Proprioception and Motor Control in Parkinson’s Disease

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ABSTRACT. Parkinson’s disease (PD) is a neurodegenerative disorder that leads to a progressive decline in motor function. Growing evidence indicates that PD patients also experience an array of sensory problems that negatively impact motor function. This is especially true for proprioceptive deficits, which profoundly degrade motor performance. This review specifically address the relationship between proprioception and motor impairments in PD. It is structured around 4 themes: (a) It examines whether the sensitivity of kinaesthetic perception, which is based on proprioceptive inputs, is actually altered in PD. (b) It discusses whether failed processes of proprioceptive-motor integration are central to the motor problems in PD. (c) It presents recent findings focusing on the link between the proprioception and the balance problems in PD. And (d) it discusses the current state of knowledge of how levodopa medication and deep brain stimulation affect proprioceptive and motor function in PD. The authors conclude that a failure to evaluate and to map proprioceptive information onto voluntary and reflexive motor commands is an integral part of the observed motor symptoms in PD.

Keywords: basal ganglia, kinaesthesia, movement disorder, sensorimotor integration

Movement abnormalities such as tremor, bradykinesia, rigidity, and postural problems constitute the clinical hallmarks of Parkinson’s disease (PD). They are thought to arise primarily from the loss of dopamine producing neurons and subsequent dysfunction of the basal ganglia-thalamocortical pathway. Yet, a growing body of research demonstrates that PD also is associated with an array of perceptual deficits, such as odor and tactile discrimination and detection (Herting, Schulze, Reichmann, Haehner, & Hummel, 2008; Mesholam, Moberg, Mahr, & Doty, 1998; Prátorius, Kimmeskamp, & Milani, 2003; Sathian, Zangaladze, Green, Vitek, & DeLong, 1997; Zia, Cody, & O’Boyle, 2003), weight and pain perception (Maschke, Tuite, Krawczewski, Pickett, & Konczak, 2006; Nolano et al., 2008), or the perception of visual depth (Maschke, Gomez, Tuite, Pickett, & Konczak, 2006). Recent evidence suggests that kinaesthesia is especially affected by PD and that such loss of kinaesthetic sensitivity is closely linked to the motor deficits (Adamovich, Berkinblit, Hening, Sage, & Poizner, 2001; Contreras-Vidal & Gold, 2004; Demirici, Grill, McShane, & Hallett, 1997; O’Suilleabain, Bullard, & Dewey, 2001).

Kinaesthesia is commonly defined as the conscious awareness of body or limb position and motion in space. It is based on sensory information derived from receptors in the muscles, tendons, and joint capsules. These receptors provide information about muscle length, contractile speed, muscle tension, and joint position. Collectively, this latter information is also referred to as proprioception or muscle sense. According to the classical definition by Goldscheider (1898) the four properties of the muscle sense are (a) passive motion sense, (b) active motion sense, (c) limb position sense, and (d) the sense of heaviness. Alternatively, some use the term proprioception to indicate the limb position sense and kinaesthesia to refer to limb motion sense (Gardner, Martin, & Jessell, 2000), a definition we do not adopt. Within the framework of this review, we use the term kinaesthesia to refer to the conscious perception of limb and body motion. We use the term proprioception to refer to the unconscious processing of proprioceptive signals used for reflexive and postural motor control while recognizing that proprioceptive information also forms the basis for kinaesthesia. The importance of proprioception for motor function such as reaching and grasping, static balance, and locomotion has been well documented (Butler et al., 2004; Diener, Dichgans, Guschlbauer, & Mau, 1984; Dietz, 2002; Sainburg, Ghilardi, Poizner, & Ghez, 1995). Patients with a loss of proprioception are still able to execute motor tasks, yet their motor behavior is gravely compromised. Goal-directed movements lack precision and postural and spinal reflexes are altered leading to problems with balance and gait (Dietz, Ghez, Gordon, & Ghilardi, 1995; Rothwell et al., 1982).

