation of the right cerebral hemisphere areas was correlated with measures of body balance stability. From these findings, the authors propose that a neural circuitry in the right hemisphere is specialized for processing of proprioceptive signals from the feet and ankles to control body balance. Further investigation in individuals with stroke has offered some support for the notion of right hemisphere specialization for lower limb proprioception processing. Duclos et al. [67] compared the disruption to upright balance control between right and left hemisphere lesions by applying vibrations to the peroneal and Achilles tendons of either leg. They found that postural stability was more disrupted when vibration was applied to the Achilles tendon of the nonparetic leg. Balance of individuals with left hemisphere lesions was perturbed at vibrations onset, but they progressively recovered balance stability afterwards. Right hemisphere lesions, on the other hand, led to minor perturbation in the initial phase of vibrations, but postural instability increased during the course of the vibrations. Following the end of bilateral tendon vibrations, individuals with right hemisphere lesions took longer to recover the initial balance stability. These results suggest that lesions of right hemisphere induce a deficit in down weighting the unreliable proprioceptive afference and weighting up the other sensory sources relevant for balance control.

Beyond sensory processing deficits potentially provoked by cerebral stroke, further research has indicated that more dramatic disruption of balance control by right hemisphere lesions can be due to neural processes responsible for the generation of motor commands to regulate upright posture. Fernandes [32] performed a series of experiments to further understand the differential deficits between individuals with stroke lesions to the right versus left cerebral hemispheres, taking healthy age-matched controls as reference. In Experiment 1, participants were evaluated in conditions of quiet posture on a rigid surface, contrasting the conditions of full vision and visual occlusion. Analysis indicated the highest values of amplitude and velocity of CoP sway for right hemisphere lesions, the lowest values for healthy controls, and intermediate values for left hemisphere lesions. No effect of vision manipulation was found, suggesting that sensory information was processed similarly across groups to maintain balance stability. To evaluate the role of distortion of proprioceptive information, in Experiment 2 the three groups

were assessed in quiet standing on a malleable surface. In comparison with balance control on a rigid surface, a malleable support surface is thought to disrupt regular tactile afference from the feet soles, making that sensory information less reliable for postural regulation. As the malleable surface is deformed during body sway, it is also expected to lead to reduced muscle stretch in the legs due to body balance, making that information less reliable to indicate body oscillation to the central nervous system. Results indicated a relationship between the groups similar to that seen in Experiment 1, with poorer balance control in individuals with right in comparison with left hemisphere lesions. This effect was evidenced through higher values of body sway amplitude and velocity in the mediolateral direction in the right hemisphere lesion as compared to left hemisphere lesion and control participants. Visual occlusion led to equivalent decrement of balance stability across the three groups. These results are in agreement with the conceptualization of right hemisphere specialization for quiet balance control, but are contradictory to the notion that hemispheric specialization is associated with processing of sensory information, either vision or proprioception.

In Experiment 3, Fernandes assessed the extent to which the conclusion of right hemisphere specialization for quiet standing control is extensive to the production of large-scale postural responses to unanticipated stance perturbations. The hemisphere lesion groups and controls were evaluated on a task requiring balance recovery following sudden release of a load attached to the trunk leading to forward sway. Vision availability was manipulated the same way as in the previous experiments of this series. Results confirmed the hypothesis of right cerebral hemisphere specialization for balance control by showing that the RHL group had lower velocity of anterior CoP displacement and a delayed reversion of forward CoP sway following the postural perturbation. These results suggest a poor capacity to apply fast and vigorous torques at the ankles to revert forward body oscillation in the RHL group, a critical characteristic to prevent loss of balance. These deficits of the postural responses in RHL were associated with increased angular motion at the hip, suggesting a proportionally stronger perturbation for this group. Electromyographic analysis showed longer latencies of muscular activation onset in the RHL's paretic leg than controls and lower magnitude of muscular activation in comparison with both LHL and controls, with lack of significant differences between the latter groups. Similarly to the previous experiments in this series, no differential effect of vision was found between groups. The fact that lesions took place at different cerebral areas across participants within group suggests that body balance control is organized in a highly distributed network. Infarction of a delimited cortical region, then, seems to affect the output of the right hemisphere network leading to reduced balance stability in both quiet and perturbed stances.

Impaired capacity to stabilize upright standing and to correct unanticipated postural perturbations from right cerebral hemisphere lesions are in agreement with the theorization that the right cerebral hemisphere is specialized for impedance control [68–71]. Based on studies of manual actions in healthy individuals, Sainburg and colleagues have proposed that the specialization for impedance control of the right hemisphere ensures steady state stability and positional

stabilization in response to unanticipated mechanical perturbations of movements. Evaluation of individuals who suffered a unilateral stroke in the performance of ipsilateral aiming actions has given support for this theorization [72–75]. Results from those investigations have demonstrated that right but not left cerebral hemisphere lesions lead to deficits in the final hand positioning over a spatial target. In situations in which initial directional errors were increased by means of visuomotor rotations, performance of individuals with right hemisphere lesions was impaired in online corrections to hand displacement toward the target [74]. Similar deficit in producing online corrections in aiming has been found also in the contralesional arm in individuals with stroke [76]. While the tasks evaluated in the cited experiments are distinct from body balance control, fundamental mechanisms of postural regulation might be advocated to be similar between hand and whole body stabilization. Impedance control has been proposed to be implemented by adjusting proprioceptive reflex gains and thresholds [77, 78], in addition to modulation of the stiffness- and viscous-like properties of the movements [79]. Evidence has been presented that reflexes can be modulated online to produce adaptive corrections to unanticipated perturbations to a segmental posture [80, 81] or voluntary movements [77]. These findings suggest that downward cerebral signals are continuously adjusting reflexes to incorporate higher order output into fast peripheral feedback loops. Generalizing this proposition to body balance control, steady state stability is considered to be a critical characteristic allowing for regulation of quiet stance. Positional stabilization in response to an unanticipated perturbation is essential for producing well-calibrated reactive responses to recover the upright orientation of the body, repositioning the center of mass over the basis of support. From this conceptualization, steady state regulation and corrective movements associated with impedance control may underlie functional asymmetries between the cerebral hemispheres relevant for body balance control.

19.5 Concluding Remarks

The reviewed literature in this chapter indicates that uni-hemispheric cerebral stroke leads to interlateral asymmetries in body balance control because of the reduced capacity of the paretic leg to control forces applied to the ground in the search for stance stabilization, in addition to delayed and weaker muscular responses of the paretic leg to balance perturbation. These interlateral asymmetries can be thought to underlie the low synchronization between forces applied through the legs on the ground to maintain upright body balance, and the increased weight-bearing on the nonparetic leg to compensate for the control deficits of the paretic leg. Deficits in balance control have been shown to become more critical when individuals with stroke fail to compensate appropriately for the reduced contribution of the paretic leg for balance control by means of increased participation of the nonparetic leg. The described deficits of the paretic leg, leading to interlateral asymmetries between the legs, are probably associated with the increased rate of falls in individuals with stroke.

Different evaluations of quiet and perturbed stance, as well as voluntary body motion over the basis of support, have indicated that balance deficits are more critical when lesions take place in the right cerebral hemisphere. Consistency of this finding across several studies has led to the notion of right hemisphere specialization for balance control. That specialization has been proposed to be based on right hemisphere preeminence for processing of visual and proprioceptive information. Here I delineate a distinct theorization based on the notion of right hemisphere specialization for impedance control. These explanations are not exclusive, and might converge to understand the causes of interlateral asymmetries of balance control following events of stroke to a single cerebral hemisphere.

References

1. Mertz L, Docherty C. Self-described differences between legs in ballet dancers: do they relate to postural stability and ground reaction force measures? J Dance Med Sci. 2012;16 (4):154–60.
2. Huurnink A, Fransz DP, Kingma I, Hupperets MD, van Dieen JH. The effect of leg preference on postural stability in healthy athletes. J Biomech. 2014;47(1):308–12.
3. Teixeira LA, de Oliveira DL, Romano RG, Correa SC. Leg preference and interlateral asymmetry of balance stability in soccer players. Res Q Exerc Sport. 2011;82(1):21–7.
4. Gstottner M, Neher A, Scholtz A, Millonig M, Lembert S, Raschner C. Balance ability and muscle response of the preferred and nonpreferred leg in soccer players. Motor Control. 2009;13(2):218–31.
5. Mihara M, Miyai I, Hatakenaka M, Kubota K, Sakoda S. Role of the prefrontal cortex in human balance control. Neuroimage. 2008;43(2):329–36.
6. Mihara M, Miyai I, Hattori N, Hatakenaka M, Yagura H, Kawano T, et al. Cortical control of postural balance in patients with hemiplegic stroke. Neuroreport. 2012;23(5):314–9.
7. Moro SB, Bisconti S, Muthalib M, Spezialetti M, Cutini S, Ferrari M, et al. A semi-immersive virtual reality incremental swing balance task activates prefrontal cortex: a functional near-infrared spectroscopy study. Neuroimage. 2014;85:451–60.
8. Ferraye MU, Debû B, Heil L, Carpenter M, Bloem BR, Toni I. Using motor imagery to study the neural substrates of dynamic balance. PLoS One. 2014;9(3), e91183.
9. Fujimoto H, Mihara M, Hattori N, Hatakenaka M, Kawano T, Yagura H, et al. Cortical changes underlying balance recovery in patients with hemiplegic stroke. Neuroimage. 2014;85:547–54.
10. Marlin A, Mochizuki G, Staines WR, McIlroy WE. Localizing evoked cortical activity associated with balance reactions: does the anterior cingulate play a role? J Neurophysiol. 2014;111(12):2634–43.
11. Mierau A, Hulsdunker T, Struder HK. Changes in cortical activity associated with adaptive behavior during repeated balance perturbation of unpredictable timing. Front Behav Neurosci. 2015;9(272):1–12.
12. Ouchi Y, Okada H, Yoshikawa E, Nobezawa S, Futatsubashi M. Brain activation during maintenance of standing postures in humans. Brain. 1999;122(Pt 2):329–38.
13. Bolton DA, Williams L, Staines WR, McIlroy WE. Contribution of primary motor cortex to compensatory balance reactions. BMC Neurosci. 2012;13:102.
14. Papegaaij S, Taube W, van Keeken HG, Otten E, Baudry S, Hortobagyi T. Postural challenge affects motor cortical activity in young and old adults. Exp Gerontol. 2016;73:78–85.
15. Taube W, Schubert M, Gruber M, Beck S, Faist M, Gollhofer A. Direct corticospinal pathways contribute to neuromuscular control of perturbed stance. J Appl Physiol. 2006;101(2):420–9.

16. Corriveau H, Hebert R, Raiche M, Prince F. Evaluation of postural stability in the elderly with stroke. Arch Phys Med Rehabil. 2004;85(7):1095–101.
17. de Haart M, Geurts AC, Huidekoper SC, Fasotti L, van Limbeek J. Recovery of standing balance in postacute stroke patients: a rehabilitation cohort study. Arch Phys Med Rehabil. 2004;85(6):886–95.
18. Yu J, Jung J, Cho K. Changes in postural sway according to surface stability in post-stroke patients. J Phys Ther Sci. 2012;24(11):1183–6.
19. Yu E, Abe M, Masani K, Kawashima N, Eto F, Haga N, et al. Evaluation of postural control in quiet standing using center of mass acceleration: comparison among the young, the elderly, and people with stroke. Arch Phys Med Rehabil. 2008;89(6):1133–9.
20. Dickstein R, Abulaffio N. Postural sway of the affected and nonaffected pelvis and leg in stance of hemiparetic patients. Arch Phys Med Rehabil. 2000;81(3):364–7.
21. Mansfield A, Danells CJ, Inness E, Mochizuki G, McIlroy WE. Between-limb synchronization for control of standing balance in individuals with stroke. Clin Biomech. 2011;26(3):312–7.
22. Mansfield A, Mochizuki G, Inness EL, McIlroy WE. Clinical correlates of between-limb synchronization of standing balance control and falls during inpatient stroke rehabilitation. Neurorehabil Neural Repair. 2012;26(6):627–35.
23. Singer JC, Mansfield A, Danells CJ, McIlroy WE, Mochizuki G. The effect of post-stroke lower-limb spasticity on the control of standing balance: inter-limb spatial and temporal synchronisation of centres of pressure. Clin Biomech. 2013;28(8):921–6.
24. Garland SJ, Stevenson TJ, Ivanova T. Postural responses to unilateral arm perturbation in young, elderly, and hemiplegic subjects. Arch Phys Med Rehabil. 1997;78(10):1072–7.
25. Garland SJ, Ivanova TD, Mochizuki G. Recovery of standing balance and health-related quality of life after mild or moderately severe stroke. Arch Phys Med Rehabil. 2007;88 (2):218–27.
26. Garland SJ, Willems DA, Ivanova TD, Miller KJ. Recovery of standing balance and functional mobility after stroke. Arch Phys Med Rehabil. 2003;84(12):1753–9.
27. Di Fabio RP, Badke MB, Duncan PW. Adapting human postural reflexes following localized cerebrovascular lesion: analysis of bilateral long latency responses. Brain Res. 1986;363 (2):257–64.
28. Ikai T, Kamikubo T, Takehara I, Nishi M, Miyano S. Dynamic postural control in patients with hemiparesis. Am J Phys Med Rehabil. 2003;82(6):463–9.
29. Marigold DS, Eng JJ, Timothy IJ. Modulation of ankle muscle postural reflexes in stroke: influence of weight-bearing load. Clin Neurophysiol. 2004;115(12):2789–97.
30. Marigold DS, Eng JJ. Altered timing of postural reflexes contributes to falling in persons with chronic stroke. Exp Brain Res. 2006;171(4):459–68.
31. Salot P, Patel P, Bhatt T. Reactive balance in individuals with chronic stroke: biomechanical factors related to perturbation-induced backward falling. Phys Ther. 2016;96(3):338–47.
32. Fernandes CA. Influence of the injured brain hemisphere and of sensory deficits on body balance post-stroke. University of São Paulo, Master thesis; 2014.
33. Marigold DS, Eng JJ. The relationship of asymmetric weight-bearing with postural sway and visual reliance in stroke. Gait Posture. 2006;23(2):249–55.
34. Roerdink M, Geurts AC, de Haart M, Beek PJ. On the relative contribution of the paretic leg to the control of posture after stroke. Neurorehabil Neural Repair. 2009;23(3):267–74.
35. Bensoussan L, Viton JM, Schieppati M, Collado H, de Bovis VM, Mesure S, et al. Changes in postural control in hemiplegic patients after stroke performing a dual task. Arch Phys Med Rehabil. 2007;88(8):1009–15.
36. Niam S, Cheung W, Sullivan PE, Kent S, Gu X. Balance and physical impairments after stroke. Arch Phys Med Rehabil. 1999;80(10):1227–33.
37. Meyer PF, Oddsson LI, De Luca CJ. The role of plantar cutaneous sensation in unperturbed stance. Exp Brain Res. 2004;156(4):505–12.
38. Thompson C, Belanger M, Fung J. Effects of plantar cutaneo-muscular and tendon vibration on posture and balance during quiet and perturbed stance. Hum Mov Sci. 2011;30(2):153–71.

39. Oliveira CB, Medeiros IR, Greters MG, Frota NA, Lucato LT, Scaff M, et al. Abnormal sensory integration affects balance control in hemiparetic patients within the first year after stroke. Clinics (Sao Paulo). 2011;66(12):2043–8.
40. Marigold DS, Eng JJ, Tokuno CD, Donnelly CA. Contribution of muscle strength and integration of afferent input to postural instability in persons with stroke. Neurorehabil Neural Repair. 2004;18(4):222–9.
41. Bonan IV, Guettard E, Leman MC, Colle FM, Yelnik AP. Subjective visual vertical perception relates to balance in acute stroke. Arch Phys Med Rehabil. 2006;87(5):642–6.
42. Bonan IV, Hubeaux K, Gellez-Leman MC, Guichard JP, Vicaut E, Yelnik AP. Influence of subjective visual vertical misperception on balance recovery after stroke. J Neurol Neurosurg Psychiatry. 2007;78(1):49–55.
43. Baggio JA, Mazin SS, Alessio-Alves FF, Barros CG, Carneiro AA, Leite JP, et al. Verticality perceptions associate with postural control and functionality in stroke patients. PLoS One. 2016;11(3), e0150754.
44. Mansfield A, Danells CJ, Zettel JL, Black SE, McIlroy WE. Determinants and consequences for standing balance of spontaneous weight-bearing on the paretic side among individuals with chronic stroke. Gait Posture. 2013;38(3):428–32.
45. Barra J, Oujamaa L, Chauvineau V, Rougier P, Perennou D. Asymmetric standing posture after stroke is related to a biased egocentric coordinate system. Neurology. 2009;72(18): 1582–7.
46. Bohannon RW, Larkin PA. Lower extremity weight bearing under various standing conditions in independently ambulatory patients with hemiparesis. Phys Ther. 1985;65(9):1323–5.
47. Dickstein R, Nissam M, Pillar T, Scheer D. Foot–ground pressure pattern of standing hemiplegic patients: major characteristics and patterns of improvement. Phys Ther. 1984;64 (1):19–23.
48. Sackley CM. Falls, sway, and symmetry of weight-bearing after stroke. Int Disabil Stud. 1991;13(1):1–4.
49. Rougier PR, Genthon N. Dynamical assessment of weight-bearing asymmetry during upright quiet stance in humans. Gait Posture. 2009;29(3):437–43.
50. de Kam D, Kamphuis JF, Weerdesteyn V, Geurts AC. The effect of weight-bearing asymmetry on dynamic postural stability in healthy young individuals. Gait Posture. 2016;45:56–61.
51. Genthon N, Rougier P, Gissot AS, Froger J, Pélissier J, Pérennou D. Contribution of each lower limb to upright standing in stroke patients. Stroke. 2008;39:1793–9.
52. van Asseldonk EH, Buurke JH, Bloem BR, Renzenbrink GJ, Nene AV, van der Helm FC, et al. Disentangling the contribution of the paretic and non-paretic ankle to balance control in stroke patients. Exp Neurol. 2006;201(2):441–51.
53. Di Fabio RP. Lower extremity antagonist muscle response following standing perturbation in subjects with cerebrovascular disease. Brain Res. 1987;406(1–2):43–51.
54. Pollock CL, Ivanova TD, Hunt MA, Garland SJ. Behavior of medial gastrocnemius motor units during postural reactions to external perturbations after stroke. Clin Neurophysiol. 2015;126(10):1951–8.
55. Perennou DA, Mazibrada G, Chauvineau V, Greenwood R, Rothwell J, Gresty MA, et al. Lateropulsion, pushing and verticality perception in hemisphere stroke: a causal relationship? Brain. 2008;131(9):2401–13.
56. Rode G, Tiliket C, Boisson D. Predominance of postural imbalance in left hemiparetic patients. Scand J Rehabil Med. 1997;29(1):11–6.
57. Peurala SH, Kononen P, Pitkanen K, Sivenius J, Tarkka IM. Postural instability in patients with chronic stroke. Restor Neurol Neurosci. 2007;25(2):101–8.
58. Perennou DA, Benaim C, Rouget E, Rousseaux M, Blard JM, Pelissier J. Postural balance following stroke: towards a disadvantage of the right brain-damaged hemisphere. Rev Neurol. 1999;155(4):281–90.
59. Laufer Y, Sivan D, Schwarzmann R, Sprecher E. Standing balance and functional recovery of patients with right and left hemiparesis in the early stages of rehabilitation. Neurorehabil Neural Repair. 2003;17(4):207–13.

60. Ioffe ME, Chernikova LA, Umarova RM, Katsuba NA, Kulikov MA. Learning postural tasks in hemiparetic patients with lesions of left versus right hemisphere. Exp Brain Res. 2010;201 (4):753–61.
61. Ustinova KI, Chernikova LA, Ioffe ME, Sliva SS. Impairment of learning the voluntary control of posture in patients with cortical lesions of different locations: the cortical mechanisms of pose regulation. Neurosci Behav Physiol. 2001;31(3):259–67.
62. Ishii F, Matsukawa N, Horiba M, Yamanaka T, Hattori M, Wada I, et al. Impaired ability to shift weight onto the non-paretic leg in right-cortical brain-damaged patients. Clin Neurol Neurosurg. 2010;112(5):406–12.
63. Tasseel-Ponche S, Yelnik AP, Bonan IV. Motor strategies of postural control after hemispheric stroke. Neurophysiol Clin. 2015;45(4–5):327–33.
64. Hugues A, Di Marco J, Lunven M, Jacquin-Courtois S, Rossetti Y, Bonan I, et al. Long-lasting reduction in postural asymmetry by prism adaptation after right brain lesion without neglect. Cogn Process. 2015;16 Suppl 1:371–5.
65. Manor B, Hu K, Zhao P, Selim M, Alsop D, Novak P, et al. Altered control of postural sway following cerebral infarction: a cross-sectional analysis. Neurology. 2010;74(6):458–64.
66. Goble DJ, Coxon JP, Van Impe A, Geurts M, Doumas M, Wenderoth N, et al. Brain activity during ankle proprioceptive stimulation predicts balance performance in young and older adults. J Neurosci. 2011;31(45):16344–52.
67. Duclos NC, Maynard L, Abbas D, Mesure S. Hemispheric specificity for proprioception: postural control of standing following right or left hemisphere damage during ankle tendon vibration. Brain Res. 1625;2015:159–70.
68. Duff SV, Sainburg RL. Lateralization of motor adaptation reveals independence in control of trajectory and steady-state position. Exp Brain Res. 2007;179(4):551–61.
69. Sainburg RL. Convergent models of handedness and brain lateralization. Front Psychol. 2014;5:1092.
70. Yadav V, Sainburg RL. Motor lateralization is characterized by a serial hybrid control scheme. Neuroscience. 2011;196:153–67.
71. Yadav V, Sainburg RL. Limb dominance results from asymmetries in predictive and impedance control mechanisms. PLoS One. 2014;9(4), e93892.
72. Haaland KY, Prestopnik JL, Knight RT, Lee RR. Hemispheric asymmetries for kinematic and positional aspects of reaching. Brain. 2004;127(Pt 5):1145–58.
73. Schaefer SY, Haaland KY, Sainburg RL. Ipsilesional motor deficits following stroke reflect hemispheric specializations for movement control. Brain. 2007;130(Pt 8):2146–58.
74. Schaefer SY, Haaland KY, Sainburg RL. Dissociation of initial trajectory and final position errors during visuomotor adaptation following unilateral stroke. Brain Res. 2009;1298:78–91.
75. Schaefer SY, Mutha PK, Haaland KY, Sainburg RL. Hemispheric specialization for movement control produces dissociable differences in online corrections after stroke. Cereb Cortex. 2012;22(6):1407–19.
76. Mani S, Mutha PK, Przybyla A, Haaland KY, Good DC, Sainburg RL. Contralesional motor deficits after unilateral stroke reflect hemisphere-specific control mechanisms. Brain. 2013;136(Pt 4):1288–303.
77. Mutha PK, Boulinguez P, Sainburg RL. Visual modulation of proprioceptive reflexes during movement. Brain Res. 2008;1246:54–69.
78. Pruszynski JA, Scott SH. Optimal feedback control and the long-latency stretch response. Exp Brain Res. 2012;218(3):341–59.
79. Shadmehr R, Arbib MA. A mathematical analysis of the force-stiffness characteristics of muscles in control of a single joint system. Biol Cybern. 1992;66(6):463–77.
80. Crevecoeur F, Kurtzer I, Bourke T, Scott SH. Feedback responses rapidly scale with the urgency to correct for external perturbations. J Neurophysiol. 2013;110(6):1323–32.
81. Kurtzer IL, Crevecoeur F, Scott SH. Fast feedback control involves two independent processes utilizing knowledge of limb dynamics. J Neurophysiol. 2014;111(8):1631–45.

Postural Control and Somatosensory Information: Effects of Aging and Parkinson's Disease

20

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Abstract

Sensory information from visual, vestibular, and somatosensory systems plays an important role in postural control. Deficits in postural control performance are the primary cause of the increased number of falls during aging. Postural instability in older adults is related to a decline in sensory and motor function and this can be more evident when associated with Parkinson's disease (PD). Among the declines in the sensory systems, deficits in the somatosensory function have been identified in older adults and these appear to be even higher in PD patients. Evidence supporting the importance of the somatosensory system in postural control comes from experiments that artificially reduced sensitivity in the soles of the feet and studies involving individuals with compromised plantar sensation. In an attempt to improve somatosensory input, previous research has shown that artificially enhancing cutaneous information can improve postural control. The aim of this chapter is to describe the postural control of healthy older adults and patients with PD and highlight the importance of somatosensory information to postural control in both populations. In addition, we will discuss some therapies using enhanced somatosensory information in order to improve postural control and, consequently, prevent or reduce falls.

Keywords

Postural control • Somatosensory system • Parkinson's disease • Older adults • Insole therapy • Cutaneous information • Additional somatosensory information • Postural instability • Falls • Balance • Plantar sensation

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20.1 Introduction

Falls have been reported as one of the primary causes of hospitalization of older adults and are the main consequence of changes in postural control [1, 2]. The relation between the sensorial and motor systems is extremely important for individuals to demonstrate appropriate orientation and postural balance. However, significant alterations in these systems are observed during the aging process [3]. Older adults can present functional and structural changes and these can interfere directly in posture and position of body segments, setting the body in a forward posture position called stooped posture [4]. In addition, changes in kinetic parameters related to postural control are observed, such as an increase in the frequency and amplitude of center of pressure (CoP) in the anteroposterior and mediolateral directions [5].

Added to problems due to the aging process, diseases such as Parkinson's disease (PD) can further highlight motor and sensory deficits and, consequently, greater losses in postural control are observed. Postural instability is one of the main signs of PD and its causes are multifactorial. Changes in integration of sensory information are suggested as one of the possible causes [6]. As older adults, patients with PD can present a forward posture (stooped posture), causing a displacement in CoP to near the anterior boundary of the base of support. Also, other symptoms such as rigidity and bradykinesia can be related to the physiopathology present in the postural instability.

Changes in integration of sensory information from visual, somatosensory, and vestibular systems can negatively impact on the postural control [3] of healthy older adults and patients with PD. Specifically with regard to the somatosensory system, mechanoreceptors in the sole of the foot play an important role in postural control. Research with artificial decreases in plantar sensitivity demonstrates this importance [7]. It was observed greater speed and root mean square (RMS) of the velocity of the CoP after an immersion of the foot of healthy people in ice water compared to trials before cooling [7].

Alterations in plantar sensitivity like a decline in capacity to detect stimulus and greater sensitivity threshold have been commonly related to aging and PD [8, 9]. These changes have an important impact on postural instability and an increase in the number of falls [10]. Based on this, some studies have investigated the effects of increased somatosensory information of the sole of the foot on the postural control of older adults and patients with PD [11, 12]. These studies are based on the fact that increased sensorial input could promote greater feedback to motor output. The augmented somatosensory information was able to decrease the CoP surface area and the anteroposterior RMS in healthy older adults and decrease medial-lateral sway and medial-lateral sway standard deviation in patients with PD [11, 12].

In this context, the aim of the current chapter is to describe the main alterations in postural control due to the aging process and the presence of PD. Regarding the somatosensory systems, we will focus specifically on the role of the receptors in the sole of the feet to postural control in both older adults and patients with PD.

In addition, we will present changes in plantar sensitivity and their association with postural control. Lastly, we will present intervention strategies with a focus on increased sensory input through stimulation of the sole of the foot, aiming to improve postural control, specifically through insoles.

20.2 Postural Control in Healthy Older Adults

In the age group from 18 years to 60 years, postural control presents a stabilization phase. At this stage, improvements can be observed depending on the stimulus provided, where individuals who are more exposed to balance activities or orientation of posture may present better postural control. After this age and due to genetic (primary aging) and environmental factors (secondary aging) [13, 14], structural and functional alterations are observed [1, 15]. These factors determine changes in the nervous and musculoskeletal systems, among others, directly affecting postural control [16, 17] and reflecting in an increased risk of falls and musculoskeletal complaints [1].

Postural control is defined as the body's position of dominance in space in order to maintain the proper relationship between body segments and the environment (orientation and postural alignment) and the ability to maintain the body in balance (postural stability) through the complex interaction between neural and musculoskeletal components [14, 18]. In postural control, guidance and stability are two distinct sensorimotor processes and act depending on the type of activity, performance, or biomechanical needs; some aspects can be common to both [3].

Aging deteriorates the neuronal components of both motor and sensory systems and their integrative processes, which are essential to ensure the anticipation and adaptation of postural control [14]. Changes are observed in the somatosensory, visual, and vestibular systems, resulting in adaptation of the senses to postural control, necessary for the selection of sensory and motor strategies for a particular task or environment [3, 19]. For example, people with proprioceptive impairments modify the weight for each sensory input to control posture, relying more on vision and vestibular senses.

The somatosensory system consists of a large number of receivers spread throughout the body and is responsible for capturing the position and speed of all body segments, muscle length, the contact with external objects, including the base of support [19]. With age this system is functionally and structurally affected, having a strong association with postural instability. Older adults show decreased tactile sensitivity mediated by Meissner's corpuscles and Pacinian corpuscles, due to the decrease in the number of receptors [20, 21]. These receptors are responsible for information on the distribution of the muscle power during balance activities. The sensory information of muscles receptors, tendons, and articulation that provide the feedback about the joints position and the movement detention sense are affected by age, especially at the lower limbs [20]. The visual system also presents aging-related alterations. This system is affected by multiple changes in the structure and functionality of the eye (eye threshold is lower for observing an

object due to the smaller quantity of light transmitted to the retina). As a result, there is a decrease in the visual field and in the perception of contrast (contour and depth) providing difficulties in detecting body movements (body position and center of mass travel speed) for a specific environment and an increase in the body oscillation. With regard to the vestibular system, the absolute reference system becomes less reliable due to a decline on vestibular function, resulting in difficulty of the nervous system to deal with the information coming from the visual and somatosensory systems. This degeneration in the vestibular system can lead to changes in automatic postural adjustments, endangering balance [14, 19, 21].

Age-related impairments of musculoskeletal components of postural control can also occur. These include reduction in overall height due to changes in the intervertebral discs (such as dehydration of the nucleus pulpous) [22] and a decrease in flexibility of the spine. In older adults, flexibility is affected by the formation of cross-bridges between collagen fibers, decreasing the elastic muscle component (elastin fibers) and brittle cartilage, cracked and hardened. This decrease represents around 20 % between 20 and 65 years (increasing after this period). Associated with this there is a decrease in joint mobility (50 %) and neuromuscular coordination (90 %) [23, 24]. The deterioration in muscle function is associated not only with reduced strength and endurance but also with maximum and rapid forces [20, 25], and the biomechanical relationships between body segments and the environment.

Postural control involves the complex and dynamic relationship between sensory information (perception) and motor actions (action) [19, 21]. Older adults may have difficulty in changing the weighting of the various sensory modalities, as well as accommodating variations in the environment and goals of the movement. For this reason, individuals on a firm, stable surface that primarily depend on somatosensory information, or individuals on unsteady air foil that depend more on vestibular and visual information, may struggle to judge which sensory modality is dominant, depending on the conditions of postural support and specific motor behavior [3].

The declines caused by aging induce the older adults to adopt, for example, a flexed or tilted forward position [13, 14], which results from difficulty in minimizing the spontaneous inclination of the vertical posture. In older adults, the change in position of body segments is especially pronounced in the sagittal plane, forming a characteristic postural pattern (stooped posture) often seen in older adults [4]. This postural pattern is characterized by a forward head, protruding shoulders, increased thoracic kyphosis, lumbar lordosis, and reduction in hip and knee flexion [14, 17, 26–28]. This posture may result in a larger displacement of the center of mass (CoM) and higher oscillation of the CoP, making it difficult to maintain stability (static or dynamic) or affecting functional performance [17, 29, 30].

Other issues affected by aging are the kinetic parameters, such as changes in the ability to respond effectively to threats to balance of different magnitudes and velocities. In older adults changes in the CoM occur, and these changes cause modifications in the ground reaction force (GRF) that influences the maintenance of

balance. Regarding the kinetic analysis, changes can be observed in CoP parameters showing increase on displacement, frequency, mean velocity, and amplitude in the anterior posterior and mediolateral directions. Furthermore, older people show an increase in elliptical area formed by 95 % of the coordinates of the CoP [5]. These changes have been highlighted for some studies that indicated older adults had greater oscillation than young adults during the maintenance of upright posture in both eyes open and closed conditions [2, 19, 31, 32]. Previous researches also observed greater amplitudes of CoP in the anteroposterior direction in older adults compared with young adults [5, 14].

In postural control some balance maintenance strategies are observed in response to unexpected (compensatory strategies) and expected disturbances (anticipatory strategies). The strategies most commonly studied in this population are the hip strategy, ankle strategy, and step strategy. These are characterized by muscle synergies which enable the maintenance of balance and/or regaining stability [3, 19], allowing repositioning of the body using the moment of inertia to oppose CoM, controlling the oscillation speed and CoP [33, 34].

The hip strategy is one of the most commonly used strategies in older adults. It is characterized by activation of the anterior and posterior muscles in a distal proximal sequence to seek relocation of the center of gravity (CoG) by extending and flexing the hip joint. In this strategy, the muscular action is associated with backward balance corrections, avoiding a fall in that direction. The hip strategy is used for rapid and long disturbances or when the support surface is small. Another strategy used is the ankle strategy, which is characterized by activation of the anterior and posterior muscles of the ankle, thigh, and trunk, proximal to distal sequence. In this strategy, older adults swing an inverted pendulum and achieve a change in position of the CoG by rotating around the ankle joint with a minimum movement of the hip and knee. This strategy can be observed in small, slow disturbances and when the support surface is large and firm. Finally, the step strategy can be observed in situations where sufficiently strong disturbances move the CoG outside the base of support. It is characterized by the body's response through lower limb flexion, displacing the CoG downward with forward displacement of the foot, reconfiguring a new base of support. This strategy is used when the previous two strategies are ineffective or during very long and very fast disruptions [3, 19, 20, 33, 34].

As shown, changes in postural control can interfere directly in quality of life of older adults and the main problem caused by these changes is falling. Added to the normal aging process, diseases can emerge. The second most prevalent neurodegenerative disease in the older population is PD and postural instability is one of the cardinal signs of this disease. The next topic will present some of the alterations in postural control of patients with PD and some explanations about the possible causes of these alterations.

20.3 Postural Control in Patients with Parkinson's Disease

Approximately 0.3 % of the world population is affected by PD, the second most common neurodegenerative disease [35]. Projections indicate that by 2030, the number of individuals with PD worldwide will reach 8.7 million [36]. PD occurs due to progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta of the brain where dopamine is produced, a neurotransmitter regulating the activity of the motor cortex [14, 37]. Such a decrease in dopamine levels inhibits the thalamus cortical motor systems and those located in the brain stem, compromising other brain structures [38] and causing motor damage [39]. PD is characterized by four cardinal symptoms: resting tremor, rigidity, bradykinesia, and postural instability [40–46].

Postural instability predominantly appears during the advanced stages of PD and can be affected by various causes. Other symptoms, such as rigidity and bradykinesia, can interfere in postural reactions, leading to slower and reduced response to balance perturbation. In addition, other factors such as fear of falling and deficits in the sensory system, like the decrease in integrity of some receptors, are possible causes of postural instability [39, 47]. Many individuals in moderate and advanced stages of PD present a posture with the upper body leaning forward and the head forward to the trunk, which is called the stooped posture. This position may be associated with postural instability as it moves the CoP near the limit of the base of support [48].

As stated above, postural control can be defined as a complex motor skill, based on the interaction of sensorimotor dynamic processes [18]. To maintain balance in static and dynamic activities, it is necessary to maintain the CoM of the body within the area of the supporting base, which is determined by the distance between the feet borders. Another strong influencing factor for equilibrium is the stability limit, which varies between individuals and relates to the oscillation of the CoP within the base of support: the larger the oscillation without imbalance, the greater the stability limit of the individual [49, 50]. Changes in posture control are multifactorial in PD; the change in the integration of sensory information being a major reason. The proprioceptive system provides inside information on tensions and stretching of the muscle and joint positions and can contribute to the orientation of the body in relation to gravity. Thus, it is believed that the deficit in sensory motor integration could be responsible for the stability deficit in PD [6]. Individuals with PD present a decline in the ability to adequately control the body orientation through failure in integration of sensory information.

Another problem based on postural control in PD is hypometria, which can contribute to changes in balance recovery after a postural perturbation [51]. This change can be observed even after the use of drugs for dopaminergic replacement, where the pharmacological treatment does not seem to reduce the postural instability of PD patients, as has been well described in the literature [52–54]. Resulting from these observations, the hypothesis was raised that the main reason for the deficit in proprioceptive integration is in the supplementary motor area of the cerebral cortex (important for planning and execution of motor

actions), since this area has great dopaminergic innervation and connection to the basal ganglia, degenerating only in the later stages of the disease, when the largest deficit in postural instability is observed, thus, these could be related [51]. Among the additional evidence of proprioceptive integration in PD is the dependence on visual information for posture maintenance [55]. This greater dependence is evident from the fact that individuals cannot differentiate the importance of sensory information in the absence of vision, increasing the rigidity of the lower oscillation system by the CoP. In the absence of visual sensory integration, declines are more evident, demonstrating that individuals with PD have difficulty to properly use proprioceptive information to create movement strategies [56].

Lower limits of stability can also be observed in individuals with PD, possibly caused by postures characteristic of the disease, such as hip, knee, and ankle flexion requiring greater antigravity forces for posture control [57]. In addition, other symptoms such as rigidity and bradykinesia may be related to the pathophysiology of postural instability. Bradykinesia is related to decreased dopamine in the basal ganglia [58]. This can be characterized by slowness of movement [59–62], where the individual finds it difficult to recruit muscle fibers properly to perform the movement [63], this having an important influence on the musculoskeletal system for the reaction of postural responses. Due to the dysfunction of the basal ganglia caused by PD, changes are made in the automatic movements such as postural control, demanding more compensation from the cortex. Thus, people with PD allocate more attentional resources than healthy individuals for the performance of movements, even "automatic" movements, hindering the achievement of other concurrent tasks. Despite the lack of knowledge on the pathophysiology of postural instability, postural control is due to the involvement of different afferent and efferent neural structures [64, 65]. The causes of postural control impairments are multifactorial and specifically for people with PD are diverse, with an inhomogeneous effect on postural control [66].

Studies on static and dynamic postural control in PD are very important for a better understanding of the changes caused by the disease, for example, the increase in asymmetry in postural control in these patients [67–71] and finding strategies to improve the quality of life of these individuals. Beretta and colleagues [68] found increases in asymmetry in postural control in individuals with PD, especially during challenging tasks. Another study found that asymmetry in challenging tasks increases according to the effects of PD medication and disease severity [69]. Furthermore, studies on asymmetry in postural control in PD are important as this can be associated with falls. Adequate postural control using both left and right limbs, together on double support and separately on single support, is needed in daily life situations that require challenging postural tasks. However, this behavior is affected in patients with PD. Possibly the postural control is worse when a perturbation happens during the use of most affected limb, which could cause a fall in these patients. Impaired postural control is a major cause of the increased risk of falls in this population [53, 54, 65, 66, 72, 73], the number of falls increases by two or three times in PD [74, 75], and complications of falls are the second leading

cause of hospitalization [76]. Williams and colleagues [77] showed that 16.9 % of falls in patients with PD resulted in fractures.

With regard to dynamic postural control, PD patients present inadequate responses to postural disturbances, an inability to modulate postural responses, delays or reduction in compensatory responses, inadequate arm movements to maintain balance, and deficits in somatosensory integration [53, 54, 72, 73]. Furthermore, inadequate muscular responses were found after posture disturbance [49, 72, 78, 79].

Older adults and patients with PD can present changes in integration of sensory information. Specifically in relation to the somatosensory system, some studies have shown that decreases in integrity of the receptors of the sole of the foot can influence postural control. The next topic will present changes in plantar sensibility caused by aging and PD and how these changes can negatively impact on postural control. In addition, some somatosensory therapies will be presented, especially using insoles, with a focus on the benefits to postural control.

20.4 Somatosensory Information and Postural Control in Older Adults and Patients with Parkinson's Disease: Role, Changes, and Therapies

As mentioned previously, changes in postural control can be multifactorial. Declines in the integration of sensory information from visual, somatosensory, and vestibular systems can play an important role in these changes. Specifically regarding the somatosensory system, receptors in the sole of the foot play an important role in postural control. These receptors, the mechanoreceptors, are responsible for sending tactile information to the central nervous system related to changes in pressure distribution of the sole of the foot. Frequently, this kind of information is related to changes in vertical body position [80, 81].

In a study by Kennedy and Inglis [80], the authors evaluated the distribution and activity of the receptors of the sole of the foot and classified these mechanoreceptors according to the response of the stimuli and receptive field size. The authors were able to classify the receptors into four types: (a) slowly adapting type I; (b) slowly adapting type II; (c) fast adapting type I; and (d) fast adapting type II. Receptors of type I presented numerous receptive points and distribution in a small receptive field and receptors of type II presented a single receptive point, however, with large fields. The distribution of the receptors and the sensation they provide enables the somatosensory system to provide information of great importance for balance control, especially considering the fact that in humans the feet are the only part in contact with the environment [82, 83]. This contact allows the plantar receptors to perceive information on temporal and spatial contact pressure of the feet with the ground, facilitating, for example, the control of compensatory reactions [84].

Considering the characteristics of the receptors of the sole of the foot and the importance of the information they provide, some studies have used the approach of reducing plantar sensory (mechanoreceptors) feedback to investigate the role of these receptors for postural control. Billot and colleagues [7] investigated the effects of plantar sensitivity reduction, achieved through immersion of the feet in ice (cooling procedure), on balance control in healthy adults. For the cooling procedure, the feet of the participants were immersed in water between 0 °C and 2 °C for a total of 12 min. Next, the participants performed the balance control protocol on a force plate in the bipedal position. Seven trials of thirty seconds each were performed. The authors found that, after the cooling procedure, short term changes were observed in balance control. There was an increase in CoP velocity and RMS of the velocity of CoP in the anterior posterior and medial-lateral directions. In addition to this study, findings of other studies in patients with reduced somatosensation (e.g., diabetic neuropathy) also found changes in postural control [85]. As observed by Billot and colleagues [7], losses in somatosensory information could be the primary cause of these changes.

As shown so far, the integrity of the somatosensory system, especially in the sole of the foot, has an important role in postural control. However, due to the aging process, changes in the somatosensory system can be observed. Some studies show that older adults have higher thresholds of perception of vibratory stimulus and may experience difficulties in mild touch perception in some regions of the plantar surface, when compared with young adults [8]. The main aspects that can influence these differences are: a) reduction in the total number of receptors; b) irregular distribution of receptors; c) morphological changes in receptors; and d) reduction in skin elasticity, due to the smaller quantities of elastin and collagen [8, 86]. Also, age-related changes were found to vibration thresholds. There is a sharp increase in these vibration thresholds as from seventy years old [8].

Based on the highlighted changes and considering the important role of mechanoreceptors of the sole of the foot in postural control, some studies have investigated the association between age-related changes in plantar sensibility and postural control. Menz and colleagues [87] verified that tactile plantar sensibility was strongly related with postural sway. In another study, Ueda and Carpes [88] verified a positive correlation between plantar sensitivity and postural control in older adults, specifically in the area and anteroposterior displacement of CoP. Individuals with less plantar sensitivity demonstrated a greater area and displacement of CoP. It is also important to highlight that changes in plantar sensitivity have been associated with an increased number of falls in older adults [10] and, in addition to this decrease in sensitivity, older adults have difficulty in processing and integrating sensory information [89].

Older adults are more likely to have diseases that alter both sensitivity and integration of sensory information, with ultimate consequences to posture control. Diseases such as atherosclerosis and diabetes can damage the somatosensory system and, consequently, reduce the peripheral sensitivity [90]. Patients with PD present more pronounced deficits in both sensory integration and peripheral sensory function when compared to healthy people [9]. With regarding to plantar sensitivity

in patients with PD, Prätorius, Kimmeskamp, and Milani [9] found that individuals with PD have higher sensitivity thresholds (or impaired sensitivity) when compared with neurologically healthy individuals. The same authors also found a correlation between the somatosensory system and motor deficits in patients with PD, where patients with greater motor impairment presented higher plantar sensitivity thresholds. As in other studies, the authors highlighted the fact that deficits in somatosensation can directly affect balance control in patients with PD and patients in later stages could present more pronounced deficits. In general, deficits in peripheral sensory function generate less sensory input. Due to this decreased input and added to the fact that patients have deficits in motor-sensory integration [91] less feedback to the motor output is generated, possibly explaining some deficits in the postural control of patients.

Considering the impact of reduced plantar sensation on postural control, some studies have investigated the benefits of an increase in sensorial information of the sole of the foot on the postural control of healthy older adults and those with PD. These studies were based on the fact that an increase in sensory input would promote better feedback to the motor output and, consequently, benefits to postural control.

In healthy older adults, Palluel, Nougier, and Oliver [11] verified the immediate and temporary benefits of an increase in sensory input on postural control with eyes closed. To increase sensory input, the authors used sandals with an insole covered with an array of spikes. The evaluations of postural control with eyes closed were conducted immediately after the use of sandals and after two different protocols: (i) 5 min standing quietly in an upright position wearing sandals with the insole and 5 min without sensory stimulation; (ii) 5 min walking wearing sandals with the insole and 5 min walking without sensory stimulation. The authors did not find immediate effects of using the sandals with the insoles in healthy older adults. After 5 min of use during standing quietly in an upright position, a decrease in CoP area and anteroposterior RMS was verified. A decrease in CoP velocity and mediolateral RMS was also found for both the 5 min standing quietly in an upright position protocols and the 5-min walking protocol using an insole covered with an array of spikes. The authors suggest that the insole provides important tactile information about corporal position in relation to verticality.

Qiu and colleagues [92] investigated the effects of different densities of insoles (barefoot, soft, and hard) with granulations on postural control of older adults. The older adults were evaluated in two different conditions of standing surface (firm and foam), with eyes open and closed. The authors verified a reduction in CoP area (especially on the foam surface using a soft insole), in path length and anteroposterior sway (both textured insoles on a foam surface), and in mediolateral sway (both insoles on both surface conditions). With eyes closed, older adults presented a decrease in mediolateral sway, path length, and CoP area, for both insoles on the foam surface. Based on these findings, the authors concluded that insoles with sensorial stimulation are able to reduce postural sway, especially in more challenging tasks, such as an unstable surface and eyes closed. As a possible explanation, Qiu and colleagues [92] suggested that increased plantar pressure

produced by textured insoles promoted higher stimulation of mechanoreceptors in this region. Consequently, there was an increase in feedback of cutaneous receptors to the central nervous system and, thus, an improvement in postural control.

Although some studies demonstrate the great potential of the use of additional somatosensory information on postural control of older adults, the use of this paradigm has been more restricted in research with patients with PD. Qiu and colleagues [12] investigated postural control in patients with PD and older adults in different insole conditions (barefoot, smooth insole, and textured insole), with and without visual information, on a firm and foam surface. The authors verified that for patients with PD, there was a reduction in mediolateral sway when patients were using the smooth insole or textured insole. Only the textured insole promoted a benefit to mediolateral sway and standard deviation during postural task on both firm and foam surfaces, with eyes open and closed. However, greater benefits were observed in patients with PD when they used a textured insole, on a foam surface with eyes closed.

As shown through this section, healthy older adults show significant alterations in plantar sensibility and these changes directly affect postural control. Added to the aging process, the emergence of some diseases, such as PD, can further negatively affect the role of mechanoreceptors in the sole of the foot as well as the processing of information coming from the somatosensory system. Strategies that offer an increase in somatosensory information, such as insole therapies, seem to be efficient for older people and patients with PD. It is important to highlight that the results presented by the literature are encouraging in relation to applicability of insole therapy.

20.5 Final Considerations

This chapter provides important information about changes in postural control of older adults and patients with PD, highlighting the fact that the causes of the emergence of these changes are multifactorial. Deficits in processing and integrating somatosensory information are among the main causes.

The role of plantar receptors and the importance of their integrity to postural control were evidenced. Both, older adults and patients with PD can present structural changes in mechanoreceptors distributed on the plantar surface, higher thresholds of stimuli perception, and difficulty in integration of somatosensory information. These changes negatively impact postural control of both healthy older adults and patients with PD.

We presented studies that used additional somatosensory information in the sole of the foot (insole) as an intervention to benefit postural control in older adults and patients with PD. Although some studies were found using this kind of intervention in older adults, studies with patients with PD are still limited. In both cases, the reported results are encouraging regarding the contributions from this type of intervention. The use of insoles seems to be an efficient, low cost and, principally, simple intervention that can be easily used in the daily life of older adults and

patients with PD. However, further studies are needed and should explore the chronic use of insoles in order to detect possible adaptations of the receptors to continuous stimulus.

