4 Gait in Vascular Cognitive Impairment

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4.1 Introduction

Gait function is an important factor in maintaining activities of daily living (ADL) in patients with older age or cognitive impairment. Abnormalities in gait and balance are found in about 35% of elderly over the age 70. Falls due to gait disturbances induce severe injury including fractures and traumatic brain injuries to the elderly, thereby increasing health costs and further promoting cognitive decline [1]. People with gait disturbances also show a faster rate of cognitive decline. However, there is not much research on gait compared to cognitive function. Their clinical features differ according to the degree and characteristics of the vascular insult. For example, when a brain lesion occurs in a nigral or brain stem region, parkinsonism can appear but unilateral, and gait disturbances are not prominent. White matter hyperintensities (WMH), on the other hand, is known to cause slow progression of gait disturbance [2]. Therefore, in this chapter, we describe the characteristics of gait [3] in vascular cognitive impairment and consider its pathophysiological mechanisms.

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4.1.1 Gait Cycle (Fig. 4.1)

- Right heel strike: gait cycle begins when one heel strikes the ground.
- Left toe off: supported by the stance of the right leg, body weight shifts forward as the left leg flexes at the hip and knees.
- Left leg swing: left leg swings forward.
- Left heel strike: left heel strikes the ground.
- Right toe off: weight then shifts forward onto the left leg, right leg flexes at the hip and knees.
- Right leg swing: right leg swings forward.
- Right heel strike: again, the right heel strikes the ground.

4.1.2 Examination of Gait

Observe individuals as they walk in a straight line and note if there is any difficulty in rising from a chair, initiating gait, or turning.

- Record the components below:
  - Velocity: distance covered in a given time
  - Cadence: steps per minute
  - Stride length: distance covered by the gait cycle
  - Step length: distance covered during the swing phase of a single leg
  - Step width or base: distance between the left and right feet while walking

S.-H. Lee, J.-S. Lim (eds.), *Stroke Revisited: Vascular Cognitive Impairment*, Stroke Revisited,
https://doi.org/10.1007/978-981-10-1433-8_4

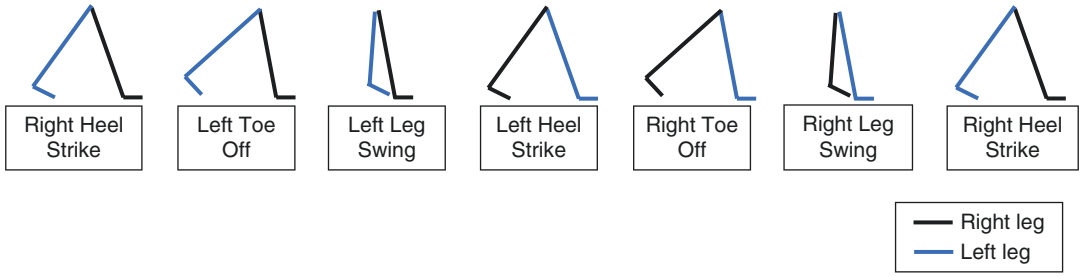

Fig. 4.1 Gait cycle

- Observe posture, arm swing, the height of each step, leg stiffness, or side-to-side lurching.
- Check muscle strength and tone in the legs, sensation, and reflexes.
- Test for the Romberg sign, tandem gait, and heel or toe walking.

4.2 Relationship Between Gait and Cognition

Decreases in gait are associated with declines in cognitive function [4] and might be an early sign of dementia [5]. Similarly, a decrease in gait velocity could predict persistent cognitive impairment [6]. Unsteady gait, frontal gait, and hemiparetic gait are related to the risk of vascular dementia in particular [7].

4.2.1 Cognitively Normal Elderly

Executive function is associated with decreased gait velocity, slower pace, reduced cadence, and gait variability. Especially, the degree of stride time variability, which is a sensitive marker of gait stability, is related to executive dysfunction. Immediate memory is linked to gait dysfunction and falls. Worse memory function is associated with reduced cadence and impaired gait velocity in single and dual task conditions. A meta-analysis showed there is also a correlation between faster gait speed and better performance on memory tests. However, the relationship between memory function and gait ability is not consistent. Visuospatial function is correlated with gait velocity and double support phase variability, which is associated with fall risk. Slower processing speed is associated with poor performance on many gait measures, including gait velocity, rhythm, step time, step length, and double support phase variability [8].

4.2.2 Cognitively Impaired Elderly

Executive dysfunction is associated with reduced gait speed and increased variability in patients with mild cognitive impairment (MCI) and Alzheimer's disease (AD). Only the executive function component of cognitive function is associated with a longitudinal decrease in gait speed. Lower episodic memory is associated with increased dual task cost in both patients with amnestic and non-amnestic MCI, which represents deterioration in motor performance [8].

