IMPAIRED VERTICAL POSTURAL CONTROL AND PROPRIOCEPTIVE INTEGRATION DEFICITS IN PARKINSON’S DISEASE

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Abstract—The aim of the present study was to investigate how the orientation and stabilization components of postural control may be affected as the result of the impaired proprioceptive integration possibly occurring in Parkinson’s disease.

To determine the proprioceptive contribution to postural control, parkinsonian patients and control subjects were asked to maintain vertical stance while very slow sinusoidal oscillations were being applied in the lateral and antero-posterior planes to the platform on which they were standing. The amplitude and frequency of their movements were kept below the semicircular canal perception threshold. Data were collected with the ELITE automatic motion analyzer and the two postural components (orientation and segmental stabilization) were analyzed at head and trunk levels while the subjects were performing the task with their eyes open and closed.

The results show that 1) the parkinsonian groups’ performances were affected in terms of both the postural orientation and stabilization components in comparison with the control group, 2) the use of vision improved the parkinsonian patients’ postural performances, and 3) both parkinsonian patients and control subjects achieved better postural performances when antero-posterior perturbations rather than lateral perturbations were applied to the foot support.

 These results suggest that Parkinson’s disease is associated with proprioceptive impairment, which may be an important factor contributing to these patients’ postural deficits. On the basis of these results, the visual dependence observed in parkinsonian patients is re-defined as an adaptive strategy partly compensating for the impaired proprioception. © 2007 IBRO. Published by Elsevier Ltd. All rights reserved.

Key words: postural orientation, segmental stabilization, proprioceptive integration, Parkinson’s disease.

Postural control involves adopting an appropriate body orientation, which is generally aligned with the gravity vector, and maintaining this posture despite the perturbing effects of gravity and other external forces. Amblard et al. (1985) have suggested the existence of a dual postural control system, part of which deals with body orientation with respect to gravity and the other, with body stabilization. These two postural control systems do not operate independently but probably interact, providing a stable physical basis for perception and action. When organizing a given movement, the CNS has to coordinate the control of body balance and that of body orientation.

Erect postural control (orientation and stabilization) is known to depend on vestibular, visual and somesthetic information, arising from sensory sources such as muscles, skin, and joints. In a previous study, Vaugoyeau (2001) established that in the absence of visual information, oscillatory perturbations applied to the foot support below the vestibular perception threshold did not affect healthy young subjects’ ability to control vertical posture. The authors concluded that somesthetic cues suffice to control postural orientation and stabilization in normal young adults. Mittelstaedt (1983) has reported that proprioception provides highly accurate information contributing to the maintenance of body verticality. Darling and Hondzinski (1997) have suggested that somatosensory cues may contribute more usefully than otolithic cues to the perception of body orientation in normal young adults. Higashiyama and Koga (1998) have reported that healthy subjects’ perception of body tilt may be subserved by a single sensory process based on messages arising from cutaneous and muscle proprioceptors rather than on vestibular and joint proprioceptive cues. Experiments performed under microgravity conditions have also shown that the absence of static otolithic inputs does not affect the control of body verticality (Amblard et al., 1997).

Parkinson’s disease, which has usually been described as a motor disorder, is characterized by a triad of clinical symptoms, including bradykinesia and hypokinesia, tremor, and rigidity, resulting from nigrostriatal denervation (Barbeau, 1986). Parkinsonian patients have difficulty in performing various motor tasks, such as walking, writing and speaking. Their postural control is also impaired, and the postural instability which occurs during the later stages of the disease, which is poorly DOPA-responsive, is one of the main symptoms of the disease. The control of postural stabilization has been widely studied at both the overall (Schieppati et al., 1994; Horak et al., 2005) and segmental levels (Mesure et al., 1999), probably because falling has such serious effects on the daily life of parkinsonian patients. Little attention has been paid so far, however, to the other component of posture, namely the control of body verticality, in Parkinson’s disease, although clinical findings and experimental studies have recently provided evidence that the control of verticality is impaired in Parkinson’s disease. Parkinsonian patients have a characteristically stooped posture, since they tend to bend the head and trunk forward. The fact that bent spinal posture
was found by Nieves et al. (2001) to be associated with lenticular lesions suggests that the basal ganglia may play an important role in the maintenance of axial posture. On the other hand, defective control of axial movements has been described in Parkinson’s disease. Steiger et al. (1996) have reported that PD patients have difficulty in coordinating the orientation of the superimposed axial segments along the spinal axis. Schenkman et al. (2001) have observed a decrease in parkinsonian patients’ ability to perform longitudinal spinal rotation while seated during the performance of a reaching task. Vaugoyeau et al. (2001) recently described the time course of body reorientation control impairments in parkinsonian patients.

