Neurophysiologie Clinique/Clinical Neurophysiology (2008) **38**, 459—465

Disponible en ligne sur www.sciencedirect.com

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REVIEW/MISE AU POINT

Postural disorders in Parkinson's disease

Anomalies de la posture dans la maladie de Parkinson

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Received 28 July 2008; accepted 29 July 2008
Available online 21 August 2008

KEYWORDS
Parkinson's disease;
Posture;
Falls

MOTS CLÉS
Maladie de Parkinson ;
Posture ;
Chutes

Summary Posture is often affected in Parkinson's disease. Postural abnormalities belong to the motor axial involvement. Generally, postural dysfunction induces clinical impairment at the latest stages of the disease, except in late-onset idiopathic Parkinson's disease and in atypical parkinsonian syndromes. Posture may be affected in its orientation component (stooped posture, camptocormia, Pisa syndrome) or in its balance component (loss of postural reflexes). Overall, postural impairment is poorly improved by levodopa, which implies that it is unlikely due to the nigrostriatal dopaminergic denervation. Several methods of investigation have been proposed but are generally not available in clinical practice. Medical treatment and deep brain stimulation (DBS) of the subthalamic nucleus or globus pallidus pars interna are less efficient on axial than on distal motor signs. The pedonculopontine nucleus seems promising as a new target for DBS in combination with the subthalamic nucleus. Physical therapy is, in most cases, the best way to improve postural dysfunction.

Résumé Les atteintes de la posture sont fréquentes dans la maladie de Parkinson. Elles peuvent toucher les deux composantes du contrôle postural, trouble de l'orientation avec une tendance à la flexion du tronc, une camptocormie, un antecollis ou encore un Pisa syndrome ou trouble de l'équilibration dû à la perte des réflexes de correction posturale. Des liens entre ces deux dysfonctions sont probables mais encore mal établis. Les troubles posturaux surviennent souvent tardivement dans l'évolution de la maladie, à l'exception des formes à début tardif et sont peu améliorés par les agents dopaminergiques, ce qui implique la présence d'autres lésions que la dénervation dopaminergique nigrostriatale. La stimulation cérébrale profonde du noyau sous-thalamique ou du pallidum interne est également peu efficace sur les troubles posturaux. Une nouvelle cible est proposée depuis peu, le noyau pédonculopontin,

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0987-7053/$ - see front matter © 2008 Published by Elsevier Masson SAS.
doi:10.1016/j.neucli.2008.07.006

dont la stimulation à basse fréquence combinée à celle du noyau sous-thalamique permettrait d'améliorer les troubles de la posture et de la marche. La rééducation fonctionnelle trouve ici une indication importante.

Introduction

Parkinson's disease (PD) is a chronic neurodegenerative disorder, which is characterized by three cardinal features: rest tremor, rigidity, and bradykinesia. PD is also characterized by postural disorders affecting the two main components of postural control: orientation and stabilization. The perturbations of the trunk and neck orientation may be due essentially to rigidity, which is often detected in the distal parts of the limbs but may also affect axial muscles. Concerning stabilization, the perturbations of postural reflexes generally occur later on in the course of the disease, therefore, postural instability is a sign of more advanced PD, potentially responsible for an important loss of autonomy due to falls and injuries.

Postural deformities

Among several axial deformities, stooped posture with a moderate flexion of the knees and trunk, with elbows bent and arms adducted is the most common one. This posture has been associated with lenticular lesions suggesting that the basal ganglia play an important role in the maintenance of axial posture [38]. Dorsal cyphosis is classical and was already described by James Parkinson (1817): ''A propensity to bend the trunk forward'' and ''the chin is now almost immovably bent down upon the sternum''. Lateral flexion of the trunk is also frequent and appears in the late stage of the disease. It is either a regular lateral curve extended from sacrum to the neck or a limited deformity of the dorsal column. The head is generally inclined in the same direction than the trunk. Others postural abnormalities include extreme neck flexion, Pisa syndrome and camptocormia.

