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Research Article

# Recurrent fall in Parkinson Disease: Possible Role of the Proprioceptive and Vestibular Systems

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**Abstract**

Falls are a major risk for Parkinson disease patients (PD). We sought to understand why some patients fall once and some more than once (recurrent fallers). Recurrent fallers had PD significantly longer, 12.6 + 7.0 versus 5.9 + 4.5 years, had significantly higher motor MDS UPDRS scores" 31.2 + 12.7 versus 19.7 + 8.3, and walked with significantly shorter steps: 0.37 + 0.18 meters versus 0.52 + 0.19 meters than single fallers.

The major difference between single fallers and recurrent fallers was an inability of recurrent fallers to stand on one leg for < 3 seconds: 95% versus 11 %, odds ratio 178 CI 95% 39.5-801.2 We attribute this inability to stand on one leg for < 3 seconds to an impairment of proprioceptive function and or possibly vestibular (utricle and sacculus) function. When a person stands on one leg, they effectively decrease their proprioceptive input by 50%.

In two studies of tremor in PD we implicated proprioceptive and vestibular impairments. Such impairments were demonstrated more than 50 years ago by J Purdon Martin, the eminent English neurologist, in post-encephalitic Parkinson patients. Based on our studies of recurrent falls in PD we believe proprioceptive and vestibular impairments may be major risk factors in falls.

**Keywords:** Parkinson disease; Falls; Proprioception

## Introduction

Falls, especially recurrent falls, are a major cause of disability in Parkinson disease (PD) [1,2]. In a study of 761 hospital admissions for PD only 15% were for the management of PD, while 39% were for falls [1]. A major risk for recurrent falls is a previous history of a fall. PD patients who fall once may do so because of PD or because of factors such as poor eyesight, leg weakness, and environmental hazards. They do not differ, substantially from PD patients who don't fall in regard to age, duration or severity of PD [3-5]. Some PD patients fall repeatedly [3]. They do differ from patients with PD who do not fall in regard to age, duration and severity of PD [5].

There is variability in the reported prevalence of falls in PD: from 11% to 68% [3-14]. The variability depends on whether specific fall risk factors are excluded. These include visual loss, neuropathy resulting in leg weakness or proprioceptive loss, orthostatic hypotension resulting from anti-hypertensives or imbalance resulting from tranquillizers, sedatives or alcohol. The variability also includes whether persons with evolving atypical Parkinson disorders such as Progressive Supranuclear Palsy (PSP) or Multiple System Atrophy (MSA) are excluded. Although patients with these disorders represent only a small number of patients a high percentage of them fall [6].

## Methods

We only analyzed serious falls where all 4 limbs, the skull or buttocks hit the ground, with patients sustaining a fracture or soft tissue injury. Patients who had a serious fall sought attention, within 24-48 hours, in an Emergency Room, an Urgent Care Center, their local physician, or us.

All patients were examined using the Movement Disorder Society (MDS) motor portion and its sub-tests part of the Unified Parkinson Disease Rating System [15] and selected subtests: freezing of gait (FOG) subtest, postural instability (PI), utilizing the "pull test". All patients were studied using the BNI balance scale [16]. This included the ability of a patient to stand on one leg for at least 3 seconds: the One-Legged Stance, a test of postural instability (PI). All patients walked 7.63 meters (25 feet). The number of steps taken was counted, divided by 7.63 meters, and an average was obtained.

We excluded patients with dementia, Mini-Mental Status Examinations, MMSE, < 24. Although dementia can be a risk for falling, many PD patients with dementia are without a care-giver for at least 4 hours. Thus we were uncertain if they reported their falls. We excluded patients who were legally blind. We excluded patients with orthostatic hypotension. Although orthostatic hypotension can be part of PD, it can also result from the use of anti-hypertensives, diuretics or dehydration [17]. We excluded patients with neuropathy when it resulted in impaired proprioception or weakness and we excluded patients with major orthopedic problems [13,14].

In our study of single versus recurrent fallers [3], we examined 452 patients. We excluded 51 patients for the reasons enumerated above. We next excluded patients who did not fall. We then compared 161 single fallers with 44 recurrent fallers, a total of 205 patients.

