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Kinematics of Goal-Directed Arm Movements in Neglect: Control of Hand Velocity

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Do patients with unilateral neglect exhibit direction-specific deficits in the control of movement velocity when performing goal-directed arm movements? Five patients with left-sided neglect performed unrestrained three-dimensional pointing movements to visual targets presented at body midline, the left and right hemispace. A group of healthy adults and a group of patients with right-hemispheric brain damage but no neglect served as controls. Pointing was performed under normal room light or in darkness. Time-position data of the hand were recorded with an opto-electronic camera system. We found that compared to healthy controls, movement times were longer in both patient groups due to prolonged acceleration and deceleration phases. Tangential peak hand velocity was lower in both patient groups, but not significantly different from controls. Single peak, bell-shaped velocity profiles of the hand were preserved in all right hemispheric patients and in three out of five neglect patients. Most important, the velocity profiles of neglect patients to leftward targets did not differ significantly from those to targets in the right hemispace. In summary, we found evidence for general bradykinesia in neglect patients, but not for a direction-specific deficit in the control of hand velocity. We conclude that visual neglect induces characteristic changes in exploratory behavior, but not in the kinematics of goal-directed movements to objects in peripersonal space. © 1998 Academic Press

INTRODUCTION

A prerequisite for performing goal-directed behavior in extrapersonal space is the intact interaction between perceptual and motor systems. In unilateral neglect this interaction is disrupted in a characteristic manner. For

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example, patients suffering from neglect fail to explore and react to stimuli located in the contralesional part of space. While the perceptual deficits associated with this syndrome have been studied extensively, the concurrent impairments of motor behavior have not yet received the equivalent degree of attention. Yet clinical and experimental findings indicate that patients suffering from neglect may experience motor problems affecting spatial and also temporal coordination among limb segments. (We here define *spatial* as related to position and orientation of body segments and *temporal* as related to reaction time and movement speed). The present paper addresses the issue of temporal control of movement during pointing, while we discussed the spatial aspects of coordination elsewhere (Karnath, Dick, & Konczak, 1997).

Concerning temporal movement parameters, several studies have demonstrated that patients with neglect exhibit a delayed reaction time when performing movements to the contralesional side. This is true for upper limb (Heilman, Bowers, Coslett, Whelan, & Watson, 1985; Mattingley, Bradshaw, & Phillips, 1992; Meador, Watson, Bowers, & Heilman, 1986) as well as for eye movements (Girotti, Casazza, Musicco, & Avanzini, 1983; Karnath, Schenkel, & Fischer, 199 1). Increased reaction times can be indicative of a deficit in motor planning (e.g., shifting attention and locating targets). This specific deficit in temporal control is termed *directional hypokinesia* (Heilman et al., 1985; Mattingley, Phillips, & Bradshaw, 1994). The second facet of temporal coordination pertains to the control of velocity during execution. Neglect patients may reveal abnormally slowed movement speeds— an impairment that Mattingley et al. (1994) named *directional bradykinesia.* In contrast to general forms of bradykinesia (such as in Parkinson's disease), a direction-specific bradykinetic deficit implies that movements toward the neglected side should be more affected than movements away from it.

Given the previous experimental findings, it remains unclear whether a gradient in velocity dyscontrol is expected from the intact to the neglected side. One might assume that speed control at the ipsilesional side is largely preserved and does degrade the more the intended motion is directed toward the neglected side. To date, evidence for direction-specific impairments of velocity control during execution is still rare (Mattingley et al., 1992, 1994). Mattingley et al. (1994) examined 14 patients with right-hemispheric damage and neglect performing six consecutive pen strokes with a stylus on a digitizing tablet. The actual movement amplitudes were small, ranging between 6.3 and 12.5 cm. Based on the recorded time-position data of the stylus, higher order kinematics of the endpoint were calculated. (We refer to *endpoint* as the most distal part of the arm, i.e., the hand or a tool manipulated with the hand). The authors did find an effect of movement direction on peak velocity only in patients with severe neglect. In addition, they report that leftward strokes revealed a prolonged acceleration time and contained more movement units (epochs of acceleration and deceleration).

The present study extends on these previous findings. In comparison to the small amplitude, planar movements investigated by Mattingley et al. (1994), the present experiment focuses on the question of how patients with left-sided neglect control movement speed during large amplitude, three-dimensional multijoint arm movements.

We know that during unrestrained reaching and pointing human adults produce stereotyped kinematic patterns (Abend, Bizzi, & Morasso, 1982; Morasso, 198 1). Adult hand trajectories during reaching are approximately straight and tangential hand velocity shows a single bell-shaped configuration (unimodal) for a wide variety of movement speeds and loads (Morasso, 1983). We also know that in patients with right-hemispheric damage but no neglect this unimodal pattern of tangential hand velocity is largely preserved when performing reaches with their right hand. Fisk and Goodale (1988) examined patients with right-hemispheric brain damage in a multijoint pointing task. When compared to healthy adults, these patients required a similar time period to transport the hand to the final position, achieved the same peak endpoint velocity, and spent similar amounts of time in acceleration and deceleration.

The present study addresses two questions: First, how well are unimodal velocity patterns preserved in patients with neglect when they perform three-dimensional pointing movements to targets within their workspace? Do right-hemispheric brain-damaged patients with and without visuospatial neglect differ in that respect? Second, do we find evidence for directional velocity dyscontrol during execution? That is, are endpoint velocity profiles to targets located on the contralesional, left side of egocentric space more impaired than hand movements to targets on the ipsilesional side? To account for the role of vision in guiding the hand through space, pointing movements were performed in light and darkness. Under the latter condition the motor system cannot rely on visual feedback and is presumably operating in an "open-loop" mode, which requires that large parts of the trajectory are preplanned before execution (Harvey, Milner, & Roberts, 1994; Prablanc, Pelisson, & Goodale, 1986).

METHODS

Subjects

Five patients with unilateral right-hemispheric lesions and spatial neglect participated in the study (mean age, 62 years, SD 14.5). Five patients with unilateral right-hemispheric lesions without neglect (RH group) and six healthy subjects served as control groups. The average age of the healthy control group was 56 years (SD 10.4) and for the RH group 52.8 years (SD 27.2). Clinical and demographic characteristics of all patients are listed in Table 1. All subjects were right-handed.

TABLE 1
Demographics and Clinical Data for the Two Patient Groups

Patients	Gender	Age (years)	Etiology	Lesion localization	Days since first clinical symptoms	Hemianopia	Neglect	Letter cancellation		Line bisection	Copying		Clock face	
								Left	Right		Left	Right	Left	Right
RH1	F	76	Lymphoma	Temporoparietal	15	No	No	30	30	0.4	+	+	+	+
RH2	F	48	Meningioma	Temporal	-360	No	No	28	30	0.8	+	+	+	+
RH3	F	84	Infarct	Basal ganglia	4	No	No	24	27	0.1	+	+	+	+
RH4	M	38	OP grade IV glioma	Frontal	15	No	No	25	25	0.3	+	+	+	+
RH5	M	18	OP grade IV glioma	Frontal	12	No	No	23	26	0.1	+	+	+	+
NP1	M	63	Infarct	Temporoparietal	7	No	Yes	11	22	3.5	–	(+)	+	+
NP2	M	43	Infarct	Fronto-temporoparietal		Yes	Yes	11	29	1.9	–	+		
NP3	F	69	Infarct	Temporoparietal	10	No	Yes	0	23	1.4	+		+	+
NP4	F	54	Infarct	Frontoparietal	30	No	Yes	0	2		–	(+)	–	+
NP5	F	81	Hemorrhage	Thalamus	28	No	Yes	0	7		–	(+)	–	+

Note. Letter cancellation (Mesulam, 1985): number of correct target letters on each half of the test sheet (30 max. on either side). Line bisection: average rightward deviation of horizontal lines with 20 and 24 cm length (three lines per length). NP4 and NP5 did not perform this task. Copying[a]: + complete copy of all objects; (+) neglect of object elements located at the objects' left side; – complete neglect of all objects. Clock face: + all numerals positioned; – omission of numerals. OP, operation/surgery.

[a] The copying task featured four different objects (car, flower, house, tree). Two objects were placed on the right and two on the left side of the page. If objects were not copied at all, or essential object elements were not redrawn, we scored this as an omission.

Apparatus and Procedure

Arm movements were recorded at a sampling frequency of 100 Hz with an optoelectronic 3D camera system (BTS ELITE, Milano, Italy). Infrared spherical markers were attached to the shoulder, elbow (lateral epicondyle), wrist (ulnar styloid process), and to the base of the index finger (second metacarpal). During the experiment, subjects sat in front of a table (80 x 80 cm) and performed unrestricted three-dimensional pointing movements with their right hand to three targets. Light-emitting diodes (LED) served as targets. LEDs were attached to the end of a 20-cm-long wire hanging from a vertically mounted metal rod. Upon contact, LEDs could swing freely. This setup allowed hypermetric motion and prevented hand movements from being slowed down passively by a rigid support panel. The three LEDs were positioned in front of the subject at eye level and arranged in a straight line. A central LED was aligned to each subject's sagittal head/trunk midplane (see Fig. 1). The two other LED's were located at the level of the right and left shoulders (with respect to the mediolateral axis). Subsequently, we determined how far the three LED's had to be placed in front of the body (distance along the anterior-posterior axis). This distance was body-scaled to 95% of total arm length when the subject was pointing to the leftward target. The above procedure assured that all targets were reachable without requiring flexion or rotation of the trunk, yet guaranteed a substantial movement amplitude of the hand.

