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Tactile/proprioceptive integration during arm localization is intact in individuals with Parkinson's disease

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Abstract

It has been theorized that sensorimotor processing deficits underlie Parkinson's disease (PD) motor impairments including movement under proprioceptive control. However, it is possible that these sensorimotor processing deficits exclude tactile/proprioception sensorimotor integration: prior studies show improved movement accuracy in PD with endpoint tactile feedback, and good control in tactile-driven precision-grip tasks.

To determine whether tactile/proprioceptive integration in particular is affected by PD, nine subjects with PD (off-medication, UPDRS motor=19-42) performed an arm-matching task without visual feedback. In some trials one arm touched a *static* tactile cue that conflicted with *dynamic* proprioceptive feedback from biceps brachii muscle vibration. This sensory conflict paradigm has characterized tactile/proprioceptive integration in healthy subjects as specific to the context of tactile cue mobility assumptions and the intention to move the arm.

We found that the individuals with PD had poorer arm-matching acuracy than age-matched control subjects. However, PD-group accuracy improved with tactile feedback. Furthermore, sensory conflict conditions were resolved in the same context-dependent fashion by both subject groups. We conclude that the somatosensory integration mechanism for prioritizing tactile and proprioception feedback in this task are not disrupted by PD, and are not related to the observed proprioceptive deficits.

Keywords

Parkinson's disease; proprioception; touch; sensory integration

INTRODUCTION

Individuals with PD show deficits in proprioceptive acuity and impaired performance in motor tasks under proprioceptive control [21]. Individuals with PD make more errors than controls in matching one arm's posture with the other [33], discriminating passive arm-joint movement

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direction (≤5°), and maintaining constant arm positions [6,17]. PD degrades movement accuracy under proprioceptive guidance (without visual feedback)[1,29,30]. Individuals with PD also show sensory integration deficits in visually-guided tasks [8,24] and tendencies resembling spatial neglect [7]. Impaired movement adaptation to altered visual feedback has suggested that PD affects sensorimotor integration, leading to spatial processing and motor control deficits [1].

In contrast to evidence that PD affects sensorimotor integration, adaptive responses and appropriate anticipatory motor planning in multi-modal tasks involving tactile feedback appear to be intact. Although individuals with PD show reduced tactile resolution [32], tactile cues improve their motor performance. PD affects reproducing arm postures without endpoint contact, but not pointing to locations on one's own skin [14]. In precision-grip control, adapting to object weight and texture (proprioceptive and tactile feedback) are intact in PD [11,13]. This suggests that PD may not impair the tactile aspect of sensorimotor processing, and that individuals with PD use tactile information in movement control.

To better understand how tactile feedback may help the affected proprioception of individuals with PD, and also address whether PD affects tactile/proprioceptive integration, like visuomotor integration [1] in visually-guided tasks [8,24] and in cases resembling neglect [7], we compared the performance of individuals with PD to that of age-matched controls in reproducing the orientation of one arm ("cue-arm") with the other ("report-arm"). Some conditions included a conflicting pattern of *static* tactile cues (fingertip contact with a stationary surface) and *dynamic* muscle stretch cues (transcutaneous biceps brachii muscle-spindle vibration, causing dynamic elbow-extension illusions, and **t**onic **v**ibration stretch-**r**eflex muscle contraction (TVR) [9,10]).

Previously, this paradigm demonstrated in healthy subjects how *stationary* external tactile cues can attenuate *dynamic* proprioceptive feedback from muscle vibration, depending on whether the tactile cue was thought to be stationary or mobile (cue *context*). In the paradigm, notions of the cue-surface's location were updated from 'stationary' to 'mobile' when subjects touched the cue-surface as it moved. After this experience, subjects reported that the touched stationary cue-surface moved during biceps vibration, even though the cue-surface was actually stationary [27]. We tested subjects under *passive* cue-arm control (held stationary by the experimenter) and *active* cue-arm control (without external restraint) to determine whether somatosensory/motor-set (muscle activation and reafference) integration to estimate elbow orientation also depends on context. The response pattern generalized across active and passive cases, suggesting that the *intention to extend* the cue-arm elbow to maintain fingertip contact with the cue-surface thought to be mobile contributed to the elbow extension perception (although both cue-arm and surface were actually stationary) [28].