It was recently established that motor deficits are not isolated deficits and that Parkinson’s disease is also associated with sensory integration impairments. The first evidence showing the existence of impaired proprioceptive integration abilities in these patients was provided by a study by Tatton and Lee (1975) on the stretch reflex, in which the latter component of this reflex was found to be enhanced in parkinsonian patients. It was also established in this study that the contribution of proprioceptive feedback information to static position and movement perception decreases in parkinsonian patients (Rickards and Cody, 1997; Zia et al., 2002; Keijsers et al., 2005).

Since the proprioceptive mode is thought to contribute mainly to the orientation component of posture, it seemed to be worth investigating the possibility that sensorimotor integration deficits may also partly account for the postural impairments observed in Parkinson’s disease. With this possibility in mind, an original experimental procedure was used, which makes it possible to dissociate the postural orientation and stabilization components and to isolate the somesthetic information, including proprioceptive, cutaneous and articular information. This procedure consists in subjecting standing subjects to small angular sinusoidal perturbations applied to the supporting platform at frequencies below the perception threshold of the semicircular canal system. Under these conditions, the postural control strategies used by the subjects with their eyes closed are mainly based on the use of somesthetic information.

First it was proposed to analyze the two postural components, i.e. orientation and stabilization, independently in parkinsonian patients in order to check the hypothesis that the postural orientation component is that which is mainly impaired in Parkinson’s disease, at least in the earlier stages of the disease.

Secondly, it was proposed to check the hypothesis that proprioceptive integration deficits are responsible for the postural orientation and postural stabilization difficulties encountered by patients with Parkinson’s disease.

**EXPERIMENTAL PROCEDURES**

**Subjects and tasks**

Eleven parkinsonian patients (aged 60.1 ± 6.4 years) and 10 control subjects (aged 58.1 ± 6.2 years) took part in these experiments, which were approved by the local ethical committee. All the subjects gave their informed consent prior to the study. The patients’ characteristics are given in **Table 1**.

**Subjects stood on a motor-driven one-directional rotating platform with their eyes closed, and the platform was rotated sinusoidally at a frequency of 0.01 Hz (10° peak to peak) in either the pitch or roll direction, depending on the subjects’ position on the platform. They had to maintain a vertical posture as steadily as possible, keeping their feet 15 cm apart without flexing their knees. The trial lasted for 106 s, including a complete cycle of angular platform movement. The maximum angular accelerations of the platform were thus well below the vestibular detection threshold, namely 0.2°/s² (Henn et al., 1980). Therefore, if any angular head accelerations occurred beyond this threshold value, they would not result directly from the platform movements and would not be involved in correcting the experimentally induced postural disturbances.

**Fig. 1** gives the characteristics of the supporting platform movements in terms of the angular displacements, angular velocity and angular acceleration. The maximum inclination was 5°, the maximum velocity was 0.31°/s and the maximum acceleration was 0.02°/s².

**Data collection and kinematic analysis**

Data collection was performed with the ELITE automatic motion analyzer (BTS, Milan, Italy) working at 100 Hz, using passive body markers (Ferrigno and Pedotti, 1985). System accuracy was assessed to be 1/3000 of the maximum value of the useful acquisition volume, which amounts to an error of less than 1 mm in the markers’ three-dimensional position.

