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Kinesthesia Is Impaired in Focal Dystonia

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Abstract: Parkinson's disease (PD) and focal dystonia (FD) are both predominantly characterized by motor symptoms. Also, recent research has shown that sensory processing is impaired in both movement disorders. FD is characterized by involuntary movements and abnormal limb postures; thus, abnormal kinesthesia could be involved in the pathogenesis. We examined passive index finger movements in patients with FD (n = 12) and PD (n = 11) and in age-matched healthy controls (n = 13). Compared to healthy controls, patients with PD and FD were significantly impaired in the correct detection of the movement direction. The perceptual thresholds for 75% correct responses of movement direction were 0.21 degrees for FD and 0.28 degrees for PD patients compared to 0.13 degrees in control subjects. Subjects with PD and FD were also significantly impaired when they had to judge consecutive amplitudes. Results of the present study point to impaired kinesthesia in FD. Defective sensory processing could be involved in the pathophysiology of the disease and may influence dystonic contractions. © 2006 Movement Disorder Society

Key words: kinesthesia; Parkinson's disease; focal dystonia; basal ganglia; proprioception

Kinesthesia is defined as the conscious perception of motion and direction of movements[1] and constitutes one aspect of proprioceptive processing. It relies on intact peripheral sensory input involving predominantly muscle spindles.[2] Kinesthesia is crucial in the context of movement execution and maintaining posture. Knowledge about what structures play a role in secondary processing of proprioceptive signals arriving at the somatosensory cortex is limited, but there is evidence that the basal ganglia are involved. Electrophysiological and psychophysical studies revealed that the dysfunction of the basal ganglia in PD leads to impaired kinesthesia.[3–9] Studies using somatosensory evoked potentials (SEPs) suggested a normal function of the peripheral structures in basal ganglia disorders[10–12] and normal early processing but a deficit in late processing of proprioceptive signals.[13]

Like PD, focal dystonia (FD; writer's cramp, torticollis, and blepharospasm) is regarded as a movement disorder. Knowledge on the underlying pathophysiology of FD is limited. Imaging studies provided evidence for a cerebral dysfunction as the pathophysiological basis of the disease.[14,15] Recent results from neurophysiological, neuroimaging, and animal studies suggest that sensory afferent activity is not adequately processed in FD.[16–19] Whereas previous SEP studies led to the speculation that a deficit in inhibition of sensory signals at a supraspinal level ("sensory overflow") could drive the motor disorder[20–24] other studies provided evidence for abnormalities of sensory afferent pathways from the muscle spindle ending.[16–18,25]

In the present study we sought to determine whether FD patients exhibit kinesthetic deficits. We used a psychophysical paradigm examining the detection of passive motion and the discrimination between consecutive amplitudes of passive finger movements. Because a kinesthetic deficit can be considered as an established feature of PD, PD patients serve as a second control group to validate the paradigm and apparatus.

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TABLE 1. *Characteristics of dystonia patients*

Patient no.	Age (yr)	Sex	Disease duration (yr)	Diagnosis	Fahn Torticollis Scale	Blepharospasm Rating Scale	BTX dose (units)
01	48	F	5	Bleph.		11	220
02	45	F	0.5	TC left	11		0
03	43	M	24	AC	12		480
04	54	M	8	TC left	21		300
05	53	F	9	TC right	32		1000
06	42	M	11	Bleph.		7	80
07	62	M	7	TC left	19		300
08	53	F	7	TC	14		340
09	43	M	4	Bleph.		8	120
10	64	F	15	TC left	24		950
11	44	F	0.5	TC right	23		0
12	56	M	8	TC right	17		1100

The Fahn Torticollis Scale and Blepharospasm Rating Scale were applied according to Bara-Jimenez.[24]
Bleph., blepharospasm; TC, torticollis; AC, anterocollis; BTX dose, cumulative dose Dysport per treatment every 12 weeks.

SUBJECTS AND METHODS

Subjects

Three different age-matched groups were examined: (1) 12 patients with idiopathic focal dystonias (blepharospasm n = 3, cervical dystonia n = 9), (2) 11 patients with idiopathic Parkinson's disease, and (3) 13 control subjects. Patient groups were recruited from the movement disorders outpatient clinic of the Department of Neurology, University Duisburg-Essen. All patients had a confirmed diagnosis of either idiopathic PD or a form of idiopathic FD. None of the patients suffered from psychiatric comorbidity.