At the beginning of each trial, the right index finger rested on a marked position at the right, close end of the table. Each subject's vertical head and body axes were aligned. During each trial one of the three LEDs was illuminated. The order of LED presentation was pseudo-random. Upon an acoustic signal, which served as a preparatory signal, subjects pointed to the lighted LED with a "comfortable" movement speed. The target LED began to light up 400 ms after the preparatory signal and stayed "on" for 8 s. Pointing movements were carried out either under normal room light or in complete darkness. The "darkness" condition was employed to restrict vision of the hand during the transport phase. Subjects pointed to each LED target seven times in both lighting conditions, performing a total of 42 arm movements. Patient NP4 was unable to fulfill the complete protocol. She performed movements only in darkness.

Data Analysis

We recorded a total of 651 trials across all subjects. Of those, 593 were used for the subsequent kinematic analysis. In the remaining 58 trials the hand marker was obscured for periods exceeding 100 ms during execution. In these cases we did not interpolate the missing time-position data but discarded the complete trial. Trials with missing data were evenly spread among all patients. The three-dimensional time-position data of each joint marker

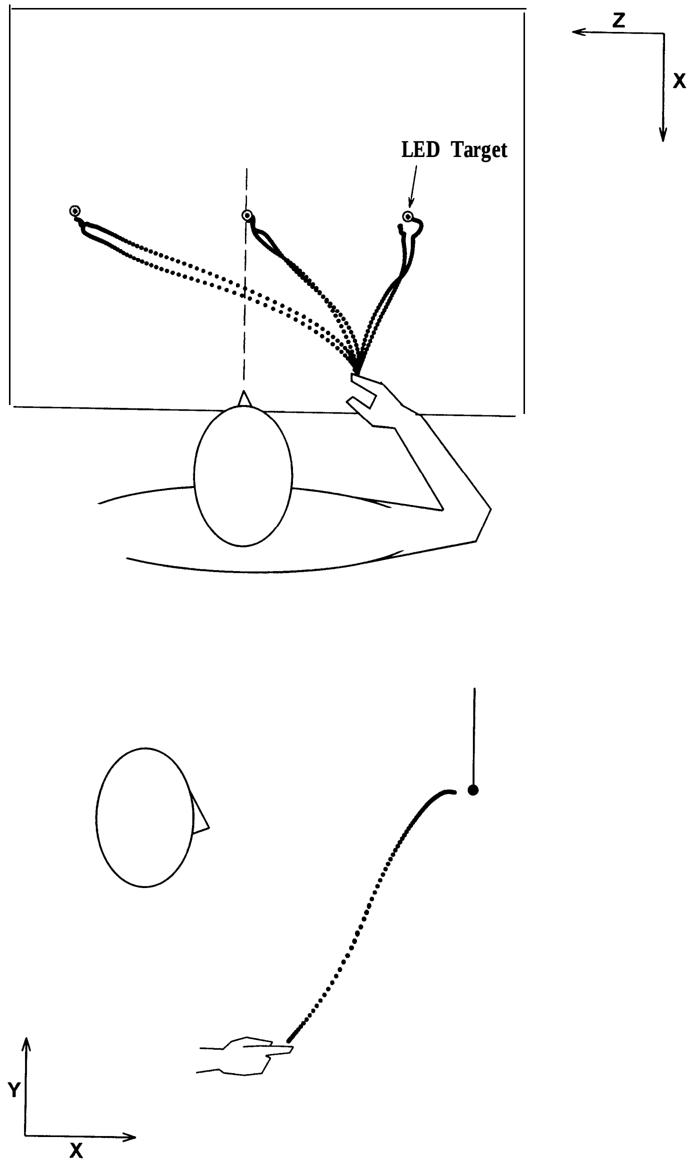

FIG. **1.** Schematic drawing of the experimental setup. Top graph represents a view from above (transverse plane), bottom graph presents a side view (sagittal plane). Position of the nasion was aligned with respect to the central LED. Right and left LED were positioned at the respective shoulder level (tip of the acromion). Shown are hand paths of a healthy subject.

were filtered applying the automatic model-based bandwidth selection procedure LAMBDA (D' Amico & Ferrigno, 1992). Within the scope of this paper, we report only on the kinematics of the hand marker, representing the most distal part of the arm.

The following kinematic parameters were obtained: First, based on the time-position data we derived tangential hand velocity, using a three-point differentiation technique. With the help of a customized digital signal processing software, we subsequently determined the *peak tangential velocity* (V_{max}) and *time to peak hand velocity* (TV_{max}). Second, we computed *total movement time* (MT) as the time between movement onset and the kinematic end of movement. We defined the "kinematic end" of each movement as the time when hand velocity decreased to 5% of V_{max} after reaching its peak. Applying this criterion allowed us to clearly distinguish between movement time and holding time after the target was reached. Third, we obtained the time from peak velocity to the kinematic end by subtracting TV_{max} from MT. This period represents the *deceleration time* (DT) of the hand.

Statistical Analysis

We computed means and standard deviations for each kinematic parameter. Three-way repeated measures ANOVAs were performed on the factors GROUP, TARGET, and LIGHTING. GROUP refers to healthy controls, the RH group, and the group of neglect patients. TARGET refers to the three LED positions: left, center, right. LIGHTING refers to room illumination: darkness or room lights. Two-sample *t* tests were employed for post-hoc mean comparisons. In these tests, the critical significance level of .05 was adjusted according to the Bonferroni-Holm method.

RESULTS

Figure 2 shows exemplary hand paths for each experimental group. In this particular figure, the density of consecutive data points was greater in the neglect trajectories than in the control group, indicating that these patients took longer to reach the desired target. The corresponding group means of MT corroborate this assessment. While neglect patients did not take significantly longer to terminate the movement than right-hemispheric patients, both patient groups had a larger MT with respect to the healthy controls ($t_{0.05,10} = -3.42$, $p < .008$; $t_{0.05,10} = -4.45$, $p < .002$). All subjects needed significantly more time to point in the darkness than in the lighted condition ($F(1, 25) = 31.07$, $p < .0001$), but did not reveal significant differences in MT as a function of target location (see Table 2). That is, subjects kept MT relatively constant across all three targets.

Figure 3 depicts profiles of tangential hand velocities that correspond to the hand paths shown in Fig. 2. Unimodal velocity profiles were preserved in the RH patient for all target directions, a finding in line with the results

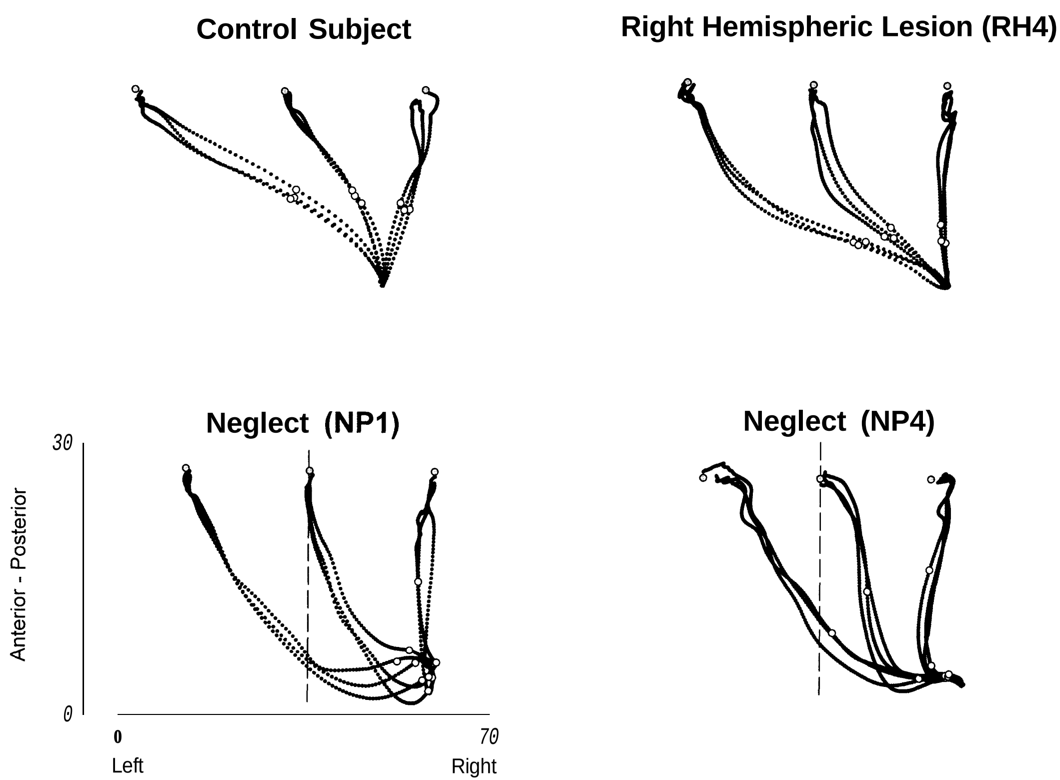

FIG. 2. Exemplar hand paths of four subjects during pointing. The trajectories of two neglect patients serve to illustrate the range of kinematic responses seen in these patients. Time interval between successive data points is 10 ms. Hand paths are projected in the transverse plane (view from above). The white circles indicate the time of V_{max}.