In a second and separate study, we distinguished the tremor of PD from the tremor of Essential Tremor [18]. Although the tremor of PD usually, but not always, differs from essential tremor (ET), there is no simple bedside test to distinguish PD from ET. We studied 50 consecutive tremor-dominant PD patients (mean age: 63.4 years;




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**Table 1:** Single-fallers vs recurrent fallers.

| | Single –Fallers 161 patients | Recurrent Fallers 44 patients | p-value |
|---|---|---|---|
| Age (years) | 67.4 ± 8.7 | 69.7 ± 9.3 | NS |
| PD Duration (years) | 5.9 ± 4.5 | 12.6 ± 7.0 | 0.001* |
| MDS UPDRS Motor Score | 19.7 ± 8.3 | 32.3 ± 12.6 | 0.01* |
| Step length Meters/ Step | 0.52 ± 0.12 | 0.31 ± 0.12 | <0.0* |

mean disease duration: 4.9 years) and 35 consecutive ET patients (mean age: 64.1 years; mean disease duration: 12.5 years). Among PD patients, 31 had a bilateral tremor and among ET patients, 29 patients had a bilateral tremor. Patients sat opposite the examiner and pointed both index fingers at the examiner's index fingers. Then they closed their eyes. Within 15 seconds, one or rarely both of the patient's index fingers moved, were displaced, either upward or laterally.

In a third and separate study, we examined the above phenomenon in 104 PD patients: 72 without a tremor and 32 with a minimal tremor to see if the displacement is related to the disease or the tremor [19]. Sixty-eight of the 72 patients without tremor, 94%, exhibited finger displacement suggesting the phenomenon is related to PD. None of the 104 patients were demented.

All patients were informed that the information collected could be used for research but that they personally could not be identified. Approval for the analysis was obtained by the St. Joseph's Hospital institutional review board. No patients were compensated. As the evaluations were part of the patient's routine care no special consent other than the standard signed consent obtained from all patients at the time their visit was obtained.

Continuous variables were analyzed using t-tests and categorical variables were analyzed using chi-square tests.

## Results

In our study of single versus recurrent fallers [3] 87.5 % of single fallers were on levodopa, among recurrent fallers 100 % were on levodopa [3]. 40% of single fallers had dyskinesias, 56% of recurrent fallers had dyskinesias. Although a higher percent of recurrent fallers had dyskinesias we cannot comment on the contribution of dyskinesias to falls. This is because we recorded only the presence, not the severity of dyskinesias.

We studied 205 patients: 113 men, 92 women of whom 161 (79%) fell once and 44 (21%) fell more than once. See Tables 1, 2.

## Discussion

The prevalence of falls in our study is comparable to that reported by others [4-10]. It's estimated that 33% of all people over age 65 years fall at least once a year. Thus approximately 55 % more people with PD fall, at least once per year, than people without PD. Fall-related injuries in addition to reducing mobility and independence result in hospitalization [1,2]. The costs associated with fall-related injuries are high and estimated to reach $30 billion by 2020 by the Rand Corporation [20]. These figures will be proportionately higher in PD, because a higher percent of people with PD fall and are more likely to fall repeatedly.

If falls, and especially recurrent falls, can be reduced in PD, they possibly can be reduced in other disorders. The main measures, at present, for reducing falls are education and gait and balance training [20-24]. However, before newer measures can be undertaken it is important to understand why people with PD fall.

**Table 2:** Single vs recurrent fallers chi- square critical level p< 0.05.

| | Single Fallers 161 people | Recurrent Fallers 44 people | Odds Ratio |
|---|---|---|---|
| MDS UPDRS FOG Sub Test ≥1 | 9 patients 5.6% | 21 patients 47.7% | 15.4 CI95% 6.3-37.7 <0.001* |
| MDS UPDRS Pull Test Sub Test ≥3 | 35 patients 22.0% | 29 patients 65.9% | 7.0 CI95% 3.4-14.4 <0.001* |
| Inability Stand One Foot < 3 seconds | 17 patients 10.8% | 42 patients 95.4% | 177.9 CI95% 39.5-801.2 <0.0001* |

Recurrent fallers have PD longer and are more disabled. However, in an individual PD patient who falls, the duration of PD may be insufficient to predict another fall. And in an individual PD patient the MDS UPDRS motor score may be insufficient to predict another fall. This is so in part because the MDS UPDRS is weighted toward upper extremity rigidity, tremor, and bradykinesia, metrics that are less likely to predict falls.

Fallers take significantly shorter steps. This "cautious" gait is, in our opinion, an inappropriate adaptation to postural instability. Short steps, and especially the short steps of freezing of gait (FOG) may create a "mismatch" between the displacement of the upper versus the lower body, resulting in the upper bodies "tipping over", and the patient falling [25-27].