Clinical characteristics of FD patients (mean ± SD: age, 50.6 ± 7.6 years; range, 42–64 years; 6 women, 6 men) are summarized in Table 1. FD patients (10 of 12, 2 without therapy) were tested 12 weeks after the last injection. Apart from botulinum toxin (BTX) therapy, none of the FD patients received any disease modulating or centrally acting medication.

All PD patients (mean ± SD: age, 55.9 ± 11.8 years; range, 41–75 years; 4 men, 7 women; Hoehn & Yahr scale score 1.5–3, Unified Parkinson's Disease Rating Scale; range, 14–75). We did not test any PD patients with juvenile PD nor with a known mutation in the Parkin gene. Of the 11, 9 were tested on dopaminergic medication according to their regular schedule. They did not experience fluctuations. The remaining 2 patients were newly diagnosed with PD and had not received any medication.

The group of age-matched healthy controls (mean ± SD: age, 48.9 ± 8.9 years; range, 41–73 years; 8 women, 5 men) had no medical problems.

Neither patients nor control subjects demonstrated sensory deficits on routine neurological examination. Routine median nerve SEP studies were performed in most of the FD patients (10 of 12). The N20/P25 components showed normal latencies and symmetrical amplitudes in all FD patients tested. None of the subjects showed any cognitive impairment as revealed by Mini-Mental State Examination (MMSE; mean ± SD MMSE score: PD patients, 29.1 ± 1.4; FD patients, 29.4 ± 0.7; controls, 29.4 ± 0.7). All subjects were right-handed. All subjects gave written informed consent before the investigations. The study was approved by the local ethics committee.

Testing Apparatus

The testing apparatus consisted of a plastic platform on which the palm of the subject's hand rested in a relaxed position (Fig. 1). The construction prevented any motor vibrations. A motor (DC servo motor, 4.8 V) was placed below the platform. The motor extended or flexed the index finger at the metacarpophalangeal (MCP) joint at a speed of 50 degrees/sec, the minimum range for both directions was 0.2 degrees, and maximum was 4 degrees. Neither acoustic information (constant pink noise of 75 db over headphones) nor visual information was available. If any voluntary muscle contraction was detected, the trial was excluded from further analysis.

Procedure

We tested the index finger of both hands. Index fingers were moved passively with 45 consecutive movements with MCP displacements of 0.2 degrees, 0.4 degrees, 0.6 degrees, 1 degree, 1.5 degrees, 2 degrees, 2.5 degrees, 3 degrees, and 4 degrees in a pseudorandomized order. After each displacement, subjects were asked if they had detected a movement or not. When the subject noticed a movement, we asked the subject if he/she had noticed the direction of the movement ("up" or "down"; Task 1: detection of movement direction). Answers were scored

FIG. 1. Testing apparatus. The forearm was placed on a comfortable cushion as an extension of the platform. **A:** The palm of the subject's hand rested on a padded plastic platform. **B:** The index finger was surrounded by a soft cotton strap, which attached the finger to the moveable part of the apparatus. The metacarpal joint of the index finger and the joint of the moveable platform arm were positioned exactly vertical to each other. B: The DC motor was placed below the platform. To prevent acoustic information, the subjects wore headphones. The curtain prevented the use of visual information. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

correct or incorrect according to the actual direction of the displacement. Furthermore, subjects were asked to compare the amplitude of two consecutive displacements (Task 2: discrimination of movement amplitudes). Allowed answers were "smaller", "larger", or "the same"; replies were rated correct or incorrect. When subjects answered "can't tell" or "don't know" to any of the questions mentioned above, responses were scored incorrect.

Statistical Analyses

The percentage of correct responses for both tasks and per angular displacement was calculated for each of the three groups. Group differences in the percentage of correct responses both for movement direction (Task 1) and comparison of movement amplitudes (Task 2) were determined using χ^2 tests for each displacement (*SPSS for Windows* v. 10.0.7). If significant differences were found, χ^2 tests were performed to elucidate further which difference (FD vs. controls, PD vs. controls, FD vs. PD) was significant. To control for multiple comparisons, P values < 0.02 were considered significant. Furthermore, following Fechner's technique for determining sensory thresholds,[26] we defined as the threshold for correct responses the point midway between guessing and a perfect response. A curve-fitting procedure (Box Lucas exponential fit) was performed to determine the threshold for 75% correct responses. The model equation was as follows:

$$y = a[1 - e^{(-bx)}]$$

were y is the number of correct responses (%), x is the displacement (degrees), a and b are coefficients, and e is Euler's number (2.718. . .).