TABLE 2
Movement Time (in Milliseconds) for Each of the Three Targets

	Left				Center				Right			
	Light		Dark		Light		Dark		Light		Dark	
Healthy controls	930	(262)	1211	(271)	850	(173)	1207	(323)	850	(209)	1272	(325)
RH group	1275	(164)	1692	(467)	1201	(178)	1454	(236)	1127	(223)	1612	(333)
Neglect	1390	(334)	2175	(607)	1289	(172)	2141	(635)	1324	(220)	2187	(596)

Note. Values represent group means. Respective standard deviations are in parentheses. Large standard deviations in the neglect group are due to slowed performance of two patients (NP4, NP5), who needed up to 3800 ms to point to a target in darkness.

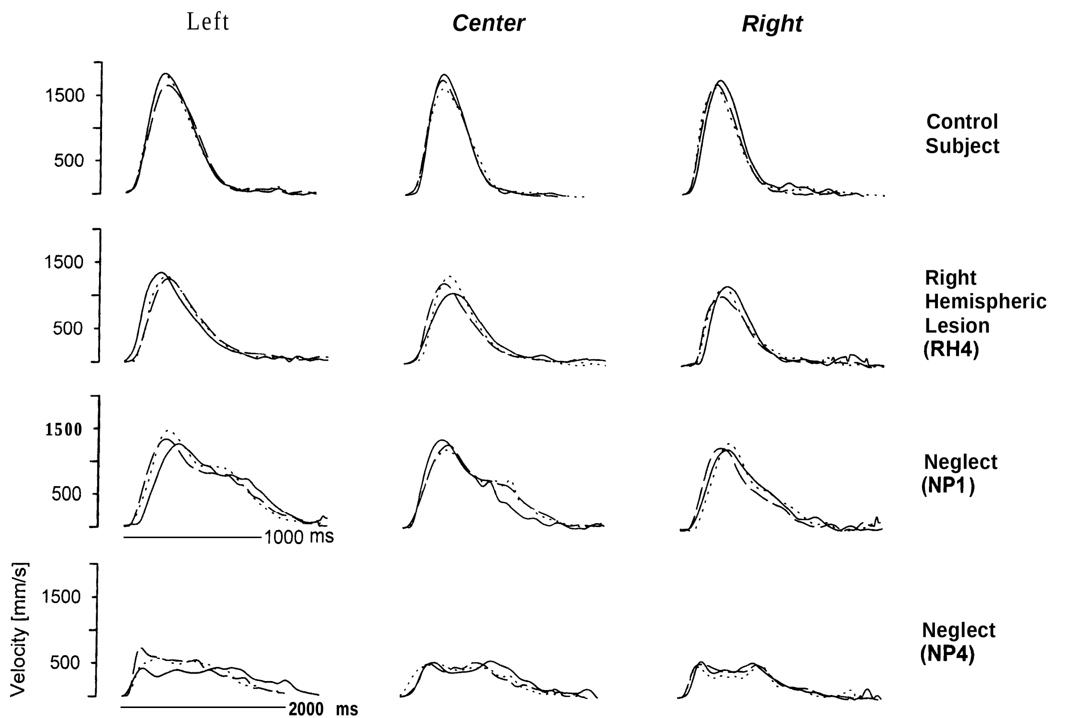

FIG. 3. Exemplar profiles of tangential velocity of the hand during pointing. Data correspond to the hand paths shown in Fig. 1. The right-hemispheric patient and both neglect patients attained lower V_{max}, yet a clear gradient in performance from right to left targets was not observed. The bell-shaped profile during the transport phase was basically preserved in all patients with the exception of NP4 and NP5.

TABLE 3
Tangential Peak Hand Velocity (V_{max}) for Each of the Three Targets during Pointing in the Dark

	Left		Center		Right	
Healthy controls	1465	(272)	1324	(256)	1259	(311)
RH group	1156	(178)	1096	(135)	1069	(140)
Neglect	1148	(298)	1076	(282)	1040	(340)

Units are in (mm/s). Values represent group means. Standard deviations are in parentheses. Corresponding mean values during pointing in the lighted condition were higher by 50-300 mm/s in all groups.

of Fisk and Goodale (1988). This was also true for three neglect patients (NP1, NP2, NP3). However, unimodality was lost in the profiles of two patients who exhibited extremely severe forms of neglect (NP4, NP5). Besides revealing a largely decreased peak velocity, the reaches of the latter patients were accompanied by multiple velocity peaks, giving rise to more segmented endpoint paths.

The ANOVA analysis yielded a significant GROUP effect for V_{max}. However, post-hoc tests revealed no significant differences between any of the groups once critical a was adjusted. The corresponding mean peak velocities are shown in Table 3. The main effect of V_{max} for TARGET was not significant (p > .05). That is, in our sample, peak hand velocity did not change systematically between right and left target presentations. There was, however, a slight, yet not significant, trend for V_{max} to increase from right to left targets. This rise in V_{max} was expected given that movement distance increased from right to left. Accounting for the differences in movement distance between targets by computing a measure of kinematic scaling (V_{max}/ movement distance) did not provide further insights to our questions. None of the groups showed a significant degree of scaling among the three target or the lighting conditions. Further, the main effect for LIGHTING was not significant (p > .05), implying that early visibility of the hand during the transport phase did not influence the amplitude of V_{max}.

