REVIEW ARTICLE

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The Relationship between Saccades and Locomotion

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ABSTRACT

Human locomotion involves a complex interplay among multiple brain regions and depends on constant feedback from the visual system. We summarize here the current understanding of the relationship among fixations, saccades, and gait as observed in studies sampling eye movements during locomotion, through a review of the literature and a synthesis of the relevant knowledge on the topic. A significant overlap in locomotor and saccadic neural circuitry exists that may support this relationship. Several animal studies have identified potential integration nodes between these overlapping circuitries. Behavioral studies that explored the relationship of saccadic and gait-related impairments in normal conditions and in various disease states are also discussed. Eye movements and locomotion share many underlying neural circuits, and further studies can leverage this interplay for diagnostic and therapeutic purposes.

Key Words
Gait; posture; saccade; fixation; locomotion; deep brain stimulationpe.

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INTRODUCTION

Visual information from the environment is gathered through quick eye movements, which consist of a series of saccades and fixations. Saccades align the fovea with an object of interest.[1] Once an object is foveated, it is held stationary during a fixation, allowing time for the visual information to be collected.[1] Efficient locomotion is dependent upon visual information that is gathered by these quick eye movements. Understanding the relationship among fixations, saccades and locomotion may provide insight into how these seemingly parallel and potentially integrated systems work together.

When studied independently, saccadic and locomotor parameters (Table 1) can be measured precisely. It is difficult to reach the same level of precision when measuring both parameters simultaneously. To get around this, most studies in the literature have correlated saccadic eye movement or gait-related parameters[2,3] with a given disease state or functional impairment.

In this review, we explore the literature for correlations made between saccades and locomotion. We present the neural circuitry of saccadic and gait-related circuitry and the similarities between them. We highlight brain regions that have been found in animal studies that potentially integrate these two networks. Lastly, we review neurodegenerative diseases that manifest saccadic and gait-related impairments.

NEURAL COMPONENTS OF SACCADES, FIXATIONS AND LOCOMOTION

Fixation, saccades and locomotion are served by specific areas and networks of the brain. It is particularly interesting to compare the neural components of saccades and locomotion because there are many overlapping brain areas, suggesting a potential integrated neural network between them.

The most relevant areas that support neural integration between saccades and locomotion would likely be at the level of the brainstem and the cerebellum (Figure 1). Afferent inputs between these two parallel networks differ greatly, in that spinal cord pathways provide the majority of sensory information for locomotion, while the geniculate and extrageniculate pathways are important for saccades. On the other hand, modulating structures such as the cerebral cortex, basal ganglia and thalamus are common to all sensorimotor networks and are nonspecific to locomotion and saccades. Saccades and locomotion are primitive functions, are well-developed in lower species,[4,5] and are more likely to be preserved in primitive integrating brain areas, such as the brainstem and cerebellum, more specifically, the mesopontine tegmentum and the cerebellar vermis.

Table 1. Eye movement/fixation parameters and gait/balance parameters

Saccadic/fixation parameters[2]	
Fixation duration	Duration of time that the eyes remain fixated. Measured in milliseconds to seconds.
Saccadic duration	Duration of time between saccadic initiation and the saccadic endpoint.
Saccadic latency	Time taken for the eyes to move (saccade) after the target appears. Measured in milliseconds or seconds.
Saccadic amplitude	Arc distance of rotational movement made during a saccade. Sometimes called saccadic size. Measured in degrees or minutes.
Saccadic peak velocity	During a saccade, it is the highest velocity attained. Measured in degrees/seconds.
Saccadic intrusions	Series of irregular interruptions by fast eye movements during primary fixation.
Saccadic gain	Ratio of the actual saccadic amplitude over the intended saccadic amplitude.
Main sequence	Relationship among saccadic peak velocity, duration and amplitude.
Gait/balance parameters[3]	
Step length	Distance between initial ground contact of one foot and initial ground contact of the opposite foot.
Step time	Time in seconds between initial ground contact of one foot and initial ground contact of the opposite foot.
Step width	Lateral distance between the centers of the heels when both feet are on the ground (i.e., double stance).
Stride length	Distance between initial ground contact of one foot and initial ground contact of the same foot, constituting the distance of one gait cycle.
Stride time	Time between initial ground contact of one foot and initial ground contact of the same foot, constituting the time of one gait cycle.
Postural sway	Horizontal movement of the center of gravity while standing still.
Swing phase	Remaining 40% of the gait cycle, when the foot no longer is in contact with the ground, spanning from initial swing phase to initial contact.
Cadence	Steps per minute.
Stance phase	Initial 60% of the gait cycle, when the foot is in contact with the ground, spanning from initial contact to terminal double stance.