Moreover, scores for the severity of the disease were correlated with the percentage of correct responses (bivariate correlations computing Spearman's rho). For correlations, P values < 0.05 were assumed significant.

RESULTS

There were no statistically significant differences in the percentage of correct responses between passive extension and flexion movements of the index finger ($P > 0.2$). Therefore, we collapsed results of both movements to simplify analyses and presentation of results.

Task 1: Detection of the Movement Direction

The threshold for correct responses was defined as the point midway between guessing and a perfect response (i.e., 75%). According to this definition, control subjects showed no difficulties in detecting the direction of even the smallest tested displacement (87.5% correct responses at 0.2 degrees displacements of the index finger). Correct responses further increased promptly with greater angular displacements (Fig. 2).

Compared to control subjects, PD patients showed a clear impairment in this task. They detected the correct direction of angular displacements of 0.2 degrees in 62.5% of trials ($P < 0.001$). Performance improved with increasing angular displacements, but at 0.4 degrees displacement correct responses were still significantly lower if compared to control subjects ($P = 0.018$). At displace-

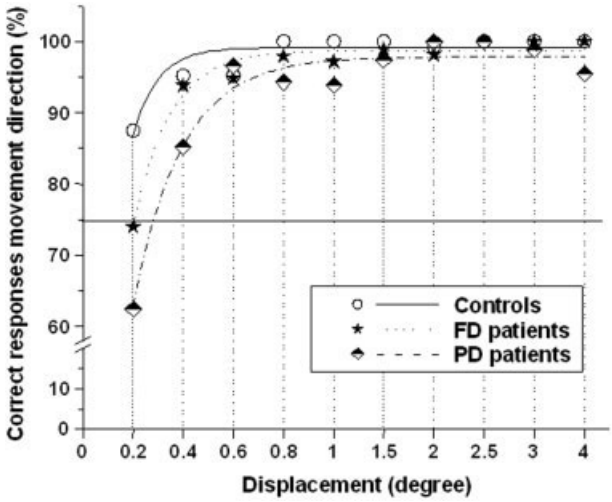

FIG. 2. Percentages of correct responses for each displacement shown for each group. Patients with Parkinson's disease (PD) showed the most prominent impairment in the detection of the movement direction. The threshold for 75% correct responses was 0.13 degrees in control subjects. For focal dystonia (FD), the threshold was nearly two-fold (0.21 degrees), for patients with PD nearly threefold higher (0.28 degrees) compared to control subjects.

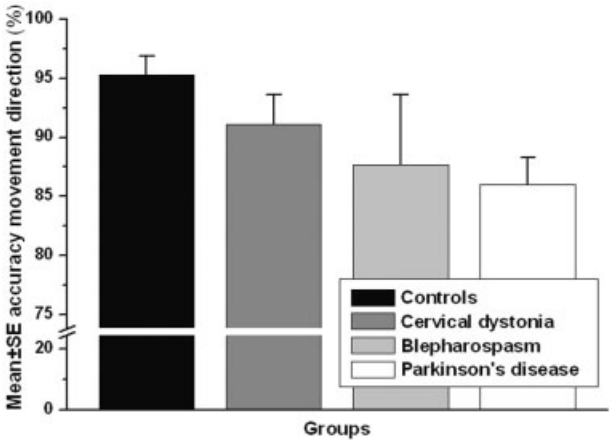

FIG. 3. Mean ± SE accuracy of the detection of movement direction of displacements below 1 degree is shown for each group. Control subjects answered correctly in 95.3% of the trials, whereas the Parkinson's disease group was impaired in accuracy of the detection of the movement direction (86% correct responses). Overall, patients with focal dystonia gave 90.2% correct responses (no significant difference between patients with blepharospasm and cervical dystonia, $P > 0.2$).

ments of 1 degree and greater, no significant differences between PD patients and control subjects were observed.

The performance of patients with FD was also significantly impaired compared to control subjects at angular displacements of 0.2 degrees, with only 74% correct responses ($P = 0.015$). At displacements of 0.4 degrees and above, results did not significantly differ from control subjects ($P = 0.66$). Mean number of correct responses for 0.2 degrees and 0.4 degrees displacements appeared higher in FD than in PD patients, although the group comparison was not significant ($P = 0.1$).