It is the purpose of this review to summarize the current knowledge of the extent of kinaesthetic deficits in PD. The review is guided and structured around four main questions: First, is there evidence that kinaesthetic deficits are altered in PD? Second, are the motor problems in PD the result of failed processes of proprioceptive-motor integration? Third, what is the link between the proprioception and the balance problems in PD? And fourth, how does levodopa medication and deep brain stimulation (DBS) affect proprioceptive and motor function in PD? Before addressing these four questions, we briefly review neurophysiological evidence that links proprioceptive function to neural processes in the basal ganglia.
ganglia and describe how the neural output of the basal ganglia is altered by PD.

**Proprioception and the Basal Ganglia**

Animal studies have long established that many basal ganglia neurons have proprioceptive receptive fields, responding both to passive and active joint motions (Crutcher & DeLong, 1984a, 1984bb; DeLong, Crutcher, & Georgopoulos, 1985). In humans, single cell recordings in PD patients submitted to neurosurgery revealed that a third of the neurons in the nucleus subthalamicus responded to passive or active movements of limbs, the oromandibular region, or the abdominal wall (Rodriguez-Oroz et al., 2001). These neuronal responses are joint specific with several reports showing that the vast majority of neurons in the monkey globus pallidus internal (GPI) respond to passive motion of a single joint (Boraud, Bezard, Bioulac, & Gross, 2000; Filion, Tremblay, & Bedard, 1988). However, when these monkeys were made parkinsonian through 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) treatment, the number of cells responding to passive movement increased, with most neurons now responding to movements of several joints. Researchers also observed this loss in neuronal response specificity in the efferent projections of the basal ganglia to the thalamus (Pessiglione et al., 2005) and supplementary motor area (Escola et al., 2002). Therefore, this disturbance of neural function is propagated throughout the striato-pallido-thalamo-cortical system. For example, in intact monkeys, thalamic neurons receiving basal ganglia afferents mostly respond to movement around a single joint. Following MPTP-induced parkinsonism, the tuning of the proprioceptive receptive fields of these thalamic neurons was markedly broadened thus providing much noisier and less differentiated proprioceptive information to cortical motor regions.

In general, the neurodegenerative processes associated with PD may lead to abnormal neural hyper- or hypoaacitivity in the basal ganglia and could act as a constant facilitator or brake on its efferent target structures (Pessiglione et al., 2005). The classical view regards an imbalance between the direct and indirect pathways projecting from the striatum to the globus pallidus internus/substantia nigra pars reticulata (GPI/SNr) as the major cause of the increased firing rate in GPI/SNr (DeLong, 1990). This increased firing inhibits thalamic projections to cortical motor areas thus reducing motor cortex excitation (see Figure 1). The reduced activation of motor cortical neurons is then believed to be responsible for the observed *bradykinesia*, the patients’ inability to activate a selected motor program or their failure to inhibit competing motor programs (Mink, 1996). A more recent, alternative view is that basal ganglia neurons become excessively synchronized at low frequencies in PD and the MPTP model of PD (Bevan, Magill, Terman, Bolam, & Wilson, 2002; Gatev, Darbin, & Wichmann, 2006; Goldberg, Rokni, Boraud, Vaadia, & Bergman, 2004; Raz et al., 2001; Raz, Vaadia, & Bergman, 2000). This excessive neuronal synchronization means a reduced responsiveness to signals related to a particular context or action. In addition, the output may lose topographic specificity (Bergman et al., 1998). In information processing terms, this implies that the signal to noise ratio of basal ganglia neural processing is altered in parkinsonism (Bar-Gad & Bergman, 2001). Such an increase in neural noise in the basal ganglia likely impairs the facilitation and modulation of premotor regions that must act synergistically to generate accurate and well-timed movement (Graybiel, 1998, 2005; Leiguarda et al., 2000).

![FIGURE 1. The basal ganglia-thalamocortical circuitry. Degeneration of the nigrostriatal dopamine pathway (SNc → Striatum) leads changes in the two striato-pallidal projections (direct and indirect pathways). STN = nucleus subthalamicus; SNr = substantia nigra, pars reticulata; SNc = substantia nigra, pars compacta; GPe = globus pallidus externus; GPI = globus pallidus internus; PPN = pedunculo-pontine nucleus.](image)

**Altered Kinaesthetic Sensitivity in PD**

Although routine clinical examination often fails to demonstrate sensory changes, sensory alterations have been documented in PD (Snider, Fahn, Isgreen, & Cote, 1976). Early reports showed that approximately 40% of PD patients experience spontaneous abnormal sensations despite having a normal neurological examination (Koller, 1984).