4.2.3 Stroke Patients

Patients with TIA or minor stroke show worse performance on turn time (increases), step length, gait speed (slower), and double support (prolonged). That means people with TIA or minor stroke have gait and balance dysfunction despite having no obvious physiological impairments [9]. The problem is poststroke patients with gait and balance problems show an increased risk of developing cognitive dysfunction. This relationship can be explained by the hypothesis that deficits in attention, executive function, and motor processing functions induce gait dysfunction in stroke patients [10–12].

Therefore, gait speed and other motor signs can be used as predictors of future cognitive impairment development [10, 13].

On the other hand, stroke patients present with hemiparesis including foot drop, which induces poor ambulation [14]. Poor ambulation is also associated with greater cognitive impairment.

4.3 Neural Basis of Gait Disturbance in Cognitive Impairment

4.3.1 Hippocampus

Hippocampal atrophy is mainly associated with memory dysfunction, but the hippocampus is also strongly associated with gait. The hippocampus contributes to sensorimotor integration that combines internally and externally generated sensory information into voluntary motor activity [15]. Internally and externally motor-related sensory information are related to the head direction essential for spatial orientation and navigation [16]. The hippocampus also forms the orientation of the body in space and incorporates visual, vestibular and proprioceptive sensory signals, and contextual information into the spatial map [17, 18]. The hippocampus is functionally correlated with the prefrontal cortex, mediated through the entorhinal cortex and the nigrostriatal system.

4.3.2 Prefrontal Cortex

The prefrontal cortex is known as an important area for executive function including attention and working memory. The prefrontal cortex receives information from virtually all sensory systems and has preferential connections with motor processing structures. It has an important role in the cognitive control of motor performance [19]. Its function is enhanced during arousal, and it is vulnerable to normal aging. The prefrontal cortex is additionally involved in motor performance in the elderly. The elderly are more likely to depend on bilateral activation of the frontal cortices during motor performance [20]. The prefrontal cortex has a role in gait through its connections to the striatum and hippocampus.

4.3.3 Periventricular White Matter

In the periventricular white matter, there are circuits that connect to distant parts of the brain. They include cortico-cortical circuits, such as the fronto-hippocampal circuit, and cortico-subcortical circuits, such as the fronto-striatal system. Cognitive impairment due to disconnection of periventricular white matter could be a risk factor for gait disturbances. For example, patients with impaired executive function including divided attention cannot safely cross a busy street by foot [21].

4.3.4 Corpus Callosum

The genus of the corpus callosum connects to the prefrontal cortex and has a role in the cognitive function required to prepare motor responses [22, 23]. The splenium of the corpus callosum is connected to the superior parietal and occipital cortices that are important for the integration and interhemispheric transfer of visual and somatosensory inputs [23].

4.3.5 Cerebellar Peduncle

The superior cerebellar peduncle is associated with gait. The superior cerebellar peduncle has predominantly efferent projections to the premotor and primary motor cortices. In addition, it has a role in mental rehearsal of movement and motor learning. Whereas, the inferior cerebellar peduncle includes both cerebellar efferent and afferent fibers to and from the vestibular nuclei that carry information about eye movements and the orientation of the head and body as well as afferent spinal fibers carrying ipsilateral proprioceptive information important for posture, locomotion, and muscle tone control. It is associated with mobility impairment but not with gait. The middle cerebellar peduncle contains connections

from the pontine nuclei to the cerebellum carrying information from the cerebral cortex, mostly from the motor and somatosensory areas. However, the middle cerebellar peduncle is not associated with mobility impairment [24].

4.3.6 Cingulum

The cingulum is frequently associated with slowing of gait. The cingulum is interconnected with the cingulate gyrus and the dorsal and medial prefrontal cortices, which have a role in converting short-term memory to long-term memory and cognitive function [25]. The cingulum affects gait through reduced cognitive function rather than as a direct effect [26].

4.4 Gait Disturbances Associated with Small Vessel Disease

4.4.1 White Matter Hyperintensities (WMH)

WMH burden is associated with gait disorders. WMH has an effect on gait, according to location and severity. Especially, white matter lesions in the frontal lobe, the centrum semiovale, the posterior limb of the internal capsule, and the genu and splenium of the corpus callosum are related to gait dysfunction [27]. WMH is also associated with low cerebellar volume [28]. There is also a dose-effect relation between WMH severity and gait velocity [28]. A longitudinal study showed both baseline WMH and WMH progression predict increased fall risk [29, 30]. A population-based study showed that the relationship between WMH burden and gait function is constant in both middle and late ages [28].