Presently, we address theories about impaired sensory integration in PD [1] in the specific area of tactile/proprioceptive integration because previous work such as in grip control that shows the benefits of tactile feedback to individuals with PD, which seems contrary to sensory integration deficits. Therefore, we tested the hypotheses that PD affects integration of tactile cues with proprioceptive cues in limb localization according to spatial context as in healthy individuals: depending on a-priori notions about the tactile stimulus's spatial properties. To test the hypothesis that PD affects tactile/proprioceptive integration is affected by during active motor tasks, we compared haptic sensory-conflict of individuals with PD to control subjects testing under active as well as passive cue-arm control. If individuals with PD can prioritize tactile cues over proprioceptive feedback according to spatial and motor contexts as healthy subjects do it would suggest that: prior findings about tactile cue benefits to individuals with PD are further supported; sensory integration deficits in PD do not include tactile/proprioceptive integration; and neural mechanisms which contextualize tactile cues to inform

limb position sense are intact in PD, and probably are outside the BG/dopaminergic pathways affected by PD.

METHODS

Procedures accorded with the Declaration of Helsinki, institutional human subjects review board approvals, and subjects' understanding and written consent.

Subjects

Nine subjects with PD (5 male and 4 female; mean age=58.7; Table 1) between ages 48 and 72, and nine age-matched controls participated. Subjects were tested 12 hours off-medication. PD-group inclusion criteria were: capacity to follow verbal instructions; Modified Mini-Mental State Examination score>40/57; and a zero score on the UPDRS rigidity subscale for the *less* involved arm—used as the "report-arm" in our study—so that we can address the sensory processing contribution to arm mislocalization in PD, independent of rigidity. Exclusion criteria were: other chronic neurological diseases, arthritis, dementia, upper extremity weakness, and orthopedic problems. The nine control subjects (mean age=59.4; p=0.77, T-test) were screened for neurological or orthopedic symptoms and were generally healthy.

Experimental set up

Subjects sat with eyes closed and elbows on a table (Figure 1). In this position, one index fingertip (of the subject's "cue-arm") could touch a shelf ("cue-surface") on one side. The cue-surface was fixed in a horizontal orientation 20cm above the table surface during experimental trials, but it could pitch about a horizontal axis co-axial with the subject's elbows to accommodate fingertip contact during elbow movements when desired. To test whether PD affects sensory integration of arm position cues, the cue-surface was positioned for touching by the more PD-involved hand. Control subjects (all right-handed) were all tested with their left hand as the cue-arm.

Throughout muscle vibration trials, the experimenter applied a massage vibrator (Hitachi Magic Wand, Tokyo) to the cue-arm biceps brachia transcutaneously.

Experimental design

There were two blocks of eight experimental trials. The experimental conditions varied biceps vibration (~1mm amplitude vibration at 100 Hz, no-vibration), fingertip contact with the stationary cue-surface (touch, no-touch), and cue-forearm control ('active-control' in which subjects were instructed to maintain cue-arm orientation or fingertip contact with the cue-surface, 'passive-control' subjects were instructed to let the experimenter control their arm). Thus, each block's trial-conditions were: no-touch/active; touch/active; biceps−vibration/active; vibration+touch/active; no-touch/passive; touch/passive; biceps−vibration/passive; and vibration+touch/passive. Subjects were informed of these experimental manipulations before the experiment, but not of muscle vibration-related illusions or TVRs they might experience. Subjects were not informed that the cue-surface could move—until the cue-surface mobility demonstration between blocks I and II.

During the first trial block (I) subjects were naïve of the cue-surface's capacity to move. In the second block (II) subjects had been made aware of the cue-surface's capacity to move. Following block I, the cue-surface's mobility was disclosed verbally, demonstrated visually, and experienced by the subjects by touching the cue-surface without restraint or vibration while the experimenter moved the cue-surface. Then the cue-surface was again fixed (unbeknownst to subjects) and the eight conditions described above were repeated identically to block I. The cue-surface was always stationary during each block's experimental trials.

To optimize the influence of vibration conditions were performed once per block to minimize vibration fatigue effects, and for 15 seconds which was sufficient to elicit significant dynamic proprioceptive sensation in prior studies [27,28]. Trial order within blocks was counter-balanced across subjects to control for possible fatigue effects. During active-control without touch, accidentally touching the cue-surface is prevented because the cue-surface is below the hand, and in this posture TVR raises the hand.

Procedure

Every trial began with the experimenter positioning the cue-forearm ~45° from horizontal. With eyes closed, the subject moved their report-arm to match the orientation of the cue-arm. The baseline arm-matching error (E_0) was this initial discrepancy between the two forearm orientations.