Neurophysiological studies have demonstrated that patients with Parkinson’s or Huntington’s disease show severely depressed frontal somatosensory-evoked or proprioception-related potentials (Abbruzzese & Berardelli, 2003; Rossini, Filippi, & Vernieri, 1998; Seiss, Praamstra, Hesse, & Rickards, 2003). Behavioral studies have suggested that basal ganglia dysfunction leads to an altered sensitivity of arm position sense (Klockgether, Bolz, Rapp, Spieker, & Dicgans, 1995; Schneider, Diamond, & Markham, 1987; Zia, Cody, & O’Boyle, 2000, 2002). However, it has remained unclear whether these findings were solely because of impaired kinaesthesia or could be attributed to problems in visual and cognitive processing; both of which are known to be
impaired in PD (Antal, Bandini, Keri, & Bodis-Wollner, 1998; Diederich, Raman, Leurgans, & Goetz, 2002). In addition, the possibility needs to be excluded that kinaesthetic deficits are simply the result of the known motor deficits of PD or are caused by some compensatory motor strategy.

Recently, researchers in several psychophysical studies attempted to address the concerns that vision or active limb motion contributed to or was responsible for the kinaesthetic impairments. These studies investigated kinaesthetic sensitivity of PD patients under conditions of blocked vision and those in which the patients were not actively moving and muscle activation was monitored. Using a passive motion apparatus, Maschke, Gomez, Tuite, and Konczak (2003) examined the sensitivity of PD patients to detect small changes in limb position by passively displacing the forearm at velocities less than 0.5°/s. After each displacement (between 0.2–8.0°), participants indicated whether their forearm had been moved. They tested three groups: individuals with mild to moderate PD, patients with spinocerebellar ataxia, and control participants. The detection thresholds at the 75% correct response level were 1.03° for controls, 1.15° for cerebellar patients, and 2.10° for PD patients, indicating that the PD group had elevated detection thresholds that were on average twice as high as the control and the cerebellar patient group. Only at displacements greater than 5° did PD patients exhibit the same sensitivity as controls and cerebellar patients (see Figure 2). This kinaesthetic impairment significantly correlated with the severity and duration of PD (r = −0.7, for both measures). In a similar study, Putzki et al. (2006) replicated these findings for the index finger, indicating that the sensitivity for detecting changes in limb position sense is reduced at distal and proximal arm joints in PD.

The previously mentioned studies have established that PD patients have difficulties detecting or determining limb positions. Another important aspect of kinaesthesia is the ability to sense limb motion. Researchers still have an incomplete understanding to what extent the limb motion sense is altered by PD, but in a recent study, Konczak, Krawczewski, Tuite, and Maschke (2007) investigated the sensitivity to detect passive limb motion. They assessed blind-folded participants for their ability to detect motion of their arm placed in a passive motion apparatus, which horizontally extended or flexed the elbow joint at a range of velocities between 1.65°/s and 0.075°/s. PD patients needed significantly larger limb displacements before they could judge the presence of passive motion. With decreasing velocity, the detection time increased exponentially in both control and PD participants. However, in comparison with healthy controls, the detection times of the PD group were increased 92–166% for the various velocity conditions.

A third aspect of kinaesthesia, the sense of heaviness, was recently evaluated in mild to moderately affected PD patients (Maschke, Tuite, et al., 2006). The sense of heaviness is related to the perception of weight. Maschke, Tuite, et al. determined psychophysical thresholds for detecting weight sensations by applying a gradually increasing load to the index finger by means of two slings of different width (low vs. high skin pressure). Although healthy age-matched control participants sensed a load at 31–33 g, the mean thresholds for the PD group were significantly increased in both pressure conditions (48–52 g). The increase in detection thresholds correlated positively with the severity of PD as measured by the Unified Parkinson’s Disease Rating Scale, indicating that the sensitivity to detect a load decreases as the disease progresses.