Camptocormia (Fig. 1) was first described by Earle in 1815 and by Brodie in 1837. It is characterized by an abnormal posture of the trunk with marked flexion of the thoracolumbar spine. Camptocormia increases with time and fatigue during the day and during walking; it is relieved by sitting, lying in the supine position, or by volitionally extending the trunk when the patient leans against a wall [4]. It may be sometimes associated with a latero-deviation of the trunk. Among the 16 patients with camptocormia described by Azher and Jankovic [4], the most frequent aetiology was PD, accounting for 11 cases (68.8%). Nevertheless, camptocormia may be associated with various aetiologies including other parkinsonian syndromes, dystonia, vascular lenticular lesions [38], muscular and rheumatologic disorders. PD associated with camptocormia is characterized by a male predominance, an older age, a longer disease duration, a prominent axial involvement, motor fluctuations, and autonomic symptoms [32]. Camptocormia is also associated with a high prevalence of lumbar or thoracolumbar scoliosis (in 61% of patients) and a mild to moderate low-back pain in 77% of patients. The time intervals between the onset of PD and the clinical onset of camptocormia ranged from four to 14 years [8,17,32]

Another truncal deformity is the Pisa syndrome (Fig. 2), which is characterized by a tilting of the trunk, particularly when sitting or standing. Although most cases of Pisa syndrome in PD occur as a complication of neuroleptics, antiemetics or cholinesterase inhibitors [48], several patients with PD spontaneously developed Pisa syndrome, which should be considered as a rare form of axial dystonia [19]. Even, if it was suggested that a dopaminergic dysfunction or an imbalance in the cholinergic-dopaminergic tonus might cause the syndrome [52], its pathophysiology actually remains largely unexplained.

Finally, only one study has evaluated the frequency of a dropped ''head syndrome (antecollis)'' in PD [28] and found a percentage of 6% of parkinsonian patients. The mean disease duration was 5.4 ± 4.3 years and it was more frequent in women and in patients, who had primarily suffered from rigidity and akinesia. Disproportionate antecollis complicates mainly multiple system atrophy (MSA) and PD. Its aetiology is much debated, the main issue being whether it represents a primary myopathy or whether it is secondary to the underlying motor disorder.

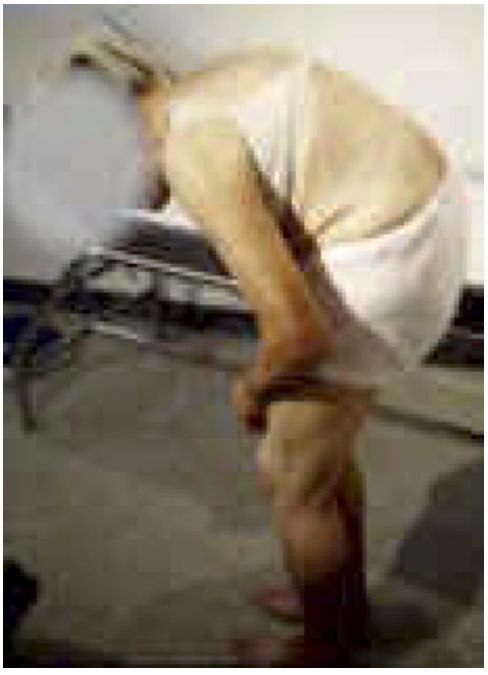

Figure 1 Camptocormia.

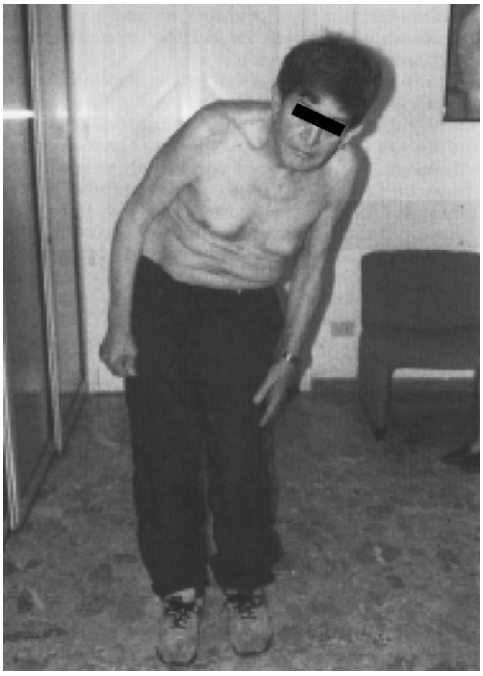

Figure 2 Pisa syndrome characterized by severe lateral flexion of the trunk and the neck.