A second aspect of temporal coordination concerned the timing of V_{max} during execution. In terms of the time to peak velocity (TV,,,), patients in the RH and the neglect groups needed longer to reach peak velocity than the controls ($t_{0\ 05\ 11}$ = -2.83 and -3.02, p's < .019). Main effects of TARGET and LIGHTING for TV,,, failed to reach statistical significance (p < .05). That is, in our sample, TV,,, did not change as a function of target position in any of the groups. Corresponding group means split by TARGET are shown in Fig. 4.

While the duration of the acceleration phase (time to peak velocity) did not vary between the two lighting conditions in any group, overall movement time increased in all subjects during pointing in darkness, effectively giving

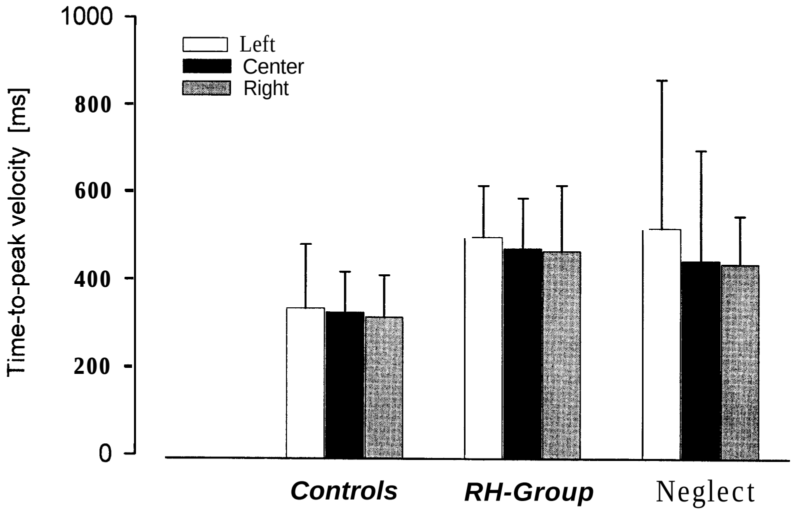

FIG. 4. Time to peak velocity as a function of target location. Shown are group means for each target position. Because pointing in the darkness did not affect the temporal onset of V_{max}, data are pooled across both lighting conditions. Error bars represent one standard deviation. Note that the absolute onset of V_{max} did not reveal systematic right-to-left changes.

rise to a significantly larger deceleration time (DT) in that condition ($F(1, 25) = 44.44$, $p < .0001$). DT differed significantly between groups. RH patients had a longer DT than controls ($t_{0.05,11} = -2.73$, $p < .023$), while neglect patients exhibited a larger deceleration phase than RH patients ($t_{0.05,10} = -3.13$, $p < .014$). The latter difference was due to extremely long DT of two neglect patients (NP4, NP5). When these two patients were subsequently excluded from the analysis, the differences between RH and neglect patients vanished. This implies that the remaining neglect patients (NP1–NP3) showed DT's similar to those of the RH group.

The GROUP*LIGHTING interaction for DT was not significant, indicating that the enlarged deceleration phase during pointing in the dark was not specific to a particular group. Finally, deceleration time did not change sys-