Then the subject was reminded to keep matching their arms matched for 15 seconds, and the trial's specific tactile/proprioceptive conflict conditions were applied. For example, in active-control trials, the experimenter would release the cue-arm. In vibration trials, the vibrator was applied to the biceps brachii of the cue-arm. For 'touch' trials, subjects flexed the cue-arm's index finger to touch the cue-surface. For 'no-touch' trials, subjects were instructed to maintain their cue-arm fingertip position, just above cue-surface.

After the trial, the experimenter flexed the subjects' cue-arm at the elbow before resting that forearm horizontally on the near table surface, and subjects rested their report-forearm flat on the near table.

Measurements and Analysis

Forearm orientations were measured using data acquisition software (60 Hz; SC/ZOOM, Umeå, Sweden) sampling 3-d position of electromagnetic sensors (Polhemus Fastrack, VT; resolution=0.75mm) fastened to the dorsal wrist and elbow surfaces.

A measure of baseline proprioceptive error, the absolute difference in elbow angles at the beginnings of experimental trials, E_0, was calculated:

$$E_0 = |C(0) - R(0)|,$$

where $C(0)$ and $R(0)$ are the initial forearm orientations. The cue-arm position change during active-control/no-touch trials, C_Δ, quantifies the TVR response during vibration and drift during no-vibration, was calculated:

$$C_\Delta = max[C(t) - C(0)],$$

where $C(t)$ is the cue-forearm orientation time-series, and $C(0)$ is the initial orientation.

To determine how tactile/proprioceptive conflict is resolved, we quantified the vibration effect strength (excess report-arm elbow extension from vibration-induced cue-arm flexion and/or reported extension) as the maximum relative change in forearm orientations. A positive error corresponds to report-arm overextension relative to the cue-arm. Subtracting the error at the trial start unbiased scores. Thus the maximum difference in arm positions, D_{max}, was calculated:

$$D_{max} = max[(C(t) - C(0)) - (R(t) - R(0))],$$

where $C(t)$ is the cue-forearm orientation time-series, $R(t)$ is the report-forearm orientation time-series, and $C(0)$ and $R(0)$ are initial orientations. Statistics were performed on across-subject within-condition mean scores.

T-tests determined significance of group effects on E_0 and C_Δ. A 2×2×2×2 ANOVA tested the significance of effects of previous experience with the tactile cue (naive vs. aware of table cue-surface motion), vibration (vibration vs. no-vibration), fingertip contact (touch vs. no-touch), cue-arm control (passive vs. active) and group (PD vs. control) on tactile/proprioceptive integration test performance (D_{max}). Where main effects or interactions were significant, Tukey pairwise comparisons evaluated individual condition differences.

RESULTS

Subjects with PD had poorer arm-matching accuracy than control subjects, but had the same response patterns during conflicting somatosensory stimuli as control subjects.

Subjects with PD had a greater baseline arm-matching error than age matched controls

At every test trial start, subjects' cue-arm was positioned by the experimenter, and subjects matched its perceived location with their report-arm. Subjects with PD had a significantly greater absolute baseline arm-matching error (6.18°±0.98° (mean±standard error)) than the age-matched control group (4.93°±0.31°; p=0.007). The PD group's cue-arm drift (C_Δ) in no-touch active-control trials (1.26°±1.30°) was also greater than controls (0.44°±0.11°; p=0.05). This was not due to marker movement on the subjects between or within trials.

Touching the cue-surface attenuated arm-matching error during biceps brachii vibration

Figure 2 summarizes D_{max} within-subject group means. No significant effects were found among no-vibration conditions (p>0.05); therefore all no-vibration condition results have been averaged within subjects. The positive bias in no-vibration trials is probably due to gravity and/or report-arm fatigue throughout the 15-second trial.

As expected, biceps brachii vibration caused elbow angle overestimation during the arm-matching task in both subject groups (p<0.001, main effect: vibration). In the passive-control no-touch condition subjects reported elbow extension perceptions by extending their report-arm. Without touch under active control vibration caused cue-arm flexion via the unchecked TVR. TVRs (C_Δ) were greater in PD subjects (5.62°±4.27°) than in controls 9.99°±2.28; p<0.021). But there was no group effect on D_{max} in these vibration conditions (subject-means ranged +8°-+19°, Figure 2).

For all subjects there was a general effect of touching the cue-surface on arm-matching error (p<0.001, main effect: touch). In the first block, touching the cue-surface reduced arm-matching errors to <5° on average (Figure 2, unfilled "T"-bars). Unlike the no-touch condition under active control, the cue-arm did not flex while subjects touched the cue-surface top.