Recurrent fallers were significantly more likely to be unable to stand on one foot for < 3 seconds: 95.4% of recurrent fallers compared to 10.8% of single fallers, odds ratio 177.9 CI 95% 39.5-801.2, p < 0.001. This is, in our experience, the single best "bedside" predictor of recurrent falls, better than FOG or the "pull test" (Table 2).

In the study comparing tremor dominant PD patients versus ET patients, finger displacement occurred only with bilateral simultaneous pointing with the patient's eyes closed [18]. All the tremor-dominant PD patients exhibited displacement of a limb and the index finger of the limb. In46 patients, finger and limb displacement occurred on the side of dominant tremor, in 4, it occurred bilaterally. In 31 of 35 ET patients, no displacement occurred. Odds ratio of distinguishing PD from ET: 89.62 at 95% confidence limits (5.31-1513.4), p = 0. 0018. Sensitivity 100%.

In a second study of finger and limb displacement in 104 non-tremor and minor tremor PD patient 90 patients displayed upward displacement (56 patients), lateral or medial displacement (34 patients) [19]. Eight patients displayed downward displacement. Fifty of the patients, 48%, had fallen, 16, 15%, had fallen more than once. All of the PD patients in both of the above studies [18,19] had intact light touch, vibration sense, joint-position sense, two point discrimination, graphesthesia, and point localization.

Locomotion, the rhythmical movement of the extremities, is generated in the spinal cord [28]. A hierarchy of supra-spinal centers in the cortex and basal ganglia signal the spinal cord. This supra-spinal command network is necessary for initiating walking, turning and stopping. Part of this locomotion *network* is under dopaminergic

control. This is impaired in PD and responds, in part, to dopaminergic therapy [3,26,29-34].

The *locomotor network* interacts with a *postural stability network* that includes inputs from pro preceptors in the joints, tendons and muscles as well as inputs from the vestibular nuclei (mainly the utricle and sacculus) and the visual system. This network is not under dopaminergic control [29-31]. Lower extremity proprioceptive inputs are an important input to the central nervous system (CNS). A patient's ability to compensate for postural instability is their ability to generate rapid, large-scale leg movements. When this is also impaired in PD, as manifested by the short step, patients fall as they are unable to counterbalance for their PI [3,26,30-33].

Similar phenomena were described by the eminent English neurologist, consulting physician to the National Hospital for Nervous Disease, Queen Square, London, in his monograph *The Basal Ganglia and Posture*, published in 1967 [35]. Purdon Martin studied 130 patients with post-encephalitic Parkinson disease, survivors of the epidemic of 1919-1925. He studied postural fixation of the limbs in patients with disorders of gait and balance. These patients were often unable to fix their hips, shoulders, head and neck on their skeletal axis. This resulted in a variety of abnormal postures: head flexed on neck, trunk flexed, anteriorly or posteriorly, hips and knees flexed. The patients were, when asked, able to correct these postured (if contractures had not occurred). Proprioception as tested with joint position sense, two point discrimination and graphesthesia was intact.

Next, when they were blind-folded, the patients resumed the abnormal postures. As the patients were not especially rigid or bradykinetic, Purdon-Martin postulated the inability to properly fix their head, neck, trunk, or limbs on their skeletal axis was related to CNS impairment of proprioceptive and vestibular inputs.

The patients, when pushed or pulled fell. Purdon Martin attributed the falls to a loss of "righting reflexes." He thought these reflexes also acted through proprioceptive impulses from muscles, joints, and tendons and from impulses from the vestibular nuclei and vestibular-spinal tracts. He thought the vestibular system was involved because the patients who fell resembled patients who had bilateral vestibular loss and were then blind-folded.

In studying postural instability under static conditions, standing, Purdon Martin wrote [35].

In the upright position man stands on a base which is narrow and small relative to his height. A manikin or dummy figure is easily knocked over and man himself is stable only as long as his center of gravity remains within the vertical projection of his base. However, he differs from the dummy in that when his stability is threatened various physiological reactions come into play to protect it and if nevertheless his equilibrium is lost and he appears about to fall other reactions come into play to restore the upright position….."

Physical instability is widespread among some of our patients and the more severely affected are remarkable in that they are unable to preserve their equilibrium (PI) not only when they are standing but also when they are sitting and even when they are on all fours (and not moving) …..".