References

1. Farinatti PTV. Envelhecimento: Promoção da saúde e exercício [Ageing: health promoting and exercise - In Portuguese]. Barueri: Manole; 2013.
2. Blaszczyk JW, Lowe DL, Hansen PD. Ranges of postural stability and their changes in the elderly. Gait Posture. 1994;2:11–7.
3. Macpherson JM, Horak FB. Posture. In: Kandel E, Schwartz J, et al., editors. Principles of neural science. 5th ed. New York: McGraw Hill Education; 2014. p. 811–22.
4. Kendall FP, McCreary EK, Provance PG, Rodgers MM, Romani WA. Muscles: testing and function, with posture and pain. 5th ed. Philadelphia: Lippincott Williams and Wilkins; 2010.
5. Lemos LFC, Ribeiro JS, Mota CB. Correlações entre o centro de massa e o centro de pressão em idosos ativos [Correlations between the center of mass and the center of pressure in elderly active]. R Bras Ci e Mov. 2015;23(1):31–9.
6. Vaugoyeau M, Viel S, Assaiante C, Amblard B, Azulay JP. Impaired vertical postural control and proprioceptive integration deficits in Parkinson's disease. Neuroscience. 2007;146 (2):852–63.
7. Billot M, Handrigana GA, Simoneaua M, Corbeila P, Teasdalea N. Short term alteration of balance control after a reduction of plantar mechanoreceptor sensation through cooling. Neurosci Lett. 2013;535:40–4.
8. Perry SD. Evaluation of age-related plantar-surface insensitivity and onset age of advanced insensitivity in older adults using vibratory and touch sensation tests. Neurosci Lett. 2006;392:62–7.
9. Prätorius B, Kimmeskamp S, Milani TL. The sensitivity of the sole of the foot in patients with Morbus Parkinson. Neurosci Lett. 2003;346:173–6.
10. Lord SR, Ward JA, Williams P, Anstey KJ. Physiological factors associated with falls in older community-dwelling women. J Am Geriatr Soc. 1994;42:1110–7.
11. Palluel E, Nougier V, Oliver I. Do spike insoles enhance postural stability and plantar surface cutaneous sensitivity in the elderly? Age. 2008;30:53–61.
12. Qiu F, Cole MH, Davids KW, Henning EM, Silburn PA, Netscher H, Kerr GK. Effects of textured insoles on balance in people with Parkinson's disease. PLoS One. 2013;8, e83309.
13. Netto MP. História da velhice no século XX: Histórico, definição do campo e temas básicos [History of the aging in 20th century: history, field definition and themes of base]. In: Freitas EV, Py L, Néri AL et al., editors. Tratado de Gerontologia: Guanabara Koogan; 2002. p. 1–12.
14. Shumway-Cook A, Woollacott MH. Motor control: translating research into clinical practice. 2nd ed. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2003.
15. Okuma SS. O idoso e a atividade física: Fundamentos e pesquisa [The older adults and physical activity: fundamentals and research – In Portuguese]. Campinas: Papirus; 1998.
16. Cadore EL, Izquierdo M. New strategies for the concurrent strength-, power-, and endurance-training prescription in elderly individuals. J Am Med Dir Assoc. 2013;14(8):623–4.
17. Valduga R, Valduga LVA, Almeida J, Carvalho GA. Relação entre o padrão postural e o nível de atividade física em idosas [Relationship between postural pattern and level of physical activity in elderly women – In Portuguese]. R Bras Ci e Mov. 2013;21(3):5–12.
18. Horak FB. Postural orientation and equilibrium: what do we need to know about neural control of balance to prevent falls? Age Ageing. 2006;35(2):ii7–11.
19. Winter DA. Human balance and posture control during standing and walking. Gait Posture. 1995;3(4):193–214.

20. Sturnieks DL, George RST, Lord SR. Balance disorders in the elderly. Neurophysiol Clin. 2008;38(6):467–78.
21. Júnior PF, Barela JA. Alterações no funcionamento do sistema de controle postural de idosos: Uso da informação visual [Changes in elderly postural control system functioning. Use of visual information]. Rev Port Cien Desp. 2006;6(1):94–105.
22. Brito J. Efeitos de um programa de treino da força, a dois níveis de intensidade, na força máxima e na massa isenta de gordura de mulheres idosas [Effects of a strength training program, at two levels of intensity, in the maximum force and mass without fat of elderly women – dissertation in Portuguese]. Faculdade de Motricidade Humana: Lisboa; 1997.
23. American College of Sports Medicine, et al. American College of Sports Medicine position stand. Exercise and physical activity for older adults. Med Sci Sports Exerc. 2009;41 (7):1510–30.
24. Coelho FG, Gobbi S, Costa JLR, Gobbi LTB. Exercício Físico no envelhecimento saudável e patológico: Da teoria à prática [Physical exercise on healthy and pathological aging: from the theory to practice – In Portuguese]. Curitiba: CVR; 2013. 462 p.
25. Hunter GR, Mccarthy JP, Bamman MM. Effects of resistance training on older adults. Sports Med. 2004;34(5):329–48.
26. Guccione AA. Fisioterapia Geriátrica [Geriatric physiotherapy – In Portuguese]. Rio de Janeiro: Guanabara; 2002. 470 p.
27. Lamotte ACS. Contribuições da musculação na postura em portadores de escoliose estrutural [Contributions of resistive training in posture in people with structural scoliosis – dissertation in Portuguese]. Brasilia: Universidade Catolica de Brasília; 2003.
28. Hinman MR. Comparison of thoracic kyphosis and postural stiffness in younger and older women. Spine J. 2004;4:413–7.
29. Faria JDC, Machala CC, Dias RC, Dias JMD. The importance of strength training programs for the rehabilitation of muscle function, equilibrium and mobility of the elderly. Acta Fisiátrica. 2003;10(3):133–7.
30. Da Silveira MM, Pasqualotti A, Colussi AE, Wibelinger LM. Human aging and body posture changes in the elderly. Rev Bras Ci Saúde. 2010;8(26):52–8.
31. Blaszczyk J, Hansen P, Lowe D. Postural sway and perception of the upright stance stability borders. Perception. 1993;22:1333.
32. Collins JJ, De Luca CJ, Burrows A, Lipsitz LA. Age-related changes in open-loop and closed-loop postural control mechanisms. Exp Brain Res. 1995;104(3):480–92.
33. Bok SK, Lee TH, Lee SS. The effects of changes of ankle strength and range of motion according to aging on balance. Ann Rehabil Med. 2013;37(1):10–6.
34. Nagai K, Yamada M, Mori S, Tanaka B, Uemura K, Aoyama T, Ichihashi N, Tsuboyama T. Effect of the muscle coactivation during quiet standing on dynamic postural control in older adults. Arch Gerontol Geriatr. 2013;56(1):129–33.
35. De Lau LM, Breteler MM. Epidemiology of Parkinson's disease. Lancet Neurol. 2006;5 (6):525–35.
36. Dorsey ER, Constantinescu R, Thompson JP, Biglan KM, Holloway RG, Kieburtz K, Marshall FJ, Ravina BM, Schifitto G, Siderowf A, Tanner CM. Projected number of people with Parkinson disease in the most populous nations, 2005 through 2030. Neurology. 2007;68 (5):384–6.
37. Saito M, Marayuama M, Ikeuchi K, Kondo H, Ishikawa A, Yuasa T, Tsuji S. Autosomal recessive juvenile parkinsonism. Brain Dev. 2000;22:S115–7.
38. Obeso JA, Rodriguez-Oroz MC, Rodriguez M, Lanciego JL, Artieda J, Gonzalo N, Olanow CW. Pathophysiology of the basal ganglia in Parkinson's disease. Trends Neurosci. 2000;23 (10):S8–19.
39. Takakusaki K, Saitoh K, Harada H, Kashiwayanagi M. Role of basal ganglia-brainstem pathways in the control of motor behaviors. Neurosci Res. 2004;50(2):137–51.
40. Nutt GJ. Motor fluctuations and dyskinesia in Parkinson's disease. Parkinsonism Relat Disord. 2001;8(2):101–8.

41. Morris EM, Huxham F, McGinley J, Dodd K, Iansek R. The biomechanics and motor control of gait in Parkinson disease. Clin Biomech. 2001;16(6):459–70.
42. Morris M, Iansek R, McGinley J, Matyas T, Huxham F. Three-dimensional gait biomechanics in Parkinson's disease: evidence for a centrally mediated amplitude regulation disorder. Mov Disord. 2005;20:40–50.
43. Pieruccini-Faria F, Menuchi MRTP, Vitório R, Gobbi LTB, Stella F, Gobbi S. Parâmetros cinemáticos da marcha com obstáculos em idosos com doença de Parkinson, com e sem efeito da Levodopa: um estudo piloto [Kinematic parameters for gait with obstacles among elderly patients with Parkinson's disease, with and without levodopa: a pilot study – In Portuguese]. Rev Bras Fisioter. 2006;10(2):243–9.
44. Vitório R, Pieruccini-Faria F, Stella F, Gobbi S, Gobbi LTB. Effects of obstacle height on obstacle crossing in mild Parkinson's disease. Gait Posture. 2010;31:143–6.
45. Vitório R, Lirani-Silva E, Barbieri FA, Raile V, Stella F, Gobbi LTB. Influence of visual feedback sampling on obstacle crossing behavior in people with Parkinson's disease. Gait Posture. 2013;38(2):330–4.
46. Barbieri FA, Rinaldi NM, Santos PC, Lirani-Silva E, Vitorio R, Teixeira-Arroyo C, Stella F, Gobbi LTB. Functional capacity of Brazilian patients with Parkinson's disease (PD): relationship between clinical characteristics and disease severity. Arch Gerontol Geriatr. 2012;54 (2):83–8.
47. Jankovic J. Parkinson's disease: clinical features and diagnosis. J Neurol Neurosurg Psychiatry. 2008;79:368–76.
48. Jacobs JV, Dimitrova DM, Nutt JG, Horak FB. Can stooped posture explain multidirectional postural instability in patients with Parkinson's disease? Exp Brain Res. 2005;166:78–88.
49. Horak FB, Dimitrova D, Nutt JG. Direction-specific postural instability in subjects with Parkinson's disease. Exp Neurol. 2005;193(2):504–21.
50. Pollock AS, Durward BR, Rowe PJ, Paul JP. What is balance? Clin Rehabil. 2000;14 (4):402–6.
51. Jacobs JV, Horak FB. External postural perturbations induce multiple anticipatory postural adjustments when subjects cannot pre-select their stepping foot. Exp Brain Res. 2007;179 (1):29–42.
52. Bloem BR, Beckley DJ, van Dijk JG, Zwinderman AH, Remler MP, Roos RA. Influence of dopaminergic medication on automatic postural responses and balance impairment in Parkinson's disease. Mov Disord. 1996;11(5):509–21.
53. Bloem BR, Grimbergen YAM, Cramer M. Prospective assessment of falls in Parkinson's disease. J Neurol. 2001;248:950–8.
54. Bloem BR, Van Vugt JP, Beckley DJ. Postural instability and falls in Parkinson's disease. Adv Neurol. 2001;87:209–23.
55. Brown LA, Cooper SA, Doan JB, Dickin DC, Whishaw IQ, Pellis SM, Suchowersky O. Parkinsonian deficits in sensory integration for postural control: temporal response to changes in visual input. Parkinsonism Relat Disord. 2006;12(6):376–81.
56. Tagliabue M, Ferrigno G, Horak F. Effects of Parkinson's disease on proprioceptive control of posture and reaching while standing. Neuroscience. 2009;158(4):1206–14.
57. Mancini M, Rocchi L, Horak FB, Chiari L. Effects of Parkinson's disease and levodopa on functional limits of stability. Clin Biomech. 2008;23(4):450–8.
58. Bergman J, Madras BK, Spealman RD. Behavioral effects of D1 and D2 dopamine receptor antagonists in squirrel monkeys. J Pharmacol Exp Ther. 1991;258(3):910–7.
59. Flowers K. Ballistic and corrective movements on an aiming task. Intention tremor and parkinsonian movement disorders compared. Neurology. 1975;25(5):413–21.
60. Horak FB, Frank J, Nutt J. Effects of dopamine on postural control in parkinsonian subjects: scaling, set, and tone. J Neurophysiol. 1996;75(6):2380–96.
61. Pfann KD, Buchman AS, Comella CL, Corcos DM. Control of movement distance in Parkinson's disease. Mov Disord. 2001;16(6):1048–65.

62. Van Gemmert AW, Adler CH, Stelmach GE. Parkinson's disease patients undershoot target size in handwriting and similar tasks. J Neurol Neurosurg Psychiatry. 2003;74(11):1502–8.
63. Berardelli A, Dick JP, Rothwell JC, Day BL, Marsden CD. Scaling of the size of the first agonist EMG burst during rapid wrist movements in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry. 1986;49(11):1273–9.
64. Boonstra TA, Van Der Kooij H, Munneke M, Bloem BR. Gait disorders and balance disturbances in Parkinson's disease: clinical update and pathophysiology. Curr Opin Neurol. 2008;21(4):461–71.
65. Błaszczyk JW, Orawiec R. Assessment of postural control in patients with Parkinson's disease: sway ratio analysis. Hum Mov Sci. 2011;30(2):396–404.
66. Benatru I, Vaugoyeau M, Azulay JP. Postural disorders in Parkinson's disease. Neurophysiol Clin. 2008;38:459–65.
67. Geurts AC, Boonstra TA, Voermans NC, Diender MG, Weerdesteyn V, Bloem BR. Assessment of postural asymmetry in mild to moderate Parkinson's disease. Gait Posture. 2011;33(1):143–5.
68. Beretta VS, Gobbi LTB, Lirani-Silva E, Simieli L, Orcioli-Silva D, Barbieri FA. Challenging postural tasks increase asymmetry in patients with Parkinson's disease. PLoS One. 2015;10, e0137722.
69. Barbieri FA, Polastri PF, Baptista AM, Lirani-Silva E, Simieli L, Orcioli-Silva D, Beretta VS, Gobbi LTB. Effects of disease severity and medication state on postural control asymmetry during challenging postural tasks in individuals with Parkinson's disease. Hum Mov Sci. 2016;46:96–103.
70. Boonstra TA, Schouten AC, van Vugt JP, Bloem BR, van der Kooij H. Parkinson's disease patients compensate for balance control asymmetry. J Neurophysiol. 2014;112(12):3227–39.
71. Boonstra TA, van Kordelaar J, Engelhart D, van Vugt JP, van der Kooij H. Asymmetries in reactive and anticipatory balance control are of similar magnitude in Parkinson's disease patients. Gait Posture. 2016;43:108–13.
72. Carpenter MG, Allum JH, Honegger F, Adkin AL, Bloem BR. Postural abnormalities to multidirectional stance perturbations in Parkinson's disease. J Neurol Neurosurg Psychiatry. 2004;75(9):1245–54.
73. Rocchi L, Chiari L, Horak FB. Effects of deep brain stimulation and levodopa on postural sway in Parkinson's disease. J Neurol Neurosurg Psychiatry. 2002;73:267–74.
74. Bloem BR, Hausdorff JM, Visser JE, Giladi N. Falls and freezing of gait in Parkinson's disease: a review of two interconnected, episodic phenomena. Mov Disord. 2004;19 (8):871–84.
75. Pickering RM, Grimbergen YA, Rigney U, Ashburn A, Mazibrada G, Wood B. A meta-analysis of six prospective studies of falling in Parkinson's disease. Mov Disord. 2007;22:1892–900.
76. Temlett JA, Thompson PD. Reasons for admission to hospital for Parkinson's disease. Intern Med J. 2006;36(8):524–6.
77. Williams DR, Watt HC, Lees AJ. Predictors of falls and fractures in bradykinetic rigid syndromes: a retrospective study. J Neurol Neurosurg Psychiatry. 2006;77:468–73.
78. Dimitrova D, Horak FB, Nutt JG. Postural muscle responses to multidirectional translations in patients with Parkinson's disease. J Neurophysiol. 2004;91(1):489–501.
79. Dimitrova D, Nutt JG, Horak FB. Abnormal force patterns for multidirectional postural responses in patients with Parkinson's disease. Exp Brain Res. 2004;156:183–95.
80. Kennedy PM, Inglis JT. Distribution and behaviour of glabrous cutaneous receptors in the human foot sole. J Physiol. 2002;538:995–1002.
81. Hijmans JM, Geertzen JHB, Dijkstra PU, Postema K. A systematic review of the effects of shoes and other ankle or foot appliances on balance in older people and people with peripheral nervous system disorders. Gait Posture. 2007;25:316–23.
82. Maki BE, Mcilroy WE. Postural control in the older adult. Clin Geriatr Med. 1996;12:635–58.

83. Eils E, Behrens S, Mers O, Thorwesten L, Völker K, Rosenbauma D. Reduced plantar sensation causes a cautious walking pattern. Gait Posture. 2004;20:54–60.
84. Perry SD, William E, Mcilroy WE, Makia BE. The role of plantar cutaneous mechanoreceptors in the control of compensatory stepping reactions evoked by unpredictable, multi-directional perturbation. Brain Res. 2000;877:401–6.
85. Kars HJ, Hijmans JM, Geertzen JH, Zijlstra W. The effect of reduced somatosensation on standing balance: a systematic review. J Diabetes Sci Technol. 2009;3:931–43.
86. Wells C, Ward LM, Chua R, Inglis JT. Regional variation and changes with ageing in vibrotactile sensitivity in the human foot sole. J Gerontol A Biol Sci. 2003;58(8):680–6.
87. Menz HB, Morris ME, Lord SR. Foot and ankle characteristics associated with impaired balance and functional ability in older people. J Gerontol A Med Sci. 2005;60A:1546–52.
88. Ueda LS, Carpes FP. Relationship between foot sensibility and postural control in the young and elderly. Rev Bras Cineantropom Desempenho Hum. 2013;15:215–24.
89. Matsumura BA, Ambrose AF. Balance in the elderly. Clin Geriatr Med. 2006;22:395–412.
90. Konrad HR, Girardi M, Helfert R. Balance and aging. Laryngoscope. 1999;109:1454–60.
91. Abbruzzese G, Berardelli A. Sensorimotor integration in movement disorders. Mov Disord. 2003;18(3):231–40.
92. Qiu F, Cole MH, Davids KW, Henning EM, Silburn PA, Netscher H, Kerr GK. Enhanced somatosensory information decreases postural sway in older people. Gait Posture. 2012;35:630–5.

Exercise and Balance in Older Adults with Movement Disorders

21

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Abstract

This chapter concerns balance, gait function, exercise, and current rehabilitation options for older adults with Parkinson's disease (PD) and stroke, within the context of normal aging. We outline the primary balance and gait deficits and their impact on fall rates for PD and stroke, with discussion regarding impairment in dual tasking and cognitive disturbances. We consider current options in physical therapy and exercise-based therapy for PD and stroke, particularly for interlimb coordination and gait function. Crossover approaches in rehabilitative techniques from PD/stroke to normal aging motor impairments often go underutilized in the clinic. Therefore, implications for older adults

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without movement disorders, who often have purely aging-related symptoms similar to those of PD, are discussed. Currently, research is especially engaged in investigating novel approaches to multimodal exercise, which have emerged as promising therapies for balance impairments including aerobic exercise, tai chi, and dance. Efficacy of such interventions is presented with respect to new research showing cognitive effects. We conclude the chapter by discussing hypotheses regarding mechanisms underlying deficit and recovery in these populations. Understanding the mechanisms for rehabilitative gains is crucial for developing targeted and efficient therapy for PD and stroke populations.

Keywords
Motor control • Gait • Parkinson's disease • Balance • Dual tasking • Exercise • Mobility • Falls • Older adults • Physical therapy • Stroke

21.1 Primary Balance and Gait Deficits in Aging and Neurodegenerative Disease

Normal aging encompasses alterations in sensorimotor processing that lead to deterioration of motor performance, including coordination impairments, increased variability of movement, slowing of movement, and gait and balance impairments. These impairments are thought to result from cumulative changes in central and peripheral nervous systems (CNS and PNS), the neuromuscular system and the peripheral vestibular system. In the PNS, older adults have decreased number and density of both articular and cutaneous mechanoreceptors and alterations in muscle spindles. In the CNS, older adults have delayed impulse conduction, decreased impulse velocity, delayed synaptic transmission, declining prevalence of all neurotransmitters, and a loss of large myelinated fibers that decreases axonal ability to transmit impulses. Cell loss from aging is most notable in the cerebral cortex lobes, cerebellar area, and frontal lobe gray matter. Thus there is a direct impact on memory, attention, awareness, motor control, coordination, and the ability to perform executive function tasks. The culmination of this overall central decline results in decreased automaticity of movement and increased reliance on workaround strategies along with a slower processing speed and deficit in the ability to handle multiple processes simultaneously.

Individuals with movement disorders resulting from disease or neurological insult must cope with impairments that go above and beyond those experienced from normal aging. This chapter will cover these impairments for the common disorders Parkinson's disease (PD) and stroke. Potentially, to address impairment stemming from normal aging, one can apply knowledge gained from research into rehabilitative approaches developed for PD and stroke.

21.1.1 Parkinson's Disease: Impact on Falls' Risk and Balance

PD is a neurological disorder characterized by rigidity, bradykinesia, postural abnormalities, resting tremor, and changes in gait. Over 70 % of persons with PD fall during the course of their disease, often resulting in decreased mobility, loss of independence, and injuries [1–4]. Lower extremity impairments contributing to gait dysfunction and postural instability also have powerful effects on self-reported motor and psychological quality of life [5, 6].

Unfortunately, gait disturbance increases in individuals with PD during performance of a secondary task (both nonmotor and motor). Camicioli et al. reported dual-task performance (verbal recitation of numbers) in people with PD led to decreased step size and gait speed [7]. Gait speed and stride length reduce significantly in PD patients while carrying a tray of glasses compared to free walking [8]. O'Shea evaluated the gait of PD participants while performing coin transference or digit subtraction. Persons with PD demonstrated a greater decrease in stride length, walking speed, and cadence during the dual-task conditions than did the matched comparison groups [9].

Gait disturbances associated with PD typically include slowed walking as well as increased gait variability. Hausdorff recently reported that dual-task performance markedly heightens gait variability and has less effect on bradykinetic manifestations, such as stride time [10]. This finding is troubling because of the strong relationship between increased gait variability and fall risk in older populations [11]. Rochester and colleagues evaluated the interference effects on walking performance by dual-motor tasks, dual cognitive tasks, and a combination motor-cognitive task (talking and carrying a tray while walking) in individuals with PD as well as the influence of cognition, depression, fatigue, and balance. Walking speed and step length decreased significantly in the dual cognitive and multitask trials. Further, cognitive function, depression, physical fatigue, and balance were significantly related to walking speed, and both cognitive function and fatigue affected dual-task performance [12]. Combined, these studies identify a dual-task interference with steady-state walking in patients with PD. Apparently, a critical level of task complexity exists after which gait deteriorates significantly.

21.1.2 Stroke: Impact on Falls' Risk and Balance

Stroke is among the four leading causes of death and disability worldwide, with about 15 million people suffering from stroke every year. The symptoms and prognosis of stroke, caused by a loss of blood supply to neural tissue, vary widely depending on the site and size of the lesion. Motor disabilities are often a consequence and can affect speech, grasp, balance, and gait, significantly impacting activities of daily living (ADLs) and quality of life (QOL). Impairments such as muscle weakness, spasticity, and decreased intra- and interlimb coordination contribute to deficits in balance and gait function. Even after discharge from rehabilitation, a majority of stroke survivors have balance dysfunction, leading to increased

risk of falls. The majority of falls occur within the first few months following discharge from rehabilitation [13–16]. Stroke survivors who experience a fall are less likely to be socially active in the community and more likely to have symptoms of depression [13]. Furthermore, post-stroke hemiparesis is accompanied by a myriad of gait deficits that further add to the risk for falls. For example, reduced flexion of the hip, knee, and ankle during paretic swing phase may prevent the paretic foot from clearing the ground during swing, which may contribute to falls.

Considerable evidence exists about the impairments in balance observed in individuals with post-stroke hemiparesis. During standing, stroke survivors demonstrate increased postural sway, with a shift in center of body sway toward the non-paretic limb, with an asymmetric distribution of body weight [17]. Interestingly, related to the increased postural sway during quiet standing, compared to able-bodied individuals, stroke participants show less between-limb synchronization, i.e., correlation between the anteroposterior and mediolateral center of pressure sway [18]. As can be expected, stroke survivors also present with deficits in dynamic balance and lower scores on clinical function tests, such as the Timed Up and Go or the Berg Balance Scale. Studies have shown that in response to a balance perturbation, stroke survivors demonstrate impairments in reactive stepping characterized by the need for assistance, inability to step with either leg, or the need for multiple-step responses [19]. Impaired stepping responses can increase the risk of falls when faced with challenges and obstacles during community ambulation. Asymmetry in weight distribution during standing is commonly observed in individuals post-stroke, with greater weight shifted toward the non-paretic limb. Interestingly, asymmetric weight-bearing during standing has been shown to correlate with asymmetry during gait [20, 21], suggesting that some common mechanisms might underlie deficits in balance and gait. A better understanding of the impairments in balance and gait that contribute to falls is warranted for the development of more effective post-stroke fall prevention programs.

21.2 Motor-Cognitive Interaction and Falls: Dual Tasking and Cognitive Disturbances

In the past, movement and cognition were thought to be controlled by isolated, proprietary, cortical regions. However, functional neuroimaging techniques have graphically demonstrated considerable overlap in brain regions for many disparate behaviors. Overlapping neural systems likely serve both cognitive and motor function [22]. Two primary regions have been identified that are activated by both cognitive and motor tasks: sensorimotor cortex and the dorsolateral prefrontal cortex (dlPFC). While it is axiomatic that motor tasks will activate sensorimotor cortices and their association cortices, action words, both verbs and nouns, can activate somatotopically appropriate locations within sensorimotor cortices [23] and supplementary motor cortex [24, 25]. The cortical representation of action word meaning includes cortex used for *enacting* a particular verb (e.g., writing, painting) or *using* an action-related noun (e.g., pencil, paintbrush) [26]. From a

behavioral standpoint, processing hand action-related words can impact finger and hand movements, but not foot movement. Conversely, hand and arm movements also impact processing of hand-related words [27]. However, both facilitation and inhibition of movement are possible following action word processing [28].

The dlPFC is a region receiving input from multiple sensory and multimodal areas with projections to and from occipital, temporal and parietal lobes, as well as sensorimotor cortices. The dlPFC may provide "processing resources" (i.e., computational resources for calculating complex behaviors) for resource-demanding tasks, including nonautomatic movement, executive function, working memory, and language. The dlPFC is vulnerable to age-related declines in function due to loss of synapses [29]. Because the dlPFC supports processing in multiple domains [30], there is often competition for processing resources during concurrent tasks. More study is necessary to determine the involvement of various cortical and subcortical areas that underlie healthy motor-cognitive integration.

21.2.1 Parkinson's Disease

In PD, declines in dlPFC functional connectivity are more pronounced than in age-matched peers [31], resulting in fewer resources available for calculating complex behaviors; therefore, people with PD often experience exaggerated dual-task impairments relative to their age-matched peers [9]. The magnitude of dual-task effects, or decrement in performance during concurrent tasks, is highly dependent on the task difficulty: as the secondary cognitive task increases in difficulty, so does the impairment in the primary motor task [32].

When considering the disease process and manifestation of PD, the link between motor and cognitive function is especially clear. Motor learning is slower and likely uses more brain activity in individuals with PD than those without [33]. Increased bradykinesia—a reliable clinical measure of the nigrostriatal lesion in PD—is associated with the presence of mild cognitive impairment (MCI) and impaired executive performance [34]. With careful concentration on critical movement aspects, those with PD can achieve close to normal movement amplitudes [35, 36]. During externally cued movements, people with PD might be able to bypass the malfunctioning basal ganglia [37] and activate the cerebellar-thalamo-cortical (CTC) neural network, in a manner similar to that noted in age-matched controls. There is also a well-known facilitating effect of cues for alleviating freezing of gait [38].

The rationale for "dual-task interference" is that performing multiple tasks requires more executive resources, which, in aging and more so in PD, are often limited. The performance decrement during the dual task, termed the dual-task cost, depends on the type of task being performed and, more importantly, the population being tested. ADLs are typically performed under modestly complex conditions and require the simultaneous performance of cognitive and motor tasks [39]. Therefore, even slight or subtle declines in cognitive function for people with PD may impact performance of routine activities, such as dressing,

grooming, cooking, walking, and driving. Even in healthy older adults, evidence suggests that the dual-task cost on cognitive and motor performance increases with age [40].

When two tasks are performed simultaneously, the more familiar task may be more reliant on basal ganglia control and processing, while the less familiar or attention-demanding task is modulated by the frontal cortex [9, 41, 42]. Therefore, impaired functioning of the basal ganglia would be hypothesized to result in automatic tasks becoming slow, reducing in amplitude or ceasing altogether. This is consistent with the lack of spontaneous movements, development of bradykinesia, and freezing of gait (FOG) seen in patients with PD. Dalrymple-Alford and colleagues examined the effects of adding a cognitive task during an upper extremity tracking task with controls and individuals with PD [43]. Those with PD showed an increase in tracking errors when asked to recall a series of digits. These data support the hypothesis that executive function deficits can be caused by disruptions of the basal ganglia circuits and contribute to increased motor performance variability under dual-task conditions.

21.2.2 Stroke

The impact of stroke on the interaction between cognitive functions and motor tasks, such as walking, has been the focus of investigation in recent studies [44]. In stroke survivors, postural sway reduces during standing balance tasks performed concurrently with a cognitive task [45]. In another study, comparison of cognitive-motor dual tasking during reactive versus intentional balance task showed that cognitive performance only decreased during the reactive balance task, suggesting the nervous system's prioritization of reactive over intentional balance post-stroke [46]. During dual-task walking, cognitive cost during a serial subtraction task performed while walking was greater for stroke survivors versus young, able-bodied individuals [47]. Another recent study tracked cognitive decline, the Timed Up and Go test, the Berg Balance Scale, dual-task performance, as well as gait speed in stroke survivors over a 20-year period, and suggested that gait and balance measures may be useful as predictors of future cognitive decline following stroke [48]. Further study of the interrelationships between cognitive-motor performance post-stroke is greatly needed.

21.3 Dual Tasking: Effects on Gait and Balance

The capacity to perform a second task during locomotion is highly advantageous. Many ADLs require multitasking, e.g., carrying objects or thinking while walking in a crowded environment, thus increasing older individuals' risk for falling [49, 50]. In fact, most falls in community-living older adults occur during routine, nonathletic activities of daily life [51]. Using the dual-task paradigm, researchers have demonstrated that maintaining balance requires attentional resources, and

demands on those resources increase with impaired balance [52]. Shumway-Cook and Woollacott demonstrated that older adults with impaired balance could maintain standing balance under a single-task condition, but fell under dual-task conditions [53]. Gait assessment under dual-task conditions may be sensitive for fall risk identification in older adults [54–56]. However, a recent review suggests that only some fallers are identified by dual-task conditions [57], so more research needs to be conducted to determine the utility of the dual-task paradigm for prediction of falls. Diverse cognitive and manual tasks have been used to assess the ability to divide attention while walking and to identify fall risk. This diversity can make comparing findings across studies challenging [57].

21.3.1 Parkinson's Disease: Impact on Gait and Cognitive-Motor Dual Tasking

Many individuals with PD use attentional strategies to maintain stability and reduce their fall risk [58, 59]. However, balance-enhancing strategies are often ineffective when presented with attention-dividing challenges. The introduction of a concurrent verbal cognitive task led to deterioration in postural stability, especially in demanding balance conditions, and this deterioration placed people with PD at a high risk of falling [58]. Similarly, greater postural sway was observed in PD fallers while completing an attention-distracting cognitive task [60].

Recently researchers have explored how the scaling and coordination aspects of motor control are affected by the disorder. Basal ganglia dysfunction causes deficits in the temporal and spatial coordination of multi-segment movements. Increased bradykinesia and rigidity have also been associated with decreased motor adaptive ability in movement coordination [61]. Although dopamine replacement is the most common pharmacological treatment of motor symptoms in PD, its effects on motor coordination are not completely understood.

Interlimb coordination is important for dynamic stability during gait and contributes to the ability to achieve a normal gait speed. However, studies show that both ipsilateral and contralateral interlimb coordination are reduced in persons with PD compared to the healthy older adults. The normal close coupling of arm and leg movements during gait is disrupted in PD, although the mechanism for this is unclear. This occurs even when the disease progression is mild [62]. In addition, an increased cognitive load (e.g., under dual-tasking conditions) is significantly correlated with increased gait variability. In people with PD, higher cognitive demands are imposed on the patient to maintain a consistent and accurate alternate stepping pattern [63].

One manifestation of dyscoordination in PD is the occurrence of motor blocks, the inability to initiate, or the sudden discontinuation in voluntary movements. This can manifest as FOG in the lower limbs. FOG occurs when a person with PD is temporarily unable to initiate or continue effective stepping, usually for only a few seconds, but can then overcome and continue walking at their normal cadence [64]. Although the mechanisms underlying FOG are poorly understood, FOG is

associated with decreased gait rhythmicity and asymmetry and bilateral dyscoordination of left-right stepping [63]. FOG also tends to manifest in functional situations requiring increased interlimb coordination, such as turning [64]. Three hundred sixty degree turning in combination with dual tasking has been shown to be a significant trigger for FOG. People who freeze tend to increase their cadence and adopt a posture second strategy in contrast to nonfreezers when presented with a dual-task situation. Freezers take significantly more steps to turn and are slower than people without FOG. ADLs are significantly impacted given that functional mobility in the home tends to involve frequent turns [65].

Motor blocks can also occur with upper extremity use. A recent study by Brown et al. also showed motor blocks during rhythmic bimanual coordination when switching between phase patterns (e.g., between inphase and antiphase). The study concluded that upper limb motor blocks occurred in PD participants both without ("off") and with ("on") dopamine replacement. Specifically, the motor blocks may be caused by difficulty shifting attention under increased cognitive demand, implicating hypoactivation in motor and prefrontal areas [66]. Studies have also shown that the rapid alternating task category in the United Parkinson's Disease Rating Scale (UPDRS) is an independent predictor of falls, suggesting a correlation between impaired intersegmental coordination and fall risk [67].

21.3.2 Stroke: Impact on Gait and Cognitive-Motor Dual Tasking

Gait impairments in people post-stroke span multiple lower extremity joints and all phases of the gait cycle. In addition to the multitude of deficits in kinematics and kinetics in post-stroke gait, asymmetry of temporal and spatial gait parameters is common post-stroke [68]. In healthy individuals, gait tends to be symmetric both spatially (between left and right joint angles and step and stride lengths) and temporally (between right and left swing, stance, step, and stride times). The unilateral deficits in the paretic limb affected by the stroke result in asymmetry, usually visibly observable as a "limp" during walking. Spatial asymmetry is manifested as a difference in step length, with the paretic leg being either longer or shorter steps compared to the non-paretic. Temporal asymmetry is reflected in reduced double support and single-leg stance duration on the paretic leg [69]. Post-stroke gait asymmetry may have important implications for gait speed, energy expenditure during gait, musculoskeletal overuse and loading on the non-paretic leg, and the incidence of falls.

Further adding to the complexity of balance and gait dysfunction observed post-stroke, deficits in adaptation and response to perturbations have been observed during dynamic balance tasks post-stroke. Post-stroke individuals with foot drop using an ankle-foot orthosis had reduced gait adaptability, as shown by reduced success with avoiding obstacles and reduced capacity to restore gait after crossing the obstacle [70]. Difficulty with safely navigating obstacles and incorporating step modifications during community ambulation would predispose stroke survivors to

falls and injuries. Post-stroke persons may also show normal obstacle-avoidance strategies, but have delayed and reduced electromyography responses, smaller joint angle deviations from unperturbed walking, and smaller horizontal margins from the foot to the obstacle, suggesting possible mechanisms that could be targeted with rehabilitation [71].

Dual-task training has also received attention in recent stroke rehabilitation research. Interventions designed to train cognitive-motor dual tasking, and incorporating visual or auditory cues, may show superior effects on post-stroke motor function compared to interventions not including dual-task practice [72, 73]. Although evidence supporting the incorporation of cognitive-motor interference tasks into stroke rehabilitation is still inconclusive, the use of dual-task training paradigms for improving community ambulation and reducing falls in post-stroke individuals merits systematic investigation [74].

21.4 Options in Physical Therapy and Exercise-Based Therapies

21.4.1 Assessment

The previous sections have highlighted the fact that a reduced capacity for dual-task walking and/or limited ability to adapt to changes in environmental context may substantially restrict the degree to which a person is able to participate in life roles. Conventional rehabilitation does not always adequately address the interaction of these two impairments, which can contribute to lower levels of participation and physical activity. As a result, the physical therapy assessment of older adults with and without neurological insult or disease should encompass a careful analysis of both coordination and dual-task ability and their impact on the patient's functional mobility.

Assessment of dual-task ability can include the Timed Up and Go Cognitive and the Timed Up and Go Manual. These tests are quick, dynamic measures for identifying individuals who are at risk for falls. Cutoff scores are available for both community-dwelling elderly [75] and for people with Parkinson's disease [76]. A 2012 study by Plummer-D'Amato et al. showed dual-task ability for community-dwelling older adults could be assessed by timing participants completing a 6 meter obstacle course under single-task and three different dual-task conditions (spontaneous speech, alphabet recitation, and coin transfer) [77]. The therapist should be aware of the different types of cognitive interference tasks and evaluate several if necessary to determine the impairments most requiring intervention. The therapist's assessment may also include looking at spatiotemporal gait parameters under single- and dual-task conditions, such as time to complete, stride length, and number of steps taken on a 10 Meter Walk Test.

A careful assessment of coordination will include detailed observation of the patient's movements during sequential tasks under various conditions. An examination of upper extremity coordination in sitting might include rapid alternating movements, finger to nose, and rapid tapping of the first finger on the crease of the

thumb. Lower extremity coordination testing could include heel-to-shin testing and timed tapping of the patient's foot into the examiner's hand. The Purdue Pegboard Test is a test of upper extremity dexterity and coordination recommended by the American Physical Therapy Association's Parkinson EDGE Task Force (PDEDGE) and takes less than 5 min to administer.

Recent work by Gammon Earhart concludes that while much insight can be gained through assessing isolated coordination of movement, there is increasing recognition that consistency of step-to-step performance across longer periods of gait provides important information about dynamic postural control. Consequently, gait analysis should play an important role in the assessment of coordination in both normal aging and in people with PD. The phase coordination index (PCI) examines temporal coordination of interlimb phasing and variability of this phasing across strides. Decreased and less consistent PCI is shown with aging, PD, and FOG. Earhart also points out that performances of more challenging gait tasks, such as backward walking and turning, are associated with reduced coordination as measured by PCI. These tasks show that older adults are more affected than younger, those with PD are more affected than age-matched controls, and those with PD and history of freezing are more affected than those with PD, but no history of freezing. Assessment of coordination and consistency over a longer duration, including when faced with dual-task challenges, is key to a comprehensive evaluation of dynamic postural control in both older adults and those with PD [78].

Gait analysis is important because gait speed has been suggested to be "almost the perfect measure". It correlates well with functional ability and balance confidence and has the ability to predict future health status and functional decline [79]. Self-selected walking speed has also been found to be reliable, valid, sensitive, and specific. Both age and sex gait speed norms have been derived, along with correlations for functional mobility that range from "household ambulator" to "crosswalk and community ambulatory". Gait speed can also be used to determine amount of assistance needed [79]. Horak et al. suggest the use of the Balance Evaluation Systems Test (BESTest) as a reliable and valid means to determine balance problems for patients and appropriately direct treatment [80]. The test is designed to identify underlying postural control mechanisms that may be responsible for poor functional balance.

21.5 Novel Approaches to Multimodal Exercise

In 2009, King and Horak introduced a novel framework of progressive sensorimotor agility exercises to delay mobility disability in people with PD [81]. Their framework has a strong evidence-based foundation and includes challenges to coordination and cognitive interference. These principles can be incorporated into an ongoing exercise program for both people with PD and those with mobility impairments as a result of stroke or from the normal aging process. The exercises should:

- Be complex, multi-segmental, and whole body and require quick selection and sequencing of motor programs
- Address axial rigidity
- Be task and environment specific
- Teach patients to increase speed, amplitude, and temporal pacing of self-initiated and reactive movements
- Involve complex gait training and in environments where freezing typically occurs
- Facilitate increased use of proprioceptive information and decreased visual reliance
- Introduce cognitive constraints to progress an agility or mobility task

Preliminary evidence shows the effectiveness of coordination and dual-task training for older adults and people with PD. A 2012 pilot randomized controlled trial found that once weekly group circuit training, with or without simultaneous cognitive tasks, resulted in significantly improved walking speed among older adults. However, group-format dual-task training once per week did not improve walking time or dual-task cost on an obstacle negotiation task [77]. Small studies and pilot studies suggest that divided attention training can lead to larger steps, increased speed, and decreased gait variability during gait under dual-task conditions in people with PD [82, 83]. A more recent randomized, controlled pilot study confirmed the efficacy of specific physical therapy intervention focused on asymmetry of motor control during gait [84].

More research has been completed on physical therapy interventions for FOG. Although the neural mechanism underlying FOG in PD is poorly understood, prolonged initial step duration is a physical predictor of FOG in PD. Therapists can easily incorporate specific interventions designed to decrease this step duration into a fall prevention training program for PD [85]. Other studies have shown that an individualized program of auditory cueing (using metronomes or similar devices) is easy and inexpensive to implement and has an immediate effect on gait. Improvements are seen in walking speed, stride length, and freezing [86–88]. Auditory and visual cues in combination with treadmill training have been shown to be effective in improving functional indicators for people with PD + FOG. The treadmill likely acts as a supplementary external cue and works to decrease asymmetry in gait [89, 90]. Another intervention target for decreasing FOG is improvement of motor set switching. For example, people with PD could repeatedly practice changing step direction from one trial to the next [91]. Because FOG is one of the most disabling symptoms of PD and tends to occur more often in the advanced disease state, therapists should also consider incorporating compensatory techniques, modification of the patient's home environment, and care partner/caregiver training into the treatment plan.

Similar to the beneficial effects of aerobic exercise shown in older adults and PD, several studies have demonstrated the benefits of treadmill-based walking exercise interventions in individuals with chronic stroke. Macko and colleagues showed that participation in a 6-month treadmill-based aerobic exercise program

resulted in greater increases in cardiovascular fitness and walking function in individuals with chronic post-stroke hemiparesis [92, 93]. Considerable evidence supports the incorporation of cardiorespiratory and aerobic exercise training within post-stroke rehabilitation programs to improve post-stroke gait and balance function [94, 95]. Single-session as well as long-term training studies in stroke survivors have shown that in addition to cardiovascular benefits obtained by aerobic exercise, such as fast treadmill walking and gait training at a faster than self-selected speed, also imparts beneficial effects on biomechanical gait impairments, gait asymmetry, and muscle activation post-stroke [96–98].

Consistent with the beneficial effects of exercise on neuroplasticity and cognition in older adults, evidence in stroke research also suggests that exercise promotes neuroplasticity by increasing brain-derived neurotrophic factor (BDNF) production and can facilitate motor learning during post-stroke rehabilitation [99, 100].

Ongoing studies are evaluating if physical exercise programs can prevent cognitive decline in individuals after a minor ischemic stroke [101]. Also, similar to PD and older adults, community-based and group exercise programs have potential for maintaining or improving post-stroke motor function [102]. Over the past decade, interest in stroke rehabilitation research has grown toward development of novel exercise interventions that capitalize on combined approaches, such as treadmill training plus functional electrical stimulation [103, 104], task-specific exercises, noninvasive brain stimulation [105], and toward treatments that maximally harness neuroplasticity.

While it is clear that exercise addresses mobility issues in older adults and those with PD, recent investigations into the cognitive effects of exercise have come to the forefront. A recent review considered six preclinical studies in rodent models of PD [106]. The mixed results revealed some positive effects of exercise on cognition in these rodents, improving long-term memory, motor learning, and short-term social memory. Along with these behavioral changes were neurobiological changes including upregulation of the neurotrophic factors, brain-derived neurotrophic factor (BDNF), and glial cell derived neurotrophic factor (GDNF) in striatum and increased dopamine in the striatum. These findings are in line with three proposed mechanisms of improved cognition via exercise specifically for individuals with PD: enhanced availability of dopamine (DA) projections to the dorsal and/or ventral striatum, increased neurotrophic factor availability, and/or decreased neuroinflammation in the basal ganglia. The same review also covered recent human studies and found eight studies that demonstrated improved cognitive function as a result of exercise [106].

Other researchers have demonstrated both motor and cognitive effects of dance exercise. Tango instruction has led to improvement in PD mobility and QOL versus other partnered dances [107], non-partnered dance [108], and generalized exercise [109]. McKee and Hackney demonstrated that, in addition to expected motor improvements, 30 h of adapted tango over 12 weeks improved disease severity and spatial cognition (as measured with the mental imagery Brooks task) in individuals with mild-moderate PD [110]. Importantly, the exercise dose in this study—30 h over 10–12 weeks—exceeds weekly exercise dosage

recommendations for deconditioned older adults with chronic illness [111], which may have contributed to cognitive and motor improvements. Tango could be considered light-moderate exercise. Participants are stepping at 60–120 beats/min (tempi of tango music) and expending at least 3 metabolic equivalent of tasks (METs) per minute [112]. Thus, cognitive gains noted in the adapted tango group could have occurred because aerobic exercise has beneficial effects upon cognition [113, 114]. Notably, the researchers in this study ruled out partnered/social learning and interaction as being responsible for gains. Thus, adapted tango elements, including structured motor components that engage memory of steps and directions while encouraging keen awareness of spatial relationships, could have contributed to cognitive gains.

Several other small studies have examined the benefits of exercise for those with PD on various aspects of cognition. Six months of generalized moderate-intensity, multimodal physical training (consisting of aerobic, resistance, coordination, and balance elements) led to improvements in the capacity for abstraction and mental flexibility as measured by the Wisconsin Card Sorting Task in ten older individuals with PD in comparison to a non-exercising control group [115]. Fifteen individuals with PD who participated in programs of anabolic and aerobic exercise two times weekly for 12 weeks showed improvements in verbal fluency [116]. Low-intensity passive cycling on a tandem bike, in a "forced exercise" situation one time per week over 4 weeks, has led to improvements by 19 people with PD on the Trail Making Test A&B, an executive function measure [117]. Dos Santos Mendes et al. assigned 16 individuals with early PD to Wii Fit training, to evaluate the motor and cognitive demands of the games on people with PD, in comparison to 11 healthy older adults. Compared to healthy controls, those with PD had no deficit in motor learning or retention on seven of the ten games, but had marked learning deficits on three games. However, the PD cohort was able to transfer motor ability gained from the games to a similar, but untrained task [118]. Muller and Muhlack investigated the effects of a single bout (rather than an intervention) of high-intensity endurance aerobic exercise (heart rate-targeted cycling) or rest following L-dopa administration on reaction time and complex movement sequence ability in 22 individuals with PD in a crossover design. Participants improved on reaction time, tapping rate, and peg insertion interval time after exercise, whereas they gave fewer correct answers after rest, and reaction time increased after rest [119].

These recent studies are encouraging and provide preliminary evidence that supports the effects of exercise on aspects of cognition in addition to motor gains, for those with PD. The findings by Uc et al. are encouraging because they agree with research supporting the beneficial effects of aerobic exercise on cognition in older adults, but these findings must be replicated in Phase III clinical trials. Further, all of these studies employed a range of cognitive outcome tasks, and improvements were limited to one or two executive function measures. The exception is the improvement noted by McKee and Hackney, which provides novel evidence that supports further study into application of complex exercise programs, like adapted tango, for improving cognition. Such study is warranted and definitely needed [120] given the prevalence of MCI in PD, which affects the spectrum of

mental function, and has deleterious effects on mobility because of a motor-cognitive interaction.

21.6 Hypotheses for Mechanisms Underlying Deficit and Recovery in Stroke and PD

Neuroplasticity is the phenomenon of changes or adaptations in structure or function of the nervous system on the basis of experience and practice. Plastic changes can occur on anatomical, molecular, genetic, structural, and functional levels within the nervous system. A possible neurobiological mechanism underlying the positive effects of exercise is the increased synthesis and release of neurotransmitters and neurotrophins, which could enhance neurogenesis, angiogenesis, and thus neuroplasticity [121]. Additionally, reorganization of cortical representations, synaptogenesis, and synaptic potentiation play a role.

Typically, increased usage of or skill acquisition by one body part is followed by increase spread in the cortical map for that body part. Some changes have been attributed to competitive plasticity, referring to competition between body parts for limited cortical space, while others attribute the changes to the unmasking of previously silent or inactive neural connections. Changes in the horizontal spread of cortical representations of a particular body segment or movement in response to a change in stimulus lead to reorganization of cortical maps. Changes in map representations with training and experience have been demonstrated in both animal and human models of injury [122, 123].

Significant progress has been made in understanding how neuroplasticity mechanisms primed through physical activity exert an effect on the brain. Exercise effects on hippocampal neuronal regeneration are especially well documented in populations including stroke, PD, and normal aging [124–127]. Erickson and colleagues longitudinally demonstrated 1–2 % increases in hippocampal volume following a year- long aerobic exercise intervention in older adults. Significantly, a non-aerobic exercise control group experienced a 1–2 % degradation of hippocampal volume over that same time course [128]. Further, researchers have examined associations between cardiovascular fitness levels and the density of gray and white matter in adults ages 55–79. Older adults with greater levels of cardiovascular fitness had significantly less atrophy of gray matter in the frontal cortex as well as significantly less tissue loss in both the anterior and posterior white matter tracts [129].

Alterations in structure may correlate to improved behavioral and functional outcomes. Neuronal regeneration in the hippocampus is critical because of its vital role in memory formation and spatial navigation. In the Erickson study, the increase in hippocampal volume following exercise was associated with more accurate performance on a spatial memory task. However, Ten Brinke and colleagues demonstrated that increased left hippocampal volume was significantly associated with greater loss in word recollection after interference. In the same sample, aerobic training significantly improved word recall after a delay as measured by The Rey

Auditory Verbal Learning Test [130]. Other factors, such as white matter degeneration, may significantly moderate the association between brain volume and cognitive performance. For example, the connectivity between the hippocampus and other brain areas may be greatly disrupted by white matter abnormalities, which is likely the case in MCI [131]. Thus, increasing hippocampal volume alone, through aerobic, other exercise, or other means, in older populations may not always result in improved memory performance.

Similarly, demonstrated enhancement in frontal connectivity associated with aerobic fitness is correlated with improvements in frontally mediated EFs. For example, aerobic exercise is associated with improvements in attention and processing speed, EF, and memory in healthy and impaired individuals, including aged individuals [132]. Seminal work demonstrated that 124 older adults randomly assigned to receive aerobic training experienced substantial improvement in performance of tasks dependent on executive control processes and the integrity of the prefrontal and frontal cortex [133]. A recent study assigned older individuals (65 years and over) to ride stationary bicycles for three "spin" sessions per week for 12 weeks in comparison to a control group. The exercise group demonstrated significantly higher category verbal fluency after the intervention. Further, follow-up analysis demonstrated significant correlations between increase in cardiovascular fitness and improvements in verbal fluency [134]. Most notably, these results report that the effects of resistance training on executive cognitive functions are selective, i.e., resistance training enhanced selective attention and conflict resolution in older women, but cognitive abilities associated with manipulating verbal information in working memory and shifting between task sets or instructions were unaffected. The mechanism for these changes comes from cross-sectional studies, which indicate that exercise may mitigate decreases in vital cortical inhibitory mechanisms in older adults [135, 136].