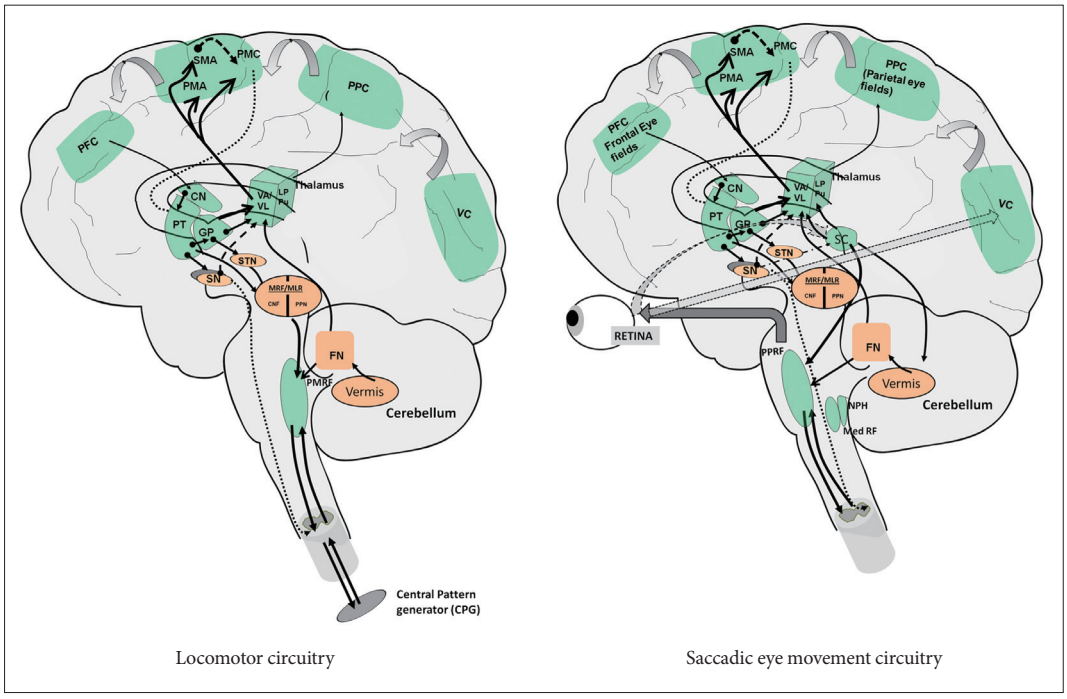

Figure 1. Brain areas involved in saccades/fixations and locomotor activities. Possible integration areas are shaded in orange. PFC: prefrontal cortex, PMA: premotor cortex, SMA: supplementary motor cortex, PMC: primary motor cortex, PPC: posterior parietal cortex, PT: putamen, CN: caudate nucleus, GP: globus pallidus, STN: subthalamic nucleus, SN: substantia nigra, SC: superior colliculus, MLR: mesencephalic locomotor region, PMRF: pontomedullary reticular formation, MRF: mesencephalic reticular formation, FN: fastigial nucleus, NPH: nucleus prepositus hypoglossi, MedRF: medullary reticular formation, PPRF: paramedian pontine reticular formation, VC: visual cortex, VA: ventral anterior, VL: ventrolateral nucleus, PPN: pedunculopontine nucleus.