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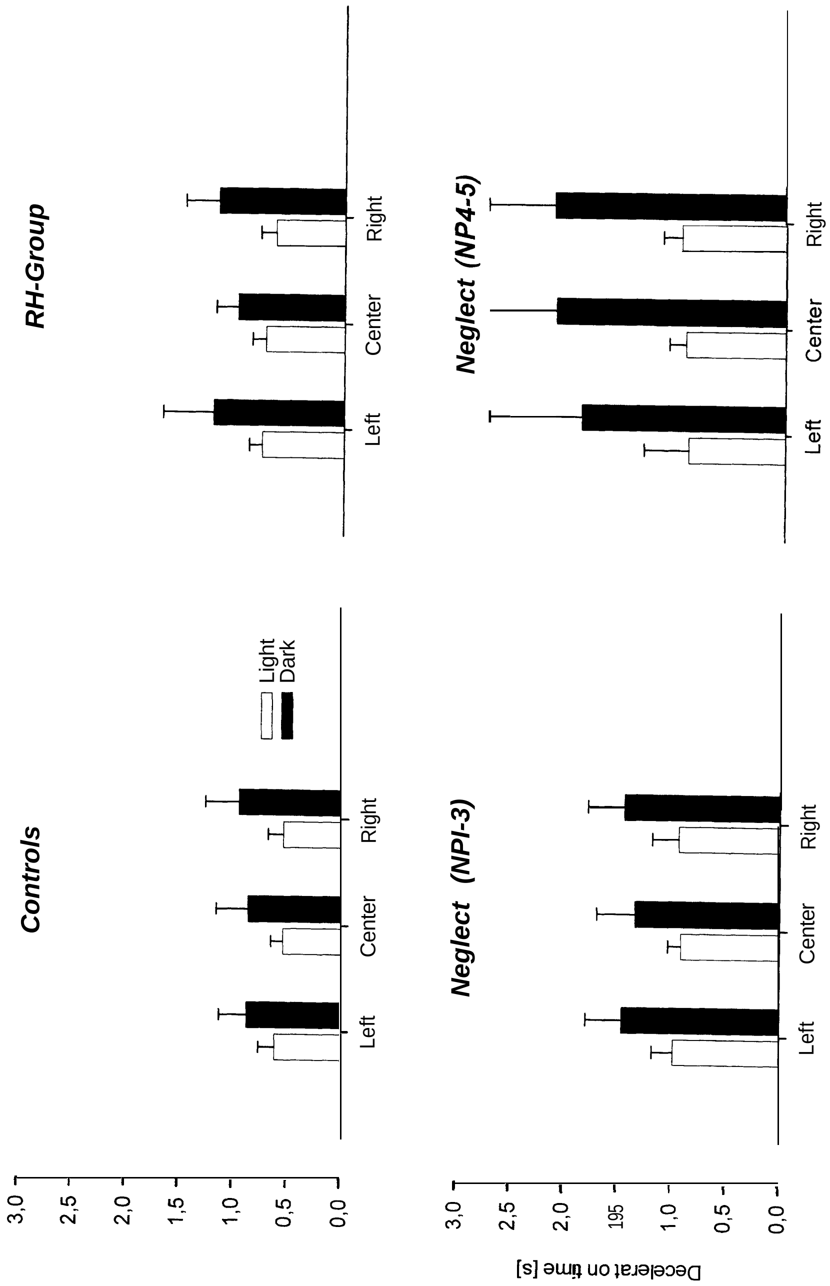
FIG. 5. Effect of LIGHTING on deceleration time. Values are groups means. Error bars represent one standard deviation. All subjects increased deceleration time when pointing in darkness. Here neglect patients are split into two subgroups. Statistically, neglect patients showed longer DTs than RH patients. This difference was due to the extremely slow performance of NP4 and 5, especially during pointing in darkness. Patients NPl-3 were not different from the RH group. No main effect of target position was found ($p > .05$).


Controls
RH-Group
Neglect (NPI-3)
Neglect (NP4-5)
Light
Dark
Left
Center
Right
Decelerat on time [s]
3,0
2,5
2,0
1,5
1,0
0,5
0,0

tematically among the three targets. That is, the subjects in our sample did not require a longer DT when pointing to the leftward target. Figure 5 presents the group means split by TARGET and LIGHTING.

DISCUSSION

Pointing with the hand to an object in the immediate surroundings represents a natural, highly overlearned task for human adults, giving rise to stereotypic kinematic patterns (Konczak, Borutta, Topka, & Dichgans, 1995; Soechting, 1989). We asked a group of five consecutively admitted patients suffering from neglect to perform such pointing movements to three targets in peripersonal space. On the basis of recent findings indicating that neglect patients reveal direction-specific impairments of movement speed (Mattingley et al., 1994), we expected to observe a systematic bias in the resulting hand velocity profiles as a function of target location.

No Evidence for Directional Bradykinesia

Our neglect patients exhibited clear signs of abnormal slowness during movement execution. Yet in most aspects of endpoint velocity control, the majority of neglect patients in our sample behaved like the patients with right-hemispheric lesions but no neglect. Both patient groups showed a reduced peak hand velocity and required a significantly longer time to terminate their arm movements. With the exception of two neglect patients, all patients still exhibited unimodal endpoint velocity profiles and neither acceleration time (time to peak velocity), nor the deceleration time revealed a right-to-left gradient. None of these parameters unequivocally indicates that neglect patients experience direction-specific deficits in velocity control that exceed the range of impairments observed in patients with right-hemispheric brain damage but no neglect. Most important, none of the kinematic parameters we analyzed presented convincing evidence for a directional bias of velocity dyscontrol.