Not all of the effects of exercise are mediated by neurogenesis in the neocortex. Exercise has effects in other CNS structures particularly implicated in PD including substantia nigra and basal ganglia [137–139], corticostriatal synapses [140], and the cerebellum [141].

In individuals with PD, neuroprotective mechanisms of exercise may be related to engagement in ongoing, vigorous exercise that increases heart rate and oxygen uptake [142]. A mechanism, through which aerobic activity engenders improvement, may be plasticity-related changes in synaptogenesis, angiogenesis, and neurogenesis [143]. "Forced exercise" has been shown to be an interesting potential mechanism of rehabilitation in the case of tandem biking [144]. The complex movement patterns experienced in adapted tango for people with PD may cause beneficial neural plasticity in neural pathways for movement. Multiple studies demonstrate that improvements in mobility, balance, spatial cognition, and disease severity may be retained for up to 3 months after adapted tango in individuals with mild-moderate PD [107–110, 145]. A link between activity, mental engagement, and neural pathways may be primed by dancing, which involves complex, unfamiliar tasks like walking backward, problem solving, and movement improvisation.

Going forward, uncovering mechanisms may be the most important area of discovery for enhancing non-pharmacological interventions that address mobility and motor-cognitive issues in older adults with and without movement disorders. The knowledge and principles gained could impact not only exercise disciplines, but also pharmacological, surgical, physical, and occupational therapy for older adults with and without neurological disorders. Future work related to neurobiological mechanisms will be crucial to informing rehabilitative interventions as well as pharmacological and surgical treatments. Imaging with positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) hold promise as well, given that lower limb neural pathways, implicated in exercise, are virtually unexplored in connection with rehabilitative efforts in PD.

Neuroplasticity mechanisms underlying recovery of sensorimotor function following stroke have been extensively investigated. During days and weeks following a stroke, changes in synaptic efficacy of cortical neurons begin to emerge in and immediately around the lesioned cortex, in distant parts of the side affected by the stroke, as well as in the contra-lesional cortex. Assessment of corticomotor excitability using transcranial magnetic stimulation (TMS) reveals depressed cortical excitability in the lesioned motor cortex early after stroke and a slow increase in corticomotor excitability over the course of recovery of post-stroke function [146]. Studies using TMS-derived measures also suggest that an abnormal imbalance of interhemispheric inhibition, i.e., increased inhibition of the lesioned hemisphere by the non-lesioned hemisphere, may be an important neural correlate underlying post-stroke motor impairment, as well as a potential target for post-stroke rehabilitation. Imaging studies reveal marked changes in brain activation during movements of the paretic limb post-stroke. Interestingly, fMRI evidence of bilateral activation during unilateral movements is also observed in older adults as well as in young individuals during performance of complex motor tasks, suggesting that overlapping neural mechanisms may be at play in aging and movement disorders, such as stroke.

Plausibly, greater activity in the unaffected hemisphere and secondary motor areas is a mechanism through which activity in residual neural networks compensates for the functions lost by the cortical tissue damaged due to the stroke (also referred to as the vicariation model of stroke recovery) [146]. In contrast, the interhemispheric competition model of stroke recovery posits that lesion to one hemisphere due to stroke disrupts the balance of interhemispheric interactions and excess activity in the non-lesioned hemisphere can be detrimental to function. Post-stroke cortical reorganization is complex with high interindividual variability, and its mechanisms and causal factors are still incompletely understood. A better understanding of the neuroplasticity mechanisms underlying post-stroke recovery can help develop new treatments and clinical decision-making protocols for maximizing sensorimotor function and QOL post-stroke.

21.7 Conclusion

Rehabilitation to restore and/or improve motor-cognitive function in older adults with neurological insult must be efficient and effective, because adherence to an exercise regimen is critical. Thus, identifying aspects of exercise and rehabilitative therapy, which are most responsible for benefits to mobility- and cognitive-mediating symptoms, i.e., axial impairment and depression, will impact adherence to exercise. Understanding these crucial aspects will help determine foundational principles of exercise training to evince motor and cognitive maintenance and/or gains. With improved and/or maintained independence via enhanced mobility, and targeted application of neuroplasticity principles, the overall goal of improving mobility and motor-cognitive function in older adults with and without neurological disorder can be reached.

References

1. Melton LJ, Leibson CL, Achenbach SJ, Bower JH, Maraganore DM, Oberg AL, et al. Fracture risk after the diagnosis of Parkinson's disease: influence of concomitant dementia. Mov Disord. 2006;21(9):1361–7.
2. Johnell O, Melton LJ, Atkinson EJ, O'Fallon WM, Kurland LT. Fracture risk in patients with parkinsonism: a population-based study in Olmsted County, Minnesota. Age Ageing. 1992;21(1):32–8.
3. Hely MA, Reid WGJ, Adena MA, Halliday GM, Morris JGL. The Sydney multicenter study of Parkinson's disease: the inevitability of dementia at 20 years. Mov Disord. 2008;23 (6):837–44.
4. Bloem BR, Hausdorff JM, Visser JE, Giladi N. Falls and freezing of gait in Parkinson's disease: a review of two interconnected, episodic phenomena. Mov Disord. 2004;19 (8):871–84.
5. Stewart KC, Fernandez HH, Okun MS, Jacobson CE, Hass CJ. Distribution of motor impairment influences quality of life in Parkinson's disease. Mov Disord. 2008;23 (10):1466–8.
6. Chen J, Devine A, Dick IM, Dhaliwal SS, Prince RL. Prevalence of lower extremity pain and its association with functionality and quality of life in elderly women in Australia. J Rheumatol. 2003;30(12):2689–93.
7. Camicioli R, Oken BS, Sexton G, Kaye JA, Nutt JG. Verbal fluency task affects gait in Parkinson's disease with motor freezing. J Geriatr Psychiatry Neurol. 1998;11(4):181–5.
8. Bond JM, Morris M. Goal-directed secondary motor tasks: their effects on gait in subjects with Parkinson disease. Arch Phys Med Rehabil. 2000;81(1):110–6.
9. O'Shea S, Morris ME, Iansek R. Dual task interference during gait in people with Parkinson disease: effects of motor versus cognitive secondary tasks. Phys Ther. 2002;82(9):888–97.
10. Hausdorff JM, Balash J, Giladi N. Effects of cognitive challenge on gait variability in patients with Parkinson's disease. J Geriatr Psychiatry Neurol. 2003;16(1):53–8.
11. Maki BE. Gait changes in older adults: predictors of falls or indicators of fear. J Am Geriatr Soc. 1997;45(3):313–20.
12. Rochester L, Hetherington V, Jones D, Nieuwboer A, Willems AM, Kwakkel G, et al. Attending to the task: interference effects of functional tasks on walking in Parkinson's disease and the roles of cognition, depression, fatigue, and balance. Arch Phys Med Rehabil. 2004;85(10):1578–85.

13. Forster A, Young J. Incidence and consequences of falls due to stroke: a systematic inquiry. BMJ. 1995;311(6997):83–6.
14. Andersson AG, Kamwendo K, Appelros P. Fear of falling in stroke patients: relationship with previous falls and functional characteristics. Int J Rehabil Res. 2008;31(3):261–4.
15. Mackintosh SF, Goldie P, Hill K. Falls incidence and factors associated with falling in older, community-dwelling, chronic stroke survivors (>1 year after stroke) and matched controls. Aging Clin Exp Res. 2005;17(2):74–81.
16. Mackintosh SF, Hill K, Dodd KJ, Goldie P, Culham E. Falls and injury prevention should be part of every stroke rehabilitation plan. Clin Rehabil. 2005;19(4):441–51.
17. Kiyota Y, Hase K, Nagashima H, Obara T, Liu M. Adaptation process for standing postural control in individuals with hemiparesis. Disabil Rehabil. 2011;33(25–26):2567–73.
18. Mansfield A, Danells CJ, Inness E, Mochizuki G, McIlroy WE. Between-limb synchronization for control of standing balance in individuals with stroke. Clin Biomech (Bristol, Avon). 2011;26(3):312–7.
19. Inness EL, Mansfield A, Lakhani B, Bayley M, McIlroy WE. Impaired reactive stepping among patients ready for discharge from inpatient stroke rehabilitation. Phys Ther. 2014;94 (12):1755–64.
20. Hendrickson J, Patterson KK, Inness EL, McIlroy WE, Mansfield A. Relationship between asymmetry of quiet standing balance control and walking post-stroke. Gait Posture. 2014;39 (1):177–81.
21. Adegoke BO, Olaniyi O, Akosile CO. Weight bearing asymmetry and functional ambulation performance in stroke survivors. Glob J Health Sci. 2012;4(2):87–94.
22. Domellof ME, Elgh E, Forsgren L. The relation between cognition and motor dysfunction in drug-naive newly diagnosed patients with Parkinson's disease. Mov Disord. 2011;26 (12):2183–9.
23. Pulvermuller F. Brain reflections of words and their meaning. Trends Cogn Sci. 2001;5 (12):517–24.
24. Kemmerer D, Castillo JG, Talavage T, Patterson S, Wiley C. Neuroanatomical distribution of five semantic components of verbs: evidence from fMRI. Brain Lang. 2008;107(1):16–43.
25. Raposo A, Moss HE, Stamatakis EA, Tyler LK. Modulation of motor and premotor cortices by actions, action words and action sentences. Neuropsychologia. 2009;47(2):388–96.
26. Grossi JA, Maitra KK, Rice MS. Semantic priming of motor task performance in young adults: implications for occupational therapy. Am J Occup Ther. 2007;61(3):311–20.
27. Rodriguez AD. Semantic-motor representations: effects on language and motor production. US: ProQuest Information & Learning; 2010.
28. Dalla Volta R, Gianelli C, Campione GC, Gentilucci M. Action word understanding and overt motor behavior. Exp Brain Res. 2009;196(3):403–12.
29. Raz N. Aging of the brain and its impact on cognitive performance: integration of structural and functional findings. In: Craik FIM, Salthouse TA, editors. The handbook of aging and cognition. 2nd ed. Mahwah, NJ: Lawrence Erlbaum; 2000. p. 1–90.
30. Cabeza R. Functional neuroimaging of cognitive aging. In: Cabeza R, Kingstone A, editors. Handbook of functional neuroimaging of cognition. Cambridge, MA: MIT Press; 2001. p. 331–77.
31. Rowe J, Stephan KE, Friston K, Frackowiak R, Lees A, Passingham R. Attention to action in Parkinson's disease: impaired effective connectivity among frontal cortical regions. Brain. 2002;125(2):276–89.
32. Al-Yahya E, Dawes H, Smith L, Dennis A, Howells K, Cockburn J. Cognitive motor interference while walking: a systematic review and meta-analysis. Neurosci Biobehav Rev. 2011;35(3):715–28.
33. Nieuwboer A, Rochester L, Muncks L, Swinnen SP. Motor learning in Parkinson's disease: limitations and potential for rehabilitation. Parkinsonism Relat Disord. 2009;15 Suppl 3: S53–8.

34. Poletti M, Frosini D, Pagni C, Baldacci F, Nicoletti V, Tognoni G, et al. Mild cognitive impairment and cognitive-motor relationships in newly diagnosed drug-naive patients with Parkinson's disease. J Neurol Neurosurg Psychiatry. 2012;83(6):601–6.
35. Baker K, Rochester L, Nieuwboer A. The immediate effect of attentional, auditory, and a combined cue strategy on gait during single and dual tasks in Parkinson's disease. Arch Phys Med Rehabil. 2007;88(12):1593–600.
36. Morris ME, Huxham FE, McGinley J, Iansek R. Gait disorders and gait rehabilitation in Parkinson's disease. Adv Neurol. 2001;87:347–61.
37. Freedland RL, Festa C, Sealy M, McBean A, Elghazaly P, Capan A, et al. The effects of pulsed auditory stimulation on various gait measurements in persons with Parkinson's disease. NeuroRehabilitation. 2002;17(1):81–7.
38. Jiang Y, Norman KE. Effects of visual and auditory cues on gait initiation in people with Parkinson's disease. Clin Rehabil. 2006;20(1):36–45.
39. Cahn-Weiner DA, Boyle PA, Malloy PF. Tests of executive function predict instrumental activities of daily living in community-dwelling older individuals. Appl Neuropsychol. 2002;9(3):187–91.
40. Li KZH, Lindenberger U, Freund AM, Baltes PB. Walking while memorizing: age-related differences in compensatory behavior. Psychol Sci. 2001;12(3):230–7.
41. Iansek R, Bradshaw J, Phillips J, Morris ME, Cunnington R. The functions of the basal ganglia and the paradox of stereotaxic surgery in Parkinson's disease. Brain. 1995;118(Part 6):1613–5.
42. Temel Y, Blokland A, Steinbusch HW, Visser-Vandewalle V. The functional role of the subthalamic nucleus in cognitive and limbic circuits. Prog Neurobiol. 2005;76(6):393–413.
43. Dalrymple-Alford J, Kalders A, Jones R, Watson R. A central executive deficit in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry. 1994;57:360–7.
44. Plummer P, Eskes G, Wallace S, Giuffrida C, Fraas M, Campbell G, et al. Cognitive-motor interference during functional mobility after stroke: state of the science and implications for future research. Arch Phys Med Rehabil. 2013;94(12):2565–74. e6.
45. Hyndman D, Pickering RM, Ashburn A. Reduced sway during dual task balance performance among people with stroke at 6 and 12 months after discharge from hospital. Neurorehabil Neural Repair. 2009;23(8):847–54.
46. Subramaniam S, Hui-Chan CW, Bhatt T. Effect of dual tasking on intentional vs. reactive balance control in people with hemiparetic stroke. J Neurophysiol. 2014;112(5):1152–8.
47. Patel P, Bhatt T. Task matters: influence of different cognitive tasks on cognitive-motor interference during dual-task walking in chronic stroke survivors. Top Stroke Rehabil. 2014;21(4):347–57.
48. Assayag EB, Shenhar-Tsarfaty S, Korczyn AD, Kliper E, Hallevi H, Shopin L, et al. Gait measures as predictors of poststroke cognitive function: evidence from the TABASCO study. Stroke. 2015;46(4):1077–83.
49. Hollman JH, Kovash FM, Kubik JJ, Linbo RA. Age-related differences in spatiotemporal markers of gait stability during dual task walking. Gait Posture. 2007;26(1):113–9.
50. Faulkner KA, Redfern MS, Cauley JA, Landsittel DP, Studenski SA, Rosano C, et al. Multitasking: association between poorer performance and a history of recurrent falls. J Am Geriatr Soc. 2007;55(4):570–6.
51. Soriano TA, DeCherrie LV, Thomas DC. Falls in the community-dwelling older adult: a review for primary-care providers. Clin Interv Aging. 2007;2(4):545–54.
52. Kerr B, Condon SM, McDonald LA. Cognitive spatial processing and the regulation of posture. J Exp Psychol Hum Percept Perform. 1985;11(5):617–22.
53. Shumway-Cook A, Woollacott MH. Attentional demands and postural control: the effect of sensory context. J Gerontol A Biol SCi Med Sci. 2000;55A(1):M10–6.
54. Melzer I, Oddsson LI. The effect of a cognitive task on voluntary step execution in healthy elderly and young individuals. J Am Geriatr Soc. 2004;52(8):1255–62.

55. Verghese J, Buschke H, Viola L, Katz M, Hall C, Kuslansky G, et al. Validity of divided attention tasks in predicting falls in older individuals: a preliminary study. J Am Geriatr Soc. 2002;50(9):1572–6.
56. Yogev-Seligmann G, Hausdorff JM, Giladi N. The role of executive function and attention in gait. Mov Disord. 2008;23(3):329–42. quiz 472.
57. Zijlstra A, Ufkes T, Skelton DA, Lundin-Olsson L, Zijlstra W. Do dual tasks have an added value over single tasks for balance assessment in fall prevention programs? A mini-review. Gerontology. 2008;54(1):40–9.
58. Morris M, Iansek R, Smithson F, Huxham F. Postural instability in Parkinson's disease: a comparison with and without a concurrent task. Gait Posture. 2000;12(3):205–16.
59. Smithson F, Morris ME, Iansek R. Performance on clinical tests of balance in Parkinson's disease. Phys Ther. 1998;78(6):577–92.
60. Ashburn A, Stack E, Pickering RM. CD. W. Predicting fallers in a community-based sample of people with Parkinson's disease. Gerontology. 2001;47(5):277–81.
61. Winogrodzka A, Wagenaar RC, Booij J, Wolters EC. Rigidity and bradykinesia reduce interlimb coordination in Parkinsonian gait. Arch Phys Med Rehabil. 2005;86(2):183–9.
62. Roemmich RT, Field AM, Elrod JM, Stegemoller EL, Okun MS, Hass CJ. Interlimb coordination is impaired during walking in persons with Parkinson's disease. Clin Biomech (Bristol, Avon). 2013;28(1):93–7.
63. Plotnik M, Giladi N, Hausdorff JM. Bilateral coordination of gait and Parkinson's disease: the effects of dual tasking. J Neurol Neurosurg Psychiatry. 2009;80(3):347–50.
64. Tanahashi T, Yamamoto T, Endo T, Fujimura H, Yokoe M, Mochizuki H, et al. Noisy interlimb coordination can be a main cause of freezing of gait in patients with little to no parkinsonism. PLoS One. 2013;8(12):e84423.
65. Spildooren J, Vercruysse S, Desloovere K, Vandenberghe W, Kerckhofs E, Nieuwboer A. Freezing of gait in Parkinson's disease: the impact of dual-tasking and turning. Mov Disord. 2010;25(15):2563–70.
66. Brown MJ, Almeida QJ, Rahimi F. The dopaminergic system in upper limb motor blocks (ULMB) investigated during bimanual coordination in Parkinson's disease (PD). J Neurol. 2015;262(1):41–53.
67. Kerr GK, Worringham CJ, Cole MH, Lacherez PF, Wood JM, Silburn PA. Predictors of future falls in Parkinson disease. Neurology. 2010;75(2):116–24.
68. Awad LN, Palmer JA, Pohlig RT, Binder-Macleod SA, Reisman DS. Walking speed and step length asymmetry modify the energy cost of walking after stroke. Neurorehabil Neural Repair. 2015;29(5):416–23.
69. Patterson KK, Parafianowicz I, Danells CJ, Closson V, Verrier MC, Staines WR, et al. Gait asymmetry in community-ambulating stroke survivors. Arch Phys Med Rehabil. 2008;89(2):304–10.
70. van Swigchem R, Roerdink M, Weerdesteyn V, Geurts AC, Daffertshofer A. The capacity to restore steady gait after a step modification is reduced in people with poststroke foot drop using an ankle-foot orthosis. Phys Ther. 2014;94(5):654–63.
71. van Swigchem R, van Duijnhoven HJ, den Boer J, Geurts AC, Weerdesteyn V. Deficits in motor response to avoid sudden obstacles during gait in functional walkers poststroke. Neurorehabil Neural Repair. 2013;27(3):230–9.
72. Choi W, Lee G, Lee S. Effect of the cognitive-motor dual-task using auditory cue on balance of surviviors with chronic stroke: a pilot study. Clin Rehabil. 2015;29(8):763–70.
73. Choi JH, Kim BR, Han EY, Kim SM. The effect of dual-task training on balance and cognition in patients with subacute post-stroke. Ann Rehabil Med. 2015;39(1):81–90.
74. Wang XQ, Pi YL, Chen BL, Chen PJ, Liu Y, Wang R, et al. Cognitive motor interference for gait and balance in stroke: a systematic review and meta-analysis. Eur J Neurol. 2015;22(3):555–e37.

75. Shumway-Cook A, Brauer S, Woollacott M. Predicting the probability for falls in community-dwelling older adults using the timed up & go test. Phys Ther. 2000;80 (9):896–903.
76. Maranhao-Filho PA, Maranhao ET, Lima MA, Silva MM. Rethinking the neurological examination II: dynamic balance assessment. Arq Neuropsiquiatr. 2011;69(6):959–63.
77. Plummer-D'Amato P, Cohen Z, Daee NA, Lawson SE, Lizotte MR, Padilla A. Effects of once weekly dual-task training in older adults: a pilot randomized controlled trial. Geriatr Gerontol Int. 2012;12(4):622–9.
78. Earhart GM. Dynamic control of posture across locomotor tasks. Mov Disord. 2013;28 (11):1501–8.
79. Fritz S, Lusardi M. White paper: "walking speed: the sixth vital sign". J Geriatr Phys Ther. 2009;32(2):46–9.
80. Horak FB, Wrisley DM, Frank J. The balance evaluation systems test (BESTest) to differentiate balance deficits. Phys Ther. 2009;89(5):484–98.
81. King LA, Horak FB. Delaying mobility disability in people with Parkinson disease using a sensorimotor agility exercise program. Phys Ther. 2009;89(4):384–93.
82. Brauer SG, Morris ME. Can people with Parkinson's disease improve dual tasking when walking? Gait Posture. 2010;31(2):229–33.
83. Yogev-Seligmann G, Giladi N, Brozgol M, Hausdorff JM. A training program to improve gait while dual tasking in patients with Parkinson's disease: a pilot study. Arch Phys Med Rehabil. 2012;93(1):176–81.
84. Ricciardi L, Ricciardi D, Lena F, Plotnik M, Petracca M, Barricella S, et al. Working on asymmetry in Parkinson's disease: randomized, controlled pilot study. Neurol Sci. 2015;36 (8):1337–43.
85. Chong RK, Lee KH, Morgan J, Wakade C. Duration of step initiation predicts freezing in Parkinson's disease. Acta Neurol Scand. 2015;132(2):105–10.
86. Ledger S, Galvin R, Lynch D, Stokes EK. A randomised controlled trial evaluating the effect of an individual auditory cueing device on freezing and gait speed in people with Parkinson's disease. BMC Neurol. 2008;8:46.
87. Lim I, van Wegen E, de Goede C, Deutekom M, Nieuwboer A, Willems A, et al. Effects of external rhythmical cueing on gait in patients with Parkinson's disease: a systematic review. Clin Rehabil. 2005;19(7):695–713.
88. Ellis T, de Goede CJ, Feldman RG, Wolters EC, Kwakkel G, Wagenaar RC. Efficacy of a physical therapy program in patients with Parkinson's disease: a randomized controlled trial. Arch Phys Med Rehabil. 2005;86(4):626–32.
89. Frazzitta G, Maestri R, Uccellini D, Bertotti G, Abelli P. Rehabilitation treatment of gait in patients with Parkinson's disease with freezing: a comparison between two physical therapy protocols using visual and auditory cues with or without treadmill training. Mov Disord. 2009;24(8):1139–43.
90. Frazzitta G, Pezzoli G, Bertotti G, Maestri R. Asymmetry and freezing of gait in parkinsonian patients. J Neurol. 2013;260(1):71–6.
91. Smulders K, Esselink RA, Bloem BR, Cools R. Freezing of gait in Parkinson's disease is related to impaired motor switching during stepping. Mov Disord. 2015;30(8):1090–7
92. Macko RF, Ivey FM, Forrester LW, Hanley D, Sorkin JD, Katzel LI, et al. Treadmill exercise rehabilitation improves ambulatory function and cardiovascular fitness in patients with chronic stroke: a randomized, controlled trial. Stroke. 2005;36(10):2206–11.
93. Silver KH, Macko RF, Forrester LW, Goldberg AP, Smith GV. Effects of aerobic treadmill training on gait velocity, cadence, and gait symmetry in chronic hemiparetic stroke: a preliminary report. Neurorehabil Neural Repair. 2000;14(1):65–71.
94. Saunders DH, Sanderson M, Brazzelli M, Greig CA, Mead GE. Physical fitness training for stroke patients. Cochrane Database Syst Rev. 2013;10, CD003316.
95. Brazzelli M, Saunders DH, Greig CA, Mead GE. Physical fitness training for patients with stroke: updated review. Stroke. 2012;43(4):e39–40.

96. Lamontagne A, Fung J. Faster is better: implications for speed-intensive gait training after stroke. Stroke. 2004;35(11):2543–8.
97. Pohl M, Mehrholz J, Ritschel C, Ruckriem S. Speed-dependent treadmill training in ambulatory hemiparetic stroke patients: a randomized controlled trial. Stroke. 2002;33(2):553–8.
98. Tyrell CM, Roos MA, Rudolph KS, Reisman DS. Influence of systematic increases in treadmill walking speed on gait kinematics after stroke. Phys Ther. 2011;91(3):392–403.
99. Mang CS, Campbell KL, Ross CJ, Boyd LA. Promoting neuroplasticity for motor rehabilitation after stroke: considering the effects of aerobic exercise and genetic variation on brain-derived neurotrophic factor. Phys Ther. 2013;93(12):1707–16.
100. Dimyan MA, Cohen LG. Neuroplasticity in the context of motor rehabilitation after stroke. Nat Rev Neurol. 2011;7(2):76–85.
101. Boss HM, Van Schaik SM, Deijle IA, de Melker EC, van den Berg BT, Scherder EJ, et al. A randomised controlled trial of aerobic exercise after transient ischaemic attack or minor stroke to prevent cognitive decline: the MoveIT study protocol. BMJ Open. 2014;4(12):e007065.
102. Moore SA, Hallsworth K, Jakovljevic DG, Blamire AM, He J, Ford GA, et al. Effects of community exercise therapy on metabolic, brain, physical, and cognitive function following stroke: a randomized controlled pilot trial. Neurorehabil Neural Repair. 2015;29(7):623–35.
103. Kesar TM, Reisman DS, Perumal R, Jancosko AM, Higginson JS, Rudolph KS, et al. Combined effects of fast treadmill walking and functional electrical stimulation on post-stroke gait. Gait Posture. 2011;33(2):309–13.
104. Awad LN, Reisman DS, Kesar TM, Binder-Macleod SA. Targeting paretic propulsion to improve poststroke walking function: a preliminary study. Arch Phys Med Rehabil. 2014;95(5):840–8.
105. Madhavan S, Shah B. Enhancing motor skill learning with transcranial direct current stimulation - a concise review with applications to stroke. Front Psych. 2012;3:66.
106. Murray DK, Sacheli MA, Eng JJ, Stoessl AJ. The effects of exercise on cognition in Parkinson's disease: a systematic review. Transl Neurodegener. 2014;3(1):5.
107. Hackney ME, Earhart GM. Effects of dance on movement control in Parkinson's disease: a comparison of Argentine tango and American ballroom. J Rehabil Med. 2009;41(6):475–81.
108. Hackney ME, Earhart GM. Effects of dance on gait and balance in Parkinson's disease: a comparison of partnered and nonpartnered dance movement. Neurorehabil Neural Repair. 2010;24(4):384–92.
109. Hackney ME, Kantorovich S, Levin R, Earhart GM. Effects of tango on functional mobility in Parkinson's disease: a preliminary study. J Neurol Phys Ther. 2007;31(4):173–9.
110. McKee KE, Hackney ME. The effects of adapted tango on spatial cognition and disease severity in Parkinson's disease. J Mot Behav. 2013;45(6):519–29.
111. Chodzko-Zajko WJ, Proctor DN, Fiatarone Singh MA, Minson CT, Nigg CR, Salem GJ, et al. American College of Sports Medicine position stand. Exercise and physical activity for older adults. Med Sci Sports Exerc. 2009;41(7):1510–30.
112. Heyward VH. Advanced fitness assessment and exercise prescription. 6th ed. Champaign, IL: Human Kinetics; 2010.
113. Kraft E. Cognitive function, physical activity, and aging: possible biological links and implications for multimodal interventions. Neuropsychol Dev Cogn B Aging Neuropsychol Cogn. 2012;19(1–2):248–63.
114. Ratey JJ, Loehr JE. The positive impact of physical activity on cognition during adulthood: a review of underlying mechanisms, evidence and recommendations. Rev Neurosci. 2011;22(2):171–85.
115. Tanaka K, Quadros Jr AC, Santos RF, Stella F, Gobbi LT, Gobbi S. Benefits of physical exercise on executive functions in older people with Parkinson's disease. Brain Cogn. 2009;69(2):435–41.
116. Cruise KE, Bucks RS, Loftus AM, Newton RU, Pegoraro R, Thomas MG. Exercise and Parkinson's: benefits for cognition and quality of life. Acta Neurol Scand. 2011;123(1):13–9.

117. Ridgel AL, Kim CH, Fickes EJ, Muller MD, Alberts JL. Changes in executive function after acute bouts of passive cycling in Parkinson's disease. J Aging Phys Act. 2011;19(2):87–98.
118. dos Santos Mendes FA, Pompeu JE, Modenesi Lobo A, Guedes da Silva K, Oliveira Tde P, Peterson Zomignani A, et al. Motor learning, retention and transfer after virtual-reality-based training in Parkinson's disease–effect of motor and cognitive demands of games: a longitudinal, controlled clinical study. Physiotherapy. 2012;98(3):217–23.
119. Muller T, Muhlack S. Effect of exercise on reactivity and motor behaviour in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry. 2010;81(7):747–53.
120. Hindle JV, Petrelli A, Clare L, Kalbe E. Nonpharmacological enhancement of cognitive function in Parkinson's disease: a systematic review. Mov Disord. 2013;28(8):1034–49.
121. Voss MW, Erickson KI, Prakash RS, Chaddock L, Kim JS, Alves H, et al. Neurobiological markers of exercise-related brain plasticity in older adults. Brain Behav Immun. 2013;28:90–9.
122. Merzenich MM, Nelson RJ, Stryker MP, Cynader MS, Schoppmann A, Zook JM. Somatosensory cortical map changes following digit amputation in adult monkeys. J Comp Neurol. 1984;224(4):591–605.
123. Buonomano DV, Merzenich MM. Cortical plasticity: from synapses to maps. Annu Rev Neurosci. 1998;21:149–86.
124. McCrate ME, Kaspar BK. Physical activity and neuroprotection in amyotrophic lateral sclerosis. Neuromolecular Med. 2008;10(2):108–17.
125. Klaus F, Amrein I. Running in laboratory and wild rodents: differences in context sensitivity and plasticity of hippocampal neurogenesis. Behav Brain Res. 2012;227(2):363–70.
126. Sahay A, Scobie KN, Hill AS, O'Carroll CM, Kheirbek MA, Burghardt NS, et al. Increasing adult hippocampal neurogenesis is sufficient to improve pattern separation. Nature. 2011;472 (7344):466–70.
127. Lau YS, Patki G, Das-Panja K, Le WD, Ahmad SO. Neuroprotective effects and mechanisms of exercise in a chronic mouse model of Parkinson's disease with moderate neurodegeneration. Eur J Neurosci. 2011;33(7):1264–74.
128. Erickson KI, Voss MW, Prakash RS, Basak C, Szabo A, Chaddock L, et al. Exercise training increases size of hippocampus and improves memory. Proc Natl Acad Sci U S A. 2011;108 (7):3017–22.
129. Colcombe SJ, Erickson KI, Raz N, Webb AG, Cohen NJ, McAuley E, et al. Aerobic fitness reduces brain tissue loss in aging humans. J Gerontol A Biol Sci Med Sci. 2003;58 (2):176–80.
130. Ten Brinke LF, Bolandzadeh N, Nagamatsu LS, Hsu CL, Davis JC, Miran-Khan K, et al. Aerobic exercise increases hippocampal volume in older women with probable mild cognitive impairment: a 6-month randomised controlled trial. Br J Sports Med. 2015;49(4):248–54.
131. Rowley J, Fonov V, Wu O, Eskildsen SF, Schoemaker D, Wu L, et al. White matter abnormalities and structural hippocampal disconnections in amnestic mild cognitive impairment and Alzheimer's disease. PLoS One. 2013;8(9):e74776.
132. Voss MW, Heo S, Prakash RS, Erickson KI, Alves H, Chaddock L, et al. The influence of aerobic fitness on cerebral white matter integrity and cognitive function in older adults: results of a one-year exercise intervention. Hum Brain Mapp. 2013;34(11):2972–85.
133. Kramer AF, Hahn S, Cohen NJ, Banich MT, McAuley E, Harrison CR, et al. Ageing, fitness and neurocognitive function. Nature. 1999;400(6743):418–9.
134. Nocera J, McGregor KM, Hass C, Crosson B. 'Spin' exercise improves semantic fluency in previously sedentary older adults. J Aging Phys Act. 2015;23(1):90–4.
135. McGregor KM, Heilman KM, Nocera JR, Patten C, Manini TM, Crosson B, et al. Aging, aerobic activity and interhemispheric communication. Brain Sci. 2012;2(4):634–48.
136. McGregor KM, Nocera JR, Sudhyadhom A, Patten C, Manini TM, Kleim JA, et al. Effects of aerobic fitness on aging-related changes of interhemispheric inhibition and motor performance. Front Aging Neurosci. 2013;5:66.

137. Smith BA, Goldberg NR, Meshul CK. Effects of treadmill exercise on behavioral recovery and neural changes in the substantia nigra and striatum of the 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine-lesioned mouse. Brain Res. 2011;1386:70–80.
138. Wu SY, Wang TF, Yu L, Jen CJ, Chuang JI, Wu FS, et al. Running exercise protects the substantia nigra dopaminergic neurons against inflammation-induced degeneration via the activation of BDNF signaling pathway. Brain Behav Immun. 2011;25(1):135–46.
139. Vucckovic MG, Li Q, Fisher B, Nacca A, Leahy RM, Walsh JP, et al. Exercise elevates dopamine D2 receptor in a mouse model of Parkinson's disease: in vivo imaging with [(1)F] fallypride. Mov Disord. 2010;25(16):2777–84.
140. Cepeda C, Cummings DM, Hickey MA, Kleiman-Weiner M, Chen JY, Watson JB, et al. Rescuing the corticostriatal synaptic disconnection in the R6/2 mouse model of Huntington's disease: exercise, adenosine receptors and ampakines. PLoS Curr. 2010;2. pii: RRN1182.
141. Ben-Ari S, Ofek K, Barbash S, Meiri H, Kovalev E, Greenberg DS, et al. Similar cation channels mediate protection from cerebellar exitotoxicity by exercise and inheritance. J Cell Mol Med. 2012;16(3):555–68.
142. Ahlskog JE. Does vigorous exercise have a neuroprotective effect in Parkinson disease? Neurology. 2011;77(3):288–94.
143. Hirsch MA, Farley BG. Exercise and neuroplasticity in persons living with Parkinson's disease. Eur J Phys Rehabil Med. 2009;45(2):215–29.
144. Alberts JL, Linder SM, Penko AL, Lowe MJ, Phillips M. It is not about the bike, it is about the pedaling: forced exercise and Parkinson's disease. Exerc Sport Sci Rev. 2011;39 (4):177–86.
145. Hackney ME, Earhart GM. Health-related quality of life and alternative forms of exercise in Parkinson disease. Parkinsonism Relat Disord. 2009;15(9):644–8.
146. Di Pino G, Pellegrino G, Assenza G, Capone F, Ferreri F, Formica D, et al. Modulation of brain plasticity in stroke: a novel model for neurorehabilitation. Nat Rev Neurol. 2014;10 (10):597–608.

Part IV

Technology and Rehabilitation Applied to Locomotion and Posture

Gait Initiation in Older People: Concepts, Clinical Implications and Methodology

22

Paula Hentschel Lobo da Costa and Marcus Fraga Vieira

Abstract

Anticipatory postural adjustments are necessary neuromuscular strategies to begin a new gait cycle and to safely take the first step forward. For this purpose, postural transients about to occur with imminent forward motion must be controlled. Impairments in this process must be understood in order to reveal pathophysiological mechanisms underlying dynamic balance problems in the older population. The aim of this chapter is twofold: to firstly explain the typical mechanism of gait initiation (GI), some aspects that affect its pattern and benefits of training interventions for the elderly population and, secondly, to describe computational methods to assess and quantify this mechanism based on force plate measurements.

Keywords

Gait initiation • Anticipatory postural adjustments • Center of pressure • Dynamometry • Older people

22.1 Gait Initiation General Aspects

According to Bernstein [1], the execution of each voluntary movement is successfully accomplished by means of two control components: a focal component (related to the segmental motion that performs a goal-directed movement) and a

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postural component (related to the rest of the body involved in stabilizing reactions). The central nervous system has the task of coordinating these two components, namely movement and posture. In this regard, ***gait initiation*** **(GI)** is an interesting process to be studied from the fundamental point of view, because it involves a precise coordination between posture and movement, as well as from the clinical one, because unbalances during GI have been shown to discriminate between stable and unstable patients [2].

The transition phase from a static to a dynamic condition involves contradictory postural stabilization functions: to prepare to an action and to recover from the perturbation. During this phase, the motor system is in a state of transient disequilibrium, which means that equilibrium will be restored in the next support phase of periodic movements like walking [3]. In this regard, GI is the task used to understand the anticipatory postural adjustments needed to make the transition from standing posture to cyclic walking.

GI is a common task performed in daily life that requires a complex muscular synergy to generate the decoupling of the center of mass (COM) and center of pressure (COP) during standing, which results the COM to move forward [4]. It encompasses the period from the preparation for the first step until the reach of a steady state gait speed.

In standing position COP and COM are close to each other. Before GI begins, ankle plantar flexors of both legs are active to maintain COM in a position anterior to the ankle joints. Now the task is to move the body outside the base of support and at the same time prevent falling [5]. GI begins with the posterior and lateral displacement of the COP toward the limb that will oscillate first, namely the swing limb. This increases the anterior-posterior (AP) and medial-lateral (ML) ground reaction forces that, in turn, cause the displacement of the COM in the opposite direction, forward and toward the supporting foot [6–8]. The result is that COP and COM begin to dissociate.

It has already been demonstrated that dividing the GI into its phases provides more information related to specific mechanisms present in older people than analyzing GI by the overall time [36]. Thus to better understand the GI function the whole event has been divided into phases (Fig. 22.1). Because COP moves to the swing foot before any detectable foot movement, the first phase is called the ***anticipatory postural adjustments (APA) phase*** (1–2 in Fig. 22.1), where the anticipatory adjustments to initiate gait begin.

During the APA phase, the COP backward movement and toward the swing limb is considered an efficient strategy, because it generates the impulse needed to begin walking without requiring the COM to move forward of the base of support [9]. The second phase is called ***swing foot unloading phase*** (2–3 in Fig. 22.1) and it begins with heel-off of the swing foot and COP movement to the support limb, which is necessary for the subsequent unloading of the swing leg and for shifting body weight from the swing to the support limb, while COM is accelerated forward. By the end of the second phase, the transition from double to single support has occurred. The COP now starts to accelerate forward under the support limb [6] and this third phase is called ***support foot unloading phase*** (3–4 in Fig. 22.1),

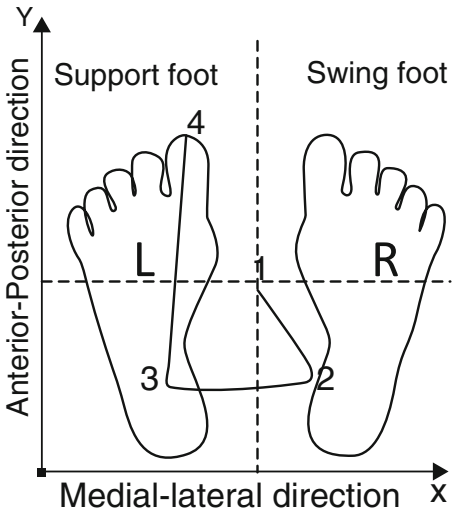

Fig. 22.1 Typical COP path during GI. 1–2: phase 1-APA phase; 2–3: phase 2-Swing foot unloading phase; 3–4: phase 3-Support foot unloading phase. "L" stands for left and "R" for right

which encompasses the roll-over of the support foot, while COP moves anteriorly until toe-off of the same foot is completed. COM moves forward through the whole GI process.

The most challenging event for stability control during GI is recognized to be the instant prior to the first contact of the swing foot [2]. GI process will be completed when the body reaches a constant walking speed, about the beginning of the third and the end of the eighth step [10]. In steady state walking or running the COM is always outside the base of support, except during the double support phase in walking [5]. During walking and running, the swing limb has a trajectory that searches balance conditions during the next support phase and for this reason this state of balance has been described as a dynamic balance [5]. Thus the main goal of GI is to forward propel the body to perform the first step of a new walking cycle while maintaining dynamic balance in order to deal with instabilities during the transition from standing to walking.

The APA in GI is mediated by a central motor program [11]. Muscles of the lower extremities are activated stereotypically and create moments of force around the ankles and hips that rotate the body like an inverted pendulum [12].

During the APA phase the stereotyped activities of ankle muscles are known to be the inhibition of m. soleus (S) and m. gastrocnemius (GA) that are followed by activation of m. tibialis anterior (TA) in both legs [13]. The anticipatory m. soleus inhibition and m. tibialis anterior activation are not observed in all young healthy adults [15] and this functional variability of APA behavior is probably influenced by initial trunk posture (backward or forward inclination), speed of the first step and initial tonic m. soleus activity [15].

In the frontal plane, the ML COP displacements during GI are controlled by coordinated action of hip abductor and adductor muscles [14]. While COP moves backward by ankle muscles synergism, swing limb hip abductors move the COP to

the swing limb [5] during the anticipatory phase. Next, support foot hip abductors move the COP to the support limb and the swing limb is unloaded. At the time of swing foot toe-off, the support limb is carrying the total weight of the body. Then, the support limb plantar flexors increase activity and the COP moves forward under this foot [5]. While the stereotyped activity of ankle muscle is well documented, we were not able to find any study that has evidenced stereotyped and invariant characteristics of muscle activities at the hip level in different populations or experimental conditions during GI, therefore, this question need to be investigated in the future.

Bilateral electromyography of m. tibialis anterior and m. gastrocnemius has revealed five evidences for the existence of this motor program: the invariant relative timing of support (1) and swing limb (2) m. tibialis anterior onsets (support limb TA action preceeds swing limb TA action), (3) the fore-aft ground reaction forces, (4) the swing limb toe-off, and (5) the swing limb heel-strike happen at 14, 16, 20, 43, and 87 % of the entire GI process, respectively, regardless of changes in speed [7]. An experiment using two force plates, foot switches, and motion analysis revealed specific limb functions during GI: since forward impulse increases rapidly after toe-off of the swing limb, the support limb is considered to be responsible for controlling forward impulse, while the swing limb is mainly concerned with weight shift from the swing foot to the support foot, because loading of the swing limb in the anticipatory phase occurs simultaneously to the unloading of the support limb and the onset of forward acceleration of the COM [7].

22.2 Influencing Factors

Many studies have investigated how the motor pattern of GI is affected by factors related to cognitive demands, foot-floor interface, speed and initial body posture among others, like aging that will be further discussed later in this chapter.

Dual tasking has been shown to affect GI in healthy older adults. Two groups of older adults, one called “fear of falling (Fof)” and another called “no - Fof group” performed GI as a single task, while counting backwards from 100 to 1. Despite both groups were not different regarding all clinical measures of functional balance (“Timed up and Go,” “10 m walking,” and “Functional Reach” tests), the Fof group produced longer GI phases during dual tasking compared to the single task, whereas for the no-Fof group no differences were found between both tasks [16]. The prolonged APA phase for the Fof group is interpreted as an attempt to stabilize the body in order to reduce the risk of falling when attention resources for initiating gait are limited [16].

Footwear type has been shown to influence the number of steps necessary to reach steady state gait: habitual shoes with foot orthoses have helped to reach steady state walking with fewer steps than when barefoot or wearing habitual shoes without orthoses [17]. When using foot orthoses ML COM range of motion is reduced during walking and GI, and this advantage has been related to a higher level of stimulation of mechanoreceptors activity and improved dynamic postural

stability [17]. On the other hand, reduction in the afferent somatosensory information under the foot by means of immersion in iced water resulted in a reduced ML COP excursion during the anticipatory phase and this loss of sensitivity was not compensated by visual input [18], indicating that plantar cutaneous afferents contribute to the control of GI. Supporting this idea, we have observed that when GI is performed barefoot, ML COP path and velocity during the anticipatory phase are higher than when using footwear, probably because of the enhanced somatosensory perception provided by the barefoot condition [19].

We have also analyzed the relative contribution of each foot to the swing foot loading and unloading processes and evidenced that when GI is performed on a soft surface made of foam the swing and support foot peak forces in the ML direction are smaller than when the subject is shod or barefoot [19]. Still regarding foam conditions, significant Spearman correlations (Fig. 22.2) between COP integral and force impulse revealed that COP shift is responsible for only about 3.8 % of ML impulse production, whereas in barefoot and shod conditions COP shift is responsible for 26.6 % and 37.4 % of impulse generation, respectively. On the other hand, COP shift is responsible for 89.5 %, 84.9 %, and 86.7 % of impulse production in the AP direction in barefoot, foam, and shod conditions, respectively.

According to our results, we can state that the production of impulse in AP direction is more dependent on the COP shift than in ML direction and thus the influence of other mechanisms for impulse generation than COP shift is stronger for the ML direction. Additionally, the foam surface limited the impulse-generating capacity of the COP shift in the anticipation phase of GI and produced smaller force peaks in ML direction, resulting in limitations to body weight transfer from swing to support foot. Thus our results suggest that footwear and soft surface affect COP patterns and force features during GI especially in ML direction of the APA phase [19].

Starting leg, whether freely chosen or predetermined, does not change the muscle activity during GI phases, but when the leading leg is predetermined the reaction time to start GI is shorter than when using a freely chosen leg for older and younger adults, with the older people needing longer reaction times [20]. Interesting is the fact that tibialis anterior (TA) anticipatory activity gradually improves with subsequent trials in a series of 10 trials [20], which may suggest a learning effect that could have implications in rehabilitation contexts.

APAs are considered to be under prospective control [21] and, as such, they are an index for the ability to feedforward control motor actions. Thus limiting factors to properly integrate posture and voluntary locomotion in standing are associated with difficulties in activating the motor program involved in the APAs for GI, especially regarding forward propulsion [22]. Concerning locomotion ability, it is likely that dynamic instabilities during step transitions may be more critical factors to reach a steady state walking speed than standing postural control.

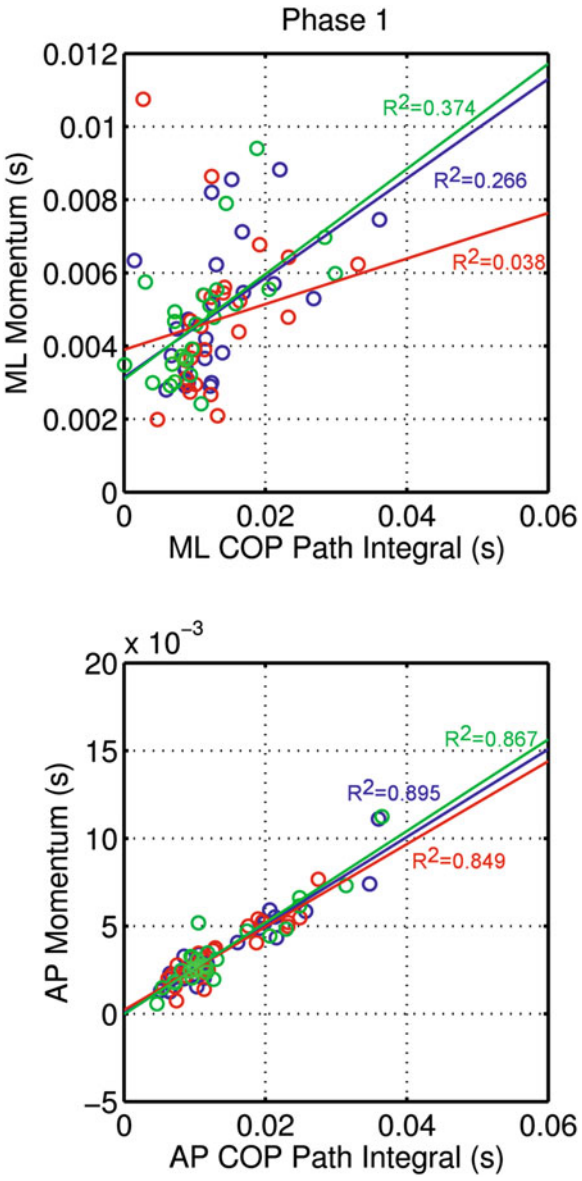

Fig. 22.2 AP Impulse × COP Path Integral graph for phase 1 of GI. See text for explanations. *Blue*: barefoot condition; *Red*: foam condition; *Green*: shod condition

22.3 GI and the Older People

The task of GI challenges the balance control system, because the person moves from a relative stable static balance to the continuously unstable gait [23]. The skills necessary to maintain stability, weight transfer, foot clearance, and propulsion

become more critical during transitions like accelerating in GI, decelerating in gait termination [17], and changing directions [24]. Individuals with postural instabilities and locomotion impairments may experience even more problems reaching equilibrium during GI. Thus GI represents a risky activity for the older people [25], considered as a fall prone population given their well-known problems with static and dynamic balance. It is accepted that the transition from standing to steady state gait is an ideal paradigm to examine the impact of advancing age on mobility [26], since those changes may not be apparent during quiet support and steady state walking tests.

GI studies in the older people have revealed some important age-related changes in this process. In the older adults, decreased COP displacements in the APA phase are evidenced [23] and despite the central motor program for GI is present, it is less frequently observed in electromyography data than in younger adults [25]. For this reason, COP displacements in the APA phase have been accepted as a sensitive indicator of balance dysfunction [2].

Two measures have revealed that the biomechanical effectiveness of the GI motor program in generating impulse (given as the time integral of GRF in a specific direction) in the older people is limited [25]:

(1) time integral of backward COP shift and the amount of forward impulse are highly correlated in young as well as in the older adults, though older people generate significantly less impulse for each increment in COP displacement;
(2) time integral of the AP COP displacement and walking speed are also highly correlated for both populations, though the correlation coefficients are significantly lower for the older group.

Together these indicators reflect that the older group may generate the impulse needed for the execution of the first step by other strategies than moving the COP backward [25]. Reduced backward COP excursions may be associated with the fact that m. soleus and m. gastrocnemius remain active while m. tibialis anterior becomes active during the anticipatory phase [25].