Mesencephalic Locomotor Region/ Mesencephalic Reticular Formation

The pedunculopontine nucleus (PPN) and the cuneiform nucleus (CNF) make up the mesencephalic locomotor region (MLR),[6] also known as the mesencephalic reticular formation (MRF). The MLR/MRF is involved in eye movement-related activity.[7-9] In addition, it promotes locomotion through the reticulospinal pathways[10] and influences postural tone and locomotor rhythmicity.[11-13] In animal studies, stimulation of the CNF has been found to be associated with locomotor initiation, while stimulation of the PPN was associated with locomotor suppression.[14] The PPN contains cholinergic, glutaminergic and GABAergic neurons; the cholinergic neurons are those closely associated with locomotion.[15] PPN cholinergic neurons are also associated with rapid eye movements in sleep.[15] The PPN directly innervates the motor neurons involved in eye movements and receives direct projections from the frontal and supplementary eye fields in the cortex.[16-20] Neuronal recordings of the PPN in primates have shown different firing patterns during fixations and saccades.[21,22] The PPN receives input from the cerebral cortex and has reciprocal connections with components of the basal ganglia, namely, the substantia nigra [both the substantia nigra pars reticulata (SNr) and the substantia nigra pars compacta (SNc)], globus pallidus and subthalamic nucleus (STN).[23-28]

Superior colliculus

The superior colliculus (SC) receives inputs from the retina and visual cortex (VC).[29-32] Neurons in the SC have projections to saccade generators in the brainstem.[33] The SC has been reported to be associated with fixation- and saccade-related activity.[34-36] There is no evidence for locomotor function related to the SC; however, the SC does receive afferents from various subcortical structures common to the locomotor network, such as the SNr, pretectum, and other nuclei in the pons and medulla. SC efferents project to the thalamus, MLR/MRF, paramedian pontine reticular formation (PPRF), cerebellar locomotor region and cerebellar vermis.[37] The PPRF is important for coordinating horizontal saccadic eye movements, but its role in locomotion has not yet been ex-

plored. The PPRF receives input from the frontal eye fields (FEF) through the contralateral SC[38] and contains burst neurons that generate horizontal saccades.[39-41]

Pontomesencephalic reticular formation

Reticulospinal neurons in the pontomesencephalic reticular formation are involved in controlling and maintaining head movements and in generating the quick phase of vestibular and optokinetic head nystagmus toward the same side.[42] Omnidirectional pause neurons (OPNs) are inhibitory interneurons in the pontomesencephalic reticular formation that are thought to stabilize fixations and saccades in the horizontal, vertical and oblique directions. OPNs are tonically active during fixations and are silent (i.e., "paused") during saccades.[43] Dysfunction in OPNs is thought to result in fixational instability, with reports of macrosaccadic oscillations, saccadic dysmetria, ocular flutter, and opsoclonus.[44,45] The pontomesencephalic reticular formation is also involved in transmitting locomotor signals to central pattern generators in the spinal cord[46] and in controlling balance, locomotion and posture.[47,48]

Cerebellar vermis

The cerebellum is involved in both locomotion[49-54] and saccades.[55-65] The fastigial nucleus (FN) of the cerebellum receives input from the vermis, which in turn receives input from the SC through the nucleus reticularis tegmenti pontis.[55,66,67] Brainstem saccade generators are driven by the FN and the vermis.[41] Studies of transcranial magnetic stimulation directed toward the cerebellar vermis have demonstrated that this area coordinates saccades ipsilateral to the side of stimulation.[68] Neuronal discharge in the FN, also known as the cerebellar locomotor region, is linked to coding of proximal movement during locomotion.[55,69] The FN is thought to act as a pacemaker during locomotion[70] and projects to the pontomedullary reticular formation in the brainstem.

Thalamus

The thalamus serves as the major relay between cortical and subcortical saccadic generators.[71-73] The internal medullary lamina, a myelinated area that divides the thalamus into the anterior, medial and lateral masses, contains nuclei that relay information among multiple areas that control saccades, namely, the frontal and parietal eye fields, SC, PPRF, striatum, cerebellum and the lateral geniculate nuclei.[71]

The lateral geniculate nuclei and pulvinar are two thalamic nuclei in the ventrolateral area that specifically process visual input. The lateral geniculate nucleus projects information from the retina to the VC. Connections between the SC and the lateral geniculate nucleus contribute to saccades that are involved in foveating objects of interest with a high degree of resolution (e.g., facial recognition).[74] The pulvinar has connections between the SC and visual cortices and is involved in visuospatial attention to areas in the visual field.[75] The pulvinar is an important relay for generating saccades toward visual targets or reflexive saccades toward or away from stimuli, and this nucleus influences visually guided behavior, including locomotion. It has been speculated that visual and motor information may be integrated in the pulvinar, allowing a distinction between changes in the visual environment caused by external sources versus self-generated visual motion (caused by eye movements or locomotion).[74]