Further evidence for altered proprioceptive sensitivity in PD comes from a study investigating haptic acuity. Using a robotic manipulandum to create virtual contours, Konczak, Li, Tuite, and Poizner (2008) assessed the ability of PD and control participants to judge, without vision, the curvature of their arm passively or actively moved in a concave or convex trajectory. Of 11 PD patients, 9 (82%) showed elevated thresholds for detecting convex curvatures in at least one test condition. The respective median threshold for the PD group was increased by 343% when compared with the control group, indicating that the acuity of the haptic sense becomes reduced in PD. That the patients in the previously mentioned studies were only mildly to moderately affected underlines the notion that haptic as well as kinaesthetic impairments may become manifest already in the early stages of the disease. That is, proprioceptive or kinaesthetic deficits can appear very early in the disease and may precede motor deficits. However, current clinical examination protocols are not sensitive enough to detect these perceptual abnormalities.

Because all of the previously mentioned psychophysical studies involved no active motion, monitored for

![FIGURE 2. Forearm position sense acuity. The forearm was passively moved to 10 different positions (elbow joint angular displacement 0.2–8°). Graph shows the sensitivity functions of healthy control participants (N = 11), a group of cerebellar patients (N = 9), and a group of mild to moderately affected Parkinson’s disease (PD) patients (N = 9). The perceptual detection threshold was defined at 75% correct response level. Each data point represents the mean response rate of each group (reprinted with permission, M. Maschke, C. M. Gomez, P. J. Tuite, & J. Konczak, 2003).](image-url)
unwanted muscle activity, and excluded vision, it is very unlikely that bradykinesia, tremor, or possible movement compensation strategies can explain the reported decline in kinesthetic sensitivity in PD. Moreover, these data argue that these kinesthetic deficits are genuine manifestations of the disease that may occur very early in the disease process and may even precede the known motor problems in PD.

**Sensorimotor Integration: The Link Between Vision, Proprioception, and Motor Function in PD**

Multiple lines of evidence indicate that the basal ganglia are important for sensorimotor integration, the mechanisms by which sensory information is processed to guide motor planning and execution. Single cell recordings in the striatum of rats, cats, and monkeys have demonstrated that the activity of these cells depends on whether sensory information is linked to movement (Alexander & Crutcher, 1990; Lidsky & Manetto, 1987; Lidsky, Manetto, & Schneider, 1985; Schneider et al., 1987; West et al., 1987). Striatal cells can be silent for a given sensory event but robustly active when that same sensory event functions as a cue for a movement (West et al., 1987). In addition, the caudate nucleus and substantia nigra contain a large proportion of cells that are multisensory, cells that could be used to integrate sensory inputs and form a multimodal representation of the environment in the basal ganglia (Nagy, Eordegh, Paroczy, Markus, & Benedek, 2006). Likewise, the primate putamen contains bimodal visual-tactile cells that provide a map of peripersonal visual space. This map of visual space is organized somatotopically and “could function to guide movements in the animal’s immediate vicinity” (Graziano & Gross, 1993, p. 96).

When basal ganglia processes become disrupted, such as in MPTP-treated parkinsonian monkeys, more pallidal neurons than normal respond to passive limb movement, suggesting an impaired gain mechanism because of dopamine depletion (Filion et al., 1988). Results from other animal studies corroborate this finding by showing that the ability to use posture-relevant sensory information decreases with striatal dopamine loss (Henderson, Watson, Halliday, Heinemann, & Gerlach, 2003; Martens, Whishaw, Miklyaeva, & Pellis, 1996). Thus, there is a wealth of evidence that the basal ganglia receive suitable inputs from vision and proprioception to play an important role in the sensorimotor integration and that dopamine depletion negatively impacts this integration process.