Although the stereotyped muscle activity of GI remains invariable in healthy older people [23], electromyography studies have demonstrated some alterations in muscle activity durations. The duration of m. tibialis anterior (TA) anticipatory activation tends to be shorter for the physically active older than for younger adults [20]. When compared to younger adults m. lateral gastrocnemius (LG) activity of the support leg starts later and the percentage of delay between TA and LG activations is greater in the older people, whereas mean duration of the simultaneous activation is shorter [20]. Thus LG activation of the support limb does not start before swing limb has left the ground in the majority of the older adults studied [20] and this delay may impair not only the propulsion, but also the control of the forward movement of the body in preparation for the swing leg to leave the ground [27].

The initial steps of GI are shorter with greater variability for older people, even when step length is normalized to body height [20]. Step lengths are even shorter in older fallers, as compared to younger adults [28].

Fear of falling is a factor that influences GI specially in older adults. Older adults transitioning to frailty are those who do not meet the criteria for either frail or vigorous based on a specific classification scheme [24]. They are highly susceptible to falls and produce smaller and less forceful COP displacements during the APA phase when compared to healthy older adults [29]. The same is known to happen with Parkinson's disease patients [23, 30], a population that perform even worse in APA phase of GI than older adults transitioning to frailty [24].

Vestibular impairments have been shown to also limit APAs and thus the dynamic balance in older adults [2]. Unilateral vestibular patients have been compared to healthy older controls during GI [34]. TA anticipatory activation is not present in most patients. Longer anticipatory phase durations, smaller backward COP shifts, larger ML COP shift toward the support foot, as well as more pronounced TA/LG co-activation for the support limb when swing limb leaves the ground (swing foot unloading phase) were observed in vestibular patients when compared to matched healthy controls. It is suggested that unilateral vestibular patients may perceive COP backward shift as too destabilizing, so that the lack of stereotyped ankle muscles activity to generate COP backward displacement indicates that these patients may use a different strategy to initiate gait. Indeed rapid unilateral arm rotation could be observed in several patients [34]. Limitations to start gait with one limb could not be related to an influence of ipsilateral vestibular loss [34], which should be further investigated.

Parkinson's Disease (PD) patients are more likely to fall than other neurological patients [31], for this reason GI has been studied to provide information about changes in dynamic postural control of this population. Patients with PD place their COP further ahead of their ankle joint than healthy older people, which means that they lean significantly further forward when standing [23]. This posture prior to GI may contribute to prolonged delays between APAs and step executions when patients with PD are tested in "ON" dopaminergic medication state. Additionally these patients have reduced COP backward displacements when compared to healthy and transitionally frail older adults [23, 24, 29]. As a result, swing foot unloading phase is prolonged and forward impulse is smaller, which may indicate that these patients have a relative inability to generate forward impulse via appropriate soleus–tibialis interactions and probably place greater emphasis on ML weight transfer to take a step. Indeed increased values of ML COP displacements and velocities [24] have been related to this situation, although decreased values for these variables have also been reported [23, 32], a fact that should be further investigated.

Reduced COP backward displacement in the APA phase has been shown to be an important pathophysiological aspect related to inefficient GI process in PD population [23], which is considered an indication that the mechanism responsible for generation of forward impulse may be more susceptible to deterioration of the complex interaction of central and peripheral changes associated with dopamine

depletion than to aging alone [24]. Very interesting is the fact that the smoothness index significantly and negatively correlates to the Unified Parkinson Disease Rating Scale and to the Hoeln and Jahr Disability Rating [24]. Together these results may reflect that PD patients are more concerned with postural stability than with forward progression and that they are less able to coordinate COP movements during GI. Difficulties or hesitation at initializing or maintaining movement, a characteristic of patients with PD, are also related to a subtype of **freezing of gait** [33], which has been shown to cause failures in starting and scaling APAs, as evidenced by the fact that absent of adequate COP ML shift toward the swing leg is more present in freezers than in non-freezers.

22.4 GI and Training Effects

Some authors have suggested training strategies that would improve GI in older people, based on results of studies that have compared groups with different characteristics. Evidences of task memorization [20] indicated that when GI is repeated in a series of successive trials this task specific repetitive training could be advised for older people in order to prevent falls. Biofeedback and relaxation exercises [25] have also been suggested to improve COP shift mechanism and thus probably prevent falls.

Other researchers have performed specific interventions and achieved improvements in GI performance.

Tai Chi training [29] as a martial art is considered a potential tool to prevent falls, because it emphasizes concentration, awareness of environment, slow weight shifting, endurance, and strength. When the typical slow transitions from double to single support in Tai Chi techniques are trained by frail older adults during 48 weeks twice weekly, these individuals improved the backward COP shift during the APA phase to values similar to those exhibited by healthy older adults [29]. Furthermore, the movement smoothness values during the swing foot unloading phase were improved in the Tai Chi training group. A comparative group engaged in wellness education program, including techniques of fall prevention, diet and nutrition advisement, mental health and other health related issues, was less efficient in improving backward COP translations and movement smoothness. These results indicate that Tai Chi may help to restore m. soleus inhibition and m. tibialis anterior activation patterns during APA phase. Additionally, the smoothness value, as a movement coordination index, is sensitive to this training. On the other hand, neither interventions improved ML COP displacements in the anticipatory and swing foot unloading phases.

Improvements in GI characteristics of patients with PD have been reported after a **Progressive Resistance Training** [35]. In a pilot study, a 10-weeks program beginning with 70 % of 1 RM (Repetition Maximum) was designed to be developed with dynamic variable resistance exercise machines. Significant increases of 29 % for COP backward displacements in the anticipatory phase were found for the exercise group, as well as improvements in stride velocity, length, and knee flexion

and extension at 1 RM. No changes were observed for the control group. For the ML direction no differences were found. On the basis of these results, it is suggested that progressive resistance training may be an interesting modality to reduce hypokinetic APAs for gait and spatiotemporal parameters of the first step in patients with PD [35].

Training effects should be further investigated to highlight how impairments in GI can be overcome in older adults, especially to prevent falls. Randomized controlled clinical studies are of interest to accomplish this goal.

The GI is frequently studied using force plates. Literature reports some common descriptors and specific variables selected by the authors to identify special aspects of GI. The next section of this chapter will deal with the computational description of the variables we consider relevant that are derived from the dynamometric method using force plates.

22.5 Forces and COP Calculations During GI

It is important to mention that electromyography and motion analysis provide complementary descriptors of the GI process, but the respective variables are not in the scope of this chapter.

Some authors use the forces recorded by a force plate in the study of GI. These variables can be associated with kinematic variables, obtained through optical systems, or electromyography (EMG) data, and some information can be extracted using multivariate statistics tools like principal component analysis (PCA) and statistical parametric mapping (SPM). However, the COP, a derived variable from the ground reaction forces, is the most used variable to study GI due to its peculiar behavior in this task, especially with respect to APAs. For this reason, we will focus on analyzing the COP in GI, although we will also comment on the ground reaction forces (GRF).

The raw data of force plate can be sampled at different frequencies (Table 22.1). When kinematic data are acquired by an optic system together with the kinetic data it is advisable to adopt a sampling frequency that is multiple of the acquisition frequency of the cameras. This will facilitate further operations like synchronization, decimation, and data normalization. However, considering that kinematic and kinetic signals of human movement are low frequency signals and they primarily occupy the lowest band of the frequency spectrum of the raw data [37], setting the force plate sampling frequency at 100 Hz will be sufficient in most cases.

It is recommended filtering the raw data to remove noise. The noise can be reduced adopting a careful protocol to collect data, including an equipment heating period. The force plate manufacturers recommend turning on the equipment for at least 1 hour before their use in data collection.

The data should be filtered by a second or fourth order low-pass-band Butterworth filter. The approximation provided by a Butterworth filter has a flatter frequency response and a phase closer to the linear in relation to other approximations such as Chebyshev or Elliptic. A linear phase is important because

Table 22.1 Sampling frequency of force plates in GI studies

Sampling frequency (Hz)	Authors
1000	[16, 18, 38, 42]
600	[30, 43, 45]
500	[8, 46, 57]
250	[58, 59]
200	[60, 61]
100	[20, 34, 62, 67]

Table 22.2 Filter cutoff frequency to process the force signal

Cutoff frequency	Sampling frequency	Authors
5	Not reported	[69]
6	1000	[16, 42]
10	500 or 1000	[18, 39, 40]
10.5	500	[49]
30	500 or 100	[46, 66]
50	600 or 1200	[45, 63]
100	500	[48, 50]

it provides an equal delay to all frequencies contained in the original signal. However, the Butterworth filter introduces a delay or phase shift in the filtered signal, and a reverse filtering should be performed to eliminate this delay or phase. In other words, the filter must be applied to the raw signal in forward and reverse direction, canceling the phase introduced by the Butterworth filter. This means that in fact a fourth or eighth order filter is applied to the raw data.

The cutoff frequency is a critical point in the filter design and depends on the sampling frequency used in data acquisition and on the maximum relative mean residual allowed [68]. There is no consensus on the cutoff frequency of the filter used in the analysis of the GI (Table 22.2) among authors and many of them omit this information.

Considering the low frequency of the signals in biomechanics [37] and the recommendations to the choice of the cutoff frequency in filter design [68], we suggest a zero-lag fourth order low-pass Butterworth filter with a cutoff frequency of 6 Hz and a sampling frequency of at least 100 Hz in GI studies using force plate.

22.6 Analyzed Variables

The data collected with one or two force plates are processed to calculate the COP trajectory during the entire task or during each phase of GI or to analyze the behavior of the ground reaction forces. In fact, the COP trajectory is a current index used in basic and clinical research to evaluate balance and locomotion control [47], being an indicator of instability to both healthy and pathological populations [62].

The behavior of the GRF is analyzed in several situations and with different populations [18, 20, 34, 38, 58, 64–66, 69–72]. Some authors used the force peak values of the swing and support limb [8], or force values in specific instants of the task [39, 70], as well as the force impulse, given by the area under the force curve [22, 41].

Most studies have considered only discrete parameters from GRF for extraction of information about the GI and other tasks, usually local peaks or minimum values, as reported above. However, different methods of multivariate analysis based on variance estimates, such as PCA and SPM, have been used in the interpretation of GI data. The PCA considers the complete pattern of GRF and can provide additional insight about GI [66], reducing the dimensionality of the data, while keeping their original variability: PCA reduces the number of variables to a subset of orthonormal factors or principal components, which maximize the representation of variance of the original data [73]. Besides, the use of PCA with a pattern recognition tool has been proven to be useful for discriminating different groups [66, 74], or for evaluating how far the gait deviates from a typical pattern [66, 75].

In order to capture features of the entire COP or force time series a vector analysis using statistical parametric mapping (SPM) methods should be conducted, as described by Pataky et al. [76]. This statistical approach captures features of the entire time series instead of a few discrete variables and may improve the effectiveness of the GI analysis. Discrete variables fail to capture sufficient portions of the data and covariance among vector components [76]. SPM analysis uses random field theory to identify field regions which co-vary with the experimental protocol [77].

To perform the SPM analysis , each component of the resultant COP or force time series is interpolated with cubic splines to contain 100 points (normalization to 100 % of the task). The average over the trials performed by the study participants should be used in the analysis. Next, the components are organized in an array with n rows, one for each participant, 100 columns and M matrices, one for each component X and Y of COP time series (or X, Y, and Z components of GRF time series). One array should be constructed for each condition analyzed in the study.

Taking the COP time series as an example, each column of the $(n \times 100)$ COP matrices is regarded as a single-vector field $\mathrm{Cop}(t) = \{\mathrm{Cop_x}(t)\ \mathrm{Cop_y}(t)\}$, where t indicates time. Within-subject mean $\mathrm{Cop}(t)$ is calculated for each ith subject at each time instant and among the different conditions analyzed, taken two by two:

$$\Delta\mathrm{Cop}(t)_i = \left(\mathrm{Cop}(t)_{\mathrm{condition}}\right)_i - \left(\mathrm{Cop}(t)_{\mathrm{condition}}\right)_i \tag{22.1}$$

where conditions are before and after an intervention, for example.

The paired Hotellings T^2 test statistic trajectory is calculated as:

$$T^2(t) = n\left(\Delta\bar{\mathrm{C}}\mathrm{op}(t)\right)W(t)^{-1}\left(\Delta\bar{\mathrm{C}}\mathrm{op}(t)^T\right) \tag{22.2}$$

where n is the number of subjects (number of rows), $\Delta\bar{C}op(t)$ is the cross-subject mean, and $W(t)$ is the 2×2 covariance matrix of ΔCop_x and ΔCop_y at time t.

For vector analysis, statistical inference is conducted by calculating the T^2 threshold taking $\alpha = 0.05$. Conditions effects are assessed using paired t tests on COPx, COPy with a Sidák correction threshold taking $\alpha = 0.05$ producing p calculated as follows:

$$p_{\text{critical}} = 1 - (1 - \alpha)^{1/N} \tag{22.3}$$

where $N = 2$ is the number of scalars for COP (x and y). Equation (22.3) produces $p = 0.0253$ for $N = 2$.

Additional information and the codes to conduct SPM analysis can be downloaded from www.spm1d.org.

Variables such as COP maximum lateral displacement and COP maximum backward displacement during the APA, as well as the APA duration [16, 30, 39, 41, 42, 52, 58, 59, 61, 78–80] are very common in the literature, as well as in studies of child development [60, 81] characterizing the motor behavior during GI of a given population [82].

Some variables can only be calculated using two force plates such as the COP displacement under each foot [34], especially in amputees [43], or the body weight on the swing foot at the beginning and at the end of APA [47, 82], or the force peak on each foot [45]. The trajectory of the COP under the swing foot and the support foot can be analyzed separately, usually normalized by foot length [81]. In the latter case, the use of two force plates is necessary. Specifically, when comparing adults and children, Malouin and Richards [81] proposed an index given by the ratio between the swing foot AP displacement and the support foot AP displacement.

Another variable used is the resultant COP trajectory [24, 63, 83, 84], or the resultant COP displacement in the AP and ML directions [18, 34, 54–57, 59, 67, 69, 70, 72, 85].

The duration of each GI phase and the COP mean velocities in the AP and ML directions, at each phase or during the entire task, are also widely used [24, 27, 29, 40, 50, 67, 86, 87] and can be calculated as suggested in Table 22.3.

The movement smoothness, a variable used to evaluate the motor performance of healthy and pathological individuals [88–90], has also been used in the study of GI [24, 29]. Maximum smoothness is obtained when the movement oscillations are minimized, indicating that the movement smoothness results from learning processes and better motor coordination [90]. Smoothness is defined as the integral of the square of the third derivative of COP trajectory in the AP and ML directions [29] as presented in Table 22.3.

Another variable used to understand the rocker behavior created by the knee-ankle-foot system is the roll-over, measured by transforming the COP from a laboratory-base coordinate system into the leg-based coordinate system [91], which can be useful in the design of prostheses.

Table 22.3 Matlab code suggestions to calculate COP descriptors

Matlab script	Description
path_x = sum (abs (diff (COPx)));	Total COP path in ML direction
path_y = sum (abs (diff (COPy)));	Total COP path in AP direction
duration = length(COPx)/Fs; or duration = length(COPy)/Fs	Duration of corresponding phase.
mean_veloc_x = displacement_x / duration;	Mean velocity in ML direction.
mean_veloc_y = displacement_y / duration;	Mean velocity in AP direction
smoothness_x = trapz((diff(COPx,3)./(1/Fs)^3).^2);	Smoothness in the ML direction
smoothness_y = trapz((diff(COPy,3)./(1/Fs)^3).^2);	Smoothness in the AP direction

COPx, COPy = COP trajectory in ML and AP directions, respectively, *Fs* = sampling frequency

22.7 Recommendations for Analytical Studies of GI

In order to standardize the analytical procedures to assess GI, some recommendations are suggested to enable future comparisons of different studies.

Table 22.4 presents Matlab code suggestions for filter design.

The *butter* command will design an infinite impulse response (IIR) Butterworth filter. Using, in this case, a second order filter means to assign a ripple smaller than 3 dB in the pass-band and a 60 dB attenuation in the stop band.

The *filtfilt* Matlab command will perform an IIR filtering using the filter coefficients calculated by the *butter* command, forward and reverse, canceling the phase introduced by the Butterworth filter. This means using, in fact, a fourth order filter, as already explained.

The COP instantaneous position is calculated by considering the ground reaction force components, the moments around the AP and ML axes, and the data concerning the true origin of the force plate, as reported by the manufacturer. It should be considered that the actual origin of the force plate does not always coincide with the force plate geometrical center due the limitations in the manufacturing process (Table 22.5).

When the plate has some kind of cover used to hide the plate to prevent the subject to aim at the plate and to modify his step, the pad thickness (e) have to be added to plate true origin z coordinate (Table 22.5).

Analyzing the equations presented in Table 22.5, we can conclude that the COP is sensitive to errors in force and moments values to very small Fz values, commonly acquired at the beginning and the end of foot contact on force plate. Hence, it is important to establish an Fz force threshold, under which the COP will not be calculated. This threshold depends of the analyzed subject. We recommend values around 2 % of the average individuals body weight.

While we use the reaction-oriented coordinate system, it is important to observe that some force plate manufactures use action-oriented coordinate system. In this case, a conversion should be done as follows (Table 22.6).

Table 22.4 Matlab code suggestion for filter design

Matlab code	Description
Fs = 100;	Sampling frequency
Fc = 6;	Cutoff frequency
order = 2;	Filter order
Wn = Fc/(Fs/2);	Cutoff frequency in radians relative to the sampling frequency
[b,a] = butter(order,	Filter coefficients
Wn,'low');	
data = filtfilt(b,a,raw_data);	Zero-lag filtering

raw_data = raw data, *data* = filtered data

Table 22.5 Matlab code suggestions for COP calculations

Matlab commands	Description
COPx = − ((My + (dz*Fx))./Fz) + dx;	ML COP
COPy = ((Mx − (dz*Fy))./Fz) + dy;	AP COP
COPx = − ((My + ((dz-e)*Fx))./Fz) + dx;	ML COP with pad on the plate
COPy = ((Mx − ((dz−e)*Fy))./Fz) + dy;	AP COP with pad on the plate
COPx_result = ((COPx1.*(Fz1./(Fz1 + Fz2)))) + (W_1/2) + ((COPx2.*(Fz2./(Fz1 + Fz2))))−(W_2/2)$^{++}$	Resultant ML COP between two plates
COPy_result = (COPy1.*(Fz1./(Fz1 + Fz2))) + (COPy2.*(Fz2./(Fz1 + Fz2)))	Resultant AP COP between two plates

Fx = ML GRF component, *Fy* = AP GRF component, *Fz* = Vertical GRF component, *Mx* = ML moment, *My* = AP moment, *dx, dy, dz* = plate true origin coordinates *x*, *y and z*, *e* = pad thickness on the plate. W_1 = force plate 1 width, W_2 = force plate 2 width. The indexes 1 and 2 refer to the first and second plates, respectively, when using two force plates. ++ The COP coordinates should be described in relation to the origin of the corresponding force plate. If the COP coordinates are described in relation to the global reference system of the laboratory, W_1/2 and W_2/2 are not necessary

Table 22.6 GRF coordinate systems

Action-oriented coordinate system	Reaction-oriented coordinate system
Axes x	Axes -x
Axes y	Axes y
Axes z	Axes -z
Fx	Fx
Fy	-Fy
Fz	Fz
Mx	Mx
My	-My
Mz	Mz

22.8 Calculating the Beginning and the End of GI

An important aspect for analyzing GI is the determination of task beginning. In some studies, the volunteers are instructed to initiate the task after a vocal command, an acoustic stimulus, a cutaneous stimulus, or a visual stimulus. However, even when the laboratory is equipped with devices capable of synchronizing the start command to data acquisition, this does not mean the volunteer will begin the task immediately after the start command. Some procedures have to be done to properly identify the task beginning. Table 22.7 presents different protocols adopted by different authors.

Therefore, for force plate protocols, data should be acquired prior to the start command. The data acquisition has to be triggered at least 2 s before the command to initiate the movement. The individual should be instructed to maintain a quiet posture on the force plate until the start command.

If two force plates are being used, the calculations for the beginning of the movement should be made from the ground reaction force of the swing foot force plate, and for the end of the movement from the support foot force plate.

The end of the movement is defined by the instant at which the support foot loses contact with the force plate, when the ground reaction force vertical component drops to zero or is smaller than the established threshold. Thus, the data can properly be cut.

Table 22.8 gives some examples of Matlab codes for these calculations considering that the data acquisition has begun 2 s before the beginning of the task, with a sampling frequency of 100 Hz and with a threshold of 15 N for the vertical component of ground reaction force.

Table 22.7 Strategies to determine task beginning in GI

Beginning of the task	Authors
When the COP medial-lateral deviation toward the swing foot is greater than three times the standard deviation of the COP average initial position during the first 1.5 s before the stimulus	[16, 42, 81]
When the COP moves beyond 10 mm laterally in relation to its initial position	[62]
When the velocity of the resultant COP is > 0.01 m/s	[49]
By the first medial-lateral COP displacement in frontal plane	[92]
When the first medial-lateral COP deviation toward the swing foot is > 4 mm	[93, 94]
By the first COP trajectory sample toward the swing foot	[32]
At the instant in which occurred a 4 mm displacement in relation to COP baseline with a duration of at least 10 ms	[95]
By an alteration in ground reaction force vertical component immediately before the anticipatory postural adjustment	[66]
By a detectable increase in swing foot ground reaction force vertical component	[34]
At the instant in which the ground reaction force vertical component exceeds its average value plus two times the standard deviation acquired during the first 0.5 s of quiet stance	[9]

Table 22.8 Matlab codes suggestions for calculating the beginning of the task[a]

Matlab script	Description
ind = find(Fz >= (mean(Fz (50:100)) + 2*std(Fz(50:100))); start = ind(1);	Task beginning: Fz >= averaged Fz during 0.5 s before the movement plus two times the standard deviation. Fz: vertical force of swing foot force plate
ind = find(Fz <= 15.0); end = ind(1);	Fz threshold = 15 N. Fz: vertical force of support foot force plate (around 2 % of average individuals body weight)
data = data(start:end,:);	Cutting the data

Fz = vertical ground reaction force component. For data acquired at 100 Hz
[a]We have used protocols suggested in literature [9, 96]

22.9 Final Remarks

Considering the importance of the GI process in older people and the studies cited in this chapter, we can point some final remarks: (1) GI studies have improved our understanding of dynamic balance in older people; (2) clinical trials are needed to verify whether GI characteristics are sensitive to interventions; (3) prospective studies are needed to identify whether improvements in GI are related to less frequent falls in the elderly population; (4) frontal plane ML COP mechanism during GI must be better understood in order to identify invariant characteristics for ankle and hip joint motions in this plane during the phases of GI; (5) results of different studies must be compared with caution, considering the respective calculation methods; (6) data processing should consider the characteristics of the process and should focus the search for the underlying mechanism that control GI.

References

1. Bernstein NA. The co-ordination and regulation of movements. Oxford: Pergamon Press; 1967.
2. Chang H, Krebs DE. Dynamic balance control in elders: gait initiation assessment as a screening tool. Arch Phys Med Rehabil. 1999;80:490–4. doi:10.1016/S0003-9993(99)90187-9.
3. Bouisset S, Do MC. Posture, dynamic stability, and voluntary movement. Neurophysiol Clin. 2008;38:345–62. doi:10.1016/j.neucli.2008.10.001.
4. Fiolkowski P, Brunt D, Bishop M, Woo R. Does postural instability affect the initiation of human gait? Neurosci Lett. 2002;323:167–70. doi:10.1016/S0304-3940(02)00158-1.
5. Winter DA. Human balance and postural control during standing and walking. Gait Posture. 1995;3:193–214.
6. Jian Y, Winter DA, Gilchrist L. Trajectory of the body COG and COP during initiation and termination of gait. Gait Posture. 1993;1:9–22.
7. Brunt D, Lafferty MJ, Mckeon A, Goode B, Mulhausen C, Polk P. Invariant characteristics of gait initiation. Am J Phys Med Rehabil. 1991;70:206–12.
8. Brunt D, Santos V, Kim HD, Light K, Levy C. Initiation of movement from quiet stance: comparison of gait and stepping in elderly subjects of different levels of functional ability. Gait Posture. 2005;21:297–302. doi:10.1016/j.gaitpost.2004.03.003.

9. Hass CJ, Buckley TA, Pitsikoulis C, Barthelemy EJ. Progressive resistance training improves gait initiation in individuals with Parkinson's disease. Gait Posture. 2012;35:669–73. doi:10.1016/j.gaitpost.2011.12.022.
10. Gormley J, Barr D, Bell A, Ravey J, Mollan R. Examination of the duration of gait initiation by use of an electrogoniometer. Gait Posture. 1993;1:85–91. doi:10.1016/0966-6362(93)90019-W.
11. Crenna BYP, Frigo C. Paolo carlo. J Physiol. 1991;437:635–53.
12. Breniere Y, Do MC. When and how does steady state gait movement induced from upright posture begin? J Biomech. 1986;19:1035–40. doi:10.1016/0021-9290(86)90120-X.
13. Crenna P, Frigo C. A motor programme for the initiation of forward-oriented movements in humans. J Physiol. 1991;437:635–53.
14. Winter DA, Patla AE, Ishac M, Gage WH. Motor mechanisms of balance during quiet standing. J Electromyogr Kinesiol. 2003;13:49–56. doi:10.1016/S1050-6411(02)00085-8.
15. Fortin A-P, Dessery Y, Leteneur S, Barbier F, Corbeil P. Effect of natural trunk inclination on variability in soleus inhibition and tibialis anterior activation during gait initiation in young adults. Gait Posture. 2015;41(2):378–83. doi:10.1016/j.gaitpost.2014.09.019.
16. Uemura K, Yamada M, Nagai K, Tanaka B, Mori S, Ichihashi N. Fear of falling is associated with prolonged anticipatory postural adjustment during gait initiation under dual-task conditions in older adults. Gait Posture. 2012;35:282–86. doi:10.1016/j.gaitpost.2011.09.100.
17. Najafi B, Miller D, Jarrett BD, Wrobel JS. Does footwear type impact the number of steps required to reach gait steady state?: an innovative look at the impact of foot orthoses on gait initiation. Gait Posture. 2010;32:29–33. doi:10.1016/j.gaitpost.2010.02.016.
18. Lin SI, Yang WC. Effect of plantar desensitization on postural adjustments prior to step initiation. Gait Posture. 2011;34:451–56. doi:10.1016/j.gaitpost.2011.06.016.
19. Vieira MF, Sacco Ide C, Nora FG, Rosenbaum D, Lobo da Costa PH (2015) Footwear and foam surface alter gait initiation of typical subjects. PLoS One. 2015;10:e0135821. doi:10.1371/journal.pone.0135821.
20. Henriksson M, Hirschfeld H. Physically active older adults display alterations in gait initiation. Gait Posture. 2005;21:289–96. doi:10.1016/j.gaitpost.2004.03.001.
21. Von Hofsten C. An action perspective on motor development. Trends Cogn Sci. 2004;8:266–72. doi:10.1016/j.tics.2004.04.002.
22. Malouin F, Richards CL. Preparatory adjustments during gait initiation in 4–6-year-old children. Gait Posture. 2000;11:239–53.
23. Halliday SE, Winter DA, Frank JS, Patla AE, Ontario NLG. The initiation of gait in young, elderly, and Parkinson' s disease subjects. Gait Posture. 1998;8:8–14.
24. Hass CJ, Waddell DE, Wolf SL, Juncos JL, Gregor RJ. Gait initiation in older adults with postural instability. Clin Biomech. 2008;23:743–53. doi:10.1016/j.clinbiomech.2008.02.012.
25. Polcyn AF, Lipsitz LA, Kerrigan DC, Collins JJ. Age-related changes in the initiation of gait: degradation of central mechanisms for momentum generation. Arch Phys Med Rehabil. 1998;79:1582–9. doi:10.1016/S0003-9993(98)90425-7.
26. Muir BC, Rietdyk S, Haddad JM. Gait initiation: the first four steps in adults aged 20-25 years, 65-79 years, and 80-91 years. Gait Posture. 2014;39:490–4. doi:10.1016/j.gaitpost.2013.08.037.
27. Michel V, Do MC. Are stance ankle plantar flexor muscles necessary to generate propulsive force during human gait initiation? Neurosci Lett. 2002;325:139–43. doi:10.1016/S0304-3940(02)00255-0.
28. Mbourou GA, Lajoie Y, Teasdale N. Step length variability at gait initiation in elderly fallers and non-fallers, and young adults. Gerontology. 2003;49:21–6. doi:10.1159/000066506.
29. Hass CJ, Gregor RJ, Waddell DE, Oliver A, Smith DW, Fleming RP, et al. The influence of Tai Chi training on the center of pressure trajectory during gait initiation in older adults. Arch Phys Med Rehabil. 2004;85:1593–8. doi:10.1016/j.apmr.2004.01.020.
30. Martin M, Shinberg M, Kuchibhatla M, Ray L, Carollo JJ, Schenkman ML. Gait initiation in community-dwelling adults with Parkinson disease: comparison with older and younger adults without the disease. Phys Ther. 2002;82:566–77.

31. Stolze H, Klebe S, Baecker C, Zechlin C, Friege L, Pohle S, et al. Prevalence of gait disorders in hospitalized neurological patients. Mov Disord. 2005;20:89–94. doi:10.1002/mds.20266.
32. Martin M, Shinberg M, Kuchibhatla M, Ray L, Carollo JJ, Schenkman ML. Gait initiation in community-dwelling adults with Parkinson disease. Phys Ther. 2002;82:1264. author reply 1264–5.
33. Delval A, Tard C, Defebvre L. Why we should study gait initiation in Parkinson's disease. Neurophysiol Clin. 2014;44:69–76. doi:10.1016/j.neucli.2013.10.127.
34. Henriksson M, Henriksson J, Bergenius J. Gait initiation characteristics in elderly patients with unilateral vestibular impairment. Gait Posture. 2011;33:661–7. doi:10.1016/j.gaitpost.2011.02.018.
35. Hass CJ, Buckley TA, Pitsikoulis C, Barthelemy EJ. Gait & posture progressive resistance training improves gait initiation in individuals with Parkinson' s disease. Gait Posture. 2012;35:669–73. doi:10.1016/j.gaitpost.2011.12.022.
36. Martin K, Blizzard L, Garry M, Thomson R, Mcginley J, Srikanth V. Gait & posture gait initiation in older people – Time to first lateral movement may be the measure of choice. Gait Posture. 2011;34:374–8. doi:10.1016/j.gaitpost.2011.06.004.
37. Winter DA. Biomechanics and motor control of human movement. Processing. 1990;2:277. doi:10.1002/9780470549148.
38. Tseng SC, Stanhope SJ, Morton SM. Visuomotor adaptation of voluntary step initiation in older adults. Gait Posture. 2010;31:180–4. doi:10.1016/j.gaitpost.2009.10.001.
39. Corbeil P, Anaka E. Combined effects of speed and directional change on postural adjustments during gait initiation. J Electromyogr Kinesiol. 2011;21:734–41. doi:10.1016/j.jelekin.2011.05.005.
40. Gélat T, Coudrat L, Le Pellec A. Gait initiation is affected during emotional conflict. Neurosci Lett. 2011;497:64–7. doi:10.1016/j.neulet.2011.04.030.
41. Caderby T, Dalleau G, Leroyer P, Bonazzi B, Chane-Teng D, Do MC. Does an additional load modify the anticipatory postural adjustments in gait initiation? Gait Posture. 2013;37:144–6. doi:10.1016/j.gaitpost.2012.06.012.
42. Uemura K, Yamada M, Nagai K, Tateuchi H, Mori S, Tanaka B, et al. Effects of dual-task switch exercise on gait and gait initiation performance in older adults: preliminary results of a randomized controlled trial. Arch Gerontol Geriatr. 2012;54:e167–71. doi:10.1016/j.archger.2012.01.002.
43. Tokuno CD, Sanderson DJ, Inglis JT, Chua R. Postural and movement adaptations by individuals with a unilateral below-knee amputation during gait initiation. Gait Posture. 2003;18:158–69. doi:10.1016/S0966-6362(03)00004-3.
44. Dibble LE, Nicholson DE, Shultz B, MacWilliams BA, Marcus RL, Moncur C. Sensory cueing effects on maximal speed gait initiation in persons with Parkinson's disease and healthy elders. Gait Posture. 2004;19:215–25. doi:10.1016/S0966-6362(03)00065-1.
45. Tokuno CD, Eng JJ. Gait initiation is dependent on the function of the paretic trailing limb in individuals with stroke. Gait Posture. 2006;24:424–8. doi:10.1016/j.gaitpost.2005.09.012.
46. Jian Y, Winter D, Ishac M, Gilchrist L. Trajectory of the body COG and COP during initiation and termination of gait. Gait Posture. 1993;1:9–22. doi:10.1016/0966-6362(93)90038-3.
47. Patchay S, Gahéry Y. Effect of asymmetrical limb loading on early postural adjustments associated with gait initiation in young healthy adults. Gait Posture. 2003;18:85–94. doi:10.1016/S0966-6362(02)00167-4.
48. Wicart P, Maton B. Body equilibrium at the end of gait initiation: importance of ankle muscular force as evidenced in clubfoot children. Neurosci Lett. 2003;351:67–70. doi:10.1016/S0304-3940(03)00619-0.
49. Do Nascimento OF, Nielsen KD, Voigt M. Influence of directional orientations during gait initiation and stepping on movement-related cortical potentials. Behav Brain Res. 2005;161:141–54. doi:10.1016/j.bbr.2005.02.031.
50. Wicart P, Richardson J, Maton B. Adaptation of gait initiation in children with unilateral idiopathic clubfoot following conservative treatment. J Electromyogr Kinesiol. 2006;16:650–60. doi:10.1016/j.jelekin.2005.11.005.

51. Welter ML, Do MC, Chastan N, Torny F, Bloch F, Tézenas du Montcel S, et al. Control of vertical components of gait during initiation of walking in normal adults and patients with progressive supranuclear palsy. Gait Posture. 2007;26:393–9. doi:10.1016/j.gaitpost.2006.10.005.
52. Colné P, Frelut ML, Pérès G, Thoumie P. Postural control in obese adolescents assessed by limits of stability and gait initiation. Gait Posture. 2008;28:164–9. doi:10.1016/j.gaitpost.2007.11.006.
53. Ruget H, Blouin J, Teasdale N, Mouchnino L. Can prepared anticipatory postural adjustments be updated by proprioception? Neuroscience. 2008;155:640–8. doi:10.1016/j.neuroscience.2008.06.021.
54. Chastan N, Westby GWM, Du Montcel ST, Do MC, Chong RK, Agid Y, et al. Influence of sensory inputs and motor demands on the control of the centre of mass velocity during gait initiation in humans. Neurosci Lett. 2010;469:400–4. doi:10.1016/j.neulet.2009.12.038.
55. Vinti M, Couillandre A, Thoumie P. Does somatosensory loss induce adaptation of the gait initiation process? Neurosci Lett. 2010;480:178–81. doi:10.1016/j.neulet.2010.06.017.
56. Yiou E, Do MC. Control of mediolateral stability during rapid step initiation with preferred and non-preferred leg: is it symmetrical? Gait Posture. 2010;32:145–47. doi:10.1016/j.gaitpost.2010.03.018.
57. Mouchnino L, Robert G, Ruget H, Blouin J, Simoneau M. Online control of anticipated postural adjustments in step initiation: evidence from behavioral and computational approaches. Gait Posture. 2012;35:616–20. doi:10.1016/j.gaitpost.2011.12.009.
58. Delval A, Bleuse S, Simonin C, Delliaux M, Rolland B, Destee A, et al. Are gait initiation parameters early markers of Huntington's disease in pre-manifest mutation carriers? Gait Posture. 2011;34:202–7. doi:10.1016/j.gaitpost.2011.04.011.
59. Yiou E, Do MC. Effects of medio-lateral postural perturbation induced by voluntary arm raising on the biomechanical organization of rapid step initiation. Motor Control. 2011;15:507–24. doi:2010-0024 [pii].
60. Ledebt A, Bril B, Brenière Y. The build-up of anticipatory behaviour. An analysis of the development of gait initiation in children. Exp Brain Res. 1998;120:9–17. doi:10.1007/s002210050372.
61. Martin K, Blizzard L, Garry M, Thomson R, McGinley J, Srikanth V. Gait initiation in older people-time to first lateral movement may be the measure of choice. Gait Posture. 2011;34:374–8. doi:10.1016/j.gaitpost.2011.06.004.
62. Jones SF, Twigg PC, Scally AJ, Buckley JG. The gait initiation process in unilateral lower-limb amputees when stepping up and stepping down to a new leveld. Clin Biomech. 2005;20:405–13. doi:10.1016/j.clinbiomech.2004.11.018.
63. Stackhouse C, Shewokis PA, Pierce SR, Smith B, McCarthy J, Tucker C. Gait initiation in children with cerebral palsy. Gait Posture. 2007;26:301–8. doi:10.1016/j.gaitpost.2006.09.076.
64. Vrieling AH, van Keeken HG, Schoppen T, Otten E, Halbertsma JPK, Hof AL, et al. Gait termination in lower limb amputees. Gait Posture. 2008;27:82–90. doi:10.1016/j.gaitpost.2007.02.004.
65. Bruyneel A-V, Chavet P, Bollini G, Mesure S. Gait initiation reflects the adaptive biomechanical strategies of adolescents with idiopathic scoliosis. Ann Phys Rehabil Med. 2010;53:372–386. doi:10.1016/j.rehab.2010.06.005.
66. Muniz AM, Nadal J, Lyons KE, Pahwa R, Liu W. Long-term evaluation of gait initiation in six Parkinson's disease patients with bilateral subthalamic stimulation. Gait Posture. 2012;35:452–7. doi:10.1016/j.gaitpost.2011.11.006.
67. Spencer LM, Van der Meer ALH. TauG-guidance of dynamic balance control during gait initiation across adulthood. Gait Posture. 2012;36:523–6. doi:10.1016/j.gaitpost.2012.05.017.
68. Yu B, Gabriel D, Noble L, An K-N. Estimate of the optimum cutoff frequency for the butterworth low pass digital filter. J Appl Biomech. 1999;15:318–29.

69. van Keeken HG, Vrieling AH, Hof AL, Postema K, Otten B. Stabilizing moments of force on a prosthetic knee during stance in the first steps after gait initiation. Med Eng Phys. 2012;34:733–9. doi:10.1016/j.medengphy.2011.09.017.
70. Liu W, McIntire K, Kim SH, Zhang J, Dascalos S, Lyons KE, et al. Bilateral subthalamic stimulation improves gait initiation in patients with Parkinson's disease. Gait Posture. 2006;23:492–8. doi:10.1016/j.gaitpost.2005.06.012.
71. Delval A, Krystkowiak P, Blatt JL, Labyt E, Bourriez JL, Dujardin K, et al. A biomechanical study of gait initiation in Huntington's disease. Gait Posture. 2007;25:279–88. doi:10.1016/j.gaitpost.2006.04.001.
72. Hirschfeld H. Motor control of every day motor tasks: guidance for neurological rehabilitation. Physiol Behav. 2007;92:161–6. doi:10.1016/j.physbeh.2007.05.018.
73. Chau T. A review of analytical techniques for gait data. Part 1: fuzzy, statistical and fractal methods. Gait Posture. 2001;13:49–66. doi:10.1016/S0966-6362(00)00094-1.
74. Muniz AM, Nadal J. Application of principal component analysis in vertical ground reaction force to discriminate normal and abnormal gait. Gait Posture. 2009;29:31–5. doi:10.1016/j.gaitpost.2008.05.015.
75. Schutte LM, Narayanan U, Stout JL, Selber P, Gage JR, Schwartz MH. An index for quantifying deviations from normal gait. Gait Posture. 2000;11:25–31. doi:10.1016/S0966-6362(99)00047-8.
76. Pataky TC, Robinson MA, Vanrenterghem J, Savage R, Bates KT, Crompton RH. Vector field statistics for objective center-of-pressure trajectory analysis during gait, with evidence of scalar sensitivity to small coordinate system rotations. Gait Posture. 2014;40:255–8. doi:10.1016/j.gaitpost.2014.01.023.
77. Pataky TC, Robinson MA, Vanrenterghem J. Vector field statistical analysis of kinematic and force trajectories. J Biomech. 2013;46:2394–401. doi:10.1016/j.jbiomech.2013.07.031.
78. Ito T, Azuma T, Yamashita N. Anticipatory control in the initiation of a single step under biomechanical constraints in humans. Neurosci Lett. 2003;352:207–10. doi:10.1016/j.neulet.2003.09.002.
79. Dessery Y, Barbier F, Gillet C, Corbeil P. Does lower limb preference influence gait initiation? Gait Posture. 2011;33:550–5. doi:10.1016/j.gaitpost.2011.01.008.
80. Roemmich RT, Nocera JR, Vallabhajosula S, Amano S, Naugle KM, Stegemöller EL, et al. Spatiotemporal variability during gait initiation in Parkinson's disease. Gait Posture. 2012;36:340–3. doi:10.1016/j.gaitpost.2012.01.018.
81. Malouin F, Richards CL. Preparatory adjustments during gait initiation in 4-6-year-old children. Gait Posture. 2000;11:239–53. doi:10.1016/S0966-6362(00)00051-5.
82. Halliday SE, Winter DA, Frank JS, Patla AE, Prince F. The initiation of gait in young, elderly, and Parkinson's disease subjects. Gait Posture. 1998;8:8–14. doi:10.1016/S0966-6362(98)00020-4.
83. Vrieling AH, van Keeken HG, Schoppen T, Otten E, Halbertsma JPK, Hof AL, et al. Gait initiation in lower limb amputees. Gait Posture. 2008;27:423–30. doi:10.1016/j.gaitpost.2007.05.013.
84. Okada Y, Fukumoto T, Takatori K, Nagino K, Hiraoka K. Abnormalities of the first three steps of gait initiation in patients with Parkinson's disease with freezing of gait. Parkinsons Dis. 2011;2011:202937. doi:10.4061/2011/202937.
85. Laudani L, Casabona A, Perciavalle V, Macaluso A. Control of head stability during gait initiation in young and older women. J Electromyogr Kinesiol. 2006;16:603–10. doi:10.1016/j.jelekin.2006.08.001.
86. Couillandre A, Brenière Y, Maton B. Is human gait initiation program affected by a reduction of the postural basis? Neurosci Lett. 2000;285:150–4. doi:10.1016/S0304-3940(00)01015-6.
87. Sasaki O, Asawa S, Katsuno S, Usami SI, Taguchi K. Gait intiation in bilateral vestibular loss. Auris Nasus Larynx. 2001;28:295–9. doi:10.1016/S0385-8146(01)00094-3.
88. Platz T, Denzler P, Kaden B, Mauritz KH. Motor learning after recovery from hemiparesis. Neuropsychologia. 1994;32:1209–23.

89. Hreljac A. Stride smoothness evaluation of runners and other athletes. Gait Posture. 2000;11:199–206.
90. Rohrer B, Fasoli S, Krebs HI, Hughes R, Volpe B, Frontera WR, et al. Movement smoothness changes during stroke recovery. J Neurosci. 2002;22:8297–304.
91. Miff SC, Hansen AH, Childress DS, Gard SA, Meier MR. Roll-over shapes of the able-bodied knee-ankle-foot system during gait initiation, steady-state walking, and gait termination. Gait Posture. 2008;27:316–22. doi:10.1016/j.gaitpost.2007.04.011.
92. Viton JM, Timsit M, Mesure S, Massion J, Franceschi JP, Delarque A. Asymmetry of gait initiation in patients with unilateral knee arthritis. Arch Phys Med Rehabil. 2000;81:194–200.
93. Melzer I, Kurz I, Shahar D, Levi M, Oddsson L. Application of the voluntary step execution test to identify elderly fallers. Age Ageing. 2007;36:532–7. doi:10.1093/ageing/afm068.
94. Melzer I, Shtilman I, Rosenblatt N, Oddsson LIE. Reliability of voluntary step execution behavior under single and dual task conditions. J Neuroeng Rehabil. 2007;4:16. doi:10.1186/1743-0003-4-16.
95. Maki BE, McIlroy WE. The role of limb movements in maintaining upright stance: the "change-in-support" strategy. Phys Ther. 1997;77:488–507.
96. Buckley TA, Pitsikoulis C, Hass CJ. Dynamic postural stability during sit-to-walk transitions in Parkinson disease patients. Mov Disord. 2008;23:1274–80. doi:10.1002/mds.22079.

Contribution of Virtual Reality (Nintendo Wii) for Exercise Training and Rehabilitation

23

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Abstract

The purpose of this chapter is to discuss the use of a commercially available video game console for intervention and/or rehabilitation. This chapter is organized into four main topics. The first one focuses on technology and basic principles and concepts of Nintendo Wii. The technology behind of the Wii gaming system will be described emphasizing the aspects regarding the relevance of such technology to motor behavior. The second topic describes the effect of Wii gaming system in older adults without movement disorders, on healthy aging. Exercise training based on the use of Wii gaming may have a positive impact on metabolic parameters and functional capacity such as balance as a result of aging process. The third topic describes the effect of Wii gaming for rehabilitation of stroke patients. The use of Wii gaming for stroke patients may bring some positive outcomes for motor recovery. Finally, the fourth topic discusses some of the relevant underlying mechanisms of motor learning related with the use of Wii gaming system for health exercising and rehabilitation.

Keywords

Virtual reality • Nintendo Wii • Health • Ageing • Balance • Intervention • Rehabilitation • Stroke patients • Motor learning • Practice

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23.1 Virtual Reality: Focus on Nintendo Wii

Virtual reality (VR) is the term mostly used to describe technologies involving the interaction between a person and a computer generated environment. The concept of virtual reality resides on the idea of presence [1]. According to Steuer, "*virtual reality is defined as a real or simulated environment in which a perceiver experiences telepresence*" [pp. 76–77]. In this context, the user becomes part of this virtual environment and through a combination of a set of devices he/she may perform a variety of actions and have sensory experiences. Products designed for educational, medical, industrial, military, and entertainment purposes are available to customers to explore the virtual world. Examples of such products are body trackers, head mounted displays, motion-sensing gloves, stereoscopic 3D display, joysticks, and virtual reality software [2].

Some studies showed that sensory experiences by virtual reality systems provide advantages according to its applications (e.g., [2, 3]). McIntire and colleagues [2], for instance, found studies (about 60 %) showing that viewing with the stereoscopic 3D display to execute a variety of tasks improved performance over the non-stereo 2D display. Another example of the usefulness of virtual reality systems is for worker safety in the industry [3]. According to these authors, an environment with virtual hands involving the use of motion-sensing gloves provides an application for risk prevention, as well as for training workers in tasks to increase their skill and safety.

The sense of existence from the user distinguishes two main types of VR, the immersive and nonimmersive [4]. According to Man [4], immersive VR systems project the user into a 3D environment produced by a sophisticated system. Immersive VR systems combine a set of devices such as head mounted display or motion-sensor gloves (e.g., PneuGlove) into an integrated system with computer and customized software. It permits the user to interact directly with the simulated world (e.g., [5]). Last generation devices for the immersive VR maximize user's experience. A stereoscopic 3D view moves relative to the user with such realism in which he/she perceives himself/herself in a real world that may be miles away from his/her actual location [6]. The idea of immersion in the artificial environment, therefore, is for complete interaction that allows the person to explore that environment fully.

Nonimmersive VR systems implement a simulated world using a high-resolution monitor. The user is not fully immersed in the artificial environment, but he/she interacts with simple devices such as computer mouse [7]. Furthermore, the interaction may be enhanced by using input devices such as motion tracker or sensor glove which the user may act in a more natural fashion [8, 9]. Both systems are widely used in a variety of applications, but immersive VR systems provide a closer experience of a real situation compared to nonimmersive VR systems. An immersive VR system is expensive and demands a sophisticated technology whereas a nonimmersive VR system is a low-cost solution for easy access, and it is still effective for many applications.

The industry of gaming and entertainment has developed a variety of low-cost nonimmersive VR systems. The Wii gaming system, a type of video game by Nintendo (Kyoto, Japan), is one of these systems, being released in 2006 as the fifth company's home console. Row [10] in his review comments the mimetic motion-control interface between user and machine, an idea that revolutionized the concept of a video game. It means that the user may control the game using physical gestures as well as pressing buttons of the controller (i.e., Wii remote or Wiimote). For example, in the Wii Sports, while playing a tennis match, the user performs the gesture of forehand (i.e., the action to strike a ball with a racket held by the hand on the same side of the body) holding the Wii remote as if he/she were holding a racquet in real tennis game. The same assumption applies to other games and exercises. The user as part of a real and virtual reality interacts with a virtual reality activating perceptual and motor systems to properly act in such virtual reality. On the other side, the Wii system is able to detect responses from the user and immediately the virtual environment alters challenging the user to a different scenario.

A relevant feature of Wii remote is a wireless motion-sensitive controller. It is used either as a handheld pointing device or as an implement serving to be a racquet, a paddle, a ball, steering wheel, and others [11]. As reported by Jones and Thiruvathukal [11], the Wii remote detects changes in direction, speed, and acceleration in three dimensions with high precision due to built-in accelerometer and gyrosensors. An optical sensor at one end of Wii remote helps to determine where it is pointing [12]. Some of the games in Wii need a second controller, the Nunchuk, which plugs into the Wii remote, requiring the two-handed coordination. The Wii remote communicates with a sensor bar placed at the center and above or below the television. The Wii remote sends via bluetooth the data regarding position, acceleration, and the button's state to the Wii console [13].

The bluetooth technology changed the concept of playing video game. The control of the games is now based on movements generated by the user (holding the Wii remote). Movements from the user holding and manipulating the Wii remote reflect on the TV display through his/her avatar. Holzwarth and collaborators [14] define avatars as, "general graphic representations that *are personified using computer technology*" (p. 20). In the Wii gaming system, the avatar is called Mii [11]. The user controls a Mii with his/her actions by holding the Wii remote giving him/her the perception of being part of such virtual world. The games in the Wii system (e.g., Wii Sports, Wii Fit, Wii Sports Resort, Wii Play – these are some of the packages of games and exercises collected in a compact disc) give to the user through a display visual feedback of the performance. Also, the games give to the user motivational feedback, positive comments, and progress information. The Wii system shows the potential to contribute to motor and perceptual development as well as motor skill acquisition and physical training.

Another wireless device that communicates via bluetooth with the Wii gaming system is the Wii Balance Board (WBB). Such a device comprises of four force transducers placed on at each corner of the platform, and then it measures the body weight and motion, precisely [15]. Also, the built-in concept of the WBB is similar

to that of a laboratory force platform that contains four force transducers that assess force distribution and the resultant movements in COP (center of pressure). The concept of this rigid platform is that the user uses the body over the WBB to control the game [11]. The user controls the balance games (Wii Fit) by adjusting the weight on either leg. A Mii (i.e., an avatar that represents the user) displayed on a screen shows the adjustments done by the user over the WBB. COP shown on the screen generally requires displacement on medial–lateral and anterior–posterior axes. The most popular game for WBB is the Wii Fit that features activities such as yoga, strength training, aerobics, and balance games. The focus of the Wii Fit is to offer the users a program that he/she can work towards personal goals to enhance health and motor control and coordination, mainly for balance and posture.

Some of the games featured in the Wii require the users to be physically active or exercise with physical effort. This style of gaming is known as "active video game" or "exergaming" that means to exercise using video games [16]. Games such as bowling and boxing (Wii Sports) require the whole body movements to participate in virtual games, although the intensity for exercising the games is still low compared to a real boxing and bowling games. Activities in the Wii Fit particularly in aerobics or strength training appear to require more energy expenditure, and therefore it would be helpful to promote an active lifestyle. Moreover, the need of a system for physical training or motor rehabilitation that is motivating and enjoyable is one of the reasons for growing interest to incorporate video game systems into clinical and fitness settings.

23.2 The Use of Wii Gaming for Healthy Aging

The regular practices of exercises contribute to a healthy aging process. According to the American College of Sports [17], physical exercises for older adults should include exercises based on aerobic capacity, muscle strengthening, and flexibility. The lack of physical activity in the adult population leads to obesity and higher body-weight conditions. These conditions are strongly associated with a sedentary lifestyle [18]. Physical activity programs for older adults must be designed to encourage them to maintain an active daily routine minimizing the effects of a sedentary lifestyle. All alternative ways must be available to adults and older adults to engage in physical activity practices, particularly to those who did not have previous physical exercise experiences and do not feel themselves motivated to begin to practice.

Video game consoles, such as Wii gaming system, have largely been incorporated into physical fitness programs. The use of technology from the entertainment industry is an enjoyable way that has recently been explored to encourage people of all ages for physical exercise. It provides at least an ambiance with fun and an opportunity to discover the level of his/her abilities (through his/her avatar) as well as to experience all sorts of motor skills. A benefit of such one-to-one interaction (i.e., user and machine) is the time available to the user to familiarize with motor skills required for playing and exercising without exposing

himself/herself to others. Therefore, the Wii gaming may constitute an opportunity to warrant an extra motivation for physical activity adherence. It would also be an excellent opportunity to those who are not accustomed to physical exercises and sports to initiate a fitness program.

Is the exergame or playing an active video game such as Wii system enough for activating energy expenditure rates for general health? Graves and colleagues [19] revealed that the physiological cost of exercising – such as heart rate, oxygen consumption – VO2, and energy expenditure – on Wii Fit games was greater than handheld video gaming. However, costs of exercising with the Wii Fit games were lower than treadmill exercise for older adults, young adults, and adolescents. Guderian and colleagues [20] found similar results showing that a session of Wii Fit games of approximately 25 min duration in middle age and older adults activates physiological and metabolic responses. In both studies [19, 20], the Wii Fit games applied to older people were considered moderate according to the metabolic equivalent of task (MET). MET expresses the energy cost of a physical activity. Therefore, exercising with Wii Fit games benefits health and is within the recommendations for improving and maintaining cardiorespiratory fitness.

The Wii Sports and Wii Fit are the most frequent games explored for research. The energy expenditure measure in the games and exercises of the Wii Fit and Wii Sports was classified as of light and moderate intensities [21]. Specifically, the mean MET values in yoga and balance exercises in the Wii Fit were 2.1 and 2.0, which reflect the activity as of light intensity. Resistance and aerobic exercise of Wii Fit and a set of games of Wii Sports (golf, bowling, tennis, baseball, and boxing) were, 3.2, 3.4, and 3.0 METs, classified as of moderate intensity. Games and exercises as of vigorous intensity were not found either on Wii Fit or on Wii Sports. Overall, these findings are relevant to design exercise training programs for the adult and elderly individuals. Both Wii Fit and Wii Sports games may contribute to enhancing cardiorespiratory fitness, but also muscle strength, motor coordination, and balance, critical for physiological capacities.

The intervention based on the Wii gaming system on motor abilities in healthy elderly has positive effects on balance. Cho and colleagues [22] showed that Wi Fit training is effective at improving the balance of elderly participants, in a program of three times per week for eight weeks. Similarly, Chao and colleagues [23] showed that Wii Fit exercise group improved in balance and mobility practicing twice a week for four weeks compared with a group that received health education program. In an elaborate research protocol, Toulotte and colleagues [24] showed that Wii Fit games improved the static balance on bipedal conditions, whereas the adapted physical activities improve the dynamic balance, after 20 weeks of one oriented session per week lasting one hour each. Furthermore, the authors showed that adapted physical activities combined with Wii Fit games did not have significant additional benefits. Overall, the Wii Fit games, featuring yoga, balance games, strength training, and aerobics activities, appear to be an alternative for older adults to exercise and improve balance.

Other attempts, however, showed little or no effects of Wii gaming system on motor abilities for older individuals. Franco and colleagues [25], for example,

found small improvements in balance using Wii Fit training program after twice a week for three weeks. The improvement was not significantly greater than that observed for the control group, who did not receive any training intervention. A serious limitation of the previous study is the number of training sessions, which might have an impact on the lack of effect. An intervention protocol comparing exercises based on videogame and physical exercise training [26] for eight weeks, two training sessions per week, showed that exercises in the Wii Fit are effective as the Tai Chi training program is on postural control and balance measures. Therefore, there was no distinction between the results of both virtual and real training program. It is possible that improvements on postural and balance measures with Wii Fit may be due to the activation of similar neuromuscular mechanisms of the physical training programs.

If the Wii Fit training is sufficient to improve postural and balance control, then such improvements may have some effect on fall prevention. A recent literature review [27] reported that for the majority of the studies examining the Wii gaming system for balance training and fall prevention in older adults both with and without balance impairment improves balance control, balance confidence, and gait measures. Furthermore, such positive effect was shown in the middle-aged adults and the hospitalized frail elderly participants. According to the authors [27], it is not evident the association between the lack of effect and any aspect of the study design for the few studies failing to show improvements. Although the research is not conclusive about the effectiveness of Wii for preventing falls, evidences of using Wii gaming as an adjunctive method show the potential to improve physical functions related to balance control in healthy older adults.

The underlying motor process of positive effects for balance control with Wii gaming in older people is still in discussion. Exercises with Wii Fit improve muscle strength because the practice of Wii gaming demands single-leg balance and body-weight resistance workouts [28]. Furthermore, the visual feedback of the center of gravity or altering body postures to meet task requirements, for instance, promotes motor learning [28]. Many of the Wii Fit activities that require static and dynamic balance demand higher postural adjustments, thus activating neural pathways at the spinal cord and cortical levels.

Is the Wii gaming viable for the older adults to engage in exercise training? There is evidence to support that the Wii gaming is safe and feasible to engage older adults to exercise training [29]. Based on previous findings, Chao and colleagues [23] described that the Wii gaming increases social interaction, alleviates symptoms of depression, improves adherence to exercises, and improves mood. Such positive effects may benefit a long-term effectiveness intervention program. In addition, older adults have reported that the Wii gaming was an enjoyable and acceptable method for exercise and that they would continue to practice after the research protocol. The use of Wii seems attractive not only for the positive results but also because of stimulating settings for both therapists and the elderly population.

23.3 The Use of Wii Gaming for Stroke Rehabilitation

The Wii gaming system is being used for motor rehabilitation in adults with movement impairments, particularly stroke survivors. Stroke is the second major cause of death worldwide and one of the leading cause of disability [30]. Among several factors, the clinical condition following stroke depends on the affected cerebral region and the extent of the injury. Often stroke patients experience sensory, motor, and cognitive impairments, which impact on their abilities to perform daily living activities. The recovery to maintain upright posture, walking, reaching, and manipulating objects with the affected arm following stroke is the objective of any rehabilitation. A range of an array of rehabilitation techniques are available, some are more effective than others [31]. There is not a gold standard treatment and research focusing on recent technologies for stroke rehabilitation is an avenue to establish effective protocols that may maximize the motor and functional recovery.

The high cost associated with stroke-related healthcare encourages the clinicians and researchers explore the effectiveness of devices of easy access, low cost, and with a broad range of environments for movements. There is a consensus that the Wii gaming for stroke patients displays a series of advantages [32]. For instance, the Wii gaming system provides repetitive task-specific training; it offers training in an enriched environment; it simulates real-world activities giving more ecological validity; and it is interesting and enjoyable encouraging patients for training. The Wii gaming appears an alternative to establishing an innovative and stimulating rehabilitation in clinical settings and at home. A further aspect of relevance is to gain some understanding on the mechanisms associated with the motor and functional amelioration in stroke.

Physical therapy based on commercial gaming consoles and off-the-shelf video games shows some benefits to stroke patients [33, 34]. A pilot study with patients less than three months after a stroke (subacute phase) tested the effect of Wii gaming therapy for upper limbs [35]. Joo and colleagues (2010) showed significant improvements in the Fugl-Meyer Assessment and Motricity Index scores on pre- and post-intervention. However, due to the lack of control group the effect of Wii gaming is uncertain. The set of games in the Wii Sports (i.e., boxing, bowling, tennis, golf, and baseball games and training) was used for six sessions of upper limb exercises, 30 min each over two weeks. During Wii gaming therapy, all patients received daily one hour of occupational therapy and one hour of physical therapy. According to the authors, the contribution of spontaneous recovery, as well as the conventional therapy administered simultaneously with Wii gaming, cannot be discarded over the positive effects on the Fugl-Meyer Assessment and Motricity Index scale. The main conclusion by the authors is that Wii gaming system appears to be viable and complementary to conventional rehabilitation in stroke patients in the subacute period with moderate impairments of upper limbs.

Similar to a previous study [35], Saposnisk and colleagues [36] showed beneficial effects of Wii gaming therapy for stroke patients in a pilot randomized controlled trial study. The Wii therapy group had a significant improvement in

the Wolf Motor Function Test compared with recreational therapy group. Patients were those with less than three months poststroke with moderate impairments. Wii gaming therapy focused on upper limbs with eight sessions of one hour each over a period of two weeks. During the Wii gaming protocol, all stroke patients were receiving conventional occupational therapy and physiotherapy. The Wii gaming group played Wii Sports games and Cooking Mama, 30 min each, whereas recreational therapy group played cards, bingo, or Jenga. The use of video game such as Wii appears to maximize gains in upper limb movement and function. According to the authors, Wii gaming practice by observing his/her representation on the screen of a context-specific task activates the mirror neuron circuit. These neurons are selectively activated by only seeing a similar action [37] helping in promoting neural plasticity and function recovery.

An elaborate study of *Wii* gaming program for stroke survivors focused on an intensive training protocol for the upper limbs [38]. The results showed improvements in the Wolf Motor Function Test and the Fugl-Meyer Assessment scores of stroke patients. Moreover, patients reported (MAL-QOM scores) using the affected hand and arm in more activities of daily living. The therapy program comprised of the one-hour session of five days per week for two weeks. The stroke patients had additional home sessions that gradually increased in duration from 30 min to three hours per day over the two weeks. Similar to previous studies, the set of games of Wii Sports was administered to patients. Mouawad and colleagues [38] argued that the positive results for stroke group substantially reflect the benefits of Wii gaming protocol. Five participants from a total of seven were chronic (i.e., more than six months poststroke) stroke patients, and they were not receiving any other type of rehabilitation. The spontaneous movement recovery would be minimal. An intensive Wii gaming therapy had an impact on limb function of chronic stroke patients. According to the authors, Wii gaming might elevate motivation facilitating compliance with the therapy. Whether intensive gaming therapy applied in clinical and home settings led to significant improvements in function and movement outcomes is a matter of further investigation.

Whereas some studies with Wii gaming system were dedicated to the upper limbs, others focused on lower limbs. In particular, Wit Fit with balance board may provide a viable means of improving postural control and balance. A randomized controlled trial study run therapy programs for 30 subacute stroke patients [39]. The authors showed improvement in postural control (Step Test and the COP measured on WBB) for patients submitted to Wii Fit training on a balance board in standing position. Whereas the control group trained with the Wii Sports and Wii Sports Resort in sitting position showed an improvement in arm function, but it was not significant. Both groups trained for three sessions per week, 45 min each, over two to four weeks, in addition to conventional physiotherapy and occupational therapy. These results strongly indicate task-specific training effects according to training programs.

Comparing two equivalent physical therapy programs related to lower limb exercises, Barcala and colleagues [40] showed beneficial effects in both groups of physical therapies for chronic stroke patients. Both the physical therapy program

combined with activities of Wii Fit and the conventional physical therapy only led to improvements in body symmetry, balance, and function. The therapy was done in two sessions per week, 60 min for each session over five weeks. Wii Fit balance training lasted an extra 30 min for the combined therapies. The combination of exercises involving muscle strengthening, static and dynamic balance training, training of functional activities (conventional physical therapy) and specific balance exercises with visual feedback that strengthen the sensory-motor integration (Wii Fit exercises) appears to enhance the effect of stroke patients rehabilitation.

Another randomized controlled trial study with chronic stroke patients demonstrated that the Wii Fit therapy group was more effective than conventional weight-shift training instability on some sensory-challenged positions [41]. However, both groups showed improvement in the Timed Up and Go Test and forward reach tests and fear of falling. Physical therapy protocols occurred twice a week, 30 min each for 12 weeks and simultaneously with another physical maintenance program. Different from a previous study, Hung and colleagues (2014) run the follow-up of three months; the Wii Fit group did not maintain the training effects in terms of stability, while the weight-shift training (i.e., control group) showed more improvement in weight-bearing symmetry than the Wii Fit group. According to the authors, the gain from Wii Fit training was not enough to be maintained possibly due to the rapid weight shifting between the legs.

Wii gaming system appears to provide some additional gain to stroke patients and may be part of rehabilitation. The results in the literature suggest that Wii gaming system may be introduced as physical therapy as soon as possible, at least in the subacute stage. The lack of high-quality evidence prevents one to conclude about the effectiveness of the Wii gaming system and other similar systems – such as PlayStation and Kinect Microsoft [33, 34]. The Wii gaming is still exploratory in clinical settings and much research is still to come to consolidate some of the preliminary findings. The protocol incorporating Wii gaming system needs further research contemplating large-scale samples, randomized control trials, comparable procedures and types of video game systems, complete assessment such as limb movement, limb function, and activities of daily living, aspects of training related with the number of trials, frequency training, intensity, duration period, and long-term follow-up.

23.4 Underlying Mechanisms of Motor Learning and Wii Gaming

The Nintendo Wii Fit and Wii Sports are widely spread both for health exercise training and rehabilitation. Studies using the Wii gaming to promote healthy aging and prevent degenerative diseases demonstrated the efficacy on energy expenditure. More recently, Wii gaming is being incorporated into rehabilitation of functional and movement recovery of stroke patients. The safety and feasibility of Wii gaming for exercise training and rehabilitation (e.g., [34]) are fairly established in research. Wii gaming system was primarily designed as consuming product for entertainment

purposes for health people. The application to healthy aging and rehabilitation should be further examined in terms of adapting the devices for individuals who do not have sufficient hand control or are not able to stand on the Wii Fit board because of balance deficits [42].

Wii gaming allows the user to interact with a computer-simulated environment in a range of games and activities. The user produces motor actions that reflect on a screen by a character, avatar, which represents him/her in the virtual environment. Playing Wii games requires sensory-motor integration and cognitive effort. The user must capture relevant visual or auditory information from a screen full of elements. Moreover, he/she must interpret such information to elaborate a plan and execute the appropriate response. In some of the Wii games, the user does not have the opportunity to correct his/her movement during a play. Frequently, in these contexts the user must plan his/her actions in advance before a movement begins and correction is made on the next attempt (e.g., Wii Sports Resorts: archery). In other games, visual or auditory cues (i.e., real-time feedback on performance) may supplement coordination and motor control during a play. The user may correct or adjust his/her movement for a better performance on ongoing task (e.g., Wii Sport: wakeboarding).

The understanding of the mechanisms that promotes improvements both on healthy aging and on motor impairments is one of the key points to consolidate Wii gaming as exercising program and rehabilitation, respectively. The motor skill performance to play games and to exercise in the Wii system demands explicit knowledge to operate Wii devices and the usage of real-time feedback to achieve game or exercise objective. Feedback plays a significant role for motor skill acquisition and learning [43]. The Wii games provide visual, auditory, and tactile (haptic) feedback [42]. The haptic (e.g., presented in the form of rumbles) and auditory feedbacks are transmitted through the Wii remote (i.e., the main game controller), while the visual feedback is showed on the screen [11]. The feedback information serves to reduce the uncertainty of the virtual environment to achieve task goal.

Two main types of feedback are available to learners [43]. The intrinsic feedback refers to the sensory information as a result of producing movement. Intrinsic feedback comes from sources outside of the body (exteroception) and inside the body (proprioception). The former informs about the events in the environment, mainly with vision and hearing. The latter informs about the own body movement such as limb position and movement using the vestibular system, muscle, joint, and cutaneous receptors. The extrinsic feedback is additional information over the content of intrinsic feedback and comes from an outside source (e.g., verbal or written instruction, and images of a video played). According to Schmidt and Lee [43], after the completion of a motor action extrinsic feedback is categorized into the knowledge of results (KR) and knowledge of performance (KP). KR is the feedback about the outcome of the movement relative to the performance's goal, whereas KP is the feedback about the movement pattern generated to achieve the goal. According to Deutsch and colleagues [42], KR is the predominant type of feedback in the Wii gaming, specifically in the Wii Sports and Wii Fit, compared to the KP.

Feedback provided by Wii gaming system is abundant and diverse. In many cases, KR may be redundant with the intrinsic feedback, and it is meaningless to the user. However, KR is not always redundant with inherent feedback. KR about the partial or total scores or the precise location of a target shooting helps to ameliorate performance. Deutsch and colleagues [42] argue that the abundance of KR in the Wii gaming may explain the positive outcomes for rehabilitation based on the assumption that KR is used to facilitate learning and skill acquisition. In the Wii Fit balance exercises, for instance, online visual feedback (KR) informs the user about how much and in what direction he/she is oscillating while performing balance exercises. Moreover, Wii gaming consistently rewards the user with positive feedback that is not linked to the performance but related to enhancing motivation and engagement [42]. For example, in the Wii games and exercises, there is cheering in the stadium, there are ribbons and pleasing music when the user wins a game or succeeds in the execution of an exercise, and stars appear to reward the winners. Feedback, mainly the positive or rewarding ones, may serve to motivate patients and individuals to rehabilitation and exercise programs.

A fundamental principle that directly affects motor skill acquisition and learning is practice. The repeated rehearsal of a movement or motor action may guarantee the motor skill acquisition, but it does not guarantee the retention of acquisition (i.e., learning) over time [43]. One of the first concerns about rehabilitation and exercise training refers to organizing practice schedule, such as the frequency per week, the duration of each practice session, and the length of a practice protocol. At first glance, one could argue that providing as much practice as possible would maximize the practice effect. However, there is a limit to the amount of practice per day for the effective learning, at least for the non-patient individuals [43].

An approach in stroke rehabilitation based on intensive massed practice (i.e., constraint-induced movement therapy – CIMT) restored substantial motor function in chronic stroke patients [44]. This protocol limits the use of the unaffected limb stimulating the utilization of the affected limb through massed practice (approximately six hours a day for two weeks) in daily activities. Although the CIMT improves upper limb function following stroke when compared to alternative or no treatment, it remains controversial in a number of issues (e.g., [45, 46]). Practice methods that do not involve physical execution may also improve learning [43]. Intensive massed practice such as CIMT for stroke rehabilitation may integrate with a practice that does not involve physical rehearsals. For example, the observation of a model executing a task takes the learner to gather all relevant information and think about his/her execution [43]. Similar processing may occur with a mental practice that requires mental effort for imagery processing.

The variable practice of a given task is better for retention and generalizability compared to constant practice [43]. According to the authors, variability of a particular task operates on a generalized motor program that defines a pattern of movement and its parameters. Neurological patients may benefit from strong and flexible generalized motor programs for functional and movement recovery over time. Wii gaming system offers a varied and enjoyable environment for exercise training and rehabilitation. In such environment, the chance of individuals to sustain the motivation to practice for an extended period is promising.

The positive effect of Wii gaming system in stroke patients is possibly due to brain plasticity [47]. A cortical reorganization and a rewiring damaged neural circuit occur as a result of training and practice. You and colleagues [48] showed that cortical reorganization associates with improvements on lower limb function for stroke patients after a VR rehabilitation. Practicing with Wii gaming facilitates motor representation and promotes reorganization of motor cortical function. Functional magnetic resonance imaging study reported related changes in cortical activity accompanied by behavioral improvements in the upper limbs of chronic stroke survivors after VR rehabilitation [49]. Similarly, physical therapy with Wii Fit modulated cortical motor excitability of stroke patients accompanied by improvement in dynamic balance and balance confidence [50]. Behavioral improvements demonstrated in clinical trials associated with rehabilitation based on Wii gaming need further understanding about the underlying neural circuit changes.

23.5 Conclusion

The finding related to the use of Wii gaming system is that the energy requirements of playing the games correspond to moderate exercise meeting the recommended guidelines for health and fitness. Wii gaming system is feasible as physical therapy to improve motor control and coordination in patients as well as to enhance physiological and functional capacities in healthy aging. Although the use of commercial video games may be complex and hard for patients, it is still an alternative for an adjunctive therapy.

References

1. Steuer J. Defining virtual reality - dimensions determining telepresence. J Commun. 1992;42 (4):73–93.
2. McIntire JP, Havig PR, Geiselman EE. Stereoscopic 3D displays and human performance: a comprehensive review. Displays. 2014;35(1):18–26.
3. Pouliquen M, Bernard A, Marsot J, Chodorge L. Virtual hands and virtual reality multimodal platform to design safer industrial systems. Comput Ind. 2007;58(1):46–56.
4. Man DWK. Common issues of virtual reality in neuro-rehabilitation. In: Kim J-J, editor. Virtual Real-London. Shanghai: China InTech; 2010. p. 419–28.
5. Connelly L, Jia YC, Toro ML, Stoykov ME, Kenyon RV, Kamper DG. A pneumatic glove and immersive virtual reality environment for hand rehabilitative training after stroke. IEEE T Neur Sys Reh. 2010;18(5):551–9.
6. Editorial. Virtual reality is nearly on our doorstep Bangkok, Thailand: The Nation; [cited 2015 20th Apr]. Available from: http://www.nationmultimedia.com/opinion/Virtual-reality-is-nearly-on-our-doorstep-30257950.html.
7. Allain P, Foloppe DA, Besnard J, Yamaguchi T, Etcharry-Bouyx F, Le Gall D, et al. Detecting everyday action deficits in Alzheimer's disease using a nonimmersive virtual reality kitchen. J Int Neuropsych Soc. 2014;20(5):468–77.
8. Olivieri I, Chiappedi M, Meriggi P, Mazzola M, Grandi A, Angelini L. Rehabilitation of children with hemiparesis: a pilot study on the use of virtual reality. Biomed Res Int. 2013.

9. Sato K, Fukumori S, Matsusaki T, Maruo T, Ishikawa S, Nishie H, et al. Nonimmersive virtual reality mirror visual feedback therapy and its application for the treatment of complex regional pain syndrome: an open-label pilot study. Pain Med. 2010;11(4):622–9.
10. Row H. Codename revolution: the Nintendo Wii platform. New Media Soc. 2015;17(2):312–4.
11. Jones SE, Thiruvathukal GK. Codename revolution: the Nintendo Wii platform. The MIT Press; 2012.
12. Lee JC. Hacking the Nintendo Wii remote. IEEE Pervas Comput. 2008;7(3):39–45.
13. Turner D. The Nintendo Wii. Technol Rev. 2007;110(4):22–3.
14. Holzwarth M, Janiszewski C, Neumann MM. The influence of avatars on online consumer shopping behavior. J Marketing. 2006;70(4):19–36.
15. Clark RA, Bryant AL, Pua YH, McCrory P, Bennell K, Hunt M. Validity and reliability of the Nintendo Wii Balance Board for assessment of standing balance. Gait Posture. 2010;31 (3):307–10.
16. Sinclair J, Hingston P, Masek M. Considerations for the design of exergames. Graphite 2007: 5th International Conference on Computer Graphics and Interactive Techniques in Australasia and Southern Asia, Proceedings; 2007. p. 289–95.
17. Chodzko-Zajko WJ, Proctor DN, Singh MAF, Minson CT, Nigg CR, Salem GJ, et al. Exercise and physical activity for older adults. Med Sci Sport Exer. 2009;41(7):1510–30.
18. Martinez-Gonzalez MA, Martinez JA, Hu FB, Gibney MJ, Kearney J. Physical inactivity, sedentary lifestyle and obesity in the European Union. Int J Obes Relat Metab Disord. 1999;23 (11):1192–201.
19. Graves LEF, Ridgers ND, Williams K, Stratton G, Atkinson G, Cable NT. The physiological cost and enjoyment of wii fit in adolescents, young adults, and older adults. J Phys Act Health. 2010;7(3):393–401.
20. Guderian B, Borreson LA, Sletten LE, Cable K, Stecker TP, Probst MA, et al. The cardiovascular and metabolic responses to Wii Fit video game playing in middle-aged and older adults. J Sport Med Phys Fit. 2010;50(4):436–42.
21. Miyachi M, Yamamoto K, Ohkawara K, Tanaka S. METs in adults while playing active video games: a metabolic chamber study. Med Sci Sport Exer. 2010;42(6):1149–53.
22. Cho GH, Hwangbo G, Shin HS. The effects of virtual reality-based balance training on balance of the elderly. J Phys Ther Sci. 2014;26(4):615–7.
23. Chao YY, Scherer YK, Montgomery CA, Wu Y-W, Lucke KT. Physical and psychosocial effects of Wii fit exergames use in assisted living residents: a pilot study. Clin Nurs Res. 2014.
24. Toulotte C, Toursel C, Olivier N. Wii Fit (R) training vs. adapted physical activities: which one is the most appropriate to improve the balance of independent senior subjects? A randomized controlled study. Clin Rehabil. 2012;26(9):827–35.
25. Franco JR, Jacobs K, Inzerillo C, Kluzik J. The effect of the Nintendo Wii Fit and exercise in improving balance and quality of life in community dwelling elders. Technol Health Care. 2012;20(2):95–115.
26. Pluchino A, Lee SY, Asfour S, Roos BA, Signorile JF. Pilot study comparing changes in postural control after training using a video game balance board program and 2 standard activity-based balance intervention Programs. Arch Phys Med Rehab. 2012;93(7):1138–46.
27. Pietrzak E, Cotea C, Pullman S. Using commercial video games for falls prevention in older adults: the way for the future? J Geriatr Phys Ther. 2014;37(4):166–77.
28. Siriphorn A, Chamonchant D. Wii balance board exercise improves balance and lower limb muscle strength of overweight young adults. J Phys Ther Sci. 2015;27(1):41–6.
29. Chao YY, Scherer YK, Montgomery CA. Effects of using nintendo wii exergames in older adults: a review of the literature. J Aging Health. 2015;27(3):379–402.
30. Donnan GA, Fisher M, Macleod M, Davis SM. Stroke. Lancet. 2008;371(9624):1612–23.
31. Langhorne P, Coupar F, Pollock A. Motor recovery after stroke: a systematic review. Lancet Neurol. 2009;8(8):741–54.
32. Laver KE, George S, Thomas S, Deutsch JE, Crotty M. Virtual reality for stroke rehabilitation. Cochrane Db Syst Rev. 2011;9.

33. Pietrzah E, Cotea C, Pullman S. Using commercial video games for upper limb stroke rehabilitation: is this the way of the future? Top Stroke Rehabil. 2014;21(2):152–62.
34. Thomson K, Pollock A, Bugge C, Brady M. Commercial gaming devices for stroke upper limb rehabilitation: a systematic review. Int J Stroke. 2014;9(4):479–88.
35. Joo LY, Yin TS, Xu D, Thia E, Chia PF, Kuah CWK, et al. A feasibility study using interactive commercial off-the-shelf computer gaming in upper limb rehabilitation in patients after stroke. J Rehabil Med. 2010;42(5):437–41.
36. Saposnik G, Teasell R, Mamdani M, Hall J, McIlroy W, Cheung D, et al. Effectiveness of virtual reality using Wii gaming technology in stroke rehabilitation: a pilot randomized clinical trial and proof of principle. Stroke. 2010;41(7):1477–84.
37. Keysers C. Mirror neurons. Curr Biol. 2009;19(21):R971–3.
38. Mouawad MR, Doust CG, Max MD, McNulty PA. Wii-based movement therapy to promote improved upper extremity function post-stroke: a pilot study. J Rehabil Med. 2011;43 (6):527–33.
39. Bower KJ, Clark RA, McGinley JL, Martin CL, Miller KJ. Clinical feasibility of the Nintendo Wii (TM) for balance training post-stroke: a phase II randomized controlled trial in an inpatient setting. Clin Rehabil. 2014;28(9):912–23.
40. Barcala L, Grecco LAC, Colella F, Lucareli PRG, Salgado ASI, Oliveira CS. visual biofeedback balance training using Wii fit after stroke: a randomized controlled trial. J Phys Ther Sci. 2013;25(8):1027–32.
41. Hung JW, Chou CX, Hsieh YW, Wu WC, Yu MY, Chen PC, et al. Randomized comparison trial of balance training by using exergaming and conventional weight-shift therapy in patients with chronic stroke. Arch Phys Med Rehab. 2014;95(9):1629–37.
42. Deutsch JE, Brettler A, Smith C, Welsh J, John R, Guarrera-Bowlby P, et al. Nintendo Wii sports and Wii fit game analysis, validation, and application to stroke rehabilitation. Top Stroke Rehabil. 2011;18(6):701–19.
43. Schmidt RA, Lee TD. Motor learning and performance. From principles to application. 5th ed. Champaign: Human Kinetics; 2014.
44. Taub E, Miller NE, Novack TA, Cook EW, Fleming WC, Nepomuceno CS, et al. Technique to improve chronic motor deficit after stroke. Arch Phys Med Rehab. 1993;74(4):347–54.
45. Krakauer JW. Motor learning: its relevance to stroke recovery and neurorehabilitation. Curr Opin Neurol. 2006;19(1):84–90.
46. Hakkennes S, Keating JL. Constraint-induced movement therapy following stroke: a systematic review of randomised controlled trials. Aust J Physiother. 2005;51(4):221–31.
47. Adamovich SV, August K, Merians A, Tunik E. A virtual reality-based system integrated with fmri to study neural mechanisms of action observation-execution: a proof of concept study. Restor Neurol Neurosci. 2009;27(3):209–23.
48. You SH, Jang SH, Kim YH, Hallett M, Ahn SH, Kwon YH, et al. Virtual reality-induced cortical reorganization and associated locomotor recovery in chronic stroke - an experimenter-blind randomized study. Stroke. 2005;36(6):1166–71.
49. Orihuela-Espina F, del Castillo IF, Palafox L, Pasaye E, Sanchez-Villavicencio I, Leder R, et al. Neural reorganization accompanying upper limb motor rehabilitation from stroke with virtual reality-based gesture therapy. Top Stroke Rehabil. 2013;20(3):197–209.
50. Omiyale O, Crowell CR, Madhavan S. Effect of Wii-based balance training on corticomotor excitability post stroke. J Motor Behav. 2015;47(3):190–200.

Deep Brain Stimulation in Parkinson's Disease: Effects on Gait and Postural Control

24

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Abstract

Patients with Parkinson's disease (PD) commonly suffer from gait and postural disorders (Bloem et al., Mov Disord 19:871–84, 2004), often leading to falls and injuries (Segev-Jacubovski et al., Expert Rev Neurother 11:1057–75, 2011; Montero-Odasso et al., J Am Geriatr Soc 60:2127–36, 2012). When the symptoms of the disease become resistant to drugs, a routine neurosurgical treatment is implantation of electrodes for deep brain stimulation (DBS). Whereas DBS is most effective in alleviating the symptoms of tremor and rigidity, it has also been demonstrated to reduce gait variability, thus improving stability (Hausdorff et al., Mov Disord Off J Mov Disord Soc 24:1688–92, 2009). We therefore begin by reviewing the effect of DBS on gait and posture when electrodes are implanted in the most common targets: the subthalamic nucleus (STN) and the globus pallidus internum (GPi) and with the commonly used high frequency parameters (around 130 Hz), considering the

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possible mechanisms underlying its therapeutic effect, negative effects, and the deterioration in therapeutic effect over time. We continue by examining a recently suggested change of parameters, reduction of stimulation frequency to 60 or 80 Hz, in order to specifically treat gait impairment. Another phenomenon that has been addressed by DBS in recent year is freezing of gait, which is a particularly debilitating phenomenon experienced by a subpopulation of patients with PD. To alleviate freezing, the pedunculopontine nucleus (PPN) has been suggested as the target brain area. Mixed evidence as for the effectiveness of this treatment and the exact symptoms it affects imply that more studies are necessary in order to establish PPN DBS as a routine treatment. We conclude with a brief summary of open questions and work that should be addressed in future studies.

Keywords
Deep brain stimulation • DBS • Gait • Walking • Posture • Stability • Parkinson's disease • Basal ganglia • Subthalamic nucleus • Neurosurgery

Abbreviations

DBS	Deep brain stimulation
fMRI	Functional magnetic resonance imaging
GPi	Globus pallidus internum
PD	Parkinson's disease
PIGD	Postural instability and gait disturbances
PPN	Pedunculopontine nucleus
STN	Subthalamic nucleus
UPDRS	Unified Parkinson's disease rating scale

24.1 Introduction

Deep brain stimulation was introduced in 1987 for the treatment of the extrapyramidal tremor in Parkinson's disease (PD) [5]. Today, it has become routinely used for treatment of PD, dystonia, and essential tremor, and is also applied for chronic pain, obsessive-compulsive disorder, and even epilepsy. Here we briefly describe the evidence regarding the effects of deep brain stimulation (DBS) on gait and postural disorders in PD, the pros and cons of low versus high frequency stimulation, and stimulation of the pedunculopontine nucleus (PPN) for treatment of gait and posture disorders.

Gait disturbances, both those that are continuous and those that are episodic, in PD severely affect the quality of life and functional independence [1, 2, 3,

6]. Surgical treatment by deep brain stimulation (DBS) reduces the symptoms of the disease and the dyskinetic effects of levodopa [4, 7]. To date, multiple brain areas have been suggested as targets for implantation of DBS. No doubt, the subthalamic nucleus (STN) is the most common target. The increased tendency of burst discharges in the STN in PD may by itself serve as a direct cause of parkinsonian locomotor deficits, even in the absence of impaired dopaminergic innervations [8]. The relationship between STN activity and gait is further supported by functional magnetic resonance imaging (fMRI) of PD patients with freezing of gait. These patients have increased STN activation during bilateral finger movements with respect to PD patients having no freezing of gait and healthy controls [9]. When implanted in the STN, DBS may improve, among other things, gait and postural symptoms of PD to an almost normal level [10] (see [11–15] for additional reviews of the impact of DBS on gait and mobility). Among the improved gait parameters are stride length, gait velocity [16, 17], angular leg excursion, reduced double-stance duration and normalization of the leg muscles pattern, and frequency of the center of foot pressure (CoP) as well as increased amplitude of arm and leg swing movements. Stimulation also reduces the spatial foot position asymmetry, stride-to-stride variability, and inter-limb coordination. The effects on locomotor activation of lower limb muscles in PD exhibit differential topographical (distal versus proximal) and stride-phase (stance versus swing) consistency and absence of the lateralized actions typically observed for the clinical signs of the disease [18]. However, evidence exists that stimulation improves gait performance during turns but not gait velocity or stride length during forward walking [19]. Treatment by combined STN DBS and levodopa improved gait speed, reduced gait variability (enhanced stability), and yielded lower Unified Parkinson's Disease Rating Scale (UPDRS) scores [4]. UPDRS scores improved with DBS alone, but not gait variability. The effect of STN DBS on posture includes reduction of the forward trunk-bending and significant decrease of the inclination of the shank, which induces a reduction of the mechanical moment at the hip and ankle. Bilateral STN DBS improved gait and postural stability, as measured using UPDRS III scores, by about 50 % [20].

Mixed evidence exists in the literature for the effect of STN DBS on freezing of gait, an episodic gait disturbance. Whereas for some patients STN DBS failed to reduce the freezing [16], for others it reduced the occurrence and severity of the phenomenon at 6 months postsurgery with largely sustained effects at 12 months follow-up [21–23]. However, the DBS-mediated effects are modest on gait impairment and extremely variable depending on the clinical phenotype and individual clinical profile, suggesting that the indication for DBS should become more individually tailored [24, 25].

24.2 Possible Mechanisms

DBS has been developed empirically during thalamotomy operations, when stimulation of the thalamus has been observed to be effective in stopping the extrapyramidal tremor [5]. As such, the mechanisms behind its therapeutic effect are still

unclear today, as are the mechanisms underlying the disorders it targets. Gait as well as other disorders of PD appear to result from pathological output of the basal ganglia which disrupts the activity in thalamocortical and brainstem networks. Accumulating evidence suggests that stimulation of the STN or GPi does not restore normal basal ganglia function, but rather replaces the abnormal basal ganglia output with a more tolerable pattern, which helps to restore the functionality of downstream networks [26]. More specifically, DBS dissociates input and output signals, resulting in the disruption of abnormal information flow through the stimulation site [27]. A number of alternative hypotheses are suppression of abnormal activity, striping basal ganglia output of misinformation, reduction of abnormal stochastic resonance effects due to increased noise in the disease state, and reinforcement of dynamic modulation of neuronal activity by resonance effects [28]. A systems-level approach suggests that DBS could be achieving its therapeutic effects by overriding pathological network activity [29]. It has been proposed that the overall effect of DBS is to inhibit the neural activity in the region stimulated [30, 31]. However, the effect of extracellular stimulation on cellular activity in the stimulated site appears to depend also on the neurotransmitter of the afferent fibers and axons projecting to that site [32, 33].

24.3 Negative Effects

Notwithstanding, STN DBS may also have adverse effects on gait [34]. Six months of DBS in the STN impaired anticipatory postural preparation for step initiation [35]. STN DBS resulted in increased rate of falls, postural instability, and gait disturbances in comparison with the best medical therapy [36]. The number of falls was significantly greater in STN DBS patients also in comparison with GPi DBS patients [37, 38], and so were gait disorders, disequilibrium, falls, and balance disturbances, 4 year postoperatively [39]. In the latter study, these disorders were not found in any of the GPi DBS patients.

Although a number of studies reported that DBS enhances gait (as described above), some found little or no improvement to gait and posture following STN DBS [40]. Despite overall improvements in motor function and reduction of dyskinesia following STN DBS, Kelly et al. [41] found no significant group effects of unilateral or bilateral stimulation on gait and mobility compared to pre-surgical function.

24.4 Deterioration of the Therapeutic Effect Over Time

Over time, patients with DBS often develop levodopa-resistant symptoms including freezing of gait and postural instability [42, 43]. In a meta-analysis of the long-term effects of DBS, St. George et al. [44] showed that DBS initially improved postural instability and gait disturbances (PIGD) compared to the presurgery OFF medicated state, but performance declined over time. Within an average of 9 years postsurgery, subjects would reach presurgery levels again. When ON medication,

DBS improved PIGD to a much better level than before the implantation. In this state, the site of stimulation, STN or GPi, had an important effect on the postoperative decline in PIGD. With STN DBS, PIGD progressively declined and within 2 years became worse than its presurgery level. In contrast, GPi DBS did not cause significant long-term decline in PIGD. Thus, GPi DBS in combination with levodopa seemed to preserve PIGD better than did STN DBS. In contrast with PIGD, DBS improved cardinal signs in both medication states and at both sites for about 5 years.

Following surgery, STN DBS improved postural stability and gait, which deteriorated between the first and fifth years postoperative [45]. However, after 5 years, postural stability and gait were still at a better level than the pre-operative baseline. Notwithstanding, gait was still improved with respect to the pre-operative condition even 8 years after surgery, whereas postural stability worsened at that time [46–48]. These findings suggest that GPi stimulation is more appropriate for PIGD than STN DBS, but regardless of the implantation site, the therapeutic effect is limited and may last several years only. The aforementioned literature raises new questions as to the causes of the deterioration. Is it due to adaptation of the STN, change in STN neuronal properties due to the long-term electrical current it receives, or the treatment is no longer effective as the disease advances further? Does the course of the disease change in advanced stages, rendering electrical stimulation no longer effective? How can DBS be modified to prevent or slow down the deterioration? Additional studies are needed to address these questions.

24.5 Low Frequency Stimulation

Empirically, it has been observed that different parameters of stimulation may improve different symptoms. Recently, Picillo et al. [49, 50] suggested specific algorithms for programming the DBS in order to target gait impairments. An especially important parameter in targeting gait disturbances by STN DBS is the frequency of stimulation. In a comparison between high (130 Hz) and low (60 Hz) frequency stimulation, only the low frequency reduced the number of freezing episodes [51–53]. The improvement persisted after 8 months for 11 patients, but 2 switched back to the high frequency due to deterioration of movements. Ricchi et al. [54] evaluated patients who developed gait impairment several years after surgery by switching their DBS parameters to low frequency stimulation at 80 Hz. This change had an immediate positive effect on gait in STN DBS treated patients, but the improvement was not maintained over time. Of 11 patients, 3 switched back to 130 Hz because of unsatisfactory control of motor symptoms, 3 showed no change, and 5 exhibited global improvement. The reduction in stimulation frequency seems to have produced better results for the non-tremor dominant subpopulation of patients. In 2 patients who developed freezing immediately following the activation of STN DBS at 130 Hz, the reduction of frequency to 60 Hz, with no change in contacts, voltages, or pulse widths,

immediately alleviated the freezing of gait [55]. The effect was maintained in a 10 months follow-up. Thus, recent studies [51, 54–56] suggest that low frequency subthalamic stimulation is beneficial for treatment of gait impairments in a subpopulation of PD patients, especially the non-tremor dominant, and its use should be tailored to the individual patient. The therapeutic effect of STN DBS may be explained in part by the indirect effect its stimulation has on the firing patterns in the PPN [57, 58] (see below).

In contrast to the above studies, a prospective study by Sidiropoulos et al. [22] found no significant improvement in UPDRS scores, and axial and gait subscores in 45 advanced PD patients when switching from high (130–185 Hz) to low (60–80 Hz) stimulation. Voltage was increased at the same time in an attempt to keep the total electrical energy delivered at a comparable level, but not kept constant. The percentage of tremor dominant patients in the population was not reported, which makes it difficult to compare with the previous studies. Another limitation of this prospective study is that patient evaluation was not uniform, with different evaluators at different patient visits.

In another study, 20 postoperative PD patients were randomly assigned to either 60 Hz or 130 Hz stimulation sessions of 1 hour, following which they were evaluated primarily for stride length, with sit-walk-stand test, UPDRS III, and gait parameters as second outcomes [59]. The study found no significant difference between the 60 Hz and 130 Hz stimulations, using the same voltage for both.

Low frequency stimulation appears to fit better PIGD, whereas tremor or rigidity is better improved by high frequency stimulation. It is for future research to investigate the different mechanisms and the different basal ganglia outputs induced by each type of stimulation, and the underlying neuronal mechanisms. If indeed DBS generates more tolerable basal ganglia patterns of activity as mentioned above, it seems that the different frequencies generate different patterns, each more tolerable to a different part of the cortico-basal-ganglia-thalamo-cortical network. Deeper investigation of the parameters of stimulation may allow to enrich our ability to generate patterns of activation of the basal ganglia, to explore which part of the network responds to them, and which symptoms they target. This may lead to both improved DBS and better understanding of the network physiology.

24.6 PPN Stimulation for Gait and Posture Disorders

Anatomically, the pedunculopontine nucleus (PPN) is a part of the mesencephalic locomotor region [60]. Physiologically, it is considered part of the gait control network, and specifically, the control of postural muscle tone and locomotion [61, 62]. Its single neurons were found to be phase-locked to alpha oscillations, and to respond to limb movements and imagined gait by dynamically changing network activity and decreasing alpha phase locking [63]. Different synchronous networks were activated during initial motor planning and actual motion. Electrophysiological studies suggest that “bursting” glutamatergic PPNd (pars dissipatus)

neurons are related to the initiation of programmed movements while non-bursting cholinergic PPNc (pars compacta) neurons are related to the maintenance of steady-state locomotion [64]. See [65, 66] for a review of PPN physiology. Lesions to this area in rats [67] and monkeys [68] resulted in gait and balance disorders. Therefore, the PPN has been suggested as a therapeutic target to improve gait in certain parkinsonian patients [42, 64, 69, 70].

In particular, PPN stimulation has been proposed as a therapy to alleviate freezing of gait [71–76] and improve balance function [77] (see [78] for a short review). When freezing of gait persists despite drugs, it is usually not improved by STN stimulation [79, 80]. Recently, concomitant stimulation of the STN and substantia nigra pars reticulata (SNr) was demonstrated to improve freezing of gait, but not balance impairment or UPDRS scores (Scale II items 13–15, Scale III items 27–31) [81]. However, gait may be improved by low frequency stimulation of the PPN [80, 82–84]. Notwithstanding, the limited studies of PPN DBS in treating gait symptoms have had mixed clinical outcomes, which likely reflect targeting variability and the inherent challenges of targeting a small brainstem structure that is both anatomically and neurochemically heterogeneous [85, 86]. A recent meta-analysis showed significant improvement in postural instability and motor symptoms of PD on and off medications, but failed to show improvement in freezing of gait [87]. A recent comparison of the therapeutic effect of DBS implanted in the STN vs. one implanted in PPN was demonstrated in a patient implanted in both locations [88]. The PPN-DBS provided modest improvements in the gait disorder and freezing episodes, while the STN-DBS failed to improve the dominant problems (rigidity and mild tremors). Nevertheless, the PPN has been examined as a DBS target to alleviate gait disturbances also in progressive supranuclear palsy [89, 90].

24.7 Conclusions and Future Directions

Unlike rigidity or tremor, which is routinely examined during the DBS implantation surgery in search for an optimal implantation site, gait and postural symptoms, for example, freezing of gait, are far more difficult to assess intraoperatively due to the supine posture of the patient. As such, the ability to pinpoint an electrode to an optimal location is more limited. Nevertheless, the aforementioned evidence indicates that gait and postural symptoms may be improved by electrical stimulation of the STN, GPi, or PPN. As the mechanism behind the therapeutic effects achieved by DBS is yet unclear, further studies are necessary in order to:

1. elaborate our understanding of the underlying mechanisms.
2. target specific symptoms related to gait and posture by means of DBS. For example, perhaps, episodic and continuous gait changes will benefit from different stimulation settings.
3. better assess the electrode targeting intraoperatively.
4. explore the overall benefit related to gait and postural control.

5. better understand changes over time in order to enhance long-term efficacy.
6. locate and evaluate novel targets with therapeutic effects.
7. improve the effectiveness of DBS by closed-loop stimulation, i.e., stimulation that will take place only based on gait-related neuronal activity or gait-related external cues.

Some investigators refer to PD as a form of advanced aging. Indeed, many older adults share gait disturbances that are typically seen in PD (e.g., small, shuffling steps, and reduced arm swing). In the future, as DBS technology improves and we learn more about the mechanisms that are common and distinct in aging and PD, it may lead to the possibility of treating gait disturbances associated with aging using some form of DBS. Of course, much work needs to be done before this intriguing possibility can become a reality.

References

1. Bloem BR, Hausdorff JM, Visser JE, Giladi N. Falls and freezing of gait in Parkinson's disease: a review of two interconnected, episodic phenomena. Mov Disord. 2004;19:871–84.
2. Segev-Jacubovski O, Herman T, Yogev-Seligmann G, Mirelman A, Giladi N, Hausdorff JM. The interplay between gait, falls and cognition: can cognitive therapy reduce fall risk? Expert Rev Neurother. 2011;11:1057–75.
3. Montero-Odasso M, Verghese J, Beauchet O, Hausdorff JM. Gait and cognition: a complementary approach to understanding brain function and the risk of falling. J Am Geriatr Soc [Internet]. 2012 [cited 2012 Dec 2]; Available from: http://onlinelibrary.wiley.com/doi/10.1111/j.1532-5415.2012.04209.x/full.
4. Hausdorff JM, Gruendlinger L, Scollins L, O'Herron S, Tarsy D. Deep brain stimulation effects on gait variability in Parkinson's disease. Mov Disord Off J Mov Disord Soc. 2009;24:1688–92.
5. Benabid AL, Pollak P, Louveau A, Henry S, de Rougemont J. Combined (thalamotomy and stimulation) stereotactic surgery of the VIM thalamic nucleus for bilateral Parkinson disease. Appl Neurophysiol. 1987;50:344–6.
6. Giladi N, Horak FB, Hausdorff JM. Classification of gait disturbances: distinguishing between continuous and episodic changes: gait classification scheme. Mov Disord. 2013;28:1469–73.
7. Moro E, Schüpbach M, Wächter T, Allert N, Eleopra R, Honey CR, et al. Referring Parkinson's disease patients for deep brain stimulation: a RAND/UCLA appropriateness study. J Neurol. 2016;263:112–9.
8. Tai C-H, Pan M-K, Lin JJ, Huang C-S, Yang Y-C, Kuo C-C. Subthalamic discharges as a causal determinant of parkinsonian motor deficits. Ann Neurol. 2012;72:464–76.
9. Vercruysse S, Spildooren J, Heremans E, Wenderoth N, Swinnen SP, Vandenberghe W, et al. The neural correlates of upper limb motor blocks in Parkinson's disease and their relation to freezing of gait. Cereb Cortex. 2014;24:3154–66.
10. Limousin P, Pollak P, Benazzouz A, Hoffmann D, Broussolle E, Perret JE, et al. Bilateral subthalamic nucleus stimulation for severe Parkinson's disease. Mov Disord. 1995;10:672–4.
11. Piper M, Abrams GM, Marks Jr WJ. Deep brain stimulation for the treatment of Parkinson's disease: overview and impact on gait and mobility. NeuroRehabilitation. 2005;20:223–32.
12. Mahlknecht P, Limousin P, Foltynie T. Deep brain stimulation for movement disorders: update on recent discoveries and outlook on future developments. J Neurol. 2015;262:2583–95.