The ventrolateral nucleus (VL) receives all major saccade-generating afferents in the brainstem and cerebellum and projects to the frontal eye field and the supplementary eye field.[76] Similar to the pulvinar, the VL is closely involved in visually guided saccades.[77] The VL is also a major afferent to the primary motor cortex, and it is not surprising that this region is important for locomotion.[78,79]

The thalamic reticular nucleus is a thin capsule of inhibitory GABAergic neurons that surrounds the dorsolateral thalamus and functions to modulate thalamocortical and corticothalamic signals for a multitude of functions.[80] In terms of saccadic and locomotor networks, this region functions as an inhibitory modulator. The thalamic reticular nucleus sends reciprocal inhibitory signals to the lateral geniculate nucleus in response to saccade-related visual perturbations to maintain a stable image.[81] Recordings have revealed phasic bursts of activity in reticular neurons within the receptive field of distal limbs during walking tasks that are thought to fine tune ongoing locomotor activity.[82]

Basal ganglia

The basal ganglia refers mainly to the caudate and the putamen, which consist of the striatum, globus pallidus, substantia nigra and STN. The nigrostria-

tal pathway modulates the striatum, affecting all motor output, and is not specific to saccadic or locomotor control, though its influence over these functions is considerable.[83-87] The STN receives inputs from the cortex via the striatum and the globus pallidus externa (GPe) through the indirect pathway and direct connections from the cortex through the hyperdirect pathway.[88] The STN receives inputs from the brainstem, thalamus and cortex. Efferents from the STN travel mainly to the GPi and SNr.[89-91] There is evidence that patients with Parkinson disease (PD) who receive deep brain stimulation (DBS) of the STN experience a significant improvement in both saccadic performance[92,93] and locomotion[93-95] compared to patients that receive other DBS targets, such as the globus pallidus interna (GPi). GPi DBS has been shown to improve locomotion,[96] but there is less evidence supporting an improvement in saccadic performance,[97] though one study found improvement in antisaccades.[98]

ANIMAL STUDIES EXPLORING THE INTEGRATION BETWEEN EYE MOVEMENT AND LOCOMOTOR CIRCUITRY

Thus far, we have identified brain areas that are common to both saccades and gait in humans. Animal studies have provided much of the direct evidence for the integration of networks controlling saccades and gait.

Semi-intact experiments in lampreys undergoing electrical stimulation of the optic tectum have demonstrated a stimulus-dependent coordination of eye movements with steering and goal-directed behavior. Saitoh et al.[99] showed that, with increasing stimulation of the lateral optic tectum, there is a stepwise recruitment of eye movements, followed by a coordinated lateral bending of the body, and then by coordinated locomotor movements. Stimulating other areas, such as the caudomedial tectum, elicits different behaviors, such as struggling behavior, characterized by undulating body movements with antiphasic eye movements. These experiments have lent support for the role of the optic tectum (SC in primates) as a stepwise integrating interface for patterned visuomotor and locomotor behavior.[99]

The coordination between eye movements and spinal locomotor patterns is also preserved and adaptable at different stages of development. Uckerman et al.[100] demonstrated how the Xenopus laevis (XL) frog adapts visuomotor control to maintain image stabilization when swimming as it transitions from a tadpole to an adult frog. In the tadpole, propulsion is achieved with undulating tail movements, requiring conjugate left-right eye rotations to maintain a stable binocular image. In the frog, forward acceleration is achieved with rhythmic bilateral leg kicking that requires nonconjugate, convergent-divergent, eye movements. In fixed-head preparations, a strict 1:1 relationship was found between eye movements and spontaneous fictive swimming movements. Vestibular and visual input were controlled for by transecting the optic nerves and ablating the vestibular end organs. In tadpoles, the eyes rotate laterally, countering each lateral tail movement, while in frogs, the eyes converge or diverge in phase with the kick cycle. This experiment provided evidence for multimodal integration between spinal central pattern generators and eye movements during locomotion in XL. More importantly, the ability of visuomotor and locomotor networks to change in a coordinated fashion at different stages of development in XL suggested that they are integrated. This adaptability is probably evolutionarily preserved in other forms of locomotion, such as quadrupedal and bipedal ambulation. The OPN, as mentioned earlier, coordinates horizontal, vertical and oblique fixations and saccades. It is possible that the omnidirectional stabilizing capability of these interneurons provides a mechanism for the adaptability of reflexive saccades to different locomotive head perturbations across species.

Schwarz et al.[101] performed microelectrode recordings of nondopaminergic SNr neurons in cats as they received different sensory stimuli, such as mechanical skin stimulation, passive and active limb movement, and visual and vestibular stimuli. Neurons within the receptive field of each limb showed regular discharge patterns that were in phase with the step cycle during locomotion. Avoiding or navigating around an obstacle had the greatest effect on neuronal firing rates. Objects moving within the contralateral visual field modulated the firing rates of a small population of neurons related to saccades. Similar findings of saccades and neuronal discharge in the SNr have been found in monkeys.[102] The authors concluded that the SNr functions as an output

station that processes convergent multimodal sensory input (e.g., joint position, limb movement, direction and amplitude of saccades) and fine tunes spinal motor output to adequately address changing environments.

The PPN has also been suggested to serve as a multimodal integrative interface.[103] Suppression of spontaneous locomotion and rhythmic eye movements was observed with stimulation of the ventral PPN in anesthetized and acutely decerebrated cats.[14]

Saccade-related[104-106] and locomotion-related[107-109] neuronal activity has been reported in Purkinje cells in the cerebellar vermis in various studies using microstimulation and optogenetic techniques in non-human primates and other mammals.

The SC and PPRF[110,111] have been shown in rhesus monkeys to influence coordinated head-eye movements, an important component of steering during locomotion.[112]

Saleem et al.[113] showed that, in order for mice to accurately gauge their speed when navigating their environment, visual speed, derived from optic input, and running speed, derived from proprioceptive input, are integrated and encoded with weighted sums within the neurons of the V1 area of the occipital cortex. While this does not pertain to eye movements per se, it at least provides more evidence linking visual sampling (which requires adequate saccades and fixations) and locomotion.

While numerous studies have suggested a multimodal integration between saccades and locomotion, the challenge of establishing a neural basis for this interaction, especially in humans, is hindered by the technical limitations related to studying the circuitry of eye movements during the act of walking. Therefore, the level at which these circuits interact with each other in real time and how activating or inactivating various nodes within one neural circuit may affect the functions of the other are not yet known.

BEHAVIORAL STUDIES IN HEALTHY INDIVIDUALS EXPLORING THE RELATIONSHIP AMONG SACCADES, FIXATIONS AND LOCOMOTION

During ambulation, the limbs, body, head and eyes move in a coordinated manner.[112,114,115] Saccadic eye movements allow the fovea to maintain fixations on relevant objects in the environment in a dynamic manner to allow guidance of locomotion. Any problems in this fixation-saccade strategy may lead to visual and gait impairments.

The visuomotor and locomotor systems influence each other via a continuous feedback loop, though the exact network is not well delineated.[116-118] Several studies have focused on gaze fixations and saccadic eye movements during stepping[119-125] to describe how eye movements influence gait parameters. In one study, visual information gathered during the latter half of the preceding step was shown to influence the step length of the following step.[126] It has also been suggested that, while walking on uneven ground or terrain, visual information from two steps is required to direct foot placement.[127]

Marigold and Patla[128] found that, when walking on a varying terrain, participants visually fixated on areas of the ground where they eventually stepped. Additionally, fixations were frequently guided to the transition zones between the varying surfaces (e.g., solid to compliant, rocky to slippery, tilted to irregular, etc). Hollands and Marple-Horvat[129] studied the eye movements of healthy participants who were made to walk in different conditions that varied in terms of the amount visual information available to the participants as they stepped onto stepping stones. The time interval between saccadic onset and foot-lift was similar in all conditions, but the interval between saccadic onset and footfall onto the stepping target differed significantly depending on the amount of visual information present. Patla and Vickers[130] found that healthy participants fixated on footfall targets that were an average of two steps ahead. Elderly participants with a history of falls tended not to look two steps ahead but instead fixated more on the imminent footfall target.[131] This finding may be the result of impaired central processing of visually guided information in that group, as suggested in another study, in which elderly participants with a high risk of falling had longer latencies from saccadic initiation to foot-lift than elderly individuals with a low risk of falling.[132]

Saccades were also studied in individuals during turning maneuvers. These studies supported a "top-down" model, in which saccadic initiation precedes, and possibly influences, turning of the head, trunk and legs.[112,114] Imai et al.[114] observed that when participants were asked to move in a straight line and

turn 90 degrees, a saccade was made in the direction of the turn. A similar observation was made by Hollands et al.,[133] in which healthy participants made saccades in order to position their gaze in line with the endpoint of the required travel path.