The pattern of deficits in PD is consistent with a disruption of this integration mechanism. PD patients may become increasingly dependent on external stimuli to initiate and shape motor output and may be unable to effectively execute movements when deprived of critical proprioceptive information. This leads to a documented dependence on visual cues during reaching movements (Adamovich et al., 2001; Flash, Inzelberg, Schechtman, & Korczyn, 1992; Klockgether & Dichgans, 1994), grasping movements (Jackson, Harrison, Henderson, & Kennard, 1995; Muratori, McIsaac, Gordon, & Santello, 2008; Schettino et al., 2006), for the coordination of arm and trunk motion (Poizner et al., 2000), sequential arm movements (Curra et al., 1997), walking (Lewis, Byblow, & Walt, 2000), or for compensating for mechanical perturbations (Jacobs & Horak, 2006). In reaching movements, for example, PD patients are impaired when they cannot see their arm and thus must use proprioception to guide their reaches (Adamovich et al., 2001; see Figure 3).

Likewise, when holding an object, afferent information from the digits must be mapped onto motor commands specifying appropriate force levels. PD patients tend to develop elevated grip force levels when holding an object, despite the fact that they show both predictive and reactive modes of force control (Mueller & Abbs, 1990; Nowak & Hermsdorfer, 2006). Again, this suggests that their dysfunction consists of defective central processing or gating of sensory input. PD patients also show deficits in visual saccades (Briand, Hening, Poizner, & Sereno, 2001; Briand, Strallow, Hening, Poizner, & Sereno, 1999; Chan, Armstrong, Pari, Riopelle, & Munoz, 2005; Hood et al., 2007; Shaunak et al., 1999). Orientering to an environmental event with a saccade requires integration of multimodal sensory information and a mechanism that controls the integration process to select a signal (Hikosaka, Takikawa, & Kawagoe, 2000). Consistent with a deficit in sensorimotor integration, PD patients’ voluntary as opposed to reflex saccades are often markedly disrupted (Briand et al., 2001, 1999).

Thus, PD may be considered, in part, as a disorder of gain control of sensorimotor integration (Kaji, 2001; Kaji, Urushihara, Murase, Shimazu, & Goto, 2005). Within this process, dopamine may help to set the threshold for this gain control by modulating the responsiveness of the organism to the environment (Schultz, 2007). Dopamine depletion in PD then would disrupt the integration of environmental context with action planning and execution.

Although the deficient sensory gating hypothesis points to a sensory origin of the motor symptoms, it fails to explain why proprioceptive function seems to be especially affected by PD. As outlined previously, numerous studies and clinical observations confirm that PD patients may rely on visual cues to initiate or maintain movements. This visual dependence is consistent with a loss of proprioceptive function and points to a largely intact visual function in PD. Such vision-for-proprioception compensation strategy indirectly implies that a major basal ganglia function is proprioceptive-motor integration, whereas visuomotor integration is accomplished elsewhere in the brain. There is some neuroanatomical evidence to support this claim. The basal ganglia receiveafferents from the whole cortical mantle including the visual cortex, yet the projections from the dorsal visual stream to the basal ganglia are minor compared with the visual afferents to the cerebellum (Glickstein, 2000). These cerebellar and posterior parietal circuits are known to be critical for on-line visuomotor control and may be largely spared by the disease,
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FIGURE 3. Participants reached in the dark to five remembered or actual targets presented in three-dimensional space with a robot arm. The target was an illuminated LED. There were three conditions of visual feedback in which participants were provided either no vision during the movement, only vision of the moving fingertip, or only vision of the target but not the arm. Arm trajectories and endpoints across trials and conditions are presented for a control participant (top panel) and a Parkinson’s disease (PD) participant (bottom panel). Green spheres represent movement endpoints when participants reached to remembered targets without vision of their arms. Gray spheres represent movement endpoints when participants reached to remembered targets with vision of their moving fingertips, and blue spheres represent movement endpoints when participants reached to a visually present target without vision of their arms. PD patients were impaired in the two conditions in which they could not see their arms. Thus, in reaching to remembered or to actual targets, PD patients have difficulty in extracting and using critical information from proprioception to map arm positions onto spatial target locations to guide reaching movements (adapted from S. V. Adamovich, M. B. Berkinblit, W. Hening, J. Sage, & H. Poizner, 2001).

which could explain the increased reliance on vision by PD patients.