The percentage of cumulated correct responses over all displacements below 1 degrees underlined the fact that PD patients showed the greatest deficit (Fig. 3). PD patients had a mean ± SE accuracy of 86% ± 3.6% (range, 61%–98%) compared to a mean ± SE accuracy of 95.3% ± 1.6% (range, 83%–100%) in the control group. In the group with FD, overall accuracy was 5% smaller compared to the control group's performance (mean ± SE: accuracy, 90.2% ± 2.3%; range, 76%–98%). Likewise, the threshold for detecting 75% correct responses was 0.13 degrees in control subjects, 0.28 degrees in PD patients, and 0.21 degrees in FD patients (Fig. 2; Table 2).

There was neither a correlation between BTX dose and kinesthetic deficits in subjects with FD nor between levodopa equivalent doses and kinesthetic deficits in PD (all $P > 0.2$). Moreover, there was no significant difference between the results of the left and right index finger. Lastly, indecisive responses ("could not tell") were observed in the same frequency in all three groups (12% in controls, 13% in the FD group, and 16% in PD group).

Task 2: Discrimination of Movement Amplitudes

In the second task, subjects had to compare the amplitudes of two consecutive movements (differences of amplitudes were between 0.2 degrees and 3.4 degrees). This task was more difficult, resulting in greater differences between both patient groups and control subjects (Fig. 4). We pooled results in four categories (every category included four different amplitudes) to allow a structured analysis: 0.2 to 0.5 degrees, 0.6 to 1.0 degrees, 1.2 to 2 degrees, and 2.3 to 3.4 degrees. Significant differences between all three groups were detected for 0.2 to 0.5 degrees ($P = 0.011$) only. At amplitudes between 0.6 to 1 degrees and 1.2 to 2 degrees, accuracy of discrimination appeared lower in the two patient groups, although this difference was statistically not significant ($P = 0.078$ and 0.069, respectively).

For controls, it was difficult to compare amplitudes of displacements between 0.2 degrees and 0.5 degrees with

TABLE 2. *Results of curve fitting procedure*

	Threshold (degrees)	a	b	R^2
Controls	0.13	99.131	10.396	0.835
PD patients	0.21	98.753	6.937	0.973
FD patients	0.28	97.834	5.157	0.964

PD, Parkinson's disease; FD, focal dystonia.

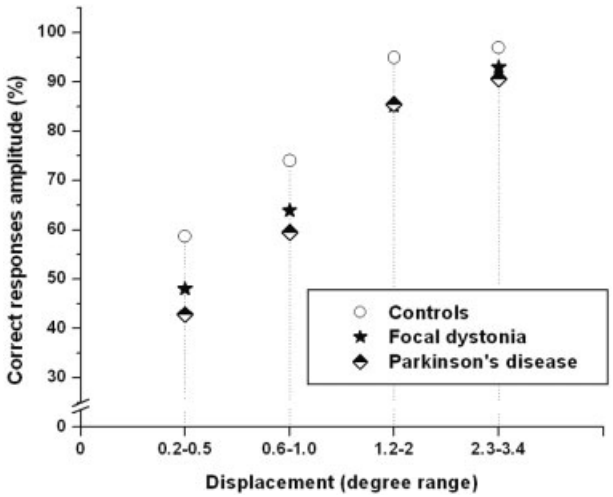

FIG. 4. Percentage of correct responses for discrimination of amplitudes of two consecutive movements. Patients with Parkinson's disease and focal dystonia revealed a clear impairment if compared to control subjects. Differences remained significant up to 2 degrees.

a rate of only 58.7% correct responses. Results improved between 0.6 degrees and 1.0 degree displacements with 74% correct responses. Above 1.2 degrees, amplitude difference correct responses appeared in 94.9% of the trials in the control group. Again, PD patients showed the greatest impairment of all groups given that they only guessed the correct response at 0.2 degrees to 0.5 degrees amplitude differences (42.8% correct responses). Correct responses were still low at amplitude differences between 0.6 degrees and 1.0 degrees, but they increased above chance level to 59.5%. Differences compared to controls were statistically significant for 0.2 to 0.5 degrees, 0.6 to 1 degrees, and 2.3 to 3.4 degrees ($P = 0.002$, $P = 0.003$, $P = 0.016$, respectively).

Performance of the FD group was found to be significantly lower than that of the control group at amplitude differences between 0.2 degrees to 0.5 degrees (48.1% correct responses, $P = 0.007$) and 0.6 to 1 degrees (63.9% correct responses, $P = 0.008$). More than 90% correct responses occurred at amplitude differences $\geq$ 2.3 degrees, and results were no longer significantly different from controls ($P = 0.085$).