Postural instability

Postural instability (PI) is one of the most disabling features of PD. It is due to a dysfunction of postural reflexes, which is generally a manifestation of the late stages of the disease, and usually occurs after the onset of non-motor symptoms [25].

In spite of their forward inclination in upright posture, PD patients tend to fall backwards very easily, with only a slight push, resulting in retropulsion [20]. Both axial rigidity and poor trunk coordination contribute to the poor stability of PD patients in response to backward body sway. Horak et al. [22] studied PD patients in their off-state and showed different stability margins for different directions of body sway. The smallest stability margin occurred for backward body sway in both narrow and wide stance, suggesting that PD patients are more vulnerable to falls in the backward direction. The reduced stability margin in PD patients was due to a slower rise and a smaller peak of their centre of pressure, when compared to control subjects. Therefore, widening the sustentation base is unlikely to help PD patients to prevent backward falls.

Beyond abnormal postural reflexes, several other factors may contribute to postural instability in PD patients as well as other parkinsonian symptoms: orthostatic hypotension, age-related sensory changes, and their ability to integrate visual, vestibular, and proprioceptive inputs.

This postural instability induces difficulties with transfers, gait disorders, inability to live independently at home, and is the major cause of falls [10]. Falls are frequent, with a 38% risk of falling found among 100 PD patients by Koller et al. [30], among these, 13% felt down more than once a week. Moreover, falls may induce adverse consequences like soft-tissue injuries, hip fractures, and fear of falling. As the disease progresses, both PI and fear of falling worsen, which leads PD patients to become increasingly immobilized. The long latency before the onset of falls differentiates PD from others neurodegenerative disorders, such as progressive supranuclear palsy (PSP) and multiple system atrophy (MSA). The average time from the onset of symptoms to the first fall was 108 months in patients with PD compared with 16.8 and 42 months in patients with PSP and MSA, respectively [54,55].

Evaluation of posture in Parkinson's disease

Static posturography

Static posturography consists in recording the displacements of the center of pression (CoP), using a force platform, during quiet stance. In these conditions, the CoP sways reflect patient instability. Reported results of static posturography in PD were often contradictory. Several studies reported that the body sway of PD patients is closed to normal under quiet stance, at least at the earlier stages of the disease [16,42]. By contrast, Horak et al. [23] reported a decrease in postural sway in PD patients, while Mitchell et al. [36] showed an increase of the postural sway in the mediolateral direction. For these authors, the mediolateral posturographic measures were also associated with a history of falls and a poor balance performance. Błaszczyk et al. [7] reported an increase of spontaneous sway indices and suggested that this method should be supplemented by additional measures of stability range such as the functional reach or maximal voluntary leaning.

The use of static posturography in PD is limited because of the heterogeneity of the results and because this method assesses only a single component of posture. Overall, these data suggest that actually the technics of postural analysis we have at our disposal are not suitable for clinical evaluation of postural troubles.

Dynamic posturography

The control of balance involves multiple components of postural control, including reactions triggered by external perturbations, antigravity muscle tone, and centrally-initiated postural adjustments preceding or accompanying voluntary movements [24]. Although balance under dynamic conditions has been rarely investigated in PD patients, some specific impairment of postural reflexes have been underlined. Analyses of postural reflexes in response to an unpredictable perturbation of the support (usually an unexpected toe-up tilt) showed that PD patients exhibit abnormal and ''inflexible'' postural reflexes, as reflected by an increase in amplitude and duration of the EMG response latency [9,40,44]. Beckley et al. [5] tested postural stability in PD during a sudden toe-up rotation of the supporting platform and found that PD patients have a fundamental difficulty in modifying the size of long-latency postural reflexes, which may be one factor contributing to their postural instability.

Using perturbations of the supporting surface in the lateral and sagittal planes, Horak et al. [22] showed that PD patients have directionally specific postural instability. Same dissociation between antero-posterior and lateral control of

posture were also demonstrated in studies assessing postural control of PD patients during slow oscillation of the support [49] and during locomotor tasks [35].

Another kind of perturbation that may be used to analyse postural control consists in continuous and predictable perturbations of the supporting platform. In this case, postural control relies on both anticipatory adjustments and sensory feedback. Postural control impairments in PD have been poorly investigated under these conditions: Nardone and Schieppati [37] compared balance performance of non-fallers and fallers using both static and dynamic approaches. They found that some indexes of stability, coordination, and anticipation significantly differed without vision particularly in PD fallers, and that the dynamic approach is the most sensitive tool for detecting instability in PD fallers.

Evaluation of postural orientation

Amblard et al. [1] suggested that the existence of a dual postural control system, one part of which deals with body orientation with respect to gravity, and the other with body stabilization. Both postural control systems do not operate independently but probably interact, which provides a stable physical basis for perception and action.

The control of postural stabilization (i.e., balance) has been widely studied in Parkinson's disease at both the overall [22,43] and segmental levels [35], probably because falling has such deleterious effects on daily life in parkinsonian patients. By contrast, little attention has been paid so far to the control of body verticality, even if clinical findings provided evidence that it is impaired in PD. Vaugoyeau et al. [49] analyzed both postural components, i.e. orientation and stabilization, independently and showed that PD patients presented a postural orientation deficit, which developed earlier than postural stabilization deficits.

Impairment of anticipatory postural adjustments (APA) and posturo-kinetic coordination analysis

Voluntary movement is usually accompanied or preceded by an adjustment of posture aimed at preventing the disequilibrium generated by the movement [34]. Lee et al. [31] analyzed APA in a lateral leg-raising task in PD patients. They showed that in the more severely affected parkinsonian patients, the amplitude of the initial displacement of CoP was markedly reduced, the interval between the earliest force changes and the onset of leg elevation was prolonged, and the relative timing of the kinematics adjustments during this interval was disrupted. The authors concluded that abnormalities in programming APA might contribute to postural instability in Parkinson's disease.

Considerable attention has also been focused on gait initiation impairments in PD [11,18,39,51]. The main abnormality consisted in an increased duration of the postural phase and a decrease in propulsive forces during postural and movement phases. As a result, the step length was shorter and the step velocity slower [15,50]. Marchese et al. [33] analysed PD and showed that dual task interfered on postural control and that the deterioration was significantly higher if patients had a prior history of falls.

Segmental control of posture in Parkinson's disease

During quiet stance or movement, maintenance of the equilibrium is not the single function of postural control that needs to be achieved. The stabilization of the head with respect to space has to be maintained in order to serve as an egocentric reference value for maintaining balance [1,2,3,6]. Mesure et al. [35] showed that, in contrast to control subjects, PD patients used, mainly in the lateral plane, a strategy of head stabilization on the shoulder (''en bloc'' functioning), irrespective of whether they were or were not under anti-parkinsonian medication.

Sensory integration

Erect postural control (orientation and stabilization) is known to depend on vestibular, visual, and somatosenrory information. It has been recently established that motor deficits are not isolated deficits and that PD is also associated with impairments in sensory integration. Therefore, it seemed worth investigating the possibility that sensorimotor integration deficits may also partly account for the postural impairments observed in PD.

Kitamura et al. [29] reported that in the absence of visual cues, the position of the CoP shifted significantly backward in patients with PD, whereas it shifted significantly forward in age-matched control subjects. This provides further evidence that visual inputs contribute to maintaining upright posture in PD patients. Similar results were obtained by Bronstein et al. [12], who studied the postural responses to slow displacements of the visual environment. In this situation, PD patients produced exaggerated responses, probably due to hyperactivity of the visuo-postural loop.

Concerning the proprioceptive contribution to postural control in PD, Smiley-Oyen et al. [45] showed that a similar adaptation to vibration occurred in both PD patients and control subjects. The authors suggested that, in PD patients, proprioceptive impairments might affect kinesthetic abilities more than their postural control. Vaugoyeau et al. [49] assessed the proprioceptive contribution to postural control in PD by using particular perturbation of the supporting platform and showed that proprioceptive impairments contribute to disturb postural orientation control. They suggested that, in PD patients, increased visual dependence might reflect the adaptive strategy to compensate their proprioceptive deficits.