The subject performed the task in front of a pair of ELITE TV cameras in the case of lateral platform oscillations, or in side view (right profile) in the case of antero-posterior oscillations (see **Fig. 2**). In the case of lateral oscillations, the subjects wore 12 markers (15 mm in diameter) on the following anatomical landmarks: left and right mastoids (1, 2), spinal process of C7 (9), left and right acromial processes (3, 4), left and right iliac crest (5, 6), sacrum (10), left and right trochanter (7, 8) and left and right malleolus (11, 12).

In the case of antero-posterior platform oscillations, the subjects wore eight markers on the right side of the body: the meatus and infra-orbital margin (markers 1, 2), acromion (3), iliac crest (4), trochanter (5), tibial plate (6), medial malleolus (7) and 5th Meta-

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**Table 1. Patients’ characteristics**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (y)</th>
<th>Disease duration (y)</th>
<th>Hoehn-Yahr stage</th>
<th>UPDRS motor score ON</th>
<th>UPDRS motor score OFF</th>
<th>DOPA (mg/d)</th>
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<td>4</td>
<td>II</td>
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</table>

**Characteristics of the disease of the 11 parkinsonian patients: age, disease duration, Hoehn-Yahr stage, UPDRS part III in the ON state performed at the time of testing (testing was performed while the patient was in the ON state), UPDRS part III in the OFF state performed 12 h after the withdrawal of dopamine treatment, dosage of L-DOPA.**
During both tasks, two markers were placed on the platform to measure its movements. In each experiment, each subject was subjected to both types of disturbances, namely lateral and anterior-posterior platform oscillations occurring in a pseudo-random order.

**Controlled variables**

The absolute angular orientations (with respect to laboratory axes, see Fig. 3) of the various segments under consideration were computed every 10 ms during each trial. When lateral perturbations were applied to the support, the absolute angular orientation was calculated in the roll component of rotation, i.e. the rotation around the sagittal axis in the lateral plane. When antero-posterior perturbations were applied to the support, the absolute angular orientation was calculated in the pitch component of rotation, i.e. the rotation around the lateral axis in the sagittal plane.

On the basis of the angular variation as function of time, various controlled variables were calculated to determine both segmental orientation (sequential mean orientation, segmental angular dispersion) and stabilization (anchoring index (AI)) values.

**Sequential mean orientation.** For each subject a reference orientation value was obtained for each body segment during 10 s of upright stance on a stable support with the eyes open. At each trial, the mean value of the absolute angular variation as function of time was calculated for each body segment (head, shoulders, trunk and pelvis in response to lateral oscillations of the platform; and head, trunk and thigh in response to antero-posterior oscillations of the platform). This value was calculated during a whole cycle (100 s) of platform movement in the plane of the perturbation.

To obtain the mean orientation of one segment during one trial, the reference value was subtracted from the mean value of the absolute angular distribution recorded during that trial. Likewise, the sequential mean orientation of each body segment was calculated using the same procedure, during each tenth of a cycle (10 s) of platform movement, in order to assess the time course of the segmental orientation process.

**Segmental angular dispersion.** The oscillations induced in each anatomical segment by the movement of the supporting platform were assessed in terms of segmental angular dispersion: at each trial, the standard deviation (i.e. the dispersion, denoted $\sigma$) of overall angle orientation values during the considered trial was calculated. The angular dispersion gives a first indication of the oscillations of a given segment in the frontal or sagittal plane during the perturbation. This variable indirectly provides some information about the attenuation of the perturbation at the anatomical level considered. When the angular dispersion of a body segment is smaller than that of another body segment, this indicates that the first body segment has moved less than the second one, or in other words, that the perturbation is more attenuated at the former anatomical level.

**Fig. 1.** Movements of the supporting platform. The first curve gives the angular displacements of the supporting platform. The second curve gives the angular velocity of the supporting platform and the third one gives the angular acceleration of the supporting platform. The arrows indicate the peak inclination, velocity and acceleration.