Proprioceptive Deficits and Postural Control in PD

Several deficits of postural coordination observed in PD patients are consistent with the hypothesis that they have an impaired proprioceptive body map. A series of studies showed that, unlike healthy controls, PD patients do not modify postural response synergies immediately following a change in initial support conditions (Chong, Horak, & Woollacott, 2000; Chong, Jones, & Horak, 1999). For example, when healthy participants change postural support from free stance to sitting or to holding onto a stable support, their postural response synergies change immediately from leg to trunk or arm muscle activation (Horak, Nutt, & Nashner, 1992). In contrast, participants with PD do not reduce leg postural responses in the first trial after a change in initial support condition of sitting or holding, although they gradually adapt postural synergies with practice (Chong et al., 1999; Horak et al.).

Similarly, healthy participants, but not PD participants, scale up the size of their anticipatory postural adjustments prior to step initiation in the first trial following an increase in stance width (Rocchi et al., 2006). The immediate increase in lateral reactive force to unload the initial stance leg with a wide stance, compared with a narrow stance, requires proprioceptive mapping of initial body posture and modification of postural adjustments prior to any feedback from body motion. In addition, the inability to quickly increase anticipatory postural adjustments when stance width increases also may be related to bradykinetic force output. However, regardless of the mechanism, such failure to make anticipatory postural adjustments to unload the stepping leg may be responsible for the compensatory narrow stance width, start hesitation, or freezing in advanced PD.

Two recent studies showed that PD patients have abnormal postural coordination only when vision was obscured, which is consistent with impaired proprioceptive mapping compensated by use of vision of the body. In one study, healthy, age-matched participants could accurately direct their automatically triggered, compensatory steps onto a small target on the ground in response to external postural perturbations, even when they could not view their stepping leg. In contrast, PD patients consistently undershot the targets when they could not see their legs (Jacobs & Horak, 2006). Similarly, when PD patients closed their eyes, they showed a breakdown in the temporal coordination between postural adjustments and arm reaching during whole body reaching to a target (Tagliabue, Ferrigno, & Horak, 2009).

Postural kinaesthesia also is disrupted in patients with PD. Horak and coworkers tested the ability of PD patients to perceive surface inclination by asking blindfolded, standing participants to compare the relative dorsiflexion or plantarflexion of the support surface under a test foot with a 4-degree tilt of the surface under the reference foot (Wright et al., 2006). Participants with PD showed fewer percent correct trials when indicating whether the surface under the reference foot was more or less plantar-flexed or dorsiflexed than the surface under the test foot (see Figure 4). Impaired kinaesthesia of the support surface was significantly correlated with the severity of PD. It is noteworthy that levodopa medication did not improve this kinaesthetic deficit, although it generally led to better motor performance.
like the postural deficits associated with peripheral, proprioceptive deficits that cause prolonged postural response latencies that are not observed in PD (Cameron, Horak, & Herndon, 2009; Inglis, Horak, Shupert, & Jones-Rycewicz, 1994).

**Effects of Levodopa and DBS on Motor and Proprioceptive Function**

Dopamine replacement therapy is highly effective in ameliorating many symptoms in PD. It is especially effective in early PD and results in an improvement of quality in life and survival (Miyasaki, Martin, Suchowersky, Weiner, & Lang, 2002). However, the effects of levodopa on proprioception are not fully understood. Jobst, Melnick, Byl, Dowling, and Aminoff (1997) concluded that levodopa does not improve kinaesthetic deficits in PD. During their on-medication state and with vision occluded, PD patients did not improve their sensitivity to perceive differences in movement amplitudes made to a target away from the body, a task requiring the reliance on proprioceptive feedback (Jobst et al.). There is some indication that levodopa actually may have an added detrimental effect on kinaesthesia in PD. For example, when comparing PD patients between their off- and on-medication states, arm proprioception was impaired by 31% 1 hour after administration of levodopa or dopamine agonists (O’Suilleabhain et al., 2001). In contrast, psychophysical studies found no significant correlations between levodopa dosage and elevated detection thresholds for arm position sense, arm motion sense, or weight perception in a group of medicated and de novo (i.e., nonmedicated) PD patients (Maschke et al., 2003; Maschke, Tuite, et al., 2006).