13. Collomb-Clerc A, Welter M-L. Effects of deep brain stimulation on balance and gait in patients with Parkinson's disease: a systematic neurophysiological review. Neurophysiol Clin Neurophysiol. 2015;45:371–88.
14. Poortvliet PC, Silburn PA, Coyne TJ, Chenery HJ. Deep brain stimulation for Parkinson disease in Australia: current scientific and clinical status. Intern Med J. 2015;45:134–9.
15. Fasano A, Aquino CC, Krauss JK, Honey CR, Bloem BR. Axial disability and deep brain stimulation in patients with Parkinson disease. Nat Rev Neurol. 2015;11:98–110.
16. Stolze H, Klebe S, Poepping M, Lorenz D, Herzog J, Hamel W, et al. Effects of bilateral subthalamic nucleus stimulation on parkinsonian gait. Neurology. 2001;57:144–6.
17. Chastan N, Westby GWM, Yelnik J, Bardinet E, Do MC, Agid Y, et al. Effects of nigral stimulation on locomotion and postural stability in patients with Parkinson's disease. Brain. 2009;132:172–84.
18. Ferrarin M, Carpinella I, Rabuffetti M, Rizzone M, Lopiano L, Crenna P. Unilateral and bilateral subthalamic nucleus stimulation in Parkinson's disease: effects on EMG signals of lower limb muscles during walking. IEEE Trans Neural Syst Rehabil Eng. 2007;15:182–9.
19. Lohnes CA, Earhart GM. Effect of subthalamic deep brain stimulation on turning kinematics and related saccadic eye movements in Parkinson disease. Exp Neurol. 2012;236:389–94.
20. Karimi M, Golchin N, Tabbal SD, Hershey T, Videen TO, Wu J, et al. Subthalamic nucleus stimulation-induced regional blood flow responses correlate with improvement of motor signs in Parkinson disease. Brain. 2008;131:2710–9.
21. Vercruysse S, Vandenberghe W, Münks L, Nuttin B, Devos H, Nieuwboer A. Effects of deep brain stimulation of the subthalamic nucleus on freezing of gait in Parkinson's disease: a prospective controlled study. J Neurol Neurosurg Psychiatry. 2014;85:871–7.
22. Sidiropoulos C, Walsh R, Meaney C, Poon YY, Fallis M, Moro E. Low-frequency subthalamic nucleus deep brain stimulation for axial symptoms in advanced Parkinson's disease. J Neurol. 2013;260:2306–11.
23. Brosius SN, Gonzalez CL, Shuresh J, Walker HC. Reversible improvement in severe freezing of gait from Parkinson's disease with unilateral interleaved subthalamic brain stimulation. Parkinsonism Relat Disord. 2015;21:1469–70.
24. Galati S, Stefani A. Deep brain stimulation of the subthalamic nucleus: All that glitters isn't gold? Mov Disord Off J Mov Disord Soc. 2015;30:632–7.
25. Katz M, Luciano MS, Carlson K, Luo P, Marks WJ, Larson PS, et al. Differential effects of deep brain stimulation target on motor subtypes in Parkinson's disease. Ann Neurol. 2015;77:710–9.
26. Wichmann T, DeLong MR. Deep brain stimulation for movement disorders of basal ganglia origin: restoring function or functionality? Neurotherapeutics. 2016;13(2):264–83.
27. Chiken S, Nambu A. Mechanism of deep brain stimulation inhibition, excitation, or disruption? Neuroscientist. 2015;1073858415581986.
28. Montgomery Jr EB, Gale JT. Mechanisms of action of deep brain stimulation (DBS). Neurosci Biobehav Rev. 2008;32:388–407.
29. McIntyre CC, Hahn PJ. Network perspectives on the mechanisms of deep brain stimulation. Neurobiol Dis. 2010;38:329–37.
30. Dostrovsky JO, Lozano AM. Mechanisms of deep brain stimulation. Mov Disord. 2002;17:S63–8.
31. Benabid AL, Benazzous A, Pollak P. Mechanisms of deep brain stimulation. Mov Disord. 2002;17:S73–4.
32. Vitek JL. Mechanisms of deep brain stimulation: excitation or inhibition. Mov Disord Off J Mov Disord Soc. 2002;17 Suppl 3:S69–72.
33. Johnson MD, Miocinovic S, McIntyre CC, Vitek JL. Mechanisms and targets of deep brain stimulation in movement disorders. Neurotherapeutics. 2008;5:294–308.
34. Baizabal-Carvallo JF, Jankovic J. Movement disorders induced by deep brain stimulation. Parkinsonism Relat Disord. 2016;25:1–9.
35. Rocchi L, Carlson-Kuhta P, Chiari L, Burchiel KJ, Hogarth P, Horak FB. Effects of deep brain stimulation in the subthalamic nucleus or globus pallidus internus on step initiation in Parkinson disease. J Neurosurg. 2012;1–9.

36. Weaver FM, Follett K, Stern M, et al. Bilateral deep brain stimulation vs best medical therapy for patients with advanced Parkinson disease: a randomized controlled trial. JAMA. 2009;301:63–73.
37. Follett KA, Weaver FM, Stern M, Hur K, Harris CL, Luo P, et al. Pallidal versus subthalamic deep-brain stimulation for Parkinson's disease. N Engl J Med. 2010;362:2077–91.
38. Follett KA, Torres-Russotto D. Deep brain stimulation of globus pallidus interna, subthalamic nucleus, and pedunculopontine nucleus for Parkinson's disease: which target? Parkinsonism Relat Disord. 2012;18(Supp 1):S165–7.
39. Hariz MI, Rehncrona S, Quinn NP, Speelman JD, Wensing C, Multicentre Advanced Parkinson's Disease Deep Brain Stimulation Group. Multicenter study on deep brain stimulation in Parkinson's disease: an independent assessment of reported adverse events at 4 years. Mov Disord. 2008;23:416–21.
40. Kim SD, Allen NE, Canning CG, Fung VSC. Postural instability in patients with Parkinson's disease. CNS Drugs. 2013;27:97–112.
41. Kelly VE, Israel SM, Samii A, Slimp JC, Goodkin R, Shumway-Cook A. Assessing the effects of subthalamic nucleus stimulation on gait and mobility in people with Parkinson disease. Disabil Rehabil. 2010;32:929–36.
42. Bronstein JM, Tagliati M, Alterman RL, Lozano AM, Volkmann J, Stefani A, et al. Deep brain stimulation for Parkinson disease: an expert consensus and review of key issues. Arch Neurol [Internet]. 2011 [cited 2016 Mar 15]; 68. Available from: http://archneur.jamanetwork.com/article.aspx?doi=10.1001/archneurol.2010.260.
43. Merola A, Zibetti M, Angrisano S, Rizzi L, Ricchi V, Artusi CA, et al. Parkinson's disease progression at 30 years: a study of subthalamic deep brain-stimulated patients. Brain. 2011;134:2074–84.
44. St. George RJ, Nutt JG, Burchiel KJ, Horak FB. A meta-regression of the long-term effects of deep brain stimulation on balance and gait in PD. Neurology. 2010;75:1292–9.
45. Krack P, Batir A, Van Blercom N, Chabardes S, Fraix V, Ardouin C, et al. Five-year follow-up of bilateral stimulation of the subthalamic nucleus in advanced Parkinson's disease. N Engl J Med. 2003;349:1925–34.
46. Fasano A, Romito LM, Daniele A, Piano C, Zinno M, Bentivoglio AR, et al. Motor and cognitive outcome in patients with Parkinson's disease 8 years after subthalamic implants. Brain. 2010;133:2664–76.
47. Albanese A, Romito L. Deep brain stimulation for Parkinson's disease: where do we stand? Front Neurol [Internet]. 2011 [cited 2016 Mar 15]; 2. Available from: http://journal.frontiersin.org/article/10.3389/fneur.2011.00033/abstract
48. Li J, Zhang Y, Li Y. Long-term follow-up of bilateral subthalamic nucleus stimulation in Chinese Parkinson's disease patients. Br J Neurosurg. 2015;29:329–33.
49. Picillo M, Lozano AM, Kou N, Puppi Munhoz R, Fasano A. programming deep brain stimulation for Parkinson's disease: the Toronto western hospital algorithms. Brain Stimulat [Internet]. 2016 [cited 2016 Mar 15]; In press. Available from: http://www.sciencedirect.com/science/article/pii/S1935861X16300183.
50. Picillo M, Lozano AM, Kou N, Munhoz RP, Fasano A. Programming deep brain stimulation for tremor and dystonia: the Toronto Western Hospital algorithms. Brain Stimulat. 2016.
51. Moreau C, Defebvre L, Destee A, Bleuse S, Clement F, Blatt JL, et al. STN-DBS frequency effects on freezing of gait in advanced Parkinson disease. Neurology. 2008;71:80–4.
52. Jia F, Guo Y, Wan S, Chen H, Hao H, Zhang J, et al. Variable frequency stimulation of subthalamic nucleus for freezing of gait in Parkinson's disease. Parkinsonism Relat Disord. 2015;21:1471–2.
53. Xie T, Vigil J, MacCracken E, Gasparaitis A, Young J, Kang W, et al. Low-frequency stimulation of STN-DBS reduces aspiration and freezing of gait in patients with PD. Neurology. 2015;84:415–20.
54. Ricchi V, Zibetti M, Angrisano S, Merola A, Arduino N, Artusi CA, et al. Transient effects of 80 Hz stimulation on gait in STN DBS treated PD patients: a 15 months follow-up study. Brain Stimulat. 2012;5:388–92.

55. Xie T, Kang UJ, Warnke P. Effect of stimulation frequency on immediate freezing of gait in newly activated STN DBS in Parkinson's disease. J Neurol Neurosurg Psychiatry. 2012;83:1015–7.
56. Ramdhani RA, Patel A, Swope D, Kopell BH. Early use of 60 Hz frequency subthalamic stimulation in Parkinson's disease: a case series and review. Neuromodulation J Int Neuromodulation Soc. 2015;18:664–9.
57. Sitti I, Acar G, Zisakis AK, Özdemir M, Acar F, Burchiel KJ. Effect of subthalamic nucleus stimulation on pedunculopontine nucleus neural activity. Stereotact Funct Neurosurg. 2016;94:54–9.
58. Weiss PH, Herzog J, Pötter-Nerger M, Falk D, Herzog H, Deuschl G, et al. Subthalamic nucleus stimulation improves Parkinsonian gait via brainstem locomotor centers. Mov Disord Off J Mov Disord Soc. 2015;30:1121–5.
59. Phibbs FT, Arbogast PG, Davis TL. 60-Hz frequency effect on gait in Parkinson's disease with subthalamic nucleus deep brain stimulation. Neuromodulation Technol Neural Interface. 2014;17:717–20.
60. Windels F, Thevathasan W, Silburn P, Sah P. Where and what is the PPN and what is its role in locomotion? Brain J Neurol. 2015;138:1133–4.
61. Takakusaki K, Oohinata-Sugimoto J, Saitoh K, Habaguchi T. Role of basal ganglia–brainstem systems in the control of postural muscle tone and locomotion. In: Research B-P in B, editor. Elsevier; 2004 [cited 2016 Mar 20]. p. 231–7. Available from: http://www.sciencedirect.com/science/article/pii/S0079612303430239.
62. Takakusaki K, Chiba R, Nozu T, Okumura T. Brainstem control of locomotion and muscle tone with special reference to the role of the mesopontine tegmentum and medullary reticulospinal systems. J Neural Transm. 2015;1–35.
63. Tattersall TL, Stratton PG, Coyne TJ, Cook R, Silberstein P, Silburn PA, et al. Imagined gait modulates neuronal network dynamics in the human pedunculopontine nucleus. Nat Neurosci [Internet]. 2014 [cited 2014 Feb 25]; advance online publication. Available from: http://www.nature.com/neuro/journal/vaop/ncurrent/full/nn.3642.html.
64. Pahapill PA, Lozano AM. The pedunculopontine nucleus and Parkinson's disease. Brain. 2000;123:1767–83.
65. Garcia-Rill E, Luster B, D'Onofrio S, Mahaffey S, Bisagno V, Urbano FJ. Pedunculopontine arousal system physiology - deep brain stimulation (DBS). Sleep Sci São Paulo Braz. 2015;8:153–61.
66. Garcia-Rill E, Luster B, D'Onofrio S, Mahaffey S, Bisagno V, Urbano FJ. Implications of gamma band activity in the pedunculopontine nucleus. J Neural Transm Vienna Austria. 1996;2015.
67. Jimenez-Martin J, Blanco-Lezcano L, González-Fraguela ME, Díaz-Hung M-L, Serrano-Sánchez T, Almenares JL, et al. Effect of neurotoxic lesion of pedunculopontine nucleus in nigral and striatal redox balance and motor performance in rats. Neuroscience. 2015;289:300–14.
68. Grabli D, Karachi C, Folgoas E, Monfort M, Tande D, Clark S, et al. Gait disorders in parkinsonian monkeys with pedunculopontine nucleus lesions: a tale of two systems. J Neurosci. 2013;33:11986–93.
69. Pierantozzi M, Palmieri MG, Galati S, Stanzione P, Peppe A, Tropepi D, et al. Pedunculopontine nucleus deep brain stimulation changes spinal cord excitability in Parkinson's disease patients. J Neural Transm. 2008;115:731–5.
70. Martinez-Ramirez D, Hu W, Bona AR, Okun MS, Shukla AW. Update on deep brain stimulation in Parkinson's disease. Transl Neurodegener [Internet]. 2015 [cited 2016 Mar 21]; 4. Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4529685/.
71. Moreau C, Defebvre L, Devos D, Marchetti F, Destée A, Stefani A, et al. STN versus PPN-DBS for alleviating freezing of gait: toward a frequency modulation approach? Mov Disord. 2009;24:2164–6.

72. Ostrem JL, Christine CW, Glass GA, Schrock LE, Starr PA. Pedunculopontine nucleus deep brain stimulation in a patient with primary progressive freezing gait disorder. Stereotact Funct Neurosurg. 2010;88:51–5.
73. Wilcox RA, Cole MH, Wong D, Coyne T, Silburn P, Kerr G. Pedunculopontine nucleus deep brain stimulation produces sustained improvement in primary progressive freezing of gait. J Neurol Neurosurg Psychiatry. 2011;82:1256–9.
74. Acar F, Acar G, Bir LS, Gedik B, Oğuzhanoğlu A. Deep brain stimulation of the pedunculopontine nucleus in a patient with freezing of gait. Stereotact Funct Neurosurg. 2011;89:214–9.
75. Nonnekes J, Snijders AH, Nutt JG, Deuschl G, Giladi N, Bloem BR. Freezing of gait: a practical approach to management. Lancet Neurol. 2015;14:768–78.
76. Wen P, Li M, Xiao H, Ding R, Chen H, Chang J, et al. Low-frequency stimulation of the pedunculopontine nucleus affects gait and the neurotransmitter level in the ventrolateral thalamic nucleus in 6-OHDA Parkinsonian rats. Neurosci Lett. 2015;600:62–8.
77. Yousif N, Bhatt H, Bain PG, Nandi D, Seemungal BM. The effect of pedunculopontine nucleus deep brain stimulation on postural sway and vestibular perception. Eur J Neurol. 2016;23:668–70.
78. Fytagoridis A, Silburn PA, Coyne TJ, Thevathasan W. Understanding the human pedunculopontine nucleus in Parkinson's disease. J Neural Transm Vienna Austria. 1996;2016.
79. Faist M, Xie J, Kurz D, Berger W, Maurer C, Pollak P, et al. Effect of bilateral subthalamic nucleus stimulation on gait in Parkinson's disease. Brain. 2001;124:1590–600.
80. Benabid AL, Chabardes S, Mitrofanis J, Pollak P. Deep brain stimulation of the subthalamic nucleus for the treatment of Parkinson's disease. Lancet Neurol. 2009;8:67–81.
81. Weiss D, Walach M, Meisner C, Fritz M, Scholten M, Breit S, et al. Nigral stimulation for resistant axial motor impairment in Parkinson's disease? A randomized controlled trial. Brain. 2013;136:2098–108.
82. Mazzone P, Lozano A, Stanzione P, Galati S, Scarnati E, Peppe A, et al. Implantation of human pedunculopontine nucleus: a safe and clinically relevant target in Parkinson's disease. Neuroreport. 2005;16:1877–81.
83. Stefani A, Lozano AM, Peppe A, Stanzione P, Galati S, Tropepi D, et al. Bilateral deep brain stimulation of the pedunculopontine and subthalamic nuclei in severe Parkinson's disease. Brain. 2007;130:1596–607.
84. Plaha P, Gill SS. Bilateral deep brain stimulation of the pedunculopontine nucleus for Parkinson's disease. Neuroreport. 2005;16:1883–7.
85. Morita H, Hass CJ, Moro E, Sudhyadhom A, Kumar R, Okun MS. Pedunculopontine nucleus stimulation: where are we now and what needs to be done to move the field forward? Front Neurol [Internet]. 2014 [cited 2016 Mar 20]; 5. Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4255598/.
86. Welter M-L, Demain A, Ewenczyk C, Czernecki V, Lau B, El Helou A, et al. PPNa-DBS for gait and balance disorders in Parkinson's disease: a double-blind, randomised study. J Neurol. 2015;262:1515–25.
87. Golestanirad L, Elahi B, Graham SJ, Das S, Wald LL. Efficacy and safety of pedunculopontine nuclei (PPN) deep brain stimulation in the treatment of gait disorders: a meta-analysis of clinical studies. Can J Neurol Sci J Can Sci Neurol. 2016;43:120–6.
88. Liu H-G, Zhang K, Yang A-C, Zhang J-G. Deep brain stimulation of the subthalamic and pedunculopontine nucleus in a patient with Parkinson's disease. J Korean Neurosurg Soc. 2015;57:303–6.
89. Doshi PK, Desai JD, Karkera B, Wadia PM. Bilateral pedunculopontine nucleus stimulation for progressive supranuclear palsy. Stereotact Funct Neurosurg. 2015;93:59–65.
90. Kondziolka D. Comment on the paper by Doshi et al. Entitled "bilateral pedunculopontine nucleus stimulation for progressive supranuclear palsy". Stereotact Funct Neurosurg. 2015;93:66.

Using Virtual Reality to Advance the Understanding and Rehabilitation of Gait Impairments in Parkinson's Disease

25

Kaylena A. Ehgoetz Martens, James M. Shine, and Simon J.G. Lewis

Abstract

Virtual reality (VR) provides a unique platform to study the complex interactions between an individual's movement and their environment. Although this innovative technology has not yet been widely used in Parkinson's disease (PD) research, it has tremendous potential to advance both our understanding and treatment of gait impairments. In this chapter, we will first outline the variety of virtual reality systems available and contrast the associated advantages and disadvantages that warrant consideration for using virtual reality in experimental and/or therapeutic settings. We will then discuss the utility of VR in the scientific exploration of mechanisms that underlie gait impairments in PD. Finally, we will examine the effectiveness of using VR in a therapeutic setting based on the current research, and provide future directions for therapeutic interventions for gait in PD utilizing virtual reality.

Keywords

Virtual reality • Parkinson's disease • Gait • Sensorimotor • Cognition • Freezing of gait • Rehabilitation

25.1 Assessing the Utility of Virtual Reality

Virtual reality can be defined as a high quality computer interface which enables a person to interact with a virtual environment generated by a computer in real-time [1]. Similar to the real world, where we explore our environment with our senses, a virtual environment (VE) offers many comparable perceptual experiences, where

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our senses are able to gather information using vision, auditory, and somatosensory feedback. This information from the virtual environment, in combination with natural sensory (e.g., proprioception) inputs, is then used to guide movement within the virtual environment, and in most instances creates an immersive and realistic virtual experience for the user [1, 2]. Due to the compelling reality that an interactive and immersive virtual experience can elicit, this tool allows investigators to move beyond traditional laboratory experiments which focus on a specific component of behavior (in an artificial setting), and begins to examine the complexity of the brain and resultant behavior in a way that it is much closer to its actual form.

The key elements that contribute to an immersive virtual experience are: (1) delivering a 3-dimensional high-resolution image which incorporates stereoscopic display, and (2) integrating the user's movement (using a movement tracking system) to display relevant sensory feedback about movement in real-time. Without these key elements, a less immersive experience is achieved that is more akin to looking through a window and observing a scene rather than being present and involved [1]. Therefore, the quality of the virtual reality system utilized will have an impact on the immersive presence that can be elicited (see Table 25.1, for examples of different virtual reality systems).

For example, a head-mounted display that allows stereoscopic viewing paired with motion tracking that simulates movement in the VE will provide a much more immersive sensory experience for the user compared to a 2-D computer display monitor with a mouse/keyboard to navigate a virtual environment. Nonetheless both systems (and everything in between) have advantages and disadvantages that should be weighed and considered. While the latter is convenient, easy to use/setup, relatively cheap, less likely to produce cybersickness (a form of motion-sickness that can occur as a result of VR), without a motion tracking system it has limited utility for the study of gait since it lacks the most relevant feedback (i.e., vision, proprioception, vestibular). Recent research has advanced this more basic virtual reality setup by constructing toe tapping pedals that were paired with a desktop monitor to facilitate the study of "gait" in virtual reality [3–5]. Although pressing pedals does not simulate the complexities of walking, it provided an opportunity to examine "gait" in virtual reality while simultaneously capturing functional magnetic resonance imaging or recording electroencephalography in order to study the neural underpinnings of severe gait impairments in PD. Interacting with a projection on the floor, such as walking to step over virtual obstacles or transverse

Table 25.1 Range of virtual reality setups that vary in immersive presence

VR system	Stereoscopic vision	Motion tracking	Immersion
Head-mounted display	✓	✓	High
Projections into real environment	×	✓	Moderate
Wall projections + treadmill walking	×	✓	Moderate
Computer with keyboard or joystick	×	×	Low

lines, is another relatively cheap, convenient, and easy to use format of virtual reality, although might be argued to be less immersive since it remains heavily rooted in the real-world rather than being surrounded and moving through a virtual environment. Finally, a wireless head-mounted display (HMD) with motion tracking (e.g., vicom, optotrak, wearable accelerometers) is arguably the most immersive virtual experience one can achieve. It allows stereoscopic 3-dimensional viewing achieved with small monitors in front of each eye. Additionally, the user's viewpoint can be synchronized to their head movement so that the user can explore the entire environment by "looking" and moving around. When paired with full body kinematics, avatars can be used to represent the person's body position and movements in real-time providing even more detailed feedback about self-motion. Thus, the HMD virtual reality system can provide rich sensory feedback making the user's virtual experiences strikingly immersive. However, this system is also more likely to result in greater cybersickness due its intensity [6, 7], and is problematic for individuals with PD that have dyskinesia or severe head tremor since this excessive movement would disrupt one's ability to view the virtual environment and cause even greater cybersickness.

In addition to the features and limitations inherent to the virtual reality system, there are also many strengths from both an experimental and therapeutic perspective that makes virtual reality a sought after technology to investigate and rehabilitate gait in PD. Experimentally, virtual reality brings ease in creating controlled and safe, yet complex and dynamic environments and also extends the capabilities that the real world limits. For example, it is possible to:

- Create busy sidewalks for individuals to navigate through (which might normally be very difficult to capture rich kinematic data)
- Create stressful situations in order to observe how/whether gait breaks down (which would normally be potentially dangerous to perform in the real-world)
- Provoke gait impairments which are typically difficult to elicit in experimental settings (such as freezing of gait)
- Quantify information processing of stimuli and other cognitive processes, while walking in ecologically valid virtual environments which resemble tasks of daily living
- Isolate, manipulate and even reweight sensory systems, or provide faulty sensory feedback (which can be insightful in trying to further understand visual control or sensorimotor integration necessary for adequate gait control).

Overall, interactions in virtual reality maximize gait and cognition simultaneously (e.g., requiring planning, attention, information processing of stimuli in the virtual environment, sensory integration) [1, 2, 8–10] (see Fig. 25.1), which is why this novel and innovative technology has the potential to advance the understanding of gait impairments.

Therapeutically, virtual reality offers many additional perks. Beyond being safe and affordable, training in virtual reality has been argued to provide similar if not

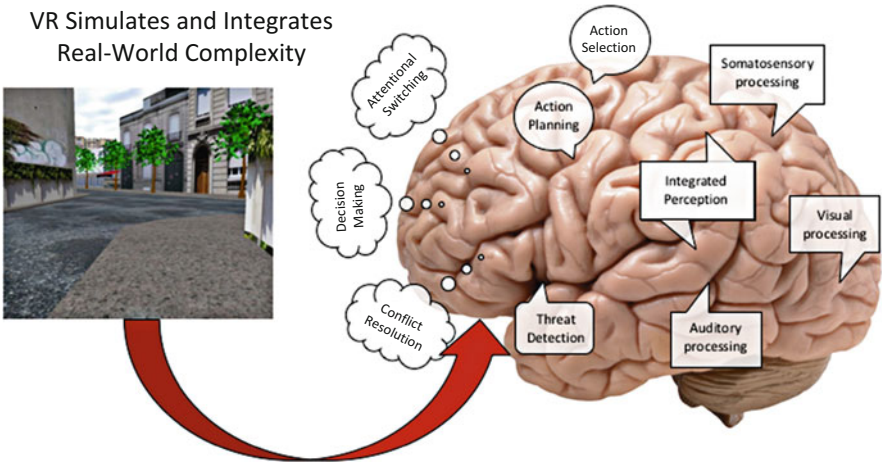

Fig. 25.1 Virtual reality has the capacity to simulate and integrate real-world complexity in both cognitive and motor domains

greater benefits when contrasted with real-world equivalent training programs [1, 2, 8, 11–14]. These greater benefits have been suggested to result from enhanced motor learning through the use of augmented feedback, knowledge of performance, and knowledge of results which virtual reality can easily capitalize and exploit. Some additional reasons virtual reality may be superior are that it often eliminates distracting elements that are difficult to escape in rehab settings, it can be delivered in a personalized manner—adapted for those who need it, and it could even be delivered within the home by using gaming consoles. Therefore, virtual reality may also be a valuable rehabilitative tool for gait interventions in PD.

25.2 Using Virtual Reality to Understand Underlying Mechanisms of Gait Impairments in Parkinson's Disease

Although limited research has utilized virtual reality to study gait in PD, there has been three main arms of scientific investigation that have benefitted from this innovative technology which has contributed to the advancement in understanding underlying mechanisms of gait impairments in Parkinson's disease. The first has focused on understanding the role of sensory feedback and examining deficits in sensorimotor integration which might contribute to gait impairments in PD. The second has examined how gait deficits are exacerbated in complex situations which require additional cognitive abilities (such as planning, attention, and task-switching) or interpretation of environmental demands (such as obstacles or threatening situations). The final contribution has arguably had the most dramatic impact on the understanding of a severe gait phenomenon, known as freezing of gait (FOG), commonly experienced in PD (and thus received a substantial amount of

attention in this chapter). Although the underlying cause and mechanism of freezing of gait remains unknown, virtual reality paired with brain imaging techniques such as fMRI and EEG has allowed research to make gigantic leaps in understanding FOG and developing devices to predict or prevent FOG episodes altogether.

25.2.1 Visual Control and Sensorimotor Integration During Gait

It has been well-established that individuals with PD are highly dependent on vision, particularly dynamic visual information (i.e., optic flow) during locomotion [15–19]. This excessive dependence on visual feedback during gait in PD has been suggested to reflect abnormal sensorimotor integration as a result of the disease [20–23]. Evidence of this has been provided by early studies which examined PD patients walking on a treadmill at a trained pace while manipulating the optic flow speed using virtual reality [24, 25]. In healthy older adults it is expected that increases in optic flow will lead to increases in gait speed, but eventually individuals demonstrate an adaptive shift in dependence from visual to proprioceptive feedback due to the "false" information delivered through VR [24]. Individuals with PD, however, demonstrate an exaggerated response in relation to changes in optic flow, which provides additional support that PD has increased susceptibility to depend on visual information to modulate gait [18]. There was also little evidence that PD patients actually reweighted visual and proprioceptive feedback like healthy older adults did. Instead, many PD did not even notice the incongruity, leading the authors to conclude that a discrepancy between the two kinesthetic signals (optic flow and proprioception) was likely already present. Corroborating these findings, another virtual reality study (utilizing HMD with kinematic movement tracking) manipulated optic flow speed in effort to further understand whether sensory-perceptual information contributed to gait impairments [26]. Previous research has noted that individuals with PD show distance estimation deficits only when required to walk to a target (compared to pointing or being passively moved with VR or in a wheelchair) [27, 28]. A follow-up study was done to disentangle whether delayed perceptual processing versus corrupt sensory integration was responsible for impairments in self-motion perception during gait. Similar to Schubert and colleagues, optic flow speed was increased and decreased relative to the participants' gait speed; however, PD participants did not notice the discrepancy between the various forms of sensory feedback [26]. More interestingly, the study found that decoupling sensory feedback made older adults perform similarly to PD, in fact speeding up the optic flow speed made older adults display similar judgment accuracy and variability as PD at baseline (when optic flow matched gait speed). However, decoupling did not significantly influence PD participants' estimations [26]. Taken together, these research studies (amongst others) suggest that proprioceptive deficits along with delayed sensory processing result in a mismatch of competing sensory information which not only disrupts normal sensorimotor integration in PD, but the erroneous signal (proprioception) eventually

becomes suppressed in favor of the more appropriate information (vision), and gait suffers and is more vulnerable [24, 26, 27].

Other researchers have also used virtual reality to exploit visuoperceptual deficits such as impaired perception of optic flow [17, 29]. It has been argued that asymmetrical dopamine levels between the brain hemispheres might lead to the compression of one visual field more than the other (depending on side of asymmetry) [29]. As a result of this asymmetrical compression of the visual field, individuals with PD can misperceive optic flow since it appears that one hemi-field is moving faster than the other, and thus misrepresenting the speed of movement and heading [17, 29, 30]. Nonetheless PD participants are still highly dependent on optic flow to control gait, such that when the richness in optic flow information was reduced (Fig. 25.2), gait in PD was also shown to worsen (i.e., slower and smaller

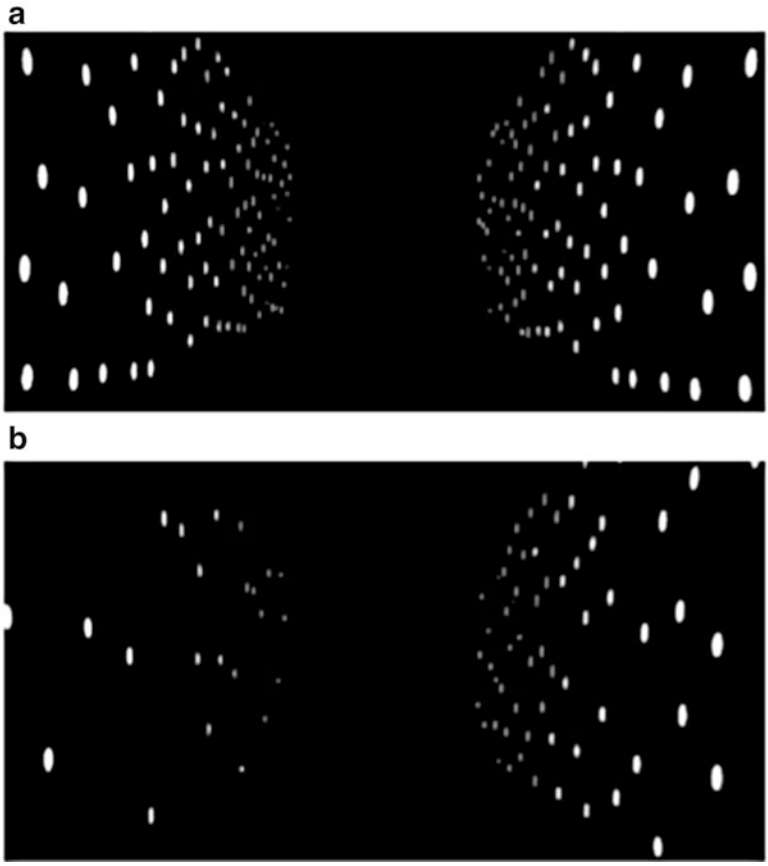

Fig. 25.2 A virtual hallway where optic flow speed was manipulated separately on the right wall from the left wall, but dot density was equal (**a**); whereas (**b**) shows a manipulation of optic flow richness, where the dot density is significantly less on the left wall compared to the right. (Adopted from: [29])

steps) [29]. Considering that even the *visual* form of kinesthesia (optic flow) may be disrupted in PD, and knowing how heavily individuals with PD rely on vision during gait to compensate for proprioceptive deficits, it is not surprising that these sensory-perceptual impairments could play a primary role in the breakdown of gait and balance in PD. Furthermore, accurate sensory feedback is essential for gait planning, selecting the correct motor program, making online gait adjustments, and correctly distributing attention. Taken together, gait impairments may become only more problematic when faced with navigating complex and threatening environments.

25.2.2 Gait in Complex Environments

One of the key features of virtual reality is that it provides an ecologically valid platform for cognition and movement to be assessed and further understand how they interact in PD. Up until recently, cognitive functions were primarily assessed with pen and pencil type assessments which not only lacked the complexity of the everyday world and thus lacked ecologic validity [31, 32], but they are also limited in the generalizability and meaningfulness of their findings to reflect the capabilities in tasks of daily living [33]. For this reason, researchers have utilized virtual reality to create "everyday" environments in order to assess daily tasks that include different cognitive functions [32–34]. For example, one study constructed a virtual supermarket where participants were given a grocery list of items that needed to be bought (Fig. 25.3) [34]. This type of task focused on evaluating action planning, and showed that individuals with PD needed more time to execute this task

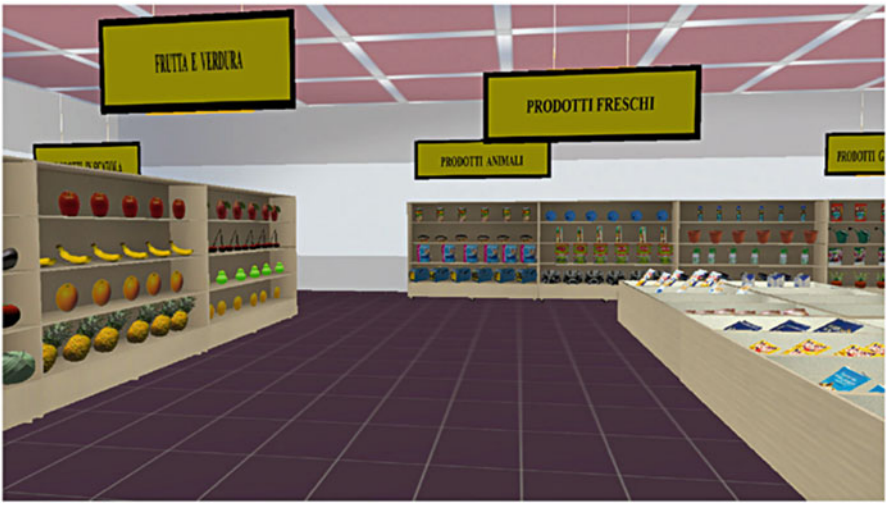

Fig. 25.3 An example of the virtual environment used in the virtual multiple errands test. (Adopted from: [33])

(even though they moved at a constant speed using a joystick), covered a longer distance, and made more hesitations and stops. The authors concluded that this behavior demonstrated that individuals with PD had slower action planning, and were often inefficient at using contextual elements in the environment to aid the search for products. A similar paradigm was also employed where participants were asked to "walk" around an apartment (viewing the VE in a helmet and moving using a joystick) remembering objects and rooms that they had viewed in order to estimate cognition [32]. Interestingly, virtual narrow spaces were found to exaggerate reduced speeds in early non-freezing PD participants (even though they were not actually walking), something commonly seen in the real-world in PD and known to provoke FOG. Although this research has great potential in improving the assessment of cognition in a more ecological manner, more research studies are needed to know whether this will be useful, reliable, and clinically meaningful. Once this is established it would be useful to use these cognitive tasks to assess and quantify changes in gait in order to understand gait impairments outside of the laboratory and potentially prevent falls that are common in the home.

Gait in PD has been shown to deteriorate in more complex situations which encompass a threat or secondary tasks, for example. The current theory as to why individuals with PD are so susceptible to interference in these more complex environments goes back to how PD consciously monitor and control gait with vision as a compensatory strategy to deal with the loss of useful proprioceptive feedback which is necessary for gait to be automatic. Because PD patients constantly having to pay attention to their walking, it has been proposed that this limits their attentional capacity to do other tasks and monitor other relevant information in the environment. By using virtual reality to create a carefully controlled complex environment, one can evaluate aspects of gait that are susceptible to interference and make recommendations for therapeutic interventions.

Stressful situations are commonly reported to elicit gait problems, falls, and freezing of gait in PD. In a series of studies, virtual reality was used to create a stressful and "virtually dangerous" environment to understand if and how threat/anxiety influenced gait in PD and whether dopaminergic replacement therapy mediated this relationship [35–38]. These studies were performed using an HMD integrated with full body kinematics. Participants were asked to walk in virtual environments with and without threat (i.e., across an elevated plank versus a plank located on the ground) (Fig. 25.4). Across all studies, all participants (PD and healthy older adults) had greater levels of anxiety (self-reported and skin conductance levels) when they walked across the elevated plank compared to the plank on the ground. All participants also walked with a slower velocity, smaller steps, and greater step-to-step variability across the elevated compared to the ground plank. These findings were similar to what has been found when older adults were asked to walk across an elevated plank in the real-world, confirming that the experimental manipulation of threat with VR was effective, immersive, and realistic enough to elicit more cautious gait. The most interesting results of this work found that the elevated plank provoked a greater number of freezing of gait

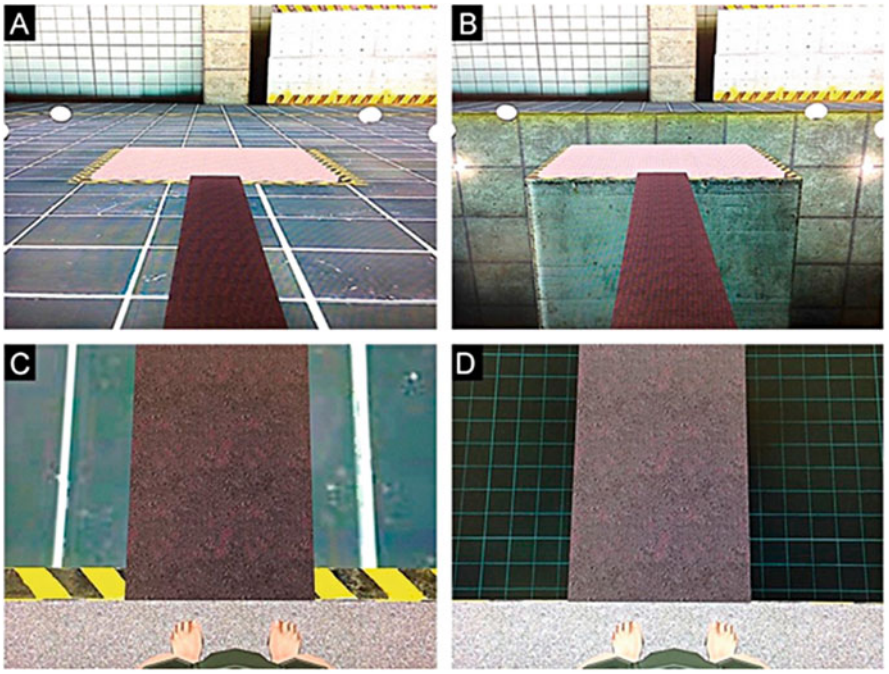

Fig. 25.4 Virtual environments that have been used to induce anxiety by asking participants to walk across a plank that is located on the ground (**a**) compared to a plank located above a deep pit (**b**). Virtual visual feedback about the lower limbs was also manipulated (**c**, **d**) and synchronized using a full kinematic motion tracking system. (Adopted from: [41])

episodes in PD freezers and significantly more variable gait specifically in freezers compared to non-freezers [38] and in those with PD who had high trait anxiety (non-freezers) compared to those with PD who had low trait anxiety (non-freezers) and healthy older adults [35, 36]. Highly trait anxious PD also appeared to be less able to use visual feedback about their lower limbs when it was provided to improve gait, especially when walking across the elevated plank [35]. Notably, the frequency of FOG in freezers and step-to-step variability (among other gait parameters) in highly trait anxious PD were improved with dopaminergic replacement therapy [35, 36, 38]. Furthermore, dopaminergic medication also improved step time variability in highly trait anxious PD when visual feedback about their lower limbs was available. Taken together, this research provided strong evidence to suggest that anxiety influenced PD gait, possibly by demanding shared processing resources at the level of the basal ganglia, which may interfere with other processes (such as processing sensory feedback) necessary to control gait [39]. Researchers postulated that dopaminergic replacement therapy might have improved information processing within the basal ganglia and thus alleviated some of the interference due to the competition for shared resources [40]. In conclusion, this VR approach allowed investigators to evaluate the impact anxiety has on gait in

PD, and offer a mechanistic explanation for how anxiety exacerbates gait impairments in PD. Overall, walking in threatening environments likely demands greater voluntary control and as result also demands more attentional resources (similar to being distracted by a dual-task while walking), however, given the limited resources available in individuals with PD, anxiety and threatening environments may overload the system and manifest gait deficits.

25.2.3 Using VR to Uncover Neural Correlates of Freezing of Gait

Other studies have used a VE that requires participants to navigate a virtual corridor using binary response buttons while performing a modified stop-signal task to elicit a behavioral surrogate of FOG that occurs in either the hands [42] or feet [4, 5]. Although the task does not require balance, postural adjustments, or utilization of the muscles of the upper legs, by manipulating cognitive and perceptual load (through the administration of a complex stop-signal task and through the navigation of embedded narrow doorways, respectively), the task (displayed in Fig. 25.5) has been shown to elicit many of the known clinical characteristics of FOG,

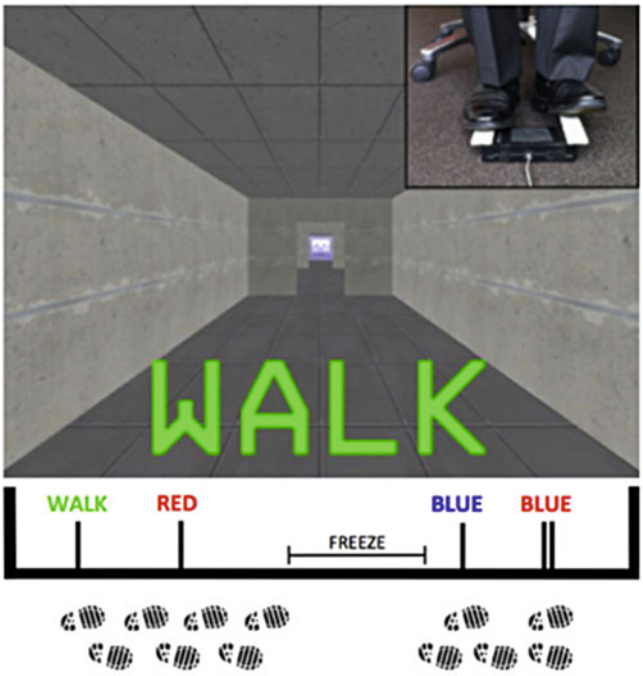

Fig. 25.5 An example of the virtual environment used during fMRI. The configuration of the foot pedals is shown in the *top right corner inset* which enabled a simulation of virtual "gait." The *bottom image* depicts the varying complexity of the task/cues and an example of a patient's footstep pattern as these cues are presented. (Adopted from: [5])

including paroxysmal cessations in ankle flexion [5], heightened step-to-step variability [3], and worsening of motor output in the presence of cognitive [43] and perceptual load [44].

Importantly, the gait-based VE has been successfully combined with functional neuroimaging to improve our understanding of the neural mechanisms underlying the manifestation of freezing behavior. In an early fMRI study, Shine and colleagues contrasted paroxysmal freezing episodes with epochs of normal "walking," and found that episodes of freezing were related to an increased BOLD response in prefrontal cortical regions, with a decreased BOLD response in motor cortex and bilateral striatum [45]. These results suggested the hypothesis that freezing of gait is related to impaired functional connectivity between the striatum and frontoparietal cortex, a prediction that was later confirmed using the same VE task [46] in combination with a novel method for estimating fluctuations in coupling strength between neural regions over time [47]. Subsequently, the same VE paradigm was used to delineate the patterns of abnormal BOLD response associated with cognitive load [48] and the completion of turns [49], both of which are known to exacerbate freezing episodes in susceptible individuals [50]. Together, these results highlight the importance of utilizing VE tasks to understand symptoms of neurodegenerative disorders that would otherwise be difficult to interrogate using neuroimaging analyses.

In sum, it should be emphasized that virtual reality has advanced researchers' ability to examine more complex gait as well as cognition in greater detail than ever before. In the last decade, these lines of research have provided data that has helped progress the current understanding of gait impairments in PD, and also re-assess existing pathophysiological models of gait deficits (such as freezing). For example, prior to examining gait with fMRI, mechanisms underlying freezing of gait were initially discussed using broad concepts such as executive dysfunction, sensory-perceptual deficits, decoupling of anticipatory postural adjustments, and so on (see these reviews for more detail [50, 51]). In fact, only in the past decade have various hypotheses begun to incorporate more detailed levels of neurobiology and consideration for damaged circuitry. One of the most widely accepted theoretical views focuses primarily on the dopaminergic depletion within the basal ganglia, which was theorized to limit information processing of complementary yet competing inputs from different cortico-striatal pathways (see Fig. 25.6). However, after taking into consideration recent neuroimaging findings that highlight reduced functional connectivity and decoupling between key neural networks during virtual gait, researchers have revised theoretical models of gait impairment in PD to be more sophisticated [52], and identified a key paradigm shift that is necessary in order to fully understand underlying neural mechanisms of gait (Fig. 25.6a, b). Therefore, virtual reality has played a tremendous role in advancing our understanding mechanisms of gait impairments in PD.

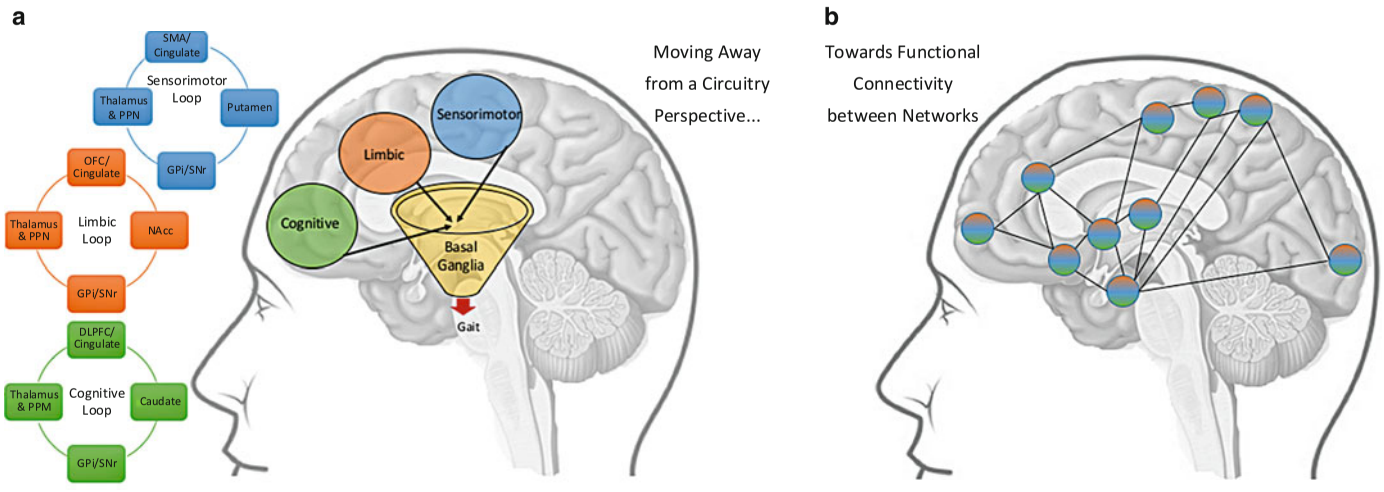

Fig. 25.6 A schematic illustrating a paradigm shift from previously focusing on faulty circuitry underlying gait abnormalities in PD towards understanding functional connectivity between neural networks which underlie gait impairments in PD (e.g., FOG). Note the abbreviations: *SMA* supplementary motor area, *GPi* globus pallidus interna, *SNr* substantia nigra reticulate, *PPN* pedunculopontine nucleus, *OFC* orbitofrontal cortex, *NAcc* nucleus accumbens, *DLPFC* dorsal lateral prefrontal cortex

25.3 Rehabilitating Gait in Parkinson's Disease with Virtual Reality

As our understanding of gait deficits in PD becomes more comprehensive, the ultimate goal of this research is to translate the current knowledge about mechanisms underlying gait impairment into novel therapeutic interventions that will effectively improve gait in PD. One way to facilitate this translation is to create rehabilitative strategies that focus on improving or overcoming contributing factors by entraining the brain to either repair itself or compensate in through some form of neuroplasticity to restore function. As stated above, one mechanism that VR has highlighted is the role of sensory feedback and sensorimotor integration on gait. Complex situations which require cognitive abilities (planning, attention, task-switching, etc.), or interpretation of environmental demands (obstacles, threat), are also known to exacerbate gait deficits in PD. Based on these findings, recent therapeutic strategies have focused primarily in two domains: (1) sensory cueing and (2) intense motor-cognitive training (i.e., walking with continuous cognitive tasks). In recent years, by utilizing virtual reality, research has advanced therapeutic interventions and their efficacy. For example, research has shown that visual and auditory cue training is more effective when delivered in an integrated feedback fashion (e.g., closed-loop) rather than being feedforward (e.g., open-loop), which has been shown to be less effective [53, 54]. VR provides an easy, consistent, and controlled method to maximize gait and cognition simultaneously [1, 2, 8, 32, 55]. Another advantage of virtual reality is the way in which training can be implemented. It creates a fun, "game-like" atmosphere that is enjoyable and conducive to high adherence. Training in VR has even been suggested to be superior compared to real-world equivalent training programs [1, 2, 8, 11–14], since it capitalizes on principles of motor learning with enhanced and immersive feedback (such as auditory and visual feedback of successful and unsuccessful performance). This was confirmed by a recent Cochrane review which favored VR therapeutic approaches for stroke rehabilitation compared to conventional therapies [56]. Difficulty and intensity of training programs can also be easily modulated with a VR format by increasing number of stimuli, distractors, and speed, which are difficult to control and manipulate in real-world settings [8]. Finally, one of the major advantages of VR is that training can simulate daily activities. It also enables at-home training, due to the portability and current computing capacity which is capable of measuring and providing performance feedback to users during and following gait training. Overall VR training may be a practical and useful tool to target sensory and cognitive deficits that contribute to gait impairments, and thus provide new opportunities to improve gait, mobility, and ultimately quality of life in those living with PD. In the following sections we will discuss research utilizing virtual reality as a method for therapeutic intervention for gait impairments in PD.