**Fig. 2.** Left side: Arrangement of the markers used to measure the effects of the lateral disturbance applied to the supporting platform. The 12 markers were placed at the following sites: left and right mastoids (1, 2), spinal process of C7 (9), left and right acromial process (3, 4), left and right iliac spinal crests (5, 6), sacrum (10), left and right great trochanters (7, 8) and left and right lateral malleoli (11, 12). Right side: Positions of the markers used to measure the effects of antero-posterior disturbances. The eight markers were placed on the right profile of the subject at the following sites: meatus and infra-orbital margin (1, 2); acromion (3); iliac crest (4), trochanter (5); tibial plate (6), medial malleolus (7) and 5th metatarsal (8). Two additional markers were also placed on the platform to measure its lateral and antero-posterior movements (13 and 14 and 9 and 10).
AI. Segmental stabilization was defined in terms of the global AI calculated during a whole perturbation cycle (Amblard et al., 2001; Assaiante and Amblard, 1993; Assaiante et al., 1993; Borel et al., 2002; Nadeau et al., 2003; Isableu et al., 2003).

The AI was used to determine the stabilization of a given segment with respect to both space and the moving platform. With regard to the head AI for example, the angular orientation of the head relative to the support was first calculated every 10 ms during a trial using the formula:

\[ \theta_a = \theta_r - \theta_s \]

In this formula, \( \theta_a \) is the angular orientation of the head relative to the support, and \( \theta_r \) and \( \theta_s \) are the absolute head and support angular orientations, respectively.

At a given trial, the standard deviation of the relative angular distribution (\( \sigma_r \)) and the standard deviation of the absolute angular distribution (\( \sigma_a \)) were calculated for each anatomical segment.

The AI was then calculated at each trial as follows, as shown in Fig. 3.

\[ AI = \frac{\sigma_a^2 - \sigma_r^2}{\sigma_a^2 + \sigma_r^2} = 0.93 \]

Values of AI may vary between −1 and +1. A positive value of AI for a given segment indicates that a better segmental stabilization occurred with respect to space than with respect to the supporting platform, in other words, that the reference frame used to achieve postural stabilization was space, whereas a negative value indicates that a better segmental stabilization occurred with respect to the platform than with respect to space, in other words, that the reference frame used to achieve postural stabilization was the support.

Statistical analysis

The statistical analyses were performed using the Statistica software program. Descriptive statistics are given here in the form of medians and interquartiles.

Differences between control and parkinsonian groups were tested with a Mann-Whitney U test. The effects of vision were analyzed by comparing the performances of each group with and without vision, using Wilcoxon’s signed rank test for within-subject comparisons.

AIs were compared with zero, using a single-sample procedure (t-test) against the null hypothesis. Since these indices were in the −1 to +1 range, a z transform was used to convert the values into an unbiased Gaussian distribution.

Differences with a P value <0.05 were taken to be statistically significant.
Fig. 4. Medians and 1st and 3rd quartiles of head and trunk sequential orientations in response to lateral movements/oscillations applied via the supporting platform with vision (white symbols) and without vision (black symbols) in control subjects (left side) and parkinsonian patients (right side). EC, eyes closed; EO, eyes open.
Fig. 5. Medians and 1st and 3rd quartiles of head and trunk sequential orientations in response to antero-posterior oscillations applied to the supporting platform with vision (white symbols) and without vision (black symbols) in the control subjects (left side) and parkinsonian patients (right side). EC, eyes closed; EO, eyes open.
RESULTS

Control subjects versus parkinsonian patients

Postural orientation: sequential orientation and angular dispersion. The time course of the head and trunk orientation occurring in response to the perturbation applied to the supporting platform is shown in Figs. 4 and 5 (in the case of a lateral perturbation of the platform and that of an antero-posterior perturbation of the platform, respectively). In control subjects, this sequential orientation process showed few variations with time, which showed that the oscillations induced in the various anatomical segments, were efficiently attenuated. Without the use of vision, however, the control subjects tended to slightly follow the movement of the platform, especially at trunk level.

The parkinsonian patients followed the movement of the supporting platform in both perturbation planes under both visual conditions at head and trunk levels, which shows that the attenuation of the oscillations was less efficient than in the control subjects, especially in the absence of vision.