Treatment

The response of axial symptoms to levodopa treatment is usually modest. This can be explained by the fact that, when postural abnormalities and postural instability appear, patients are already at an advanced stage of the disease with severe axial symptoms and gait disorders, all symptoms known to respond poorly to levodopa, suggesting the implication of non-dopaminergic pathways [13]. Furthermore, the postural reactions to support surface perturbations are resistant to dopaminergic therapy [14]. Because of the implication of non-dopaminergic pathways in postural instability, further studies are needed to identify new therapeutic tools to reduce postural disturbances in PD.

Stereotactic neurosurgery of the basal ganglia is a therapeutic alternative for patients with advanced PD. Deep brain stimulation (DBS) of the *globus palllidus pars interna* (Gpi) or of the subthalamic nucleus (STN) is associated with significant improvement of motor symptoms in patients with severe PD. DBS of the NST improved all the cardinal features of PD and daily living activities, and reduced significantly levodopa-induced dyskinesias and motor complications. On the other hand, axial symptoms (falling, freezing, gait, standing, posture, and postural instability) are less improved by STN stimulation than distal motor involvement [53]. A multicenter study assessing long-term efficacy of either STN or GPi-DBS showed at three to four years a significant worsening in the on-medication motor states of gait in both STN and GPi groups, and of speech and postural stability in the STN-treated group. Targeting other nuclei for DBS, such as the zona incerta and pediculopontine nucleus, is currently explored to improve gait and posture in patients stimulated in the STN [46]. Six patients with unsatisfactory pharmacological control of axial signs such as gait and postural stability underwent bilateral implantation of DBS electrodes in the STN and PPN. Clinical effects were evaluated two to six months after surgery with both STN and PPN stimulation ''On or Off'', or with only one target being stimulated. Bilateral PPN-DBS at 25 Hz in Off-medication produced an immediate 45% amelioration of the motor score, followed by a decline to give a final improvement of 32% after three to six months. PPN-DBS was particularly effective on gait and postural items. In On-medication state, the association of STN and PPN—DBS provided a significant further improvement when compared to the specific benefit mediated by the activation of any single target. These findings indicate that, in patients with advanced Parkinson's disease, PPN—DBS associated with standard STN-DBS may be useful in improving gait and in optimizing the dopamine-mediated On-state, particularly in those whose response only to STN DBS has deteriorated over time. This combination of targets may also prove useful in extrapyramidal disorders, such as progressive supranuclear palsy.

Because of the poor effects of dopaminergic drugs and DBS on postural disorders, many PD patients are referred to a physiotherapist. Rehabilitation plays an important role in the treatment of PD. Most reviewed studies showed improvement of gait, everyday activities and quality of life after physical therapy. There is a general agreement about a positive influence of rehabilitation on postural instability and prevention of falls. Sensory cueing (visual, auditory or mental stimuli) reduces freezing of gait in PD [41].

Conventional physical therapy aiming at reducing parkinsonian kyphosis consists of lasting postures applied on shoulders and passive relaxation of the rachis. Moreover, rehabilitation significantly influences balance in PD patients [47]. Repetitive training of compensatory steps is an effective approach to enhance protective postural responses [26]. Another study measured the effect of two exercise-training programs on balance in PD in a randomized control trial [21,27]. Nine patients completed a balance improvement exercise program and six patients completed a balance improvement associated with a muscle-strengthening program. All patients underwent 10 weeks of high-intensity resistance training and/or balance training, three times a week on non-consecutive days. Balance was assessed before, immediately after training and four weeks later, using a computerized dynamic posturography. The results showed a significant improvement in balance score in both groups, but only the combined training induced a beneficial effecting at four weeks. Despite the small number of patients and the absence of control group, this study represents an informative pilot study. Thus, two therapeutic methods improve postural instability: repetitive training of compensatory steps and the balance and strength training [26]. The individual clinical examination helps to identify the deficit leading to fall and a therapeutic individually adapted method can be chosen.

Conclusions

Several factors contribute to postural impairments in PD patients, including disturbed postural reflexes, sensory deficits, postural-adjustment deficits, posturo-kinetic coordination impairments. Further studies are now required to determine the links among all these postural components. It still remains to be determined whether any interactions occur between the loss of postural orientation control and that of postural stabilization control. Another point, which needs to be addressed, is to what extent these larger postural deficits that were observed in several studies contribute to the unsteadiness and falls to which PD patients are particularly exposed.

Conflicts of interest

We declare that we have no conflict of interest with the present manuscript.

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