WMH is linked to gait velocity, stride length, and step width [27]. Another study reported that WMH was associated with most spatiotemporal gait parameters and was borderline significant for variability in stride length. Especially, WMH in the sub-lobar (basal ganglia, thalamus, internal and external capsule, insula), limbic areas, and frontal lobe were related to a lower gait velocity, due to a shorter stride length [28]. However, the association between WMH and cadence was not significant [26]. This suggested that cadence was less influenced by WMH than stride length, and it was a similar feature seen in Parkinson's disease and normal pressure hydrocephalus.

Periventricular WMH predominantly located in the frontal lobe was associated with lower gait velocity, shorter stride length, and broader stride width. Frontal WMH contained fibers connected to bilateral prefrontal cortex [26]. Periventricular WMH was more associated with a clinical rating gait scale than deep WMH [31].

Deep WMH were associated with dual task gait speed, and this relationship was mediated by global cognition and executive function [32]. Increased WMH volumes might result in a disruption to the deep frontal-subcortical neuronal networks that interconnect various cortical areas, which is likely to have a direct impact on gait [33].

4.4.2 Lacunar Infarction

Lacunar infarction is also associated with slower gait and a lower volume of supratentorial white matter [28]. Similar to WMH, lacunar infarction location and severity influence gait. Lacunar infarcts in the thalamus and frontal lobe are associated with lower gait velocity due to, respectively, a shorter stride length and lower step frequency. Lacunar infarction in the brain stem is also related to lower cadence [34]. There is a dose-effect relation between the severity of lacunar infarcts and gait velocity. Like WMH, lacunar infarction is also independently associated with most spatiotemporal gait parameters and is borderline significantly associated with stride length variability [28].

4.4.3 Cerebral Microbleeds (CMB)

A higher number of CMB are associated with lower gait performance. CMB are independently related to shorter stride length and borderline

significantly associated with a longer double-support percentage. CMB in the frontal and temporal lobe and basal ganglia are significantly related to shorter stride length. And CMB in the temporal lobe shows an association with lower gait velocity. CMB in the thalamus are also related to gait [35].

4.5 Gait Disturbance Associated with Vascular Cognitive Impairment and Vascular Dementia

4.5.1 Vascular Cognitive Impairment

Subcortical VCI is associated with reduced cadence, increased variability of single and double support times, and a reduced single support phase [36]. Patients with vascular cognitive impairment no dementia (VCIND), a preclinical stage of SVaD, walk more slowly and have lower static balance [37]. They show impaired dynamic balance performance, rigidity, and bradykinesia [38]. Combined with this feature, VCIND patients show gait disturbances and gait-related motor impairments.

4.5.2 Vascular Dementia

Compared to AD, vascular dementia (VD) patients show slower gait velocity and reduced step length. In a study, 79% of patients with VD showed gait and balance disorders, while 25% of patients with AD showed gait and balance disorders [39]. In a longitudinal study, all types of dementia patients showed a decline in mobility, but the progression rate was faster in VD than in AD. A faster physical decline was also observed in patients with the fastest dementia progression [40].

In SVaD, both lacunar and Binswanger types showed a gait disturbance classified as frontal gait [39]. Its manifestation was walking with a wide base, decreased velocity and step length, static and dynamic instability, truncal ataxia, disturbance in gait initiation, shuffling, and apractic-atactic gait. More severe disease was associated with more prominent gait changes [41].

4.6 Association Between Gait Disturbance and Falls in Cognitively Impaired Patients

Both cognitive dysfunction and gait disturbance are independent risk factors for falls. AD and VD patients fall more frequently than cognitively normal elderlys [42]. Interference under dual task conditions is a predictive marker for falls [43]. In cognitively normal elderlys, executive dysfunction is associated with a high risk of falling [44]. Attention, processing speed, and visuospatial performance are also related. In MCI patients, executive function and visuospatial function are linked to higher fall risk [45]. Among gait parameters, slower gait velocity and reduced stride length are the predictors of falls [46].

4.7 Treatment

4.7.1 Physical Activity

Physical activity is associated with reduced dementia risk. Physical activity improves cognitive function and gait in cognitively normal elders [47]. It also has a beneficial effect on cognition in MCI patients, while the results in dementia patients are less consistent. Moreover, there is insufficient evidence that exercise is effective in patients with VCI. Research on the effect of physical activity on gait is less frequent than on cognitive function. Larger and good quality studies are needed to identify the beneficial effects of physical activity on gait [8].

4.7.2 Cognitive Training

Several studies have shown that cognitive interventions, mainly based on dual task training, improve gait function. However, as previous

studies had small sample sizes and tasks were heterogenous, it is difficult to confirm this effect. The mechanisms and effects of cognitive training are still unclear. More systematic research is required [8].

4.7.3 Medications Used in Alzheimer's Disease

Both cholinesterase inhibitors and memantine have a lack of evidence. In AD, several studies of cholinesterase inhibitors showed gait improvement. However, its effect on VD patient gait is unclear [8].

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