Subjects with PD integrated muscle stretch feedback and tactile cues in a similar context-dependent fashion as control subjects

Touch attenuated elbow error during vibration (p<0.001, interaction: vibration×touch). This effect of touch depended on whether or not subjects had experienced unambiguous cue-surface motion (p=0.013, interaction: vibration×touch×block). This interaction of vibration, touch and assumptions about the cue-surface for both PD and control groups is consistent with previous studies with younger healthy subjects [27,28]. Initially, when subjects were unaware of the possibility of cue-surface motion (trial block I), fingertip contact with the stationary surface prevented cue-arm elbow flexion and attenuated extension perception when the cue-arm was

under passive or active control (Figure 2, unfilled "T" bars). However, after the actual cue-surface motion experience (block II), fingertip contact with the stationary cue-surface during vibration led to elbow extension perception as though the surface were moving regardless of restraint, resulting in larger arm-matching errors (figure 2, filled bars). In block I (before cue-surface motion) touching the cue-surface attenuated the vibration illusion significantly more than in block II after subjects had experienced cue-surface motion ($p<0.001$, Tukey test).

The D_{max} result patterns for PD and control groups were the same. There was no significant active vs. passive cue-arm control effect on D_{max} for either group.

DISCUSSION

Individuals with PD we tested had more drift when holding their cue-arm still and greater errors in reproducing the same static arm positions than control subjects which is consistent with previous findings in PD [cf.[1],6,14,21,33]. Movement error associated with these matching errors magnitudes impacts function in proporioception-dependent tasks [16] by contributing to bradykinesia and hypometria in patients with PD by forcing multiple movements to achieve motor goals [5,6]. It has been suggested that spatial processing deficits and poor motor control are due to impaired sensorimotor integration in PD [1]. However, the PD-group resolution of tactile/proprioceptive conflict in the present paradigm did not differ from control subjects'. Our task examined separately and combined two sensory modalities naturally linked in function. This task possibly masks other subtle deficits related to PD. Nevertheless, tactile cues could attenuate conflicting proprioceptive cues, which may be worthy of greater investigation in the context of movement based therapies. No subjects spontaneously reported experiences as out-of-the-ordinary, which suggests that vibration caused nothing out-of-the-ordinary, physiologically speaking.

Tactile feedback guided PD subjects in the same fashion as control subjects. This is consistent with previous finding showing reduction in movement error with tactile feedback [14], as well as intact grip control [11,13], in spite of proprioceptive deficits. The spatial framework associated with the tactile cue is so influential that assumptions about the tactile cue spatial properties determined whether the arm was perceived as moving or not during biceps vibration. Our results show how a tactile cue's ambiguity is resolved based on association of specific patterns of tactile and other feedback from prior experience [27]. Initially in our paradigm, touching the stationary cue-surface attenuates the elbow extension perception during biceps vibration. When subjects actually move their elbow while touching the moving cue-surface, touching the cue-surface is associated with genuine elbow motion proprioceptive feedback. After this experience, the tactile cue matches the dynamic feedback from biceps vibration, and elbow and cue-surface motion is perceived, even though both are stationary.

Outcomes generalized across active and passive cue-arm control conditions. Under active or passive arm control, the arm-matching errors during biceps vibration correspond to overestimation of the vibrated arm's elbow angle. Biceps vibration of an arm held in place externally (passive control) blocks TVR flexion; biceps vibration of an unrestrained arm (active control) triggers TVR flexion, which is underestimated because illusory biceps feedback from vibration can cancel genuine triceps stretch feedback from the TVR flexion. The effects of touch and cue-surface understanding within active-control conditions demonstrate not only the influence of prior sensory experience [18,19,23] but also movement goals and expected outcomes [e.g. 2,3,28] to estimate body state. Although PD subjects had smaller TVRs, individuals with PD did not differ significantly from controls in their tactile/proprioceptive conflict resolution under active or passive control. This suggests that tactile/proprioceptive integration is not affected by PD-affected sensorimotor processing [1] during active motor

tasks, and that the tactile/proprioceptive integration mechanisms forming body configuration perceptions are not related to PD functional proprioceptive deficits.