Why do our findings differ from those obtained by Mattingley et al. (1994)? First, there are notable differences in task demands. While Mattingley and colleagues tested small range hand motions (distance < 12.5 cm amplitude) in the transverse plane, we examined three-dimensional pointing movements with a much larger amplitude (distance >40 cm). Yet, differences in amplitude and orientation are hardly responsible for our not finding directional bradykinesia. On the contrary, impairments in motor planning and execution should become more prominent when humans are required to perform large amplitude limb movements to the outer range of their workspace, because these movements necessarily encompass rotations of large limb segments and require the involvement of complex sets of mono- and biarticular muscles. More important than mere amplitude seem to us differences in the starting point of the hand. In our experiment, all movements originated in the right hemispace and were performed to targets in both hemispaces. In contrast, Mattingley and co-workers found evidence of directional bradykinesia only when rightward movements originating in left hemispace were compared to leftward movements with a starting point in right hemispace. Previous research has shown that starting position may influence visual attention in neglect (Duhamel & Brouchon, 1990), while the results by Mattingley and co-workers then provide evidence that starting position may affect movement kinematics. Given that differences in starting position may account for the differences in results between the present experiment and the study by Mattingley et al. (1994), a critical experiment to clarify the issue would be the inclusion of movements directed rightward from the starting point of the hand.

A second issue concerns possible differences in the patient samples of the two studies. Mattingley et al. (1994) found differences only in seven patients with severe spatial neglect and not in patients with mild subclinical neglect. According to their clinical classification, all of our five patients are classified as suffering from severe spatial neglect. Consequently, it is highly unlikely that our patients did not show any signs of directional bradykinesia, because they were experiencing a milder form of neglect than those patients studied by Mattingley et al. (1994).

Two patients (NP4 and NP5) with extremely severe neglect symptoms exhibited drastically slowed motor patterns seen in neither the remaining neglect nor in the RH patients. Peak hand velocity was markedly reduced in NP4/5; their pointing movements no longer revealed unimodal velocity profiles. Their hand paths were jerky, and the corresponding velocity curves were multipeaked. Thus, we need to address the question of whether these deficits in the control of endpoint velocity observed in NP4/5 are specific to neglect. Alternatively, these impairments could just reflect manifestations of acute and/or extensive brain damage to the right hemisphere. Indeed, rather similar endpoint kinematic profiles are also observed in other neurological disorders with completely different pathomechanisms, i.e., dystonia (Inzelberg, Flash, Schechtman, & Korczyn, 1995). Thus, we cannot exclude the possibility that lesions in other brain areas will lead to similar behavioral outcomes, i.e., a general slowness of movement.[1] However, decisive for the issue of neglect-specific deficits in velocity control is the fact that our patients showed no evidence of directional bradykinesia while pointing to targets in the left and right hemispace.

Vision of the Hand Does Not Overcome General Bradykinesia in Neglect

Vision of the hand is not important during its transport phase, which comprises about 75% of total movement time. Only during the final approach

[1] More so, even healthy subjects will not exhibit unimodal velocity profiles when asked to reach very slowly.

phase does vision play a major role when the hand is visually guided to the target. In darkness, the motor system has to rely on open-loop mechanisms that are not dependent on visual feedback, but will result in longer movement times when accuracy is required (as in pointing). The underlying reason for the longer movement time is not a change in the initial acceleration phase, but a prolonged time spent in deceleration. That is, the motor system attempts to keep the transport-related parameters unchanged, but needs extra "search" time to bring the hand to the desired target. All of our subjects behaved in this way, effectively increasing deceleration time during pointing in the dark. However, none of the other movement speed parameters indicated significant differences between pointing in the dark and lighted conditions. Most importantly, the simultaneous presence of visual information about target and hand does not alleviate the bradykinetic symptoms seen in these patients. The finding that patients with right-hemispheric brain damage show the same type of light-dark adaptation when reaching with their right hand implies that the basic properties of open- and closed-loop control are still functioning. Neglect patients with right-sided lesions were not exceptional in that respect.

Concluding Remarks

We recently reported (Karnath et al., 1997) that neglect patients do not show characteristic deviations in their hand paths during pointing when compared to healthy controls. The straightness of their hand paths was similar to those of controls. Neglect patients did not exhibit systematic deviations of the path during the transport phase; they did not misreach the target in the approach phase requiring them to perform a corrective movement. A specific bias in the spatial layout of their hand trajectories seemed possible given that these patients report a 10–15° deviation of their subjective body midline. We here report that next to intact positional kinematics of the hand path, the corresponding velocity profiles of neglect patients were markedly slowed with respect to those of healthy adults. Yet, they were not different from those profiles of patients with right-hemispheric lesions who exhibited no neglect. Specifically, we did not observe differences between reaches in the left and right hemispace. That is, we found signs of a general bradykinesia in the neglect patients, but no evidence of directional bradykinesia. Considering both spatial and speed parameters of hand trajectory formation we observed that the motor impairments of neglect patients do not exceed the level seen in patients with right-hemispheric lesions but no neglect. We conclude that visual neglect induces characteristic changes in exploratory behavior, but not in the kinematics of goal-directed movements to objects in peripersonal space.

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