The attenuation of the oscillations induced in the anatomical segments was assessed in terms of the segmental angular dispersion. The median, 1st and 3rd quartile values are given in Figs. 6 and 7 (when lateral perturbations and antero-posterior perturbations were applied to the platform, respectively).

When lateral perturbations were applied to the supporting platform, with and without vision, no statistically signifi-

cant difference in the angular head dispersion was observed between groups, whereas the angular trunk dispersion was significantly greater in the parkinsonian patients under both visual conditions (U=129, P=0.01; U=149, P<0.001 with and without vision, respectively).

When antero-posterior perturbations were applied to the supporting platform, no statistically significant difference in the angular head dispersion was observed between groups when visual cues were available. Without the use of vision, the angular head dispersion was significantly greater in the parkinsonian group than in the control group (U=102, P<0.05) under the same condition. The angular trunk dispersion was significantly greater in the parkinsonian group than in the control group under both visual conditions (U=145, P<0.05; U=154, P<0.01 with and without vision, respectively).

Postural stabilization: Als. The head and trunk Als are given in Figs. 8 and 9 (in the case of lateral perturbation and antero-posterior perturbation of the supporting platform, respectively).

When lateral perturbations were applied to the foot support, the parkinsonian patients’ head Al was positive when visual cues were available (t=2.34, P<0.05) but this index did not differ from zero when no visual cues could be used. These results indicate that when deprived of vision, the parkinsonian patients could no longer use the strategies normally adopted for stabilizing the head in space. At trunk level, the Al obtained did not differ significantly from
zero when visual cues were available, but became negative when the subjects were deprived of vision.

The control subjects undergoing lateral perturbations obtained statistically positive AIs at head and trunk level when visual cues were available (t=8.01, P<0.001; t=3.48, P<0.01 for head and trunk, respectively). Without vision, the head AI was still statistically positive (t=2.84, P<0.05), while the trunk AI did not differ from zero under these visual conditions.

When antero-posterior perturbations were applied to the supporting platform, the AIs were significantly positive in all the segments investigated, both with and without vision (W=66, P<0.001 and W=58, P<0.01 at head and trunk level, respectively). No significant differences in the head AIs were observed, however, between groups either with or without vision.

Role of vision in the two groups

The effects of vision on the control subjects’ and parkinsonian patients’ postural performances were investigated.

Postural orientation: angular dispersion. Lateral perturbation of the supporting platform. Control subjects showed greater head and trunk angular dispersions without vision than with vision (W=66, P<0.001 and W=58, P<0.01 at head and trunk level, respectively).

Segmental stabilization in the frontal plane

Fig. 8. Median with 1st and 3rd quartiles of head and trunk AIs with respect to the supporting platform when lateral perturbations were applied to the supporting platform with vision (white symbol) and without vision (black symbol) in control subjects (CS) and parkinsonian patients (PP).

Segmental stabilization in the sagittal plane

Fig. 9. Median with 1st and 3rd quartiles of head and trunk AIs with respect to the supporting platform when antero-posterior perturbations were applied to the supporting platform with vision (white symbol) and without vision (black symbol) in control subjects (CS) and parkinsonian patients (PP).
A similar tendency was observed in the parkinsonian group (W = 37, P < 0.05 and W = 37, P < 0.05 at head and trunk level, respectively).

Antero-posterior perturbation of the supporting platform. In control subjects, no significant difference was found to exist in the head and trunk dispersions between the two conditions of vision.

The parkinsonian patient showed a greater trunk angular dispersion than the control subjects without vision (W = 43, P < 0.01), but the head angular dispersion was similar under both visual conditions.

Postural stabilization: AIs. Lateral perturbation of the supporting platform. Comparisons between the head AIs calculated in control subjects with and without vision did not show the existence of any significant differences, whereas the trunk AI was lower without vision (W = 55, P < 0.01); with vision, the trunk AI was positive, whereas in the absence of vision, it was not significantly different from zero.

In the parkinsonian group, contrary to the control group, the head AI was significantly reduced without vision (W = –41, P < 0.05). This index was positive with vision and negative without vision, which indicates that different modes of head stabilization were used in each case. Statistical analysis of the trunk AI also showed the existence of a significant difference between the two conditions of vision in the parkinsonian patients (W = –34, P < 0.05); this group was found to use no preferential trunk stabilization strategy with vision (the AI was almost equal to zero) and stabilized their trunk with respect to the foot support without vision, as shown by the negative value of the AI.

Antero-posterior perturbations of the supporting platform. In healthy subjects, no significant difference was observed between the head AI values with and without vision. The significant positive AI obtained by this group indicated that the segments were all stabilized in space under both visual conditions. A similar result was obtained with the trunk AI: both with and without vision, the controls subjects stabilized their trunk in space, as shown by the positive AIs values.

In the parkinsonian group, the head AI values differed significantly between the two conditions of vision (W = –24, P < 0.05). However, the significantly positive AIs obtained indicated that the head was stabilized in space both with and without vision, as in the control subjects. The trunk AI values decreased significantly without vision (W = –34, P < 0.05), but these indices were still significantly positive.

**DISCUSSION**

The aim of the present study was to investigate how the orientation and stabilization components of postural control are affected by the hypothetical loss of proprioceptive integration occurring in parkinsonian patients. For this purpose, an original procedure was used, consisting in applying slow oscillations to the platform on which the subjects were standing. The main point of this procedure is that it makes it possible to isolate the somesthetic contribution to postural control by abolishing the information processed by the semi-circular canals as well as the visual information (when the subjects’ eyes are closed). The contribution of visual inputs can then be assessed when the subjects open their eyes. The main results to emerge from this study were as follows: 1) the parkinsonian groups’ postural performances were impaired in terms of both the postural orientation and postural stabilization components under all the conditions tested, 2) vision improved the parkinsonian patients’ postural performances, and 3) both the parkinsonian and control subjects achieved better postural performances when antero-posterior perturbations were applied to the foot support when the perturbations were applied in the lateral plane.

**Specific contribution of sensory information to postural control under quasistatic conditions**

The specific contribution of somesthetic cues—arising from sensory sources such as muscle, skin, and joints—possibly along with otolithic information, was tested in the condition where the subjects performed the task with their eyes closed. In this condition, the performances of the parkinsonians were much less efficient than those of the control group. Parkinsonian patients were unable to maintain the vertical head and trunk orientation without vision; whereas the control subjects kept their body upright when deprived of visual cues and vestibular information, and the pattern of sequential mean orientation occurring in response to foot support perturbations showed few variations in this group whatever the anatomical level considered. These results show that the parkinsonian patients, unlike the control subjects, were unable to properly control their postural orientation and stabilization on the basis of the sensory information (i.e. somesthetic and otolithic information) available in this experimental condition.

**Otolithic information and postural orientation control: why do we rule out the possible contribution of an otolithic contribution to postural control under quasistatic conditions?**

The otolithic system is theoretically capable of detecting all frequencies up to null level (Nashner, 1971), and this system can therefore provide subjects with information about the vertical pull of gravity. However, it has been reported that the accurate perception of body orientation under quasi-static conditions depends mainly on somatosensory rather than otolithic information and that the vestibular inputs may become much more useful in more dynamic situations (Teasdale et al., 1999). In addition, Bringoux et al. (2002) have shown that subjects deprived of vestibular information are still able to perceive a very slow change in their body orientation in complete darkness just like normal subjects, which shows that orientation control does not require the use of the otolithic pathway. This hypothesis was confirmed by data on patients with acute unilateral vestibular disorders, whose ability to estimate the subjective postural vertical was not affected (Bronstein, 1999).
Defective proprioceptive processes in Parkinson’s disease: effects on postural control. A vestibular dysfunction might explain the postural deficits of parkinsonian patients. However, no evidence is available so far that dysfunction of the vestibular system occurs in Parkinson’s disease. Pastor et al. (1993) tested the possibility that dysfunction of the vestibular control of posture may be one of the main causes of parkinsonian instability and observed no significant differences between patients and controls in either the speed or direction of the body sway responses induced. These authors therefore concluded that vestibular dysfunction does not account for the postural deficits occurring in Parkinson’s disease.