PD's proprioceptive impairment may be related to central spindle feedback and corollary signal processing. All muscles controlling a joint influence joint proprioception. The effects of simultaneous antagonist muscle vibration differs between individuals with PD and healthy subjects during voluntary movements [4,31]. During these voluntary movements, the antagonist muscle vibration creates movement undershoots which are smaller in subjects with PD. These undershoots are due to reduced agonist activity, rather than vibrated antagonist TVR [20]. This suggests that there is a reduced spindle feedback gain in the central nervous system in PD [9].

Salience of tactile cues observed in our PD subject group may be related to peripheral mechanisms. Increased cutaneous fiber branching and improved vascularization may compensate for decreased mechanoreceptor sensitivity in early PD stages [26]. Thus, prior to significant tactile changes, proprioception receptors may be lost and therefore tactile information remains particularly useful to subjects.

We may infer from our results that tactile/proprioceptive integration mechanisms are distinct from those involving proprioception affected by PD. Substantia nigra atrophy in the basal ganglia (BG) underlies PD. The BG striatum receive cortical somatosensory projections conveying proprioceptive and tactile information. The BG projects to thalamic areas which project to motor and pre-motor areas. Substantia nigra modulatory dopaminergic projections modulate the afferent somatosensory-BG circuit [12]. While BG and dopaminergic pathway degeneration are thought to cause PD-related sensorimotor integration deficits, dopaminergic therapies does not always improve movement accuracy under proprioceptive control [22]. This suggests that sensorimotor integration is not solely mediated by BG and dopaminergic pathways. Our findings suggest that, rather than PD causing a broad sensorimotor integration deficit, PD may cause selective loss of proprioceptive functions which can be attenuated when intact sensory mechanisms engaged by tactile cues are available.

Illusory vibration induced movement perception can be blocked if considered implausible by higher order mechanisms. Such mechanism may be central. Furthermore, the block II active-control results rule out the involvement of a peripheral antagonist muscle feedback inhibition mechanism which predicts that engaging triceps (as subjects must to maintain fingertip contact during biceps TVR) would inhibit biceps stretch feedback, and therefore attenuate the illusion [eg. 15,25]. In fact the opposite occurred: perceptions of biceps stretch increased when subjects intended to extend their elbow (which requires triceps activity) in block II. Therefore, although the cue-arm never moves during experimental trials, biceps muscle stretch feedback may be affected as subjects intend to extend the cue-arm elbow to maintain fingertip contact in the active control vibration with touch condition in block II. This prediction was true half the time: specifically, when touch was not involved. When touch was involved, there active and passive conditions did not differ during vibration. This suggests a muscle spindle feedback gating at cortical rather than peripheral or spinal levels [28]. Since this was the case for PD and control subject groups, we infer that the proprioception and tactile feedback integration according to context takes place or is compensated for outside the PD-affected CNS circuit. This could partially explain the tactile feedback usefulness in individuals with PD in this and prior studies [13,14].

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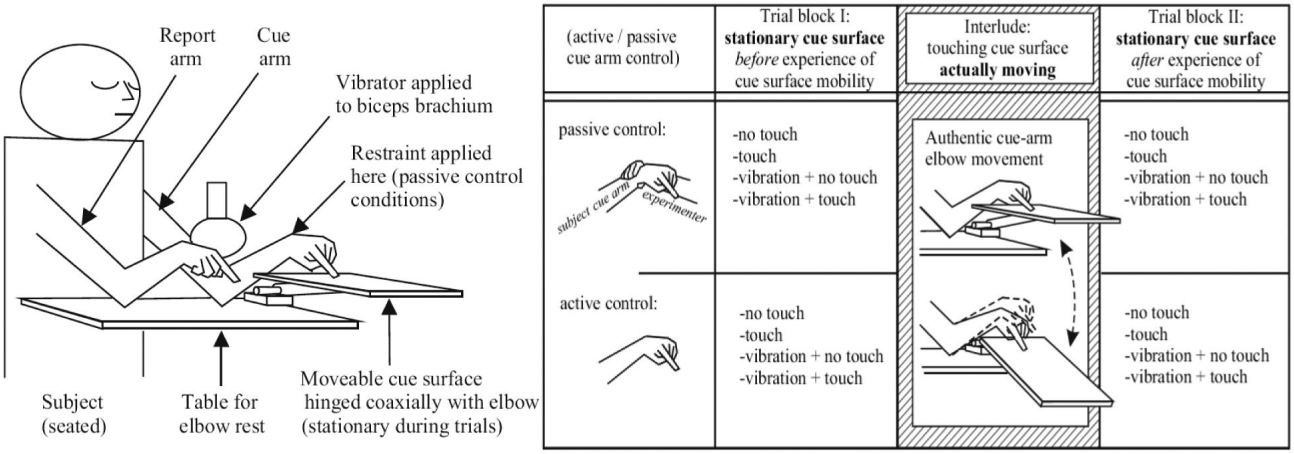

Figure 1.