25.3.1 Using Sensory Cues in VR for Gait Improvement

It has been known for some time now that using sensory cues such as a metronome or transverse lines on the ground can significantly help impaired gait in those with PD, and even alleviate FOG in some instances. The notion that sensory input can help overcome these severe gait deficits suggests that the motor program for gait might be intact but not adequately selected due to sensory impairments in PD [20, 54]. The spinocerebellar, spinothalamic, spinoreticular, and spinohypothalamic tracts carry somatosensory information/feedback (such as proprioception) up to the brain during gait [20, 54], which modulates internal timing that helps to plan and predict future movements. Internal timing has been shown to be disrupted in PD [57], as a result of striatal dopaminergic depletion since the underlying neural networks for internal timing involve the basal ganglia (BG), supplementary motor area (SMA), and primary motor cortex (PMC) [58, 59], where the BG serves the most crucial role as it generates the internal pacing required for time estimation [60, 61]. In fact, a reduction in dopaminergic innervation to the BG in PD causes slower internal pacing [60], which manifests impaired motor and perceptual timing abilities [28, 62]. Not surprisingly, research has also shown that internal timing is argued to be more affected among those PD with gait deficits (e.g., freezing of gait) compared to those without [63].

It is important to note that although PD patients have impairments with internal timing, they can still use external cues to inform temporal-based decisions such as when the next footstep should be carried out. The effectiveness of external cues is thought to be due to distinct neural networks underlying internal versus external timing [54]. External timing mainly engages automatic timing systems by recruiting the cerebellum and thus is less dependent on the faulty BG and SMA [64]. Therefore, external cues (such as metronome beats or transverse lines) can serve as a proxy for impaired internal timing [65, 66] and improve gait by inducing motor-sensory feedback signals that recalibrate internal pacing. Virtual reality provides yet another medium to deliver external cues that the BG is unable to effectively generate and thereby repair a key issue that is at the core of gait impairments in PD.

Recently, researchers have begun to develop virtual reality devices that enhance external cues for rehabilitation in both the auditory and visual domains. Although cueing has been used over the past two decades as a helpful therapy for gait in PD, recent advancements in wearable VR technology have improved the efficacy of this rehabilitation strategy. One example is "intelligent glasses," designed to be portable and have built-in headphones which enable users to listen to a metronome like auditory cue while walking [67]. In a small study of just ten PD patients these auditory cues have been shown to improve cadence, stride length, and walking speed compared to walking without auditory cues [67]. Likewise, many studies have shown that matching footsteps to visual cues such as transverse lines on the ground or using VR glasses which deliver visual cues while walking on a treadmill improves gait (increased stride length and velocity, while reduced gait variability and FOG) in PD [68–70]. One of the main limitations of fixed-tempo auditory and

fixed-equidistant visual cueing is that they require increased attentional demand to synchronize individual footsteps, which imposes a cognitive load and promotes gait breakdown [54, 71, 72]. Additionally, fixed cueing is also easy for the participant to fall out of sequence with the cues, making them less effective and potentially even more problematic, such as provoking FOG [73].

Recent research has argued that these open-loop "fixed-cueing" strategies may not be the most effective for improving gait, since these sensory signals are not generated or affected by the users' own movement which is arguably a key feature of gait-related feedback [53, 73]. Thus, recent work has begun investigating closed-loop effects of visual cues on the regulation and rehabilitation of gait [73]. Research that directly compared open- and closed-loop visual cues showed that the closed-loop configuration produced nearly twice the improvement in average walking speed and stride length. This led to the conclusions that closed-loop VR systems are more effective at improving gait in PD patients than open-loop versions [73].

WalkMate is a closed-loop device which synchronizes foot pressure sensors to auditory cues. This apparatus appears to be a promising tool based on its ability to reduce gait variability in PD patients [74]. Another recent study used VR glasses to project a virtual checkered floor into the user's visual field. As the user walks, the VR floor adapts to the user's body movements by simulating the visual effect of walking [41]. The user's goal was to match their steps with the adjacent floor tile in order to regulate gait. This closed-loop system also produced encouraging results which demonstrated improvements in walking speed and stride length.

Since limited research has implemented closed-loop VR sensory feedback of whole body movement in locomotion, further research is needed to conduct randomized control trials with sufficient power to stringently evaluate the efficacy of VR-based therapies for PD. It also remains unclear whether current VR interventions have long-lasting effects. Future research should attempt to maximize the effects of cueing by utilizing multi-sensory cueing systems that can provide responsive and dynamic cues in the auditory, visual, and tactile domains [1, 54]. If a multi-sensory and adaptive VR device could respond to patients' movements, it may also demand less attention which might lead to a superior gait therapy. Research has assessed how receptive the Parkinson's population would be to this type of virtual reality technology (e.g., smart glasses wearable computers) [75], and overall patients were very enthusiastic about this type of assistive device to facilitate daily living activities.

25.3.2 Utilizing VR to Challenge Cognitive Functioning During Gait Training

Executive functions play a critical role in the regulation of gait and freezing [76–78], especially in complex environments where decisions need to be made in real-time [18, 19, 79]. Based on the strong relationship between cognitive functioning and gait impairment, it has been suggested that motor and cognitive training combined are likely to be more efficacious compared to simple gait training or using sensory cues.

Instead of training with sensory cues, which consume attention and reduces their capacity to cope with complex situations, motor-cognitive training promotes these situations and provides an opportunity to improve planning, decision making, and online adjustments. For example, dual-tasking (DT) and obstacle negotiation both rely heavily on the availability of ample cognitive resources due to the need for motor planning and visually dependent gait regulation [80, 81]. Thus, it seems reasonable to focus training on improving one's ability to cope directly with these situations, with the objective of transferring these skills to instances encountered on a daily basis. This is the approach that Mirelman and colleagues took by designing an intense and progressive rehabilitation program. The aim of the program was to promote cognitive processing by monitoring multiple stimuli simultaneously (delivered in VR), while walking and continuously negotiating obstacles on a treadmill [8, 55]. The virtual environment was able to impose varying degrees of cognitive load which demanded attention, response selection, and several perceptual processes, while tracking participants' foot movements. This training program was performed 3 times per week for 6 weeks, and produced very promising results. Typical gait improvements were found (increased gait speed, stride length, and increased obstacle clearance) which resemble treadmill training without VR imposing a cognitive load [8]. However, the added benefit of training with VR was made evident during evaluation of DT performance at post-test. Dual-task costs improved dramatically after training (31 %) and participants also demonstrated improved performance on the trail making tasks (a test of set-shifting and processing speed). It was suggested that this intense motor-cognitive combined therapy utilizing VR improved gait and also improved attention, set-shifting, and planning, which positively impacted DT ability [8]. These impressive gains were retained for at least 4 weeks after training ceased. Limitations of this study should be noted, since training was performed with a small sample, and a historical control group was used as a comparison rather than active control group.

Finally, a major advantage of virtual reality is the possibility that these motor-cognitive therapeutic interventions could be delivered in a home setting. Off-the-shelf gaming consoles make this idea feasible, relatively cheap, and accessible. For example, the Microsoft Kinect tracks full body motion using an infra-red camera while promoting integrated simulation of motor and cognitive functions. In fact, a recent study assessed the feasibility, safety, and outcomes of playing four existing Kinect Adventures!™ games (which encompass undefined motor and cognitive training) in 7 PD participants [82]. The participants completed fourteen 1-h sessions 3 times per week. The main findings of this study were that the Kinect training was both safe and feasible for patients with Hoehn and Yahr stage 2 and 3 Parkinson's disease. Additionally, improvements in gaming performance and other gait assessments were also seen, although due to small sample and a lack of control group, no statistical results were reported. Nonetheless this study provides evidence that at-home training, which utilizes virtual reality using gaming software, is feasible and possibly effective for improving gait. However, further research is needed to investigate and develop effective at-home motor-cognitive training programs that are aimed at improving gait.

References

1. Holden MK. Virtual environments for motor rehabilitation: review. Cyberpsychol Behav. 2005;8:187–211. doi:10.1089/cpb.2005.8.187. discussion 212–9.
2. Mirelman A, Maidan I, Deutsch JE. Virtual reality an motor imagery: promising tools for assessment and therapy in Parkinson's disease. Mov Disord. 2013;28:1597–608.
3. Gilat M, Shine JM, Bolitho SJ, et al. Variability of stepping during a virtual reality paradigm in Parkinson's disease patients with and without freezing of gait. PLoS One. 2013;8:e66718. doi:10.1371/journal.pone.0066718.
4. Shine JM, Ward PB, Naismith SL, et al. Utilising functional MRI (fMRI) to explore the freezing phenomenon in Parkinson's disease. J Clin Neurosci. 2011;18:807–10. doi:10.1016/j.jocn.2011.02.003.
5. Shine JM, Matar E, Bolitho SJ, et al. Modeling freezing of gait in Parkinson's disease with a virtual reality paradigm. Gait Posture. 2013;38:104–8. doi:10.1016/j.gaitpost.2012.10.026.
6. Nichols S. Physical ergonomics of virtual environment use. Appl Ergon. 1999;30:79–90.
7. Stanney K, Kennedy R, Drexleer J, et al. Motion sickness and proprioceptive after effects following virtual environment exposure. Appl Ergon. 1999;30:27–38.
8. Mirelman A, Maidan I, Herman T, et al. Virtual reality for gait training: can it induce motor learning to enhance complex walking and reduce fall risk in patients with Parkinson's disease? J Gerontol A Biol Sci Med Sci. 2011;66A:234–40.
9. Cole S, Yoo D, Knutson B. Interactivity and reward-related neural activation during a serious videogame. PLoS One. 2012;7:e33909.
10. Messier J, Adamovich SV, Jackson D, et al. Visuomotor learning in immersive 3D virtual reality in Parkinson's disease and in aging. Exp Brain Res. 2007;179:457–74.
11. Griffin HJ, Greenlaw R, Limousin P, et al. The effect of real and virtual visual cues on walking in Parkinson's disease. J Neurol. 2011;258:991–1000. doi:10.1007/s00415-010-5866-z.
12. Todorov E, Shadmer R, Bizzi E. Augmented feedback presented in a virtual environment accelerates learning a difficult motor task. J Mot Behav. 1997;29:147–58.
13. Rose F, Attree E, Brooks B, et al. Training in virtual environments: transfer to real world tasks and equivalence to real task training. Ergonomics. 2000;43:494–511.
14. Jaffe D, Brown D, Pierson-Carey C, et al. Stepping over obstacles to improve walking in individuals with poststroke hemiplegia. J Rehabil Res Dev. 2004;41:283–92.
15. Azulay JP, Mesure S, Amblard B, et al. Increased visual dependence in Parkinson's disease. Percept Mot Skills. 2002;95:1106–14.
16. Martens KAE, Almeida QJ. Dissociating between sensory and perceptual deficits in PD: more than simply a motor deficit. Mov Disord. 2012;27:387–92. doi:10.1002/mds.24042.
17. Davidsdottir S, Wagenaar R, Young D, et al. Impact of optic flow perception and egocentric coordinates on veering in Parkinson's disease. Brain. 2008;131:2882–93. doi:10.1093/brain/awn237.
18. Iansek R, Danoudis M, Bradfield N. Gait and cognition in Parkinson's disease: implications for rehabilitation. Rev Neurosci. 2013;24:293–300.
19. Lord S, Galna B, Coleman S, et al. Cognition and gait show a selective pattern of association dominated by phenotype in incident Parkinson' s disease. Front Aging Neurosci. 2014;6:1–9. doi:10.3389/fnagi.2014.00249.
20. Patel N, Jankovic J, Hallett M. Sensory aspects of movement disorders. Lancet Neurol. 2014;13:100–12. doi:10.1016/S1474-4422(13)70213-8.
21. Kaji R, Murase N. Sensory function of basal ganglia. Mov Disord. 2001;16:593–4.
22. Abbruzzese G, Berardelli A. Sensorimotor integration in movement disorders. Mov Disord. 2003;18:231–40. doi:10.1002/mds.10327.
23. Adamovich SV, Berkinblit MB, Hening W, et al. The interaction of visual and proprioceptive inputs in pointing to actual and remembered targets in Parkinson's disease. Neuroscience. 2001;104:1027–41. doi:10.1016/S0306-4522(01)00099-9.

24. Schubert M, Prokop T, Brocke F, et al. Visual kinesthesia and locomotion in Parkinson's disease. Mov Disord. 2005;20:141–50.
25. Prokop T, Schubert M, Berger W. Visual influence on human locomotion. Modulation to changes in optic flow. Exp Brain Res. 1997;114:63–70.
26. Ehgoetz Martens KA, Ellard CG, Almeida QJ. Does manipulating the speed of visual flow in virtual reality change distance estimation while walking in Parkinson' s disease ? Exp Brain Res. 2015;233:787–95. doi:10.1007/s00221-014-4154-z.
27. Ehgoetz Martens KA, Ellard CG, Almeida QJ. Dopaminergic contributions to distance estimation in Parkinson's disease: a sensory-perceptual deficit? Neuropsychologia. 2013;51:1426–34. doi:10.1016/j.neuropsychologia.2013.04.015.
28. Ehgoetz Martens KA, Ellard CG, Almeida QJ. A closer look at mechanisms underlying perceptual differences in Parkinson's freezers and non-freezers. Neuroscience. 2014;274:162–9. doi:10.1016/j.neuroscience.2014.05.022.
29. Lin C, Wagenaar R, Young D, et al. Effects of Parkinson's disease on optic flow perception for heading direction during navigation. Exp Brain Res. 2014;232:1343–55. doi:10.1007/s00221-014-3853-9.Effects.
30. Ehgoetz Martens KA, Pieruccini-Faria F, Silveira CR, et al. The contribution of optic flow to freezing of gait in left- and right-PD: different mechanisms for a common phenomenon? Parkinsonism Relat Disord. 2013;19:1046–8. doi:10.1016/j.parkreldis.2013.06.011.
31. Chan RCK, Shum D, Toulopoulou T, et al. Assessment of executive functions: review of instruments and identification of critical issues. Arch Clin Neuropsychol. 2008;23:201–16. doi:10.1016/j.acn.2007.08.010.
32. Albani G, Pignatti R, Bertella L, et al. Common daily activities in the virtual environment: a preliminary study in parkinsonian patients. Neurol Sci. 2002;23:49–50. doi:10.1007/s100720200064.
33. Cipresso P, Albani G, Serino S, et al. Virtual multiple errands test (VMET): a virtual reality-based tool to detect early executive functions deficit in Parkinson's disease. Front Behav Neurosci. 2014;8:405. doi:10.3389/fnbeh.2014.00405.
34. Klinger E, Chemin L, Lebreton S, et al. Virtual action planning in action Parkinson's disease: a control study. Cyberpsychol Behav. 2006;9:342–7.
35. Ehgoetz Martens KA, Ellard CG, Almeida QJ. Virtually-induced threat in Parkinson's: dopaminergic interactions between anxiety and sensory-perceptual processing while walking. Neuropsychologia. 2015:1–10. doi:10.1016/j.neuropsychologia.2015.05.015.
36. Ehgoetz Martens KA, Ellard CG, Almeida QJ. Anxiety-provoked gait changes are selectively dopa-responsive in Parkinson's disease. Eur J Neurosci. 2015:1–8. doi:10.1111/ejn.12928.
37. Ehgoetz Martens KA, Ellard CG, Almeida QJ. Evaluating the link between dopaminergic treatment, gait impairment, and anxiety in Parkinson's disease. Mov Disord Clin Pract. Published Online First: 2016. doi:10.1002/mdc3.12298.
38. Ehgoetz Martens KA, Ellard CG, Almeida QJ. Does anxiety cause freezing of gait in Parkinson's disease? PLoS One. 2014;9:e106561. doi:10.1371/journal.pone.0106561.
39. Lewis SJG, Barker RA. A pathophysiological model of freezing of gait in Parkinson's disease. Parkinsonism Relat Disord. 2009;15:333–8. doi:10.1016/j.parkreldis.2008.08.006.
40. Lewis SJG, Barker RA. Understanding the dopaminergic deficits in Parkinson's disease: insights into disease heterogeneity. J Clin Neurosci. 2009;16:620–5. doi:10.1016/j.jocn.2008.08.020.
41. Ehgoetz Martens KA, Ellard CG, Almeida QJ. Virtually-induced threat in Parkinson's: dopaminergic interactions between anxiety and sensory-perceptual processing while walking. Neuropsychologia. Published Online First: 2015. doi:10.1016/j.neuropsychologia.2015.05.015.
42. Naismith SL, Lewis SJG. The specific contributions of set-shifting to freezing of gait in Parkinson's disease. Mov Disord. 2010;25:1000–4.

43. Matar E, Shine JM, Naismith SL, et al. Using virtual reality to explore the role of conflict resolution and environmental salience in freezing of gait in Parkinson's disease. Parkinsonism Relat Disord. 2013;19:937–42.
44. Matar E, Shine JM, Naismith SL, et al. Virtual reality walking and dopamine: opening new doorways to understanding freezing of gait in Parkinson's disease. J Neurol Sci. 2014;344:182–5. doi:10.1016/j.jns.2014.06.054.
45. Shine JM, Matar E, Ward PB, et al. Exploring the cortical and subcortical functional magnetic resonance imaging changes associated with freezing in Parkinson's disease. Brain. 2013;136:1204–15. doi:10.1093/brain/awt049.
46. Shine JM, Matar E, Ward PB, et al. Freezing of gait in Parkinson's disease is associated with functional decoupling between the cognitive control network and the basal ganglia. Brain. 2013;136:3671–81. doi:10.1093/brain/awt272.
47. Shine JM, Koyejo O, Bell PT, et al. Estimation of dynamic functional connectivity using multiplication of temporal derivatives. Neuroimage. 2015;122:399–407.
48. Shine JM, Matar E, Ward PB, et al. Differential neural activation patterns in patients with Parkinson's disease and freezing of gait in response to concurrent cognitive and motor load. PLoS One. 2013;8:e52602. doi:10.1371/journal.pone.0052602.
49. Gilat M, Shine JM, Walton CC, et al. Brain activation underlying turning in Parkinson's disease patients with and without freezing of gait: a virtual reality fMRI study brain activation underlying turning in Parkinson' s disease patients with and without freezing of gait: a virtual real. npj Park Dis. 2015;1:15020. doi:10.1038/npjparkd.2015.20.
50. Nutt JG, Bloem BR, Giladi N, et al. Freezing of gait: moving forward on a mysterious clinical phenomenon. Lancet Neurol. 2011;10:734–44. doi:10.1016/S1474-4422(11)70143-0.
51. Nieuwboer A, Giladi N. Characterizing freezing of gait in Parkinson's disease: models of an episodic phenomenon. Mov Disord. 2013;28:1509–19. doi:10.1002/mds.25683.
52. Lewis SJG, Shine JM. The next step: a common neural mechanism for freezing of gait. Neuroscientist 2014. doi:10.1177/1073858414559101.
53. Baram Y. Virtual sensory feedback for gait improvement in neurological patients. Front Neurol. 2013;4:138. doi:10.3389/fneur.2013.00138.
54. Ashoori A, Eagleman DM, Jankovic J. Effects of auditory rhythm and music on gait disturbances in Parkinson's disease. Front Neurol. 2015;6:1–11. doi:10.3389/fneur.2015.00234.
55. Mirelman A, Rochester L, Reelick M, et al. V-TIME: a treadmill training program augmented by virtual reality to decrease fall risk in older adults: study design of a randomized controlled trial. BMC Neurol. 2013;13:15. doi:10.1186/1471-2377-13-15.
56. Laver K, George S, Thomas S, et al. Cochrane review: virtual reality for stroke rehabilitation. Eur J Phys Rehabil Med. 2012;48:523–30.
57. Jones C, Jahanshahi M. Motor and perceptual timing in Parkinson's disease. Adv Exp Med Biol. 2014;829:265–90.
58. Chen J, Penhune V, Zatorre R. Listening to musical rhythms recruits motor regions of the brain. Cereb Cortex. 2008;18:2844–54.
59. Bengtsson S, Ullen F, Ehrsson H, et al. Listening to rhythms activates motor and premotor cortices. Cortex. 2009;45:62–71.
60. Pastor M, Artieda J, Jahanshahi M, et al. Time estimation and reproduction is abnormal in Parkinson's disease. Brain. 1992;115:211–25.
61. Coull JT, Cheng R, Meck WH. Neuroanatomical and neurochemical substrates of timing. Neuropsychopharmacology. 2010;36:3–25. doi:10.1038/npp.2010.113.
62. O'Boyle D, Freeman J, Cody F. The accuracy and precision of timing of self-paced, repetitive movements in subjects with Parkinson's disease. Brain. 1996;119:51–70.
63. Tolleson C, Dobolyi D, Roman O, et al. Dysrhythmia of timed movements in Parkinson's disease and freezing of gait. Brain Res. 1624;2015:222–31.
64. Beudel M, Galama S, Leenders K, et al. Time estimation in Parkinson's disease and degenerative cerebellar disease. Neuroreport. 2008;19:1055–8.

65. Nombela C, Hughes L, Owen AM, et al. Into the groove: can rhythm influence Parkinson's disease? Neurosci Biobehav Rev. 2013;37:2564–70.
66. Nieuwboer A, Rochester L, Muncks L, et al. Motor learning in Parkinson's disease: limitations and potential for rehabilitation. Parkinsonism Relat Disord. 2009;15:S53–8.
67. Lopez W, Higuera C, Fonoff E, et al. Listenmee and Listenmee smartphone application: sychronizing walking to rhythmic auditory cues to improve gait in Parkinson's disease. Hum Mov Sci. 2014;37:147–56.
68. Jiang Y, Norman K. Effects of visual and auditory cues on gait initiation in people with Parkinson's disease. Clin Rehabil. 2006;20:36–45.
69. Luessi F, Mueller L, Breimhorst M, et al. Influence of visual cues on gait in Parkinson's disease during treadmill walking at multiple velocities. J Neurol Sci. 2012;314:78–82.
70. Lee S, Yoo J, Ryu J, et al. The effect of visual and auditory cues on freezing of gait in patients with Parkinson's disease. Am J Phys Med Rehabil. 2012;91:2–11.
71. Yogev G, Giladi N, Peretz C, et al. Dual tasking, gait rhythmicity, and Parkinson's disease: which aspects of gait are attention demanding? Eur J Neurosci. 2005;22:1248–56. doi:10.1111/j.1460-9568.2005.04298.x.
72. Rochester L, Nieuwboer A, Baker K, et al. The attentional cost of external rhythmical cues and their impact on gait in Parkinson's disease: effect of cue modality and task complexity. J Neural Transm. 2007;114:1243–8.
73. Badarny S, Aharon-Peretz J, Susel Z, et al. Virtual reality feedback cues for improvement of gait in patients with Parkinson's disease. Tremor Other Hyperkinet Mov (N Y) 2014;4:225. doi:10.7916/D8V69GM4.
74. Hove M, Suzuki K, Uchitomi H, et al. Interactive rhythmic auditory stimulation reinstates natural 1/f timing in gait of Parkinson's patients. PLoS One. 2012;7:e32600.
75. Zhao Y, Heida T, van Wegem E, et al. E-health support in people with Parkinson's disease with smart glasses: a survey of user requirements and expectations in the Netherlands. J Parkinsons Dis. 2015;5:369–78.
76. Walton CC, Shine JM, Mowszowski L, et al. Impaired cognitive control in Parkinson's disease patients with freezing of gait in response to cognitive load. J Neural Transm. 2015;122:653–60.
77. Walton CC, O'Callaghan C, Hall JM, et al. Antisaccade errors reveal cognitive control deficits in Parkinson's disease with freezing of gait. J Neurol. 2015;262:2745–54. doi:10.1007/s00415-015-7910-5.
78. Shine JM, Naismith SL, Palavra NC, et al. Attentional set-shifting deficits correlate with the severity of freezing of gait in Parkinson's disease. Parkinsonism Relat Disord. 2013;19:388–90. doi:10.1016/j.parkreldis.2012.07.015.
79. Rochester L, Galna B, Lord S, et al. The nature of dual-task interference during gait in incident Parkinson's disease. Neuroscience. 2014;265:83–94. doi:10.1016/j.neuroscience.2014.01.041.
80. Brown LA, McKenzie NC, Doan JB. Age-dependent differences in the attentional demands of obstacle negotiation. J Gerontol A Biol Sci Med Sci. 2005;60:924–7.
81. Pieruccini-Faria F, Ehgoetz Martens KA, Silveira C, et al. Interactions between cognitive and sensory load while planning and controlling complex gait adaptations in Parkinson's disease. BMC Neurol. 2014;14. doi:10.1186/s12883-014-0250-8.
82. Pompeu JE, Arduini LA, Botelho AR, et al. Feasibility, safety and outcomes of playing Kinect Adventures! for people with Parkinson's disease: a pilot study. Physiotherapy (United Kingdom) 2014;100:162–8. doi:10.1016/j.physio.2013.10.003.

The Applicability of Inertial Motion Sensors for Locomotion and Posture

26

Mark V. Albert, Ilona Shparii, and Xiaolu Zhao

Abstract

Wearable sensors are now ubiquitous consumer devices, enabling the general population to track their physical activities - providing estimates of time in each activity and measures such as steps and calories. Clinicians can benefit from this growth in wearable devices to better evaluate patient activity and posture for diagnosis and evaluating outcomes. The technologies involved range from low-grade, inexpensive consumer-oriented sensors to high-grade clinically validated activity monitors with associated analytics suites. In this chapter, we review the relevant technologies and how they can be applied to track locomotion and posture in clinical populations. We will review the form for these devices, the enabling analytics technology, and patient-specific applications. Beyond the limitations of more traditional measures, we will see that wearable devices enable convenient, objective, and continuous information that can assist clinicians in better diagnostics to quantify the impact of therapeutic interventions.

Keywords

Wearable • Monitoring • Activity recognition • Automated measures • Accelerometer

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26.1 Introduction

Inertial sensors are small, portable devices that are capable of measuring patient movement and posture continuously. They are compact, efficient, and inexpensive enough to be a part of all standard smartphones as well as dedicated wearable devices—some of which are seamlessly integrated into everyday fashion. Combined with modern analysis tools and techniques, the depth of information available to clinicians about patient posture and movement outside the clinical setting is dramatically increasing. This will likely bring many benefits not only to evaluating therapeutic interventions, but also to assessing individuals on a more personalized level.

26.1.1 Motivation

In order to evaluate the effectiveness of therapies, it is important to collect accurate information on the patient's state before and after intervention. However, for movement and posture the best available information is often limited. In-clinic assessments present snapshots of a patient's experience and contributing lifestyle factors, while at-home journaling of symptoms is often met with low compliance, frustration by patients, and subjective scoring. This lack of quality assessment of individuals and applied therapies suggests a role for devices that can provide such information.

26.1.2 Limitations of in-Clinic Assessments

There are limitations to what can be learned of patients through in-house clinical visits. Patient histories are limited by subject memory; this presents not only general concerns of accuracy and completeness in patient reports but also concerns of subject bias in what they report. Performance measures acquired in the clinic are relatively time-consuming and costly, and only offer a snapshot of ability. For longitudinal studies, the costs are particularly high for repeated visits, not only in terms of clinician time and the associated financial burden, but also the inconvenience of travel for subjects—especially for those with mobility impairments. Due to these costs, although clinical assessments provide valuable information, they are often administered relatively infrequently for long-term care.

26.1.3 Limitations of at-Home Subject-Reported Measures

Beyond the subjective and potentially inaccurate recollections of relevant experiences that may relate to the current condition, the state of a person when visiting a clinic can be significantly different than their condition in their daily lives. To provide more accurate data of daily patient experiences, many studies rely on

patient journaling of symptoms. However, as with in-clinic patient histories, at-home patient journals can still be subject to memory and subjective bias depending on the questions asked. Also, compliance is difficult to achieve with paper journaling, and with electronic methods (e.g., tablets or web-based) the inconvenience of charting experiences daily, or ideally multiple times per day, is a burden on the subject. Though perhaps an extreme example, compliance rates can be as low as 11 % in subjects who agree to journal their symptoms study using paper journals with an embedded sensor to check when notes were made [1]. Relieving patients and research subjects of such annoying tasks would clearly be welcome.

26.2 Wearables: Objective, Continuous, and Convenient Measures

Wearable devices can provide a more complete view of subject movement and posture, particular outside the clinic, while minimizing the costs and inconvenience compared to traditional methods of assessment. Instead of relying on biased subject memories of their experiences, objective data can be collected—this is particularly relevant for lifestyle influences on posture and locomotion, which can be challenging to measure by personal recollections. To get a picture of daily fluctuation in symptoms, even patient journaling has limits of only a few times per day for assessment, whereas worn sensors can be configured to record continuously.

However, the primary advantage of inertial sensors is in convenience. This is the reason they have taken off in the consumer landscape with an expanding array of dedicated fitness sensors (e.g., Fitbit, Jawbone) and integration with wearable devices (smartphones and smartwatches). Small, portable, efficient sensors can be configured to be worn with minimal interference is a subject's normal daily experience—incorporated into clothing including shoes, belts, or necklaces. From a research perspective, this promotes very high compliance and simplifies obtaining information on much larger groups of subjects than were available before. More importantly, this improves the patient experience, both in clinical research studies and during assessment with these tools.

26.2.1 Types of Wearables

There are many different types of wearable technologies available. First, they can vary in the type of signal they measure—here we focus on accelerations and rotations, but magnetic fields, air pressure, and even the Global Positioning System (GPS) coordinates can be useful for measuring motion. In addition to the sensors themselves, there is a constellation of design aspects for any wearable—battery life, storage needs, size, and in some cases fashion. Increasingly these technologies are being integrated into devices we already carry around regularly—making some

application in medical care as simple as downloading an app. However, it is important to keep in mind that the hardware is in many cases secondary to the analyses applied to those signals to provide useful information.

26.2.2 Types of Sensors

The most common sensor used in measuring posture and locomotion is the accelerometer, which as the name implies measures linear accelerations, often along three axes. In addition to movements of the device itself, accelerometers provide device orientation relative to the measured 9.8 m/s^2 gravitational acceleration, which can be particularly valuable for measuring posture. The smallest accelerometers are microelectromechanical systems (MEMS) based, with sizes measured in millimeters, making them exceptionally portable.

Accelerometers measure translational motion, while the equivalent sensor for rotations are gyroscopes. Similar to accelerometers, gyroscopes can be equivalently small and portable; however, they do not provide static orientation information. Most device movements are a combination of linear motion and rotation, so inference can often be done on one signal alone, making accelerometers the more standard inertial sensor of choice. When both sensors are available on a device, the resolution of inferred motions can often be increased, and for quick motions (like fall detection) this can be substantial. Due to the size and complementary nature of accelerometers and gyroscopes, they are often used in conjunction. Because such sensors rely on translational or rotational inertial in their design, they are collectively referred to as inertial sensors.

In addition to inertial sensors, there are a number of additional sensors that can be used to understand patient movement, often in ways that seem counterintuitive. For example, magnetometers measure passive magnetic fields—like the Earth's magnetic field used widely in compasses. Combined with a gravitational axis from an accelerometer, this can provide a device with a 3-axis reading of orientation. Detecting the difference between stair climbing and walking can be challenging using accelerations alone, but with an air pressure sensor (e.g., altimeter) elevation changes of floors in a building can be easily estimated. Additionally, with slightly more power and size, GPS coordinates can be collected. Aside from distances traveled, a thorough analysis can estimate whether someone is walking, running, biking, or riding in any number of vehicles. Magnetometers, altimeters, and GPS sensors are only some of the many additional sensors that can be used in wearable devices.

26.2.3 Sensor Placement

Sensor placement to measure locomotion or posture depends significantly on the location of the body. Sensors in the shoe may be particular useful for measuring steps, but would clearly provide little information on posture, while sensors placed

in hats, chest straps, or belts may provide valuable postural information, but less resolution for locomotion. Accelerometers have been used to identify activities in a variety of locations including the head, chest, arm, foot, and thigh [2]. Fixing the location provides consistency, which is valuable for maximum accuracy, but attaching sensors at particular locations on the body can be impractical or inconvenient for long-term use.

One of the standard arrangements is the waist-mounted accelerometer. Depending on the activities being studied, such a simple setup can be capable of 98 % activity recognition accuracy [3–6]. However, caution should be exercised as the nature of the movement being studied can have a dramatic impact on results. For example, typically in-lab acquired motions are more standardized than at-home movements. Signals from walking, sitting, and standing are more repeatable and regular when instructed in a lab setting. For a more natural analysis, when subjects simply wore such a device for 24 h accuracy was closer to 80 % [7]. Proper sensor selection and location often depends on the activities being identified and the limitations of the subject population for compliance.

26.2.4 Integrated Device Constraints

In any sensor, there are a number of design choices that are necessary due to constraints of size and price. Memory is often limited. Clinical research-grade devices like the Actigraph can carry raw accelerometer data for months, but also cost hundreds of dollars and are bulkier than their slimmer consumer device counterparts. In many cases, the incoming sensor data is processed quickly, and only the results of processing on small timescales are stored (e.g., incrementing steps or calorie estimates). Battery life and size is also a major issue. Always-on sensors can drain inexpensive batteries and require daily charging, often negating the convenience factor. Many sensors now also attempt to offer wireless transmission, with some options for intermittent low-power Bluetooth providing convenience and efficiency, again at the sacrifice of battery life. Ultimately, modern devices make appropriate choices along these dimensions given the size, price point, and market targeted.

It should be added that in addition to dedicated wearable sensors, many are embedded in technologies that we already carry daily—e.g., mobile phones. Smartphones already have adequate memory, computing power, and battery that many sensors need, and the sensors often have alternate uses for traditional smartphone use (gaming, rotating screens, providing map directions). Because of this, many advanced processing techniques to extract useful information can be applied to smartphone wearers with only a download. However, because the position of the phone on the body must be inferred and is unreliable, the results are certainly less accurate. Mobile phones conveniently contain many of the hardware and software capabilities to create a stand-alone activity tracking system, but have limited utility due to accuracy of analytics.

26.3 Analysis of Sensor Signals

26.3.1 Standard Analytics Framework for Wearable Devices

In this section, to better understand the limitations of information that can be extracted from wearable devices, we will review a standard analytics approach used to extract information from sensor signals.

When creating and analyzing tools applied to extract useful sensor information, the standard approach is to collect sensor information during a set of known activities to be inferred. For example, if walking is to be inferred from other activities, the sensor is to be worn during observed situations when the subjects are walking and not walking—ideally with examples that may be misclassified in real-life situations. With this data, analytic tools can be created and validated, but a number of steps must be performed to get the best accuracy.

In its simplest form, any system that uses inertial sensors will record signals and extract quantifiable features from those signals for later analysis. Sometimes, the magnitude of the reading is helpful (like acceleration due to gravity, which allows accelerometers to be used to measure inclination) but generally it is the change in the sensor values over time that is most useful. For individual clips in time, features of that changing signal can be extracted. For example, the average fluctuation in acceleration over a two second window can be a useful feature for determining if someone is walking or running. Of course, there are many simpler measures that can be extracted (e.g., skew, kurtosis, squares, cross products, absolute values) and more complex measures like Fourier coefficients that can be used to estimate the periodicity of signals. At this stage, the goal is to select features that are straightforward to extract from a clip of sensory data that, when combined with other features, may help in discerning different behaviors.

After appropriate features are extracted, a machine learning algorithm is used to predict the class or type of activity from the extracted features. Any number of algorithms can be used to perform this purpose (e.g., support vector machines, random forest, logistic regression). In addition to selecting the right classifier, many of these classifiers require tuning of higher-level so-called hyperparameters for maximum accuracy. This selection and tuning is performed using the collected training data. After proper model selection and tuning, the selected model can then be trained on the data and be ready for prediction on sensor data for which the true activity is not known. It is important to note that this is a standard approach, with many variations that extend this general strategy.

26.3.2 Applications and Limitations

The above framework can be used to estimate a wide variety of useful metrics. If the type of activity the person is performing is recorded, standard machine learning classifiers can recognize the activity with varying levels of accuracy. Activities to recognize can include lying down, sitting, standing, walking, running, stair

climbing. Specific to clinical population, use of a wheelchair or walker can also be estimated with the right training data. If the inference is a number rather than an activity class a regression model can be used (e.g., for step counts or calories expended per unit time). In general, if features extracted from the sensor signal have any degree of relationship with the predicted activity, prediction systems can be made and tested.

There are a number of limitations with this general strategy to be aware of. First, relevant features need to be extracted, and in some cases that is not possible—e.g., determining if someone is sitting in a stationary chair versus a wheelchair from an accelerometer. In other cases, there are useful features that we may not perceive but the algorithms can—for example, standing can be disambiguated from sitting on a chest worn sensor depending on the amount of imperceptible sway seen in a subject over time. Second, all models are limited by the quality and quantity of data available. If there are not enough samples available in any particular class, learning will be poor. Also, it is important that the designed sensor analytics are trained and validated on the same populations for which they will be applied to assure accuracy—patients with impaired mobility often have unique movement patterns in comparison to unimpaired subjects.

26.4 Activity Recognition in Patient Populations

Although a variety of metrics can be estimated from movement data using inertial sensors, the outcomes we are most interested in are the activities of daily living that are most closely associated with quality of life [8]. This can be a challenge, as many inferred activities from sensors must be calibrated to specific populations for the best accuracy.

For example, let us examine some concerns when developing and applying sensor-based measures for patients with Parkinson's disease. Parkinson's disease (PD) responds to a variety of treatment options, including drugs and various exercise therapies. Having quality metrics on outcomes can help select and improve upon the best strategies [9–12]. Unfortunately, applying current activity recognition techniques to populations with motor disabilities is challenging. PD symptoms include tremor, slowed motion (bradykinesia), rigid muscles, loss of common automatic movements, and impaired posture [13]. These symptoms all can adversely affect activity recognition.

Activity recognition has been applied to large number of patient populations. Activity recognition strategies have been tailored specifically for the elderly [14], individuals with muscular dystrophy [15], and even PD [16]. In a study involving patients with Duchenne muscular dystrophy, a single monitoring device worn on the chest reliably quantified walking parameters and time spent performing different activities [15]. Patients with PD wore three body-fixed inertial sensors which were used to accurately identify activities including walking, sitting, standing, lying, sit to stand, and stand to sit transitions [16]. It has also been shown that a mobile phone placed in a pocket can be used to recognize activities in people with

PD [17]. In many of these studies, it is either implied or explicitly measured that there is a need to adapt the recorded data to the unique movement patterns of the population being studied to improve accuracy.

26.5 Examples in Locomotion and Posture

Accelerometers provide a non-invasive, portable method for evaluating the smoothness or variability of walking patterns. They are able to measure certain aspects of gait and detect variations in gait to provide clinically useful measures [18] including stride time, stride symmetry, and speed. The accelerations detected by the sensor during walking reflect the cyclical movement as it slows down and speeds up, rises and falls, and moves from side to side [19, 20]. One of the major benefits of using accelerometers on evaluating locomotion is to support the diagnosis of certain diseases. For example, walking symmetry and velocity can provide useful indicators that people may be suffering from neurologic or musculoskeletal disorders. Changes over time in measured movements can also alert a clinician to a subject's deteriorating health condition—for example, a sudden decrease in walking speed or stride length noting elderly frailty [21].

Bad posture is both a cause and indicator of adverse health conditions. The risks of posture deformation also increase with aging. For example, the head may lean forward and shoulders round for older adults. Such changes may affect chronic back and/or neck pain. Over time, this might lead to even more serious problems such as difficulties to maintain a posture balance, spinal deformities, and hyperkyphosis. Some treatment approaches suggest providing accurate feedback for patients about their posture, so they are able to correct their posture resulting in a long-term improvement. Some methods for posture measurement such as goniometry, photogrammetry, and video analysis have been widely used. However, many of these techniques require laboratory equipment and technicians to record data which comes at great expense [22]. Inertial sensors can provide accurate feedback about a person's posture by measuring position and orientation in space. For instance, accelerometer-based systems measure angle relative to gravity, with dedicated devices for this purpose referred to as inclinometers. Such sensors can train patients to improve their posture by giving them appropriate feedback [22, 23].

26.6 Conclusion

The ultimate objective of therapies for patients is to improve quality of life. Better outcomes data allows clinical researchers to directly compare and optimize many available therapies. Inertial sensors provide a continuous, convenient, and objective set of measures on the activities of subjects that otherwise would not be available. Such recorded information is useful for assessing individuals, or for making decisions on practices based on research studies. Through improved sensors,

analysis techniques, and validation these wearable assessment tools will continue to improve over time, enabling more convenient and higher quality information to better improve patient health.

References

1. Stone AA, Shiffman S, Schwartz JE, Broderick JE, Hufford MR. Patient compliance with paper and electronic diaries. Control Clin Trials. 2003;24(2):182–99.
2. Kavanagh JJ, Menz HB. Accelerometry: a technique for quantifying movement patterns during walking. Gait Posture. 2008;28(1):1–15.
3. Mathie MJ, Celler BG, Lovell NH, Coster ACF. Classification of basic daily movements using a triaxial accelerometer. Med Biol Eng Comput. 2004;42(5):679–87.
4. Mathie MJ, Coster ACF, Lovell NH, Celler BG. Accelerometry: providing an integrated, practical method for long-term, ambulatory monitoring of human movement. Physiol Meas. 2004;25(2):R1–20.
5. Lee M-h, Kim J, Kim K, Lee I, Jee SH, Yoo SK. Physical activity recognition using a single tri-axis accelerometer. In: Proceedings of the World Congress on Engineering and Computer Science, San Francisco, 2009.
6. Ravi N, Dandekar N, Mysore P, Littman ML. Activity recognition from accelerometer data. In: Proceedings of the Seventeenth Conference on Innovative Applications of the Artificial Intelligence, Pittsburgh, 2005.
7. Long X, Yin B, Aarts RM. Single-accelerometer-based daily physical activity classification. Conf Proc IEEE Eng Med Biol Soc. 2009:6107–10.
8. Ellis T, Cavanaugh JT, Earhart GM, Ford MP, Foreman KB, Dibble LE. Which measures of physical function and motor impairment best predict quality of life in Parkinson's disease? Parkinsonism Relat Disord. 2011;17(9):693–7.
9. Dibble LE, Addison O, Papa E. The effects of exercise on balance in persons with Parkinson's disease: a systematic review across the disability spectrum. J Neurol Phys Ther. 2009;33 (1):14–26.
10. Goodwin VA, Richards SH, Taylor RS, Taylor AH, Campbell JL. The effectiveness of exercise interventions for people with Parkinson's disease: a systematic review and meta-analysis. Mov Disord. 2008;23(5):631–40.
11. Palmer SS, Mortimer JA, Webster DD, Bistevins R, Dickinson GL. Exercise therapy for Parkinson's disease. Arch Phys Med Rehabil. 1986;67(10):741–5.
12. Schenkman M, Cutson TM, Kuchibhatla M, Chandler J, Pieper CF, Ray L, et al. Exercise to improve spinal flexibility and function for people with Parkinson's disease: a randomized, controlled trial. J Am Geriatr Soc. Blackwell Publishing Ltd; 1998;46(10):1207–16.
13. Jankovic J. Parkinson's disease: clinical features and diagnosis. J Neurol Neurosurg Psychiatry. 2008;79(4):368–76.
14. Najafi B, Aminian K, Paraschiv-Ionescu A, Loew F, Bula CJ, Robert P. Ambulatory system for human motion analysis using a kinematic sensor: monitoring of daily physical activity in the elderly. IEEE Trans Biomed Eng. IEEE. 2003;50(6):711–23.
15. Jeannet P-Y, Aminian K, Bloetzer C, Najafi B, Paraschiv-Ionescu A. Continuous monitoring and quantification of multiple parameters of daily physical activity in ambulatory Duchenne muscular dystrophy patients. Eur J Paediatr Neurol. 2011;15(1):40–7.
16. Salarian A, Russmann H, Vingerhoets FJG, Burkhard PR, Aminian K. Ambulatory monitoring of physical activities in patients with Parkinson's disease. IEEE Trans Biomed Eng. 2007;54:2296–9.
17. Albert MV, Toledo S, Shapiro M, Kording K. Using mobile phones for activity recognition in Parkinson's patients. Front Neurol. 2012;3:158.

18. Culhane KM, O'Connor M, Lyons D, Lyons GM. Accelerometers in rehabilitation medicine for older adults. Age Ageing. 2005;34(6):556–60.
19. Auvinet B, Berrut G, Touzard C, Moutel L, Collet N, Chaleil D, et al. Reference data for normal subjects obtained with an accelerometric device. Gait Posture. 2002;16(2):124–34.
20. Zijlstra W, Hof AL. Assessment of spatio-temporal gait parameters from trunk accelerations during human walking. Gait Posture. 2003;18(2):1–10.
21. Fontecha J, Navarro FJ, Hervás R, Bravo J. Elderly frailty detection by using accelerometer-enabled smartphones and clinical information records. Pers Ubiquitous Comput. Springer London; 2013;17(6):1073–83.
22. Lou E, Bazzarelli M, Hill D, Durdle N. A low power accelerometer used to improve posture. Can Conf Electr Comput Eng 2001 Conf Proc (Cat No01TH8555). 2001;2(780):1385–9.
23. Wong WY, Wong MS. Trunk posture monitoring with inertial sensors. Eur Spine J. 2008;17 (5):743–53.

Age-Related Changes in the Neural Control of Standing Balance

27

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Abstract

Controlling body sway while standing is an active process involving lower as well as higher neural centers. This chapter examines how the central nervous system controls undisturbed standing balance and summarizes the current knowledge concerning the effects of task difficulty and old age on postural control of standing. There is an age-related reorganization of neural control of standing, with decreased efficacy of Ia afferents to activate spinal motor neurons, increased cortical activation and corticospinal excitability, and reduced intracortical inhibition. Age does not affect the motor control strategy of reducing intracortical inhibition with increasing postural challenge. However, the threshold for down-modulation decreases with aging. It thus seems that motor cortical involvement in the control of standing balance becomes more prominent with age and postural task difficulty. Future studies will determine if it is beneficial and necessary through interventions to reduce the cortical involvement in the control of standing balance in healthy old adults especially if this involvement should increase in old adults with a history of falls.

Keywords

Standing • Central nervous system • Aging • Intracortical inhibition • TMS • fMRI • EEG • H-reflex

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27.1 Introduction

Undisturbed standing is a specific form of daily activity. Humans assume an upright body position as a transition between locomotor tasks. Occasionally, standing is also a purposeful and somewhat disliked activity such as when we are forced to patiently wait in the checkout line in the grocery store, wait for a bus, stand in a military formation for long minutes, or, as recently recommended, stand steadily by the office desk for bouts of 10–12 min every hour to reduce the deleterious effects of prolonged sitting [1, 2].

Maintaining an unperturbed erect posture, i.e., a body position in which ankle, knee, and hip joints are at their anatomical zero, involves seemingly passive biomechanical, physiological, and perceptual processes. The present chapter will, however, challenge the appearance of simplicity and passivity and demonstrate that against long-held views standing is very much an active process involving higher neural centers including the primary motor cortex (M1). The chapter also highlights the emerging view that even healthy aging affects the neural control of unperturbed standing, necessitating compensations for the many age-related sub-clinical structural and functional changes in the neuromuscular system [3, 4]. The involvement of higher neural centers is amplified when standing becomes less unstable due to modifications in the quality and size of the support surface under the feet or when leaning, a frequent postural challenge in daily life. The chapter examines the motor cortical, subcortical, and spinal control of undisturbed standing and summarizes the current knowledge on how healthy aging affects the neural control of standing with and without postural and surface challenges. The first part provides an overview of the control mechanisms of sway during standing in healthy young adults and the second part of the chapter summarizes recent data in healthy old adults.

27.2 How Do Healthy Young Humans Stand? Mechanical Considerations

Healthy human infants can independently stand on two feet a year after birth and stand with adult-like postural control properties by age 12 [5–8]. Healthy humans spontaneously sway while standing quietly, unsupported, and unperturbed. Routine quantification of sway indexed by center of pressure in the time and frequency domains consistently showed that sway amplitude, area, speed, and frequency decrease steeply during the period between infancy and adulthood [5–16] and then sharply increase again several fold by old age, starting at around age 60 [3, 4, 9, 14, 17–28].

The source of low frequency, ~0.5–2.5 Hz, sway is the continuous correction of the imbalance between the constant gravitational force field and the fluctuations in forces generated by lower extremity and trunk muscles. Because both sets of forces act through moment arms, standing balance is ultimately the result of the equilibrium between two counteracting torques created by gravitational and muscle force.