DISCUSSION

In this study, we examined the kinesthetic performance of patients with FD in comparison to patients with PD and to healthy controls in a passive movement task. The main finding of this study is that kinesthetic processing was impaired in FD. We also confirm previous studies showing a kinesthetic deficit in PD.

Tested Aspects of Kinesthesia

Processing of kinesthetic stimuli depends on central integration of signals deriving from mainly muscle spindle afferents that are modified by spinal γ-motoneurons.[27] Our task mainly stimulated muscle spindle afferents and joint receptors, while Golgi organ signals likely played a minor role because muscles did not develop tension throughout a trial. The high movement speed of the apparatus enabled us to test mainly the passive motion sense and only to a small extent statesthesia (position sense).[2]

Kinesthesia Is Impaired in Patients With Focal Dystonia

Compared to healthy controls, patients with FD had a nearly twofold higher perceptual threshold for detecting the correct direction of displacements of the index finger (0.13 degrees vs. 0.21 degrees). They also had problems in discriminating different finger amplitudes at threshold and suprathreshold amplitudes.

Of interest, deficits in both kinesthetic tasks were observed in dystonic patients in muscles that were clinically not affected, which is in accordance with other studies.[16,28] This finding might suggest that FD is a systemic disorder of the sensorimotor system, although the motor symptoms are expressed by specific muscle groups.

Previous studies analyzing aspects of kinesthetic processing in FD were to some extent contradictory. Kinesthesia was found to be unaffected in FD by Byl and colleagues in nonautomated clinical tests.[29] However, one may doubt that the tests might have been able to detect subtle deficits that became obvious in our study (Fig. 4). Studies by Grünewald et al. at least partly support our findings of impaired kinesthetic processing. They compared a task testing position sense compared to a task testing motion sense using a 50-Hz vibratory stimulus applied at the biceps tendon.[16] Patients with several types of FD clearly were impaired in the perception of the vibratory stimulus (i.e., motion sense) but appeared normal in sensing arm positions. The same group extended their findings by further investigations and concluded that muscle spindle afferent processing is abnormal in FD.[30,31] For patients with writer's cramp, this assumption also is supported by others.[25,32] Therefore, abnormal muscle spindle activity might contribute to an altered kinesthetic processing in FD.

Several findings support the view of a supraspinal dysfunction leading to impaired kinesthesia. As in our study, SEPs are usually normal in FD, which demonstrates that the peripheral structures and the lemniscal pathways seem to be principally intact, including the

primary input to the somatosensory cortex. However, a recent study showed an abnormal SEP response to paired stimuli in patients with dystonia.[22] This study hints at an impaired inhibition of sensory signals at spinal and cortical levels of the somatosensory system leading to a reduced filtering of irrelevant sensory signals ("sensory overflow") in the central nervous system (CNS). Moreover, our finding that not only detection but also discrimination at suprathreshold amplitudes was affected underlines that central processing of proprioceptive signals is affected in FD.

Can Other Factors Account for Kinesthetic Deficits in FD Patients?

Because the majority of the FD patients were on BTX therapy one may argue that BTX may have influenced kinesthetic processing. Effects of BTX on CNS activity have been observed. Imaging studies on a central effect of BTX are controversial.[33,34] When we performed our testing—12 weeks after the last injection—the BTX effect should have diminished. Moreover, no correlation of BTX doses with the performance was detected, and the tested limb did not receive BTX injections. Rome and Grünewald[20] compared the perception of tonic tendon vibration (stimulating predominantly Ia afferents similar to the stimulated afferents in our study) before and after BTX treatment and did not find any differences. In summary, we cannot completely rule out an effect of BTX on kinesthetic performance as no comparative data from our study with and without BTX is available, but it appears at best an unlikely possibility.

Second, cognitive deficits in FD patients could have influenced the ability to perceive stimuli correctly and to compare the amplitudes of two consecutive movements. However, no attention deficit was apparent in the FD group (same amount of "could not tell" responses as in the control group), and no obvious signs of fluctuations in attention or higher intrasubject variability were present. Groups were carefully age-matched, and cognitive function appeared normal as shown by results of the MMSE. Because cognitive dysfunction is not an associated symptom in idiopathic FD in general,[35] the MMSE can be considered an adequate test to determine relevant cognitive deficits.

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