Left: Apparatus and setup. For 'touch' conditions the cue-arm index fingertip touched the cue-surface, located 20cm above the tabletop. The cue-surface was stationary throughout experimental trials, but was rotated about a hinge in between the 2 trial-blocks to demonstrate mobility potential. For PD-group subjects, the cue-surface was moved to the more PD-involved side.

Right: Experimental design included 2 blocks with 8 conditions, separated by an 'interlude'. Left-to-right order represents protocol order.

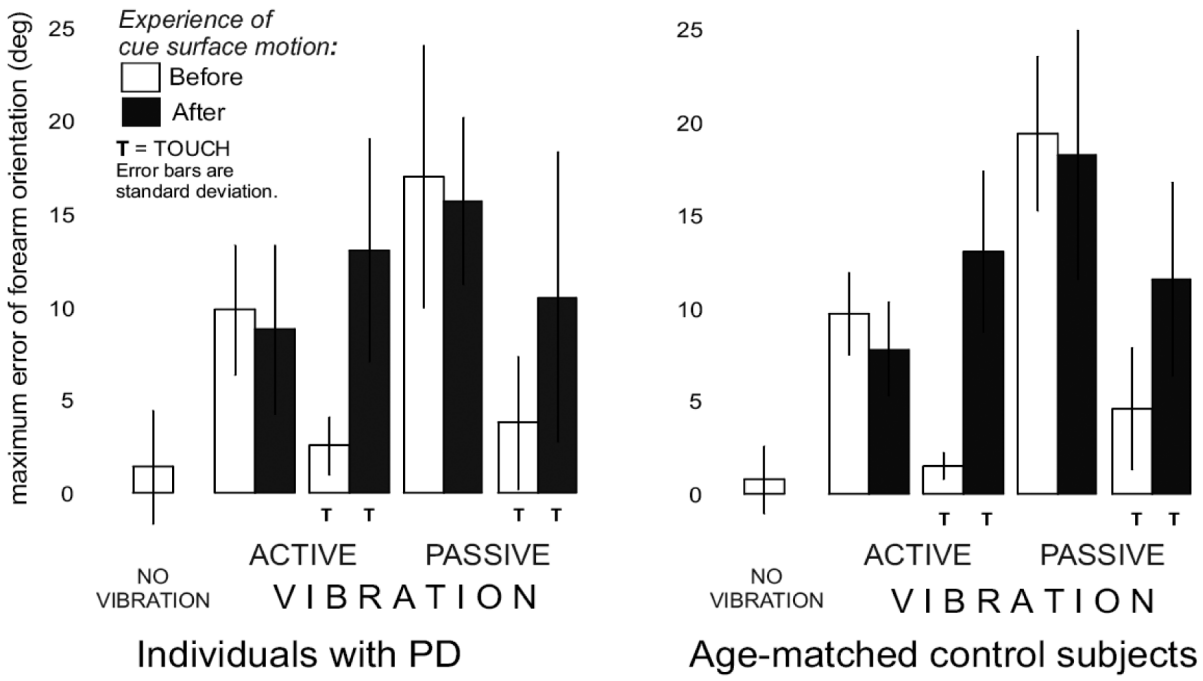

Figure 2.

Means±SD (n=9) of peak discrepancies of forearm orientation across all conditions with vibration, before (trial block I, white bars) and after (block II, dark bars) being made aware of the cue-surface's potential to move. All errors correspond to greater report-arm extension. All no-vibration results are collapsed.

Table 1

Description of subjects with Parkinson's Disease

Patient	Gender	Age	Dominant Hand	>Affected Hand	L-Dopa/ day	UPDRS (off) [a]
SL	M	59	Left	Right	0 mg	19
JG	M	58	Right	Right	75 mg	36
EK	F	52	Right	Left	75 mg	18
RJ	F	48	Right	Right	0 mg	42
SM	M	65	Right	Left	0 mg	19
AW	F	56	Right	Left	125 mg	34
KC	F	61	Right	Right	150 mg	22
TR	M	72	Right	Right	150 mg	41
DD	M	57	Right	Right	--- mg	----

[a] Items 18-31 on the UPDRS, representing the motor portion, with a possible maximum score of 108.