Even quiet, unperturbed standing requires the activation of lower extremity muscles to a level of about 5–10 % of maximum, suggesting that standing certainly is an active process [17, 18].

Quiescent standing is inherently and mechanically unstable. About 63–67 % of total body mass, with its center located at 58–62 % of body height, is controlled over a narrow base of support corresponding to a foot length of only 10–15 % of body height [29, 30]. As the center of mass sways, the toppling torque increases at the boundaries and exceeds the passive ankle torque. Mechanical analysis classifies the unstable standing equilibrium as a saddle-type, meaning that there are instances when the postural state transits close to a point of equilibrium and when the postural state falls away from vertical [31, 32]. The insufficient levels of ankle stiffness, considered as a key mechanical and passive controller of standing balance, further compounds instability while quietly standing [33, 34] because the gravitational torque, exceeding the restorative torque generated passively by the ankle, tends to topple the lower extremity joints, preventing the system from ever reaching asymptotic stability [32, 33, 35]. Studies examining neural strategies via numerical simulation challenged the ankle joint-only control hypothesis because an increase in stiffness of ankle, knee, and hip joints by co-contracting all of the muscles surrounding the three lower extremity joints was still insufficient to ensure stability, implying that in addition to mechanical factors neural control must be also strongly involved [36].

Because the linear relationship between torque and the angular acceleration it generates and because such a relationship simplifies the neural command to control sway, many studies modeled standing balance as a single-link inverted pendulum that pivots at the ankle joint in the sagittal plane to explain normal human sway during unperturbed standing [37–44]. However, it is becoming clear that muscles surrounding the knee and hip joints also become activated and a multi-joint model is more appropriate to explain normal standing sway [45–52]. Overlooked even today in the imaging and transcranial magnetic brain stimulation (TMS) literature that focuses exclusively on ankle plantarflexor and dorsiflexor function in standing and leaning [4, 17, 18, 53–59], there is long-standing experimental evidence suggesting that the angular accelerations at the hip joint are as high or even higher than those measured at the ankle during quiet standing [46, 51]. These antiphase accelerations at the ankle and hip joints tend to minimize the sway-inducing horizontal linear acceleration of the center of mass. There is also evidence for a tight coupling in the fluctuation between ankle and knee torques during standing and uncontrolled manifold analyses point to coordinated variations among the ankle, knee, and hip joints to stabilize variables important to postural control during quiet stance in healthy young adults [48–50, 60]. These observations culminate in the concept that inter-joint coordination controls sway while healthy humans stand unperturbed. That is, sway-control can be best characterized not by a one-joint inverted pendulum but by a double inverted pendulum model consisting of multiple degrees of freedom [32, 35]. The control of sway is an active rather than a passive stiffness-driven process, necessitating a neural oversight.

27.3 Need for Neural Control of Sway During Standing

There is thus a need to actively stabilize sway during undisturbed standing. An automatic, unsupervised control of active joint torques is unlikely because standing control and preparatory readiness to impending mechanical perturbations should be customized to environmental and task constraints, such as standing surface and concomitant manual or cognitive tasks [21, 61, 62]. The neural control of sway is complicated by a neural transmission delay of about 200 ms [15, 42]. Therefore, any neural control must meet the requirement of being flexible and accurately timed relative to the phase of sway. A decades-long debate has focused on intermittent and continuous neural control of sway.

According to the intermittent control model, neural activation is turned off when the sway pendulum is at or near a stable phase of sway and neural activation is turned on, with a physiologically appropriate delay, during the unstable phase of sway [37, 63]. Continuous [40, 64, 65] and discontinuous intermittent feedback control models [39, 66–68] can successfully reproduce healthy human postural sway and produce a robust yet flexible stability of erect posture through a single inverted pendulum model that operates at the ankle joint. However, these models do not and cannot account for documented actions arising from knee and especially from the hip joint. Thus, a double inverted pendulum model, consisting of upper (head, arms, trunk) and lower links (ankle, hip) and driven by an intermittent neural controller that can synchronize ankle and hip activation at a low metabolic cost would have higher ecological validity [32]. Numerical simulations revealed that such neural coupling between ankle and hip muscles [69, 70] modeled by a double inverted pendulum [32] can stabilize erect posture. The emerging idea is that perhaps there is a single neural controller of multiple muscles supervising inter-joint coordination. A reliance on local feedback within each individual joint would make the control of sway complex, perhaps redundant and an unsuitable strategy to stabilize sway [35, 71].

27.4 Neural Control of Undisturbed Standing in Young Adults

There is a remarkable lack of overlap between the body of literature reporting on the biomechanical models of postural sway that makes inferences how the nervous system stabilizes erect posture (references in previous section) and another body of literature reporting on the neural control of unperturbed standing using imaging, electroencephalography (EEG), electrophysiology, and TMS [3, 4, 17, 21, 53, 54, 56, 57, 59, 72]. While modeling studies correlate triceps surae activation and ankle muscle stiffness [39], quantify the neural coupling between ankle and hip muscles, and invoke muscle synergies as neural control mechanisms of standing balance based on surface electromyography [32, 64], a correspondence between these outcomes and those described by imaging, TMS, and electrophysiological studies is absent. On the other hand, TMS studies target specific excitatory and inhibitory

circuits during standing and imagery studies in the MRI scanner examine brain activation but do not measure muscle synergies, muscle-to-muscle couplings, or mechanical properties of muscles.

A lack of correspondence between these two bodies of literature is understandable in so much that linking model-predicted neural outcomes in the context of posture control with imaging, EEG, and TMS outcomes is at the core of the fundamental question of what M1 actually controls, i.e., individual muscles or a group of (synergistic) muscles, or activation patterns (synergies). Primate data with implanted electrodes suggest that loads only at the elbow, only at the shoulder, or at both joints modulate neural activity of certain motor cortical cells, assigning a putative role to M1 controlling not only individual muscles but also muscle synergies in a multi-joint reaching task [73, 74]. While motor modules (synergies) and their roles in gait and posture have been reviewed extensively, links with imaging, EEG, electrophysiological, and TMS studies examining M1 involvement in such synergies invariably remain elusive [75–77].

Standing balance is the result of integration of visual, vestibular, cutaneous, and proprioceptive inputs, with signal processing, motor planning, and motor execution occurring in the spinal cord, brainstem, cerebellum, basal ganglia, and M1 [3, 21, 72]. The ability of animals to stand and even modify erect posture after transections at the level of the midbrain gave rise to view that "...the whole righting apparatus...is arranged sub-cortically in the brainstem, and in this way made independent of direct voluntary influences" [78]. Extending classical studies [79], several lines of evidence converge to the idea that cerebral structures contribute to the control of unperturbed standing. Although spinal circuits have a major role in the maintenance of erect posture and in the recovery of lost abilities to stand [80], cortical recordings revealed strong activation of M1 neurons when the support surface under small quadruped mammals' paws oscillated [81]. In these animals, tonic supraspinal drive and phasic supraspinal commands tended to facilitate the otherwise weak spinal networks that receive somatosensory inputs from the limb signaling postural perturbations [81].

In intact healthy humans, evidence for M1 involvement in standing control comes primarily from magnetic stimulation studies. Such studies discovered that postural instability caused by the tilting of the surface platform modifies the state and the role of M1 in equilibrium maintenance. Indeed, the size of the motor evoked potentials (MEPs) in the soleus muscle increased 2.2 times when subjects stood on a moveable compared with stable surface [82]. When peripheral nerve stimulation was adjusted so that the peak of the H-reflex coincided with the peak of the long-latency stretch reflex evoked by posterior translation of the feet while standing, conditioning the H-reflex by subthreshold TMS resulted in a facilitation [83]. As the increase in cortical excitability contributed to the augmentation of the stretch reflex peak, these results provide evidence for a direct involvement of corticospinal projections in postural control. Moreover, motor cortical excitability is similarly [84, 85] or even more strongly [86] modulated with background electromyography (EMG) in postural compared with voluntary contractions, suggesting that these contractions require at least a similar level of cortical involvement. In a series of experiments that compared M1 involvement during postural and non-postural

contractions with varying levels of postural challenge (i.e., leaning), the area of EMG suppression was ~60 % smaller ($p < 0.05$) in unsupported vs. supported leaning and sitting, suggesting that postural challenge rather than the aim of the contraction (postural, non-postural) or posture (sitting, supported and unsupported leaning) reduced motor cortical inhibition [57]. Finally, when healthy subjects performed motor imagery and action observation of balance tasks in an MRI scanner, during the combined imagery and action observation task participants activated the putamen, cerebellum, supplementary motor area, premotor cortices (PMv/d), and M1, with imagery showing the same activation except for M1 and PMv/d. The brain activation scaled by intensity of balance task [72].

Despite these series of technically highly challenging studies pointing toward supraspinal involvement in the control of standing balance, there are also contradictory findings. The size of MEPs increased when healthy young adults stood unsupported compared with a condition in which a board provided support at the chest, relieving the central nervous system to control sway [59]. However, these, unlike the spinal and corticospinal responses were independent of sway phase. Therefore, it seems that M1 is not involved in the direct on-going control of postural sway, leaving the exact role of M1 in postural control unclear.

27.5 Structural, Functional, and Behavioral Changes with Aging in the Neuromotor System

The aging central nervous system undergoes unfavorable structural changes such as declines in quality and quantity of cortical gray and white matter [87–89]. Such structural changes may affect how the brain operates during motor tasks, resulting in functional changes (Fig. 27.1). For example, more brain activation may be required to achieve the same behavioral outcome. Structural and functional changes often lead to declines in motor performance, i.e., behavioral changes. As age-related structural changes are evident at different levels of nervous systems involved in postural control (spinal, subcortical, and cortical), functional and behavioral changes can also be expected. The following segment provides a summary and systematic review of the existing literature regarding the age-related changes in neural control of posture with a focus on standing.

27.6 Age-Related Changes in Spinal Control of Standing

Spinal reflexes can be assessed using the H-reflex, an electrical variant of the tendon stretch reflex. The amplitude of the H-reflex quantifies the efficacy of primary (Ia) afferents to activate spinal motor neurons. A consistent finding in the literature is that old compared with young adults exhibit smaller soleus H-reflexes, both in absolute units and relative to the maximal compound action potential, M-max, during standing [18, 90–94]. This age-related reduction in H-reflex may be caused by degeneration in the spinal motor neurons or Ia afferents, reduced excitability of

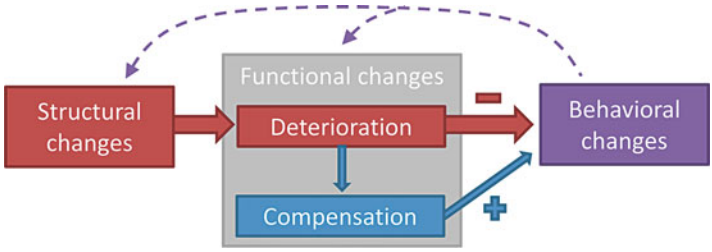

Fig. 27.1 A classification model of the different domains of age-related changes in the neuromotor system controlling postural and manual tasks. Three domains can be distinguished: structural changes, functional changes, and behavioral changes. Structural changes refer to the degeneration of brain or nerve structures with aging, whereas functional changes refer to the age-related modification in how these structures operate in the act of motor control. Behavioral changes denote the changes in performance on the motor task, which can be both a postural or a manual task. Functional changes can be divided into deterioration (as a direct result of the structural changes) and compensation (changes in function to counteract the deterioration). Structural degeneration causes functional deterioration [133], which triggers the need for functional compensation [134]. Functional deterioration has a negative impact on performance [135], whereas functional compensation has a positive impact [134]. The *dashed arrows* acknowledge the influence that acute or chronic behavioral changes, i.e., intervention or differences in lifestyle, have on structure and function of the neuromotor system [58, 136, 137]. The model can be used in future studies to systematically examine the structure-function-behavior link in the aging neuromotor system and could also be applied to other fields of research. Reproduced from Papegaaij et al. [3]

the spinal motor neurons, changes in presynaptic inhibition, or more homogeneous excitability thresholds of the afferent and efferent axons [18, 93]. How age affects the size of the H-reflex can perhaps be most insightfully ascertained by examining the interaction between age and postural task difficulty. Although most studies agree on a down-modulation of the soleus H-reflex with postural task difficulty in young adults [18, 90–93], the effect of age on this modulation remains controversial. Some studies reported an upregulation from lying or sitting to standing in old adults [90, 91], whereas others reported no modulation [92, 95], or even greater down-modulation than young adults [18]. It should be noted that only the last study controlled properly for background EMG which usually differs between conditions and age groups, probably explaining at least in part the differences in results between studies. The greater H-reflex down-modulation with task difficulty is in agreement with a previous study by the same group showing a greater increase in presynaptic inhibition with postural task difficulty in old compared with young adults [94]. Greater presynaptic inhibition was associated with greater co-contraction among lower leg muscles. In conclusion, age decreases the efficacy of Ia afferents to activate spinal motor neurons and changes the modulation of spinal reflexes between postures. The reduced spinal excitability in old adults may suggest a greater need for higher centers to control the leg muscles during standing.

27.7 Age-Related Changes in Subcortical Control of Standing

Subcortical structures such as the basal ganglia, cerebellum, and brain stem are important for postural control [96–98]. Indeed, age-related declines in white matter integrity, gray matter volume, and striatal dopaminergic activity contribute to postural instability in older adults [99–103]. However, little is known regarding the age-related functional changes in subcortical control of standing. Two fMRI studies do provide some relevant information [104, 105]. When stimulating the foot muscle spindles, older adults exhibited less activity in the right putamen [104]. Furthermore, less activity in the right putamen and pallidum was associated with greater body sway during standing with the eyes closed. In contrast, Zwergal and collaborators [105] reported no differences in activation of the subcortical structures during imaginary standing between young and old adults. It can therefore be concluded that age-related changes in subcortical structures do affect postural control but more research is needed to identify the specific functional changes.

27.8 Age-Related Changes in Cortical Control of Standing

Aging causes a reorganization of cortical control of voluntary manual movements, with an increase in brain activation and decrease in cortical inhibition [3, 106–109]. As the neural circuits affected by age-related degeneration are also involved in postural control, it is likely that changes in brain activation and inhibition also occur during standing.

Measuring BOLD responses related to postural control is a challenging task because it is not yet possible to perform fMRI during upright standing. One solution is to instruct participants to imagine that they are standing while they are lying in the scanner. Consistent with the literature on voluntary manual tasks, Zwergal and collaborators [105] reported age-related increases in activation of the multisensory vestibular cortices, motion-sensitive visual cortices (MT/V5), and somatosensory cortices (right postcentral gyrus) during imaginary standing. Focusing on the sensory aspect of postural control, Goble and collaborators [104] measured the proprioception-related neural activity by stimulating the foot muscle spindles in young and old adults and observed an age-related decrease in right putamen activation. In both age groups, greater activation in parietal, frontal, and insular cortical areas, as well as structures within the basal ganglia, was associated with better balance performance. The age-related decrease in brain activation during passive sensory processing is consistent with studies examining neural processing of visual and auditory stimuli in old adults [110, 111]. It seems likely that the increase in activation of multisensory areas observed by Zwergal and collaborators [105] is a compensation for the decline in unimodal sensory systems, a view that still awaits additional experimental confirmation.

Although the above-mentioned studies provide an interesting perspective on changes in balance-related brain activation with aging, we note that imaginary standing is quite different from actual standing. As TMS can be applied during

upright stance, this has become a popular method to assess neural activity associated with balance. Using this method, the group of Jacques Duchateau has published several papers consistently reporting an age-related increase in corticospinal excitability [18, 54, 112]. Old compared with young adults exhibited increased motor evoked potentials (MEPs) and TMS-induced facilitation of the Hoffmann (H)-reflex in the soleus muscle during quiet standing. They also reported an age-independent modulation in corticospinal excitability between postures, with greater MEP amplitude during standing as compared with sitting [18]. The major limitation of these studies is that background EMG also differed between age groups, possibly contributing to the age-related increase in corticospinal excitability. Indeed, another study reporting similar tibialis anterior and lateral gastrocnemius background EMG in young and old adults during standing also reported no difference in MEP amplitude in these muscles [113].

In addition to corticospinal excitability, TMS can also be used to assess intracortical inhibition. Old compared with young adults exhibited a shorter silent period after an MEP during standing, suggesting that intracortical inhibition decreases with aging [18, 54]. In a series of interlinked studies we assessed intracortical inhibition using paired pulse [17, 56] and subthreshold TMS [57] during postural tasks of varying difficulty in young and old adults. The first study examined whether or not age interacts with sensory manipulation during standing by measuring short-interval intracortical inhibition (SICI) in the tibialis anterior [17]. There was an overall age-related decrease in SICI. Old but not young adults reduced SICI even further when standing on foam vs. a rigid platform. As age-related sensory and motor deficits complicate postural control [114–118], standing on foam can be considered a more challenging task for old than for young adults. Therefore, the age-related difference in modulation of intracortical inhibition may have been due to the relatively higher postural challenge. In subsequent experiments we examined whether young and old adults also modulate intracortical inhibition differently when postural task difficulty is low [56] or adjusted to individual skill level [57]. Young and old adults exhibited similar down-modulation of SICI in the soleus and no modulation of SICI in the tibialis anterior from normal standing to standing with light support at the chest. In the next study, subjects were asked to lean forward to 75 % of their maximum, a position beyond which they would have fallen forward. Intracortical inhibition during leaning, as quantified by subthreshold TMS-induced suppression in the on-going soleus EMG, was compared with that during supported leaning and sitting. The area of EMG suppression was ~60 % smaller in unsupported vs. supported leaning and sitting, with no difference between these latter two conditions. Even though in absolute terms young compared with old adults leaned farther, there was no age effect or an age by condition interaction in EMG suppression. Moreover, greater reductions in intracortical inhibition were related to greater instability when standing on foam [17] and to leaning closer to the maximum during unsupported leaning [57]. Together, these results imply that postural challenge is the main factor influencing intracortical inhibition and that age does not affect the motor control strategy of reducing intracortical inhibition with increasing postural challenge. However, as

a similar task can be more challenging for old compared with young adults, the threshold for down-modulation decreases with aging.

In addition to intracortical inhibition, previous studies also examined intracortical facilitation during standing in young and old adults [17, 56]. Our finding of no age-related changes in intracortical facilitation agrees with the majority of previous studies in manual tasks [119–122], although there are sporadic reports of a decrease in intracortical facilitation in old compared with young adults [123, 124]. There was also no modulation in intracortical facilitation between the different standing tasks. Great inconsistency exists in the literature regarding modulation of intracortical facilitation between postures, with reports of reduced [84], similar [125], and increased [86] intracortical facilitation during standing as compared with sitting. Methodological differences between studies such as the muscle examined, variation in stimulation intensity and interstimulus interval, and/or the high between-subject variability in this measure could be behind the inconsistencies. Overall previous findings suggest that at least during standing motor cortical excitability is modulated through intracortical inhibition and not facilitation [17, 56].

Age-related changes in cortical activity before and after postural perturbations have been studied using EEG. Duckrow and collaborators [126] measured EEG potentials after an unexpected forward translation of the support surface in young, old-mobile, and old-frail subjects. They found that in old adults the perturbation-evoked potential was delayed and the vertex-negative component was smaller and bifid. The interval between the two components of the bifid negative peak was longer in a subgroup of old subjects who had also poorer balance. This study shows that old adults, especially those with impaired balance, exhibit delayed and abnormal sensory processing when their posture is perturbed. Other studies have used a warning signal before the support surface translation to inform subjects about an impending perturbation. This paradigm allows the assessment of contingent negative variation (CNV), a negative slow potential reflecting the cortical activity related to the motor preparation process and anticipatory attention directed to the upcoming perturbation. Both young and old adults adapt their CNV over time, although old adults need more repetitions [127, 128]. This adaptation, quantified by a greater negative peak in the late CNV, is probably related to the forward shift in center of pressure as an anticipatory postural adjustment to the forward translation.

In conclusion, there is an age-related reorganization of cortical control of posture similar to that seen in manual tasks, with different brain activation patterns, increased corticospinal excitability, and reduced intracortical inhibition. Interestingly, age does not affect the ability to modulate intracortical inhibition with increasing task difficulty. However, as for old adults the same task can be more difficult than for young adults, the threshold to reduce intracortical inhibition is lower. Lastly, EEG studies have shown that cortical activity related to postural responses to an external perturbation also differs between young and old adults. Cortical responses are delayed and motor preparation processes need more repetitions to develop.

27.9 Age-Related Functional Changes: Deterioration or Compensation?

In the classification model of the different domains of age-related changes in the neuromotor system, Fig. 27.1 shows that the functional changes can appear as deterioration (as a direct result of the structural changes) and compensation (changes in function to counteract the deterioration). An interesting and important topic of discussion is whether or not the described functional changes in postural control are due to deterioration or compensation. For example, old but not young adults down-modulate intracortical inhibition when standing on foam as compared to a rigid surface [17]. Greater down-modulation correlated with greater increases in center of pressure velocity (i.e., worse performance). One interpretation of this finding is that the down-modulation in intracortical inhibition causes the increase in center of pressure velocity, suggesting functional deterioration. Alternatively, old adults with higher center of pressure velocity had a greater need to adjust intracortical inhibition, as a compensation for the greater instability. The latter explanation was supported by subsequent experiments [57], where task difficulty was adjusted to individual skill level, and modulation of EMG suppression, presumably representing intracortical inhibition, was similar between young and old adults. Moreover, leaning closer to the maximum was correlated with greater reductions in EMG suppression; again suggesting that intracortical inhibition decreases in unstable situations. Therefore, assuming that both studies measured similar inhibitory mechanisms, the different modulation in old adults [17] was most likely a compensatory mechanism. Importantly, this discussion exemplifies the need to include at least two of the three domains in the model in order to get a better understanding of the relationships between the different domains.

27.10 Age-Related Functional Changes: Related to Co-contraction?

As in many activities of daily living and laboratory tasks [129], old adults also tend to use more co-contraction among the lower leg muscles when standing, especially when balance is challenged [130, 131]. When fitting a double inverted pendulum model to behavioral data of young and old adults, intrinsic and reflexive stiffness around the ankle joint was found to increase with age [132]. The functional changes described in the section "Age-related changes in spinal control of standing" may be related to this co-contraction. Indeed, on a spinal level, greater presynaptic inhibition was associated with greater co-contraction during standing in old adults [94]. The authors suggest that old adults shift from using muscle afferents to a simpler co-contraction strategy to control standing balance. Whether the functional changes on cortical level contribute to the co-contraction is not yet known and a promising topic for future research.

27.11 Conclusions and Recommendations

Indubitably, holding one's balance and control sway while standing is far from a passive and automated process. Even though the majority of studies focused on the ankle, it is now clear that all three lower extremity joints but especially the hip in addition to the ankle, contribute to the control of sway present in undisturbed standing. A multi-segment inverted pendulum model, for now, seems the most suitable model to predict inter-joint coordination of sway and relies on an intermittent neural controller. Behavioral, TMS, EEG, and imaging data provide convincing evidence for the involvement of M1 in the control of standing posture and that this role becomes more prominent with age and postural task difficulty. Age does not seem to affect the ability to modulate intracortical inhibition with postural challenge. Unraveling the neurophysiological mechanisms underpinning muscle synergies and inter-muscle coupling through the use of imaging, TMS, and other methods will help to better understand how and where in the mechanical and nervous systems do interventions bring about adaptations standing balance in young and old adults and in those with disability. Future studies will determine if it is beneficial and necessary through interventions to reduce the cortical involvement in the control of standing balance in healthy old adults especially if this involvement should increase in old adults with a history of falls.

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References

1. Buckley JP, Hedge A, Yates T, Copeland RJ, Loosemore M, Hamer M, et al. The sedentary office: an expert statement on the growing case for change towards better health and productivity. Br J Sports Med. 2015;49(21):1357–62.
2. Hortobágyi T. The positives of physical activity and the negatives of sedentariness in successful aging. In: Rutgers H, editor. The future of health and fitness. A plan for getting Europe active by 2025. Nijmegen: BlackBox Publishers; 2014. p. 54–62.
3. Papegaaij S, Taube W, Baudry S, Otten E, Hortobagyi T. Aging causes a reorganization of cortical and spinal control of posture. Front Aging Neurosci. 2014;6:28.
4. Taube W, Gollhofer A. Postural control and balance training. In: Gollhofer A, Taube W, Nielsen JB, editors. Routledge handbook of motor control and motor learning. London: Routledge; 2014. p. 252–80.
5. Hsu YS, Kuan CC, Young YH. Assessing the development of balance function in children using stabilometry. Int J Pediatr Otorhinolaryngol. 2009;73(5):737–40.
6. Peterka RJ, Black FO. Age-related changes in human posture control: sensory organization tests. J Vestib Res. 1990;1(1):73–85.
7. Peterson ML, Christou E, Rosengren KS. Children achieve adult-like sensory integration during stance at 12-years-old. Gait Posture. 2006;23(4):455–63.
8. Oba N, Sasagawa S, Yamamoto A, Nakazawa K. Difference in postural control during quiet standing between young children and adults: assessment with center of mass acceleration. PLoS One. 2015;10(10):e0140235.

9. Hytonen M, Pyykko I, Aalto H, Starck J. Postural control and age. Acta Otolaryngol. 1993;113(2):119–22.
10. Kirshenbaum N, Riach CL, Starkes JL. Non-linear development of postural control and strategy use in young children: a longitudinal study. Exp Brain Res. 2001;140(4):420–31.
11. Riach CL, Starkes JL. Visual fixation and postural sway in children. J Mot Behav. 1989;21 (3):265–76.
12. Riach CL, Hayes KC. Maturation of postural sway in young children. Dev Med Child Neurol. 1987;29(5):650–8.
13. Rival C, Ceyte H, Olivier I. Developmental changes of static standing balance in children. Neurosci Lett. 2005;376(2):133–6.
14. Ruffieux J, Keller M, Lauber B, Taube W. Changes in standing and walking performance under dual-task conditions across the lifespan. Sports Med. 2015;45(12):1739–58.
15. Peterka RJ. Sensorimotor integration in human postural control. J Neurophysiol. 2002;88 (3):1097–118.
16. Sveistrup H, Woollacott MH. Longitudinal development of the automatic postural response in infants. J Mot Behav. 1996;28(1):58–70.
17. Papegaaij S, Taube W, Hogenhout M, Baudry S, Hortobagyi T. Age-related decrease in motor cortical inhibition during standing under different sensory conditions. Front Aging Neurosci. 2014;6:126.
18. Baudry S, Collignon S, Duchateau J. Influence of age and posture on spinal and corticospinal excitability. Exp Gerontol. 2015;69:62–9.
19. Kuznetsov NA, Riley MA. The role of task constraints in relating laboratory and clinical measures of balance. Gait Posture. 2015;42(3):275–9.
20. Penzer F, Duchateau J, Baudry S. Effects of short-term training combining strength and balance exercises on maximal strength and upright standing steadiness in elderly adults. Exp Gerontol. 2015;61:38–46.
21. Jacobs JV, Horak FB. Cortical control of postural responses. J Neural Transm. 2007;114 (10):1339–48.
22. Woollacott MH, Shumway-Cook A. Changes in posture control across the life span – a systems approach. Phys Ther. 1990;70(12):799–807.
23. Bugnariu N, Fung J. Aging and selective sensorimotor strategies in the regulation of upright balance. J Neuroeng Rehabil. 2007;4:19.
24. Granacher U, Bridenbaugh SA, Muehlbauer T, Wehrle A, Kressig RW. Age-related effects on postural control under multi-task conditions. Gerontology. 2011;57(3):247–55.
25. Lamoth CJ, van Heuvelen MJ. Sports activities are reflected in the local stability and regularity of body sway: older ice-skaters have better postural control than inactive elderly. Gait Posture. 2012;35(3):489–93.
26. Makizako H, Furuna T, Ihira H, Shimada H. Age-related differences in the influence of cognitive task performance on postural control under unstable balance conditions. Int J Gerontol. 2013;7(4):199–204.
27. Sturnieks DL, St George R, Lord SR. Balance disorders in the elderly. Neurophysiol Clin. 2008;38(6):467–78.
28. Cofre Lizama LE, Pijnappels M, Faber GH, Reeves PN, Verschueren SM, van Dieen JH. Age effects on mediolateral balance control. PLoS One. 2014;9(10), e110757.
29. Plagenhoef S, Evans FG, Abdelnour T. Anatomical data for analyzing human motion. Res Q Exerc Sport. 1983;54(2):169–78.
30. Almeida CWL, Castro CHM, Pedreira PG, Heymann RE, Szejnfeld VL. Percentage height of center of mass is associated with the risk of falls among elderly women: a case–control study. Gait Posture. 2011;34(2):208–12.
31. Morasso PG, Sanguineti V. Ankle muscle stiffness alone cannot stabilize balance during quiet standing. J Neurophysiol. 2002;88(4):2157–62.
32. Suzuki Y, Nomura T, Casadio M, Morasso P. Intermittent control with ankle, hip, and mixed strategies during quiet standing: a theoretical proposal based on a double inverted pendulum model. J Theor Biol. 2012;310:55–79.

33. Casadio M, Morasso PG, Sanguineti V. Direct measurement of ankle stiffness during quiet standing: implications for control modelling and clinical application. Gait Posture. 2005;21 (4):410–24.
34. Loram ID, Lakie M. Direct measurement of human ankle stiffness during quiet standing: the intrinsic mechanical stiffness is insufficient for stability. J Physiol (Lond). 2002;545: 1041–53.
35. Sasagawa S, Shinya M, Nakazawa K. Interjoint dynamic interaction during constrained human quiet standing examined by induced acceleration analysis. J Neurophysiol. 2014;111(2):313–22.
36. van Soest AJ, Rozendaal LA. The inverted pendulum model of bipedal standing cannot be stabilized through direct feedback of force and contractile element length and velocity at realistic series elastic element stiffness. Biol Cybern. 2008;99(1):29–41.
37. Asai Y, Tasaka Y, Nomura K, Nomura T, Casadio M, Morasso P. A model of postural control in quiet standing: robust compensation of delay-induced instability using intermittent activation of feedback control. PLoS One. 2009;4(7):e6169.
38. Fitzpatrick RC, Taylor JL, McCloskey DI. Ankle stiffness of standing humans in response to imperceptible perturbation: reflex and task-dependent components. J Physiol (Lond). 1992;454:533–47.
39. Loram ID, Maganaris CN, Lakie M. Active, non-spring-like muscle movements in human postural sway: how might paradoxical changes in muscle length be produced? J Physiol (Lond). 2005;564:281–93.
40. Masani K, Popovic MR, Nakazawa K, Kouzaki M, Nozaki D. Importance of body sway velocity information in controlling ankle extensor activities during quiet stance. J Neurophysiol. 2003;90(6):3774–82.
41. Morasso PG, Schieppati M. Can muscle stiffness alone stabilize upright standing? J Neurophysiol. 1999;82(3):1622–6.
42. Peterka RJ. Postural control model interpretation of stabilogram diffusion analysis. Biol Cybern. 2000;82(4):335–43.
43. Winter DA, Patla AE, Prince F, Ishac M, Gielo-Perczak K. Stiffness control of balance in quiet standing. J Neurophysiol. 1998;80(3):1211–21.
44. Sasagawa S, Ushiyama J, Masani K, Kouzaki M, Kanehisa H. Balance control under different passive contributions of the ankle extensors: quiet standing on inclined surfaces. Exp Brain Res. 2009;196(4):537–44.
45. Accornero N, Capozza M, Rinalduzzi S, Manfredi GW. Clinical multisegmental posturography: age-related changes in stance control. Electroencephalogr Clin Neurophysiol. 1997;105(3):213–9.
46. Aramaki Y, Nozaki D, Masani K, Sato T, Nakazawa K, Yano H. Reciprocal angular acceleration of the ankle and hip joints during quiet standing in humans. Exp Brain Res. 2001;136(4):463–73.
47. Creath R, Kiemel T, Horak F, Peterka R, Jeka J. A unified view of quiet and perturbed stance: simultaneous co-existing excitable modes. Neurosci Lett. 2005;377(2):75–80.
48. Gunther M, Grimmer S, Siebert T, Blickhan R. All leg joints contribute to quiet human stance: a mechanical analysis. J Biomech. 2009;42(16):2739–46.
49. Gunther M, Putsche P, Leistritz L, Grimmer S. Phase synchronisation of the three leg joints in quiet human stance. Gait Posture. 2011;33(3):412–7.
50. Pinter IJ, van Swigchem R, van Soest AJ, Rozendaal LA. The dynamics of postural sway cannot be captured using a one-segment inverted pendulum model: a PCA on segment rotations during unperturbed stance. J Neurophysiol. 2008;100(6):3197–208.
51. Sasagawa S, Ushiyama J, Kouzaki M, Kanehisa H. Effect of the hip motion on the body kinematics in the sagittal plane during human quiet standing. Neurosci Lett. 2009;450 (1):27–31.
52. Zhang Y, Kiemel T, Jeka J. The influence of sensory information on two-component coordination during quiet stance. Gait Posture. 2007;26(2):263–71.

53. Baudry S, Duchateau J. Independent modulation of corticospinal and group I afferents pathways during upright standing. Neuroscience. 2014;275:162–9.
54. Baudry S, Penzer F, Duchateau J. Input–output characteristics of soleus homonymous Ia afferents and corticospinal pathways during upright standing differ between young and elderly adults. Acta Physiol (Oxf). 2014;210(3):667–77.
55. Klass M, Baudry S, Duchateau J. Modulation of reflex responses in activated ankle dorsiflexors differs in healthy young and elderly subjects. Eur J Appl Physiol. 2011;111 (8):1909–16.
56. Papegaaij S, Baudry S, Négyesi J, Taube W, Hortobágyi T. Intracortical inhibition in the soleus muscle is reduced during the control of upright standing in both young and old adults. Eur J Appl Physiol. 2016;116:959–67.
57. Papegaaij S, Taube W, van Keeken HG, Otten E, Baudry S, Hortobagyi T. Postural challenge affects motor cortical activity in young and old adults. Exp Gerontol. 2016;73:78–85.
58. Taube W, Gruber M, Beck S, Faist M, Gollhofer A, Schubert M. Cortical and spinal adaptations induced by balance training: correlation between stance stability and corticospinal activation. Acta Physiol (Oxf). 2007;189(4):347–58.
59. Tokuno CD, Taube W, Cresswell AG. An enhanced level of motor cortical excitability during the control of human standing. Acta Physiol (Oxf). 2009;195(3):385–95.
60. Hsu WL, Scholz JP, Schoner G, Jeka JJ, Kiemel T. Control and estimation of posture during quiet stance depends on multijoint coordination. J Neurophysiol. 2007;97(4):3024–35.
61. de Lima AC, de Azevedo Neto RM, Teixeira LA. On the functional integration between postural and supra-postural tasks on the basis of contextual cues and task constraint. Gait Posture. 2010;32(4):615–8.
62. Andersson G, Hagman J, Talianzadeh R, Svedberg A, Larsen HC. Effect of cognitive load on postural control. Brain Res Bull. 2002;58(1):135–9.
63. Bottaro A, Yasutake Y, Nomura T, Casadio M, Morasso P. Bounded stability of the quiet standing posture: an intermittent control model. Hum Mov Sci. 2008;27(3):473–95.
64. Kiemel T, Zhang Y, Jeka JJ. Identification of neural feedback for upright stance in humans: stabilization rather than sway minimization. J Neurosci. 2011;31(42):15144–53.
65. van dK, de Vlugt E. Postural responses evoked by platform pertubations are dominated by continuous feedback. J Neurophysiol. 2007;98(2):730–43.
66. Eurich CW, Milton JG. Noise-induced transitions in human postural sway. Phys Rev E Stat Phys Plasmas Fluids Relat Interdiscip Topics. 1996;54(6):6681–4.
67. Loram ID, van dK, Lakie M, Gollee H, Gawthrop PJ. Does the motor system need intermittent control? Exerc Sport Sci Rev. 2014;42(3):117–25.
68. Milton J, Townsend JL, King MA, Ohira T. Balancing with positive feedback: the case for discontinuous control. Philos Trans A Math Phys Eng Sci. 2009;367(1891):1181–93.
69. Kiemel T, Elahi AJ, Jeka JJ. Identification of the plant for upright stance in humans: multiple movement patterns from a single neural strategy. J Neurophysiol. 2008;100(6):3394–406.
70. Rozendaal LA, van Soest AK. Multi-segment stance can be stable with zero local ankle stiffness. J Biomech. 2007;40:S364
71. Kuo AD. An optimal state estimation model of sensory integration in human postural balance. J Neural Eng. 2005;2(3):S235–49.
72. Taube W, Mouthon M, Leukel C, Hoogewoud HM, Annoni JM, Keller M. Brain activity during observation and motor imagery of different balance tasks: an fMRI study. Cortex. 2015;64:102–14.
73. Scott SH. Role of motor cortex in coordinating multi-joint movements: is it time for a new paradigm? Can J Physiol Pharmacol. 2000;78(11):923–33.
74. Gribble PL, Scott SH. Overlap of internal models in motor cortex for mechanical loads during reaching. Nature. 2002;417(6892):938–41.
75. Ting LH, Chiel HJ, Trumbower RD, Allen JL, McKay JL, Hackney ME, et al. Neuromechanical principles underlying movement modularity and their implications for rehabilitation. Neuron. 2015;86(1):38–54.

76. Horak FB, Macpherson JM. Postural orientation and equilibrium. In: Rowell LB, Shepherd JT, editors. Handbook of physiology, Sec 12, exercise: regulation and integration of multiple systems. New York: Oxford University Press; 1996. p. 255, Äì292.
77. Rana M, Yani MS, Asavasopon S, Fisher BE, Kutch JJ. Brain connectivity associated with muscle synergies in humans. J Neurosci. 2015;35(44):14708–16.
78. Magnus R. Physiology of posture. Lancet. 1926;11:531, Äì585.
79. Sherrington CS. Flexion-reflex of the limb, crossed extension-reflex, and reflex stepping and standing. J Physiol. 1910;40(1–2):28–121.
80. Edgerton VR, Roy RR. A new age for rehabilitation. Eur J Phys Rehabil Med. 2012;48 (1):99–109.
81. Deliagina TG, Beloozerova IN, Orlovsky GN, Zelenin PV. Contribution of supraspinal systems to generation of automatic postural responses. Front Integr Neurosci. 2014;8:76.
82. Solopova IA, Kazennikov OV, Deniskina NB, Levik YS, Ivanenko YP. Postural instability enhances motor responses to transcranial magnetic stimulation in humans. Neurosci Lett. 2003;337(1):25–8.
83. Taube W, Schubert M, Gruber M, Beck S, Faist M, Gollhofer A. Direct corticospinal pathways contribute to neuromuscular control of perturbed stance. J Appl Physiol. 2006;101(2):420–9.
84. Soto O, Valls-Sole J, Shanahan P, Rothwell J. Reduction of intracortical inhibition in soleus muscle during postural activity. J Neurophysiol. 2006;96(4):1711–7.
85. Lavoie BA, Cody FW, Capaday C. Cortical control of human soleus muscle during volitional and postural activities studied using focal magnetic stimulation. Exp Brain Res. 1995;103 (1):97–107.
86. Obata H, Sekiguchi H, Ohtsuki T, Nakazawa K. Posture-related modulation of cortical excitability in the tibialis anterior muscle in humans. Brain Res. 2014;1577:29–35.
87. McGinnis SM, Brickhouse M, Pascual B, Dickerson BC. Age-related changes in the thickness of cortical zones in humans. Brain Topogr. 2011;24(3–4):279–91.
88. Ge Y, Grossman RI, Babb JS, Rabin ML, Mannon LJ, Kolson DL. Age-related total gray matter and white matter changes in normal adult brain. Part I: volumetric MR imaging analysis. AJNR Am J Neuroradiol. 2002;23(8):1327–33.
89. Sullivan EV, Pfefferbaum A. Diffusion tensor imaging and aging. Neurosci Biobehav Rev. 2006;30(6):749–61.
90. Angulo-Kinzler RM, Mynark RG, Koceja DM. Soleus H-reflex gain in elderly and young adults: modulation due to body position. J Gerontol A Biol Sci Med Sci. 1998;53(2):M120–5.
91. Koceja DM, Markus CA, Trimble MH. Postural modulation of the soleus H reflex in young and old subjects. Electroencephalogr Clin Neurophysiol. 1995;97(6):387–93.
92. Koceja DM, Mynark RG. Comparison of heteronymous monosynaptic Ia facilitation in young and elderly subjects in supine and standing positions. Int J Neurosci. 2000;103 (1–4):1–17.
93. Kido A, Tanaka N, Stein RB. Spinal excitation and inhibition decrease as humans age. Can J Physiol Pharmacol. 2004;82(4):238–48.
94. Baudry S, Duchateau J. Age-related influence of vision and proprioception on Ia presynaptic inhibition in soleus muscle during upright stance. J Physiol. 2012;590(Pt 21):5541–54.
95. Tsuruike M, Koceja DM, Yabe K, Shima N. Age comparison of H-reflex modulation with the Jendrassik maneuver and postural complexity. Clin Neurophysiol. 2003;114(5):945–53.
96. Horak FB, Diener HC. Cerebellar control of postural scaling and central set in stance. J Neurophysiol. 1994;72(2):479–93.
97. Visser JE, Bloem BR. Role of the basal ganglia in balance control. Neural Plast. 2005;12 (2–3):161–74. discussion 263–72.
98. Drijkoningen D, Leunissen I, Caeyenberghs K, Hoogkamer W, Sunaert S, Duysens J, et al. Regional volumes in brain stem and cerebellum are associated with postural impairments in young brain-injured patients. Hum Brain Mapp. 2015;36(12):4897–909.

99. Choi P, Ren M, Phan TG, Callisaya M, Ly JV, Beare R, et al. Silent infarcts and cerebral microbleeds modify the associations of white matter lesions with gait and postural stability: population-based study. Stroke. 2012;43(6):1505–10.
100. Murray ME, Senjem ML, Petersen RC, Hollman JH, Preboske GM, Weigand SD, et al. Functional impact of white matter hyperintensities in cognitively normal elderly subjects. Arch Neurol. 2010;67(11):1379–85.
101. Rosano C, Aizenstein HJ, Studenski S, Newman AB. A regions-of-interest volumetric analysis of mobility limitations in community-dwelling older adults. J Gerontol A Biol Sci Med Sci. 2007;62(9):1048–55.
102. Cham R, Perera S, Studenski SA, Bohnen NI. Striatal dopamine denervation and sensory integration for balance in middle-aged and older adults. Gait Posture. 2007;26(4):516–25.
103. Beauchet O, Barden J, Liu-Ambrose T, Chester VL, Szturm T, Allali G. The relationship between hippocampal volume and static postural sway: results from the GAIT study. Age (Dordr). 2016;38(1):19-016–9883-4.
104. Goble DJ, Coxon JP, Van Impe A, Geurts M, Doumas M, Wenderoth N, et al. Brain activity during ankle proprioceptive stimulation predicts balance performance in young and older adults. J Neurosci. 2011;31(45):16344–52.
105. Zwergal A, Linn J, Xiong G, Brandt T, Strupp M, Jahn K. Aging of human supraspinal locomotor and postural control in fMRI. Neurobiol Aging. 2012;33(6):1073–84.
106. Reuter-Lorenz PA, Jonides J, Smith EE, Hartley A, Miller A, Marshuetz C, et al. Age differences in the frontal lateralization of verbal and spatial working memory revealed by PET. J Cogn Neurosci. 2000;12(1):174–87.
107. Ward NS, Frackowiak RS. Age-related changes in the neural correlates of motor performance. Brain. 2003;126(Pt 4):873–88.
108. Cabeza R, Anderson ND, Locantore JK, McIntosh AR. Aging gracefully: compensatory brain activity in high-performing older adults. Neuroimage. 2002;17(3):1394–402.
109. Seidler RD, Bernard JA, Burutolu TB, Fling BW, Gordon MT, Gwin JT, et al. Motor control and aging: links to age-related brain structural, functional, and biochemical effects. Neurosci Biobehav Rev. 2010;34(5):721–33.
110. Cliff M, Joyce DW, Lamar M, Dannhauser T, Tracy DK, Shergill SS. Aging effects on functional auditory and visual processing using fMRI with variable sensory loading. Cortex. 2013;49(5):1304–13.
111. Cabeza R, Daselaar SM, Dolcos F, Prince SE, Budde M, Nyberg L. Task-independent and task-specific age effects on brain activity during working memory, visual attention and episodic retrieval. Cereb Cortex. 2004;14(4):364–75.
112. Baudry S, Penzer F, Duchateau J. Vision and proprioception do not influence the excitability of the corticomotoneuronal pathway during upright standing in young and elderly adults. Neuroscience. 2014;268:247–54.
113. Remaud A, Bilodeau M, Tremblay F. Age and muscle-dependent variations in corticospinal excitability during standing tasks. PLoS One. 2014;9(10):e110004.
114. McChesney JW, Woollacott MH. The effect of age-related declines in proprioception and total knee replacement on postural control. J Gerontol A Biol Sci Med Sci. 2000;55(11): M658–66.
115. Aartolahti E, Hakkinen A, Lonnroos E, Kautiainen H, Sulkava R, Hartikainen S. Relationship between functional vision and balance and mobility performance in community-dwelling older adults. Aging Clin Exp Res. 2013;25(5):545–52.
116. Granacher U, Muehlbauer T, Zahner L, Gollhofer A, Kressig RW. Comparison of traditional and recent approaches in the promotion of balance and strength in older adults. Sports Med. 2011;41(5):377–400.
117. Moreland JD, Richardson JA, Goldsmith CH, Clase CM. Muscle weakness and falls in older adults: a systematic review and meta-analysis. J Am Geriatr Soc. 2004;52(7):1121–9.
118. Serrador JM, Lipsitz LA, Gopalakrishnan GS, Black FO, Wood SJ. Loss of otolith function with age is associated with increased postural sway measures. Neurosci Lett. 2009;465 (1):10–5.

119. Smith AE, Ridding MC, Higgins RD, Wittert GA, Pitcher JB. Cutaneous afferent input does not modulate motor intracortical inhibition in ageing men. Eur J Neurosci. 2011;34 (9):1461–9.
120. Smith AE, Ridding MC, Higgins RD, Wittert GA, Pitcher JB. Age-related changes in short-latency motor cortex inhibition. Exp Brain Res. 2009;198(4):489–500.
121. Stevens-Lapsley JE, Thomas AC, Hedgecock JB, Kluger BM. Corticospinal and intracortical excitability of the quadriceps in active older and younger healthy adults. Arch Gerontol Geriatr. 2013;56(1):279–84.
122. McGinley M, Hoffman RL, Russ DW, Thomas JS, Clark BC. Older adults exhibit more intracortical inhibition and less intracortical facilitation than young adults. Exp Gerontol. 2010;45(9):671–8.
123. Kossev AR, Schrader C, Dauper J, Dengler R, Rollnik JD. Increased intracortical inhibition in middle-aged humans; a study using paired-pulse transcranial magnetic stimulation. Neurosci Lett. 2002;333(2):83–6.
124. Bashir S, Perez JM, Horvath JC, Pena-Gomez C, Vernet M, Capia A, et al. Differential effects of motor cortical excitability and plasticity in young and old individuals: a Transcranial Magnetic Stimulation (TMS) study. Front Aging Neurosci. 2014;6:111.
125. Kantak SS, Wittenberg GF, Liao WW, Magder LS, Rogers MW, Waller SM. Posture-related modulations in motor cortical excitability of the proximal and distal arm muscles. Neurosci Lett. 2013;533:65–70.
126. Duckrow RB, Abu-Hasaballah K, Whipple R, Wolfson L. Stance perturbation-evoked potentials in old people with poor gait and balance. Clin Neurophysiol. 1999;110 (12):2026–32.
127. Fujiwara K, Maekawa M, Kiyota N, Yaguchi C. Adaptation changes in dynamic postural control and contingent negative variation during backward disturbance by transient floor translation in the elderly. J Physiol Anthropol. 2012;31:12-6805–31-12.
128. Maekawa M, Fujiwara K, Kiyota N, Yaguchi C. Adaptation changes in dynamic postural control and contingent negative variation during repeated transient forward translation in the elderly. J Physiol Anthropol. 2013;32:24-6805–32-24.
129. Hortobagyi T, Devita P. Mechanisms responsible for the age-associated increase in coactivation of antagonist muscles. Exerc Sport Sci Rev. 2006;34(1):29–35.
130. Benjuya N, Melzer I, Kaplanski J. Aging-induced shifts from a reliance on sensory input to muscle cocontraction during balanced standing. J Gerontol A Biol Sci Med Sci. 2004;59 (2):166–71.
131. Nagai K, Yamada M, Uemura K, Yamada Y, Ichihashi N, Tsuboyama T. Differences in muscle coactivation during postural control between healthy older and young adults. Arch Gerontol Geriatr. 2011;53(3):338–43.
132. Engelhart D, Pasma JH, Schouten AC, Aarts RG, Meskers CG, Maier AB, et al. Adaptation of multijoint coordination during standing balance in healthy young and healthy old individuals. J Neurophysiol. 2016;115(3):1422–35.
133. Rivner MH, Swift TR, Malik K. Influence of age and height on nerve conduction. Muscle Nerve. 2001;24(9):1134–41.
134. Mattay VS, Fera F, Tessitore A, Hariri AR, Das S, Callicott JH, et al. Neurophysiological correlates of age-related changes in human motor function. Neurology. 2002;58(4):630–5.
135. Nardone A, Siliotto R, Grasso M, Schieppati M. Influence of aging on leg muscle reflex responses to stance perturbation. Arch Phys Med Rehabil. 1995;76(2):158–65.
136. McGregor KM, Zlatar Z, Kleim E, Sudhyadhom A, Bauer A, Phan S, et al. Physical activity and neural correlates of aging: a combined TMS/fMRI study. Behav Brain Res. 2011;222 (1):158–68.
137. Rovio S, Spulber G, Nieminen LJ, Niskanen E, Winblad B, Tuomilehto J, et al. The effect of midlife physical activity on structural brain changes in the elderly. Neurobiol Aging. 2010;31 (11):1927–36.

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