Twenty six of the 130 patients (20%) have not enough equilibrium to enable them to maintain the standing position, no external force being applied. Twenty one (16 %) is unable to maintain the sitting position and 13 (10 %) are unable to maintain the all-fours position these symptoms are not all proportionate to the degree of rigidity…...

The inability to maintain an erect position: standing, sitting, on all-fours, was attributed by Purdon Martin to failed CNS proprioceptive and vestibular inputs. In studying PI under dynamic conditions including after a perturbation, he wrote [35]:

Of the 130 patients, 51 (39 %), are unable to right themselves from the supine to the upright or standing position. Twenty four (18 %) are unable to roll over into the lateral position…..Most of these patients begin the movement of turning on to one side in the normal way by turning their eyes and then their head to the side but these movements are not followed as they should be by turning of the trunk.

Of the relatively non rigid patients who are unable to turn over, many show little or no reaction of the body or limbs to proprioceptive and labyrinthine stimulation as tested on a tilt-table (a see-saw). Primary proprioception and labyrinthine function are normal (as tested by joint position sense, two point discrimination, graphesthesia, and caloric responses). When the head is turned, normal impulses are presumable aroused from the neck muscles and labyrinths but they fail to evoke normal responses from the trunk and limbs. In the absence of these impulses and of visual help the patient is dependent on righting reactions (themselves dependent on proprioceptive and vestibular inputs)…..And these inputs are apparently inadequate to enable them to turn or arise…..

Fifteen of the 130 patients were tested on a tilt-table (a see-saw). The patients sat at one end while the opposite end was tilted upward. If the patient's eyes were open, the patient did not slide downward. If, however, the patients were blindfolded they slid downward---behaving like patients who had bilateral vestibular loss. Purdon-Martin wrote [35].

The reactions of these 15 patients to tilting are therefore the same as those of the subjects with no vestibular function, that is to say, they have lost those postural reactions to tiling which depend on labyrinthine stimulation (utricle and sacculus) and since their labyrinths are normal we must infer that the reactions are abolished because the reflex arcs are interrupted centrally by disease.

Finger and displacement (up, down, or laterally) with eyes closed for 15 seconds as studied by us in PD and ET requires an ability to "remember" the position of the limbs and fingers in space and to alter tone to overcome gravity. The alteration of tone depends upon an integration of proprioceptive impulses from the tendons, joints, and muscles with pyramidal and extrapyramidal motor systems in the CNS controlling the position of the limb and finger in space. The utricle, part of the vestibular system, contributes to the sense of "up" and "down." The saccule may contribute to the sense of laterality (displacement of the limb and finger laterally or medially. Thus, we believe the proprioceptive as well as vestibular system play a role, and a major role, in limb and finger displacement. At present we cannot separate the influence of the proprioceptive from the vestibular system. Similar observations were made by and conclusions drawn by Purdon Martin who studied limb and finger displacement in 69 of

130 patients (53%) and wrote [35].

By far the most common disorder seen unilaterally or bilaterally in 69 patients is a failure to maintain posture of the arm (and fingers) to perform a simple action with the hand when the patient closes his eyes. If the patient is asked to touch alternately the tips of the examiner's fore-fingers held… at the level of the of patient's shoulder…he may continue to perform the movement quite well for several minutes, but if… he closes his eyes his arm and finger almost immediately falls away…There is no evidence that the failure is due to disorder of the sense of position…The observation shows that vision plays a part in postural fixation of the upper limb…In cases of cerebellar disease (a surrogate for ET) there is an impairment of postural fixation which permits the limb to sway during a movement but closing the eyes does not aggravate these faults and does not cause the limb to fall away.

We, like Purdon Martin, have demonstrated, in the upper limbs, impairment in proprioception. We, like Purdon Martin, also believe in impairment in vestibular function, especially in of the utricle, the part of the vestibular apparatus that monitors and responds to gravity. Although we have not demonstrated it specifically, we believe, like Purdon Martin and others, of similar impairments in proprioception (and vestibular function) in the lower limbs [27, 31-33].

Postural instability, with falls, is a relatively late occurrence in the course of PD, and usually does not respond to dopaminergic drugs [3,26,30]. Postural instability and falls may respond, in part, to cholinomimetic [30], noradrenergic drugs, or serotonergic drugs. In the absence of specific and detailed knowledge of the pharmacology of the proprioceptive and vestibular systems, a main focus will continue to be on educating and gait and balance training. The training should focus on correcting the sensory deficits emphasized in this review: impaired proprioception and vestibular function.

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