The functional integrity of parkinsonian patients’ vestibular system established by Pastor et al. (1993) and our own data therefore suggest that the postural deficits occurring in these patients under quasistatic conditions may be associated with proprioceptive integration deficits rather than with vestibular impairments.

Previous studies have dealt with the lack of integration of proprioceptive information in Parkinson’s disease. Proprioceptive sensory processes have been investigated in parkinsonian patients using proprioception-related evoked potentials, for example (Seiss et al., 2003). The results of the latter study showed the occurrence of quite normal early proprioception-related potentials, but changes were observed in the cortical processing of longer-latency kinesthetic signals. Other authors have documented the difficulties encountered by parkinsonian patients in using proprioceptive information about how their limbs move in space (Klockgether and Dichgans, 1994 and Demirici et al., 1997; Klockgether et al., 1995).

Recent studies have attributed the impaired motor control performances observed in parkinsonian patients at least partly to proprioceptive integration deficits. Impaired proprioceptive integration has been observed in a precision gripping/lifting task (Fellows et al., 1998), in ankle dorsi-flexion (Khudados et al., 1999) and during locomotion toward a target (Almeida et al., 2005). However, few studies have systematically examined the contribution of vision and proprioception to postural control in Parkinson’s disease. One study, in which vibration was applied to the soleus muscle to elicit proprioceptive illusions, has focused, however, on the proprioceptive contribution to postural control in parkinsonian patients (Smiley-Oyen et al., 2002). The results showed that a similar adaptation to vibration occurred in both parkinsonian patients and control subjects. The authors suggested that proprioceptive impairments may affect parkinsonians’ kinesthetic abilities more than their postural control. The present results show, on the contrary, that impaired proprioception is responsible, at least partly, for parkinsonians’ inability to adopt and stabilize an upright body posture. Two hypotheses can be put forward to explain the discrepancies between this study and our own. First, the patients in our study had reached a more advanced stage of Parkinson’s disease: they were mostly in stage III, which means that clinical signs of postural involvement were present, while in the other study, all the patients were at stage I and II, which means that there were no clinical signs of postural dysfunction. Moreover, it has been established that the sensory deficits increase with the duration of the disease, as established by Keijzers et al. (2005).

Secondly, in the study by Smiley-Oyen et al. (2002), the authors examined the subjects’ postural sways, which involve the stabilization component more than the orientation one, whereas we ourselves focused rather on the orientation component of posture. In response to perturbations applied in both planes, these patients tended to follow the movements of the supporting platform rather than attenuating them. These results are in line with the existence of a dual system of postural control, as previously suggested by Amblard et al. (1985). They suggest first that the control of postural stabilization may depend less than the control of body verticality on proprioceptive information and secondly, that the control of body verticality may be impaired before the control of balance in Parkinson’s disease. This confirms that the distinction between these two postural components provides a promising approach to analyzing the postural disturbances occurring in Parkinson’s disease.

It can be concluded from the results of this study and those of previous studies that the processing of somesthetic information is impaired in parkinsonian patients during the later stages of the disease, and that these impairments are at least partly responsible for these patients’ postural orientation difficulties. The question as to whether these proprioceptive deficits are of central origin still remains to be answered. However, data based on the use of somatosensory evoked potentials and neurophysiological findings argue in favor of an abnormal central processing of proprioceptive cues rather than the existence of peripheral deficits (Seiss et al., 2003).