Anxiety can influence the interplay between gait and saccades. It has been suggested that early gaze transfer due to anxiety over impending obstacles is correlated with stepping inaccuracies. Investigators observed the visual and stepping behavior of an 87-year-old female when she was directed to walk along a stepping path before and after an obstacle. At the beginning of the experiment, she fixated on the stepping path before the obstacle. After falling twice, she stopped fixating on the stepping path, and instead fixated on the obstacle itself.[134] In a similar study, elderly participants with a high risk of falling were more likely to transfer their gaze early from a stepping target along a path to an impending obstacle.[135] One study indirectly showed a relationship between saccades and gait during an episode of anxiety/fear, in which participants with a fear of heights made more vertical than horizontal saccades when walking on a fire escape 20 meters above ground compared to the saccades of the controls.[136] The amygdala plays an important role in anxiety and has been found to be involved in saliency coding when scanning a visual scene.[137] States of increased anxiety may disrupt fixations and saccades through this pathway.

The relationship between saccades and gait was observed in healthy participants as they moved along a pathway with irregularly placed stepping stones, both with and without an alcohol dose. Gait impairments were observed in terms of increased step cycle durations and missed footfall targets. In terms of saccadic impairments, a large proportion of the saccades of the successive stepping stones were inaccurate and were accompanied by corrective saccades.[138] Alcohol has been shown to cause saccadic dysmetria.[139] The combination of impaired saccadic control and stepping accuracy implicates the cerebellum [See Supplementary Table 1 (in the online only Data Supplement) for summary of the studies of this section].

SACCADES AND GAIT IN NEURODEGENERATIVE DISEASES

While saccadic and gait abnormalities have been studied separately in various neurodegenerative disorders (Table 2),[140-174] simultaneous recordings of eye movements and gait in these disorders have rarely been reported.

PD is well known as having both saccadic[151] and gait abnormalities.[175] In PD, both saccades and step length can be hypometric. Side-to-side asymmetry, in terms of step length and saccadic amplitude, is often seen in PD. Nemanich and Earhart reported that, in PD, freezing of gait is associated with increased saccadic latency and variability.[176] The researchers found that PD patients with freezing of gait were slower in initiating pro- and antisaccades. Saccadic velocity and gain variability were also increased in PD with freezing of gait. Performance of antisaccades was impaired in PD patients with freezing of gait compared to patients without freezing.[177] In an-

Table 2. Separate studies showing saccadic abnormalities or gait abnormalities in essential tremor, PD, PSP, Huntington disease and cerebellar ataxia

Disorder	Saccadic abnormalities	Gait abnormalities
Essential tremor	Slow saccades and increased square-wave jerks[140]	Tandem gait difficulty[141-149]
PD	Hypometric saccades and prolonged saccadic latency[150,151]	Freezing of gait, falls, turning impairment, and decreased stride length[152,153]
PSP	Fixational saccades that are abnormally large. Square wave jerks more frequent, larger, and markedly more horizontal[154] Vertical saccades (slow and hypometric, both up and down)[155]	Hypokinetic gait characteristics: decreased velocity and step length[156] Interstep variability and asymmetry during gait. Slower cadence. Freezing of gait and frequent falls[157]
Huntington disease	Slow saccades[158-161] Increased variability in saccadic reaction times and occurrence of errors[162,163] Hypometric primary saccades[164]	Gait characteristic variation in each walk, with mean decreases in velocity, stride length, and cadence. Decreased gait velocity[165-167] Disordered regulation of footstep timing; reduced stride length[168]
Cerebellar ataxia	Square-wave jerks, saccadic dysmetria, and reduced saccadic velocity[169-171]	Decreased step length, stride length, and gait speed[172-174]

PD: Parkinson disease, PSP: progressive supranuclear palsy

other study, saccadic frequency was found to increase in both patients with PD and their age-matched controls when approaching a turn, but the PD patients made fewer preparatory saccades than the controls before the turn.[178,179] During the turn, the PD patients made more saccades, and the saccadic velocity was slower than that of the controls.[180]