In summary, there is some evidence that levodopa further degrades proprioceptive acuity with some authors arguing that levodopa enhanced proprioceptive deficits might be one key in the development of levodopa induced dyskinesia (O’Suilleabhain et al., 2001). A role of levodopa in phasic pharmacological reduction of proprioception would be further supported if nondopaminergic medication such as amantadine was shown to improve proprioception. However, the only study investigating the influence of nondopaminergic medication on proprioception revealed no influence of the NMDA-antagonist flupirtine on automatic postural responses in PD (Putzki, Maschke, Drepper, Diener, & Timmann, 2002).

Although levodopa is a very effective treatment for PD, motor complications develop over time with its use. The complications include the development of fluctuations in response to medication (i.e., motor fluctuations) and the appearance of medication-induced abnormal involuntary movements (i.e., dyskinesia). In addition, antiparkinsonian medication is not always that effective in treating tremor. Thus, as the disease progresses, a subset of patients with PD become candidates for DBS neurosurgical treatment, which can be very effective in alleviating motor certain symptoms in PD.

The same group tested the kinaesthesia of axial trunk rotation by asking participants to indicate the direction and initial detection of yaw oscillations (1/°s) of the support surface under their feet during stance (Wright et al., 2006). Axial rotation occurred either at the trunk, by stabilizing the shoulders to earth and the pelvis to the rotating surface, or hips, by stabilizing the pelvis to earth (Gurfinkel et al., 2006). The ability of PD patients to accurately detect the direction and onset of axial rotation was impaired for both trunk and hip twisting (see Figure 4), indicating that trunk position sense was compromised. This finding corroborates and extends the results of previous studies showing that upper limb position sense is altered by PD (Maschke et al., 2003; Maschke, Tuite, et al., 2005).

When vision is absent, the control of sway on a firm surface depends primarily on proprioceptive feedback, whereas control of sway on an unstable surface depends on vestibular feedback. The fact that PD patients may show larger postural sway compared with controls when standing on a firm surface than when standing on an unstable surface with eyes closed also is consistent with poor use of proprioceptive feedback but excellent use of vestibular information, to control postural sway. In fact, sway characteristics of patients with PD are consistent with increased proprioceptive feedback loop noise with abnormal feedback gain (Maurer, Mergner, & Peterka, 2004).

Thus, proprioceptive deficits may affect postural stability in PD by impairing (a) adaptation to changing support conditions, (b) accuracy of compensatory stepping, (c) coupling between postural adjustments and voluntary movement, (d) the perception of trunk and surface orientation, and (e) postural sway in stance. These postural deficits suggest a deficit in central integration of proprioception because they are un-

![Figure 4. Kinaesthesia impairment during stance in participants with Parkinson’s disease in the on- and off-levodopa state compared with age-matched controls for perception of surface inclination (A) and torso rotation (B). (A) Percent correct in judging one more degree of surface dorsiflexion in test compared with reference foot. (B) Percent correct in judging left or right direction of torso rotation.](image-url)

**FIGURE 4.** Kinaesthesia impairment during stance in participants with Parkinson’s disease in the on- and off-levodopa state compared with age-matched controls for perception of surface inclination (A) and torso rotation (B). (A) Percent correct in judging one more degree of surface dorsiflexion in test compared with reference foot. (B) Percent correct in judging left or right direction of torso rotation.
studies, it is clear that the acuity of the proprioceptive and logical mechanisms of DBS and (b) do not fully know how researchers (a) still lack a full understanding of the physiological control and upper limb function in PD patients is still poorly understood. Part of the problem of clearly discerning the improvement in postural control and speech and the increase in kinesthesia after DBS surgery is consistent with the lack of clinical improvements in postural stability commonly observed in levodopa medicated PD patients (Beuter, Hernandez, Rigal, Modolo, & Blanchet, 2008; Horak, Frank, & Nutt, 1996; O’Suilleabhain et al., 2001). In contrast, STN DBS improves proprioceptive acuity, which may explain the reported positive effect on some aspects of postural function (Guehl et al., 2006; Rocchi, Chiari, Cappello, Gross, & Horak, 2004). However, the improved kinaesthetic function because of STN DBS does not translate necessarily into improved postural function. Thus, the effects of other interventions such as STN DBS on proprioceptive sensitivity and posture require further evaluation.

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