Visual information is used by parkinsonian patients to compensate for proprioceptive impairments. To determine whether parkinsonian patients make greater use of visual information than normal subjects for postural control purposes, we compared the postural performances of our two groups of subjects with and without vision. In these experiments, the use of visual information improved the postural performances of the parkinsonian patients, in terms of both the orientation and the stabilization of the upper body segments, in both perturbation planes. The parkinsonians’ postural orientation ability to respond to oscillations applied to the supporting platform improved when their eyes were open, without reaching the performance levels of the control subjects, however. In the absence of visual cues, when lateral oscillations were applied to the foot support, these patients were unable to stabilize their head and trunk in space and adopted an “en bloc” strategy instead, as attested by the negative head and trunk AIs obtained. When the patients’ eyes were open, their postural performances improved, since they used an articulated mode of stabilization, as shown by the positive AIs obtained. It emerged quite clearly that in the absence of visual cues, the parkinsonian patients were unable to
use proprioceptive information to control their body verticality and stabilize the body segments.

Several studies on parkinsonian patients have shown that these patients rely heavily on visual information when performing tasks involving arm movements, (Klockether and Dichgans, 1994). An increased dependence on visual information was also previously observed in parkinsonian patients’ perceptual and gait control abilities (Azulay et al., 1999, 2002). In the specific case of postural tasks, Kitamura et al. (1993) have tested the effects of vision on upright posture in patients with Parkinson’s disease. These authors reported that in the absence of visual cues, the position of the center of pressure shifted significantly backward in patients with Parkinson’s disease, whereas inagematched control subjects, it shifted significantly forward. The present study provides further evidence that visual inputs contribute to maintaining upright posture in parkinsonian patients. Similar results were obtained by Bronstein et al. (1990), who studied the postural responses of parkinsonian patients to slow displacements of the visual environment. In this situation, the parkinsonian patients produced exaggerated responses, probably due to hyperactivity of the visuo-postural loop.

**Antero-posterior versus lateral postural control**

Comparisons on the subjects’ responses to the two types of perturbation (lateral versus antero-posterior oscillations) showed that control subjects and parkinsonian patients both achieved better postural performances when they had to overcome antero-posterior perturbations, mainly concerning segmental stabilization strategies. Similar results were previously obtained during locomotion, when parkinsonian patients, contrary to normal subjects, adopted an “en bloc” strategy to stabilize their posture in the lateral plane, whereas no differences were observed between parkinsonian and control groups as regards the mode of stabilization used in the antero-posterior plane (Mesure et al., 1999).

However, the use of multi-joint postural strategies has been previously described in response to perturbations in both planes (Nashner and McCollum, 1985; Lekhel et al., 1994; Mesure et al., 1997). Winter et al. (1996) have suggested that when the supporting platform is unstable, lateral equilibrium may be regained by using a stabilizing strategy at hip level, whereas antero-posterior equilibrium may be regained by operating at ankle level. In our experiments, the parkinsonian patients tended to be more rigid when responding to perturbations in the lateral plane and more flexible when responding to those in the antero-posterior plane. Parkinson’s disease might increase the difference between the subjects’ motor performances in these two planes, which are extremely slight in normal control subjects.

**Study limitations**

Our data suggest that somesthetic information contributes importantly to postural orientation. It is worth noting, however, that our results focus on postural control under quasistatic conditions. Vestibular inputs may contribute much more to postural control in more dynamic situations (when more rapid tilting of the foot support occurs). In this connection, Bringuex et al. (2003) reported that trained subjects such as gymnasts were able to use vestibular inputs when very slow oscillations were applied to the supporting platform.

**CONCLUSION**

The results of the present study confirm a reduced ability of parkinsonian patients to properly use proprioceptive feedback. Proprioceptive impairments, therefore contribute to making postural control, especially postural orientation control, difficult for these patients. Parkinsonian patients’ increased dependence on visual cues when performing perceptual and motor tasks may reflect the adaptive strategy used by these patients to partly compensate for their proprioceptive deficits. As the disease progresses and the proprioceptive deficits increase, the strategy consisting in re-weighting sensory inputs in favor of the visual sensory mode may no longer suffice. The results of the present study also show that postural orientation deficits generally develop in parkinsonian patients earlier than postural stabilization deficits.

Further studies are now required on the links between these two postural components, since 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 may require to be addressed is to what extent the postural orientation deficits observed here contribute to the unsteadiness and falls to which patients with Parkinson’s disease are prone.

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