The likely neural components affecting both saccades and locomotion in PD include the STN, the SNr, and the MLR/MFR.[175] In PD, degeneration of dopaminergic neurons in the SNc affects the direct and indirect pathways, resulting in bradykinetic movements that affect locomotion and saccades. More specifically, there is increased excitation of the STN, causing an increased inhibitory effect of the GPi and SNr through the indirect pathway. As mentioned earlier in the current review, DBS of the STN affects both saccadic and locomotor performance when compared to DBS of the GPi.[92-95] In terms of eye movements, the effect on these pathways in PD results in increased excitation of the SNr, which leads to abnormal saccade generation in the SC. There is also increased excitation of the PPN, which, as mentioned previously, has projections that are related to saccades and locomotion. In a recent imaging study, PPN alterations were suggested to be related to both saccadic and postural impairments in patients with PD.[181] It was observed that functional connectivity involving the PPN and FEF correlated with antisaccadic latencies in healthy participants but not in PD patients with postural instability. Additionally, saccadic impairment correlated with gait initiation impairment in patients.

Additional examples of neurological disorders with abnormal saccades and postural instability other than PD[182,183] include progressive supranuclear palsy,[184] cerebellar ataxia,[185] essential tremor,[186] and Huntington disease.[187,188] Some studies have reported that abnormalities in saccadic eye movements are correlated with body sway, even in healthy individuals.[189,190] These findings of these studies reflect an integration between postural dynamics and eye movements.

Patients with cerebellar ataxia have ataxic gait and dysmetric saccades. Dysmetric saccades consist of hypometric or hypermetric initial saccades, followed by a corrective saccade. TMS studies have implicated the ipsilateral cerebellar vermis in saccadic dysmetria.[68] Studies of visual fixation in patients with cerebellar ataxia have discovered the presence of dysmetric saccades. During locomotor tasks with visually guided stepping, both dysmetric saccades and ataxic gait were detected.[191,192] Other studies have found correlations between efficient footfalls and oculomotor function[127,129,130] in healthy subjects.

Studies of saccadic performance in patients with gait impairment could provide insight into how eye movements affect motor abnormalities such as freezing of gait, imbalance, turning difficulties and falls. Beyond that, saccadic eye movement training as a gait rehabilitation strategy could be an important therapeutic option. Some studies have reported saccadic eye movement training as a strategy for alleviating gait abnormalities in terms of improvement in

Table 3. Eye movement training and gait

Authors	Year	Participants	Method	Main findings
Eye movement training and gait				
Zampieri and Di Fabio[193]	2008	19 moderately affected progressive supranuclear pals patients	Balance training and eye movement exercises Eye movement training: eye movement practice on the computer screen with randomly appearing arrows on the screen	Improvements in stance time and walking speed in the treatment group
Crowdy et al[194]	2002	2 cerebellar patients	Foot placement (stepping task) Eye movement training: rehearsal of saccades for footfall targets in a stationary standing condition	Improvements in oculomotor and locomotor performance following eye-movement rehearsal
Kang and Yu[195]	2016	14 stroke patients	Foot placement (stepping task) Eye movement training: visual scanning of the picture cards, fixating gaze on a moving baton	Improvements in walking speed, step length and cadence

stance time and accuracy in stepping in patients (Table 3).[193-195]

CONCLUSIONS AND FUTURE DIRECTIONS

Eye movements and locomotion share common neural substrates and potentially have interlinked neural circuitries. The mesopontine tegmentum and cerebellar vermis are the most likely areas to have specific neural connections between these parallel networks. Physiological studies in animals and behavioral studies in healthy individuals have supported the hypothesis that these connections are preserved and adaptable across species. Many neurodegenerative disorders demonstrate coexisting eye movement and gait abnormalities. Correlations have been made in these disease states, further providing evidence of interlinked neural circuitry. As the technology of mobile eye-tracking improves, future studies exploring eye movement abnormalities in real time with simultaneous gait recording will further elucidate the interplay between these two networks. In addition, such studies may potentially serve to develop new diagnostic or disease severity markers.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.14802/jmd.18018.

Conflicts of Interest

The authors have no financial conflicts of interest.

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