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RESEARCH ARTICLE

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Recruitment and sequencing of different degrees of freedom during pointing movements involving the trunk in healthy and hemiparetic subjects

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Abstract Previous studies have shown that in neurologically normal subjects the addition of trunk motion during a reaching task does not affect the trajectory of the arm endpoint. Typically, the trunk begins to move before the onset and continues to move after the offset of the arm endpoint displacement. This observation shows that the potential contribution of the trunk to the motion of the arm endpoint toward a target is neutralized by appropriate compensatory movements of the shoulder and elbow. We tested the hypothesis that cortical and subcortical brain lesions may disrupt the timing of trunk and arm endpoint motion in hemiparetic subjects. Eight hemiparetic and six age-matched healthy subjects were seated on a stool with the right (dominant) arm in front of them on a table. The tip of the index finger (the arm endpoint) was initially at a distance of 20 cm from the midline of the chest. Wrist, elbow, and upper body positions as well as the coordinates of the arm endpoint were recorded with a three-dimensional motion analysis system (Optotrak) by infrared light-emitting diodes placed on the tip of the finger, the styloid process of the ulna, the lateral epicondyle of the humerus, the acromion processes bilaterally, and the sternal notch. In response to a preparatory signal, subjects lifted their arm 1–2 cm above the table and in response to a "go" signal moved their endpoint as fast as possible from a near to a far target located at a distance of 35 cm and at a 45° angle to the right or left of the sagittal midline of the trunk. After a pause (200–500 ms) they moved the endpoint back to the near target. Pointing movements were made without trunk motion (control trials) or with a sagittal motion of the trunk produced by means of a hip flexion or extension (test trials). In one set of test trials, subjects were required to move the trunk forward while moving the arm to the target ("in-phase movements"). In the other set, subjects were required to move the trunk backward when the arm moved to the far target ("out-of-phase movements"). Compared with healthy subjects, movements in hemiparetic subjects were segmented, slower, and characterized by a greater variability and by deflection of the trajectory from a straight line. In addition, there was a moderate increase in the errors in movement direction and extent. These deficits were similar in magnitude whether or not the trunk was involved. Although hemiparetic subjects were able to compensate the influence of the trunk motion on the movement of the arm endpoint, they accomplished this by making more segmented movements than healthy subjects. In addition, they were unable to stabilize the sequence of trunk and arm endpoint movements in a set of trials. It is concluded that recruitment and sequencing of different degrees of freedom may be impaired in this population of patients. This inability may partly be responsible for other deficits observed in hemiparetic subjects, including an increase in movement segmentation and duration. The lack of stereotypic movement sequencing may imply that these subjects had deficits in learning associated with short-term memory.

Key words Motor control · Multijoint movement · Movement synergy · Stroke · Laterality · Redundancy · Human

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Introduction

A prevalent idea in motor control theory is that the internal representation of movement is linked to the output trajectory. For example, circles drawn in the air either vertically or horizontally look similar, even though dif-

ferent muscle groups are used in the task (Bernstein 1967). A similar idea has also been suggested by studies showing that, in planar pointing movements, endpoint trajectories are close to a straight line and tangential velocity profiles are bell-shaped regardless of target location (Morasso 1981; Flash and Hogan 1985; Gordon et al. 1994; see, however, Desmurget et al. 1997). The nature of the relationship between internal movement representation and output trajectories, however, is still a matter of debate. In particular, some studies have suggested that the nervous system controls such global characteristics of movement as energy cost (Hatze and Buys 1977) or smoothness defined by the rate of change in acceleration (Flash and Hogan 1985). On the other hand it has been suggested that trajectories are properties emerging from changes in control variables influencing the equilibrium state of the system (Feldman 1966; Feldman and Levin 1995).

Another important aspect of voluntary movement is the redundancy in the number of degrees of freedom (DFs) of the body so that, in theory, a given task can be executed in different ways. This ability, called "motor equivalence," does not exclude a conservative behavior leading to stereotyped movement patterns when produced in a reproducible context. For example, studies of rhythmical movements show that successive trajectories, while never actually repeating themselves, follow a similar topological pattern (Bernstein 1967). A stereotyped arm postural configuration has also been observed in prehension movements (Desmurget and Prablanc 1997).

These two aspects of movement (internal representation of movement and redundancy) can actually complement each other. Indeed, one may say that the nervous system uses redundancy in order to produce topologically similar movement patterns by applying a similar internal representation to different effectors. In approaches to the redundancy problem, the concept of movement synergy has evolved (Bernstein 1967; Gurfinkel et al. 1971; Turvey 1990), defined as a unit of coordination of DFs fulfilling a specific functional goal.

In previous studies focusing on the redundancy problem, arm pointing movements involving the trunk have been investigated (Kaminski et al. 1995; Ma and Feldman 1995; Saling et al. 1996). In particular, Ma and Feldman (1995) observed that, in movements to a target placed within the limits of arm reaching, the addition of trunk motion did not affect the endpoint trajectory, and they hypothesized that this particular task involves two synergies: one moving the arm joints displacing the arm endpoint to the target (reaching synergy), and the other moving the trunk and arm joints without affecting the position of the endpoint (compensatory synergy). One may suggest that the changes in arm joint angles elicited by the two synergies are combined as independent actions (the principle of superposition). As a result, the trunk recruitment may be associated with substantial modifications in the arm joint angles without influencing the endpoint trajectory. The suggestion that synergies, as functionally independent units of coordination, are superimposed may be illustrative of the capacity of the brain to meet several functional requirements simultaneously. In other words, the nervous system would be able to attain two or more functional goals by combining the movement synergies required for each. The existence of a compensatory synergy was substantiated by the finding that in a majority of trials the trunk began to move before the onset and stopped moving after the offset of the endpoint movement, indicating that the effects of trunk motion were adequately compensated by movements at the elbow and shoulder. In grasping movements (Saling et al. 1996) the trunk typically stops moving after the offset of endpoint movement, also implying the use of a compensatory synergy.

In another study (Kaminski et al. 1995), seated subjects were asked to lean forward naturally in order to touch targets placed within and beyond their reach. In all conditions, the endpoint trajectory remained smooth throughout the movement, indicating that trunk motion was well incorporated in the overall goal of transporting the endpoint to the target. Subjects also showed consistent temporal coupling between articulations (shoulder and elbow, hip and shoulder), indicating the presence of adequate compensation within the joint rotations to produce a smooth endpoint trajectory. The presence of compensation was also obvious from the finding that when trunk motion was required, it started before the onset and finished after the offset of endpoint movement.

Movement synergies during multijoint tasks can be further studied by comparing the motor behavior of healthy subjects and subjects with sensorimotor deficits such as hemiparesis resulting from stroke. Neurophysiological studies in animals show that planning and sequencing of movement may be distributed throughout different areas of the brain, namely the cerebellum (Ivry and Keele 1989), the basal ganglia (Alexander and Crutcher 1990), and the supplementary motor area (Mushiake et al. 1991). Tasks involving the coordination of arm and trunk movements may involve both the premotor cortex, which plays a role in postural adjustments through the control of axial musculature (Wise and Strick 1984), and parietal area 5, in which cells are directionally tuned according to endpoint trajectory (Kalaska et al. 1990). In a recent study of regional cerebral blood flow in humans, it has been shown that the activity in the anterior cerebellum and the ventral premotor area increased during finger tapping tasks requiring coordination and rapid reversals (Winstein et al. 1997). Damage, due to stroke, to any of those structures or their pathways is liable to cause deficits in movement coordination or synchronization of different movement components. The study of movements involving multiple DFs in stroke patients may give us further insight into the locus of control of coordination in the central nervous system.

In the present study, we hypothesized that the coordination of movement synergies observed in healthy subjects would be disrupted in patients with cortical and subcortical lesions (i.e., hemiparetic subjects). This hypothesis was tested by analyzing the ability of hemiparetic subjects to compensate for trunk movement during a pointing task. Movement trajectories and timing pat-

terns were compared with those of healthy subjects. Some of the data from this study have appeared in abstract form (Archambault et al. 1997).

Materials and methods

Subjects

Six healthy controls (46±16 years old) and eight right hemiparetic (left-sided stroke) subjects (50±16 years old) participated in the experiment. All subjects were informed of the experimental procedures and signed consent forms accepted by the local Ethics Committee. Both control and hemiparetic subjects were right-hand dominant. Demographic data on the hemiparetic subjects are presented in Table 1. Hemiparetic subjects met the following inclusion criteria: (1) they had sustained a single ictal event, at least 6 months previously; (2) they had no other neurological disorders; (3) they were able to perform reaching movements with the right upper extremity (Brunnstrom stages 4–6; Brunnstrom 1970); (4) they were able to understand instructions; (5) they had no neglect or attention deficits, as measured by the Bell's test (Gauthier et al. 1989); (6) they had no shoulder subluxation or arm pain. Subjects with left-sided stroke were selected in order to avoid problems of visual neglect often associated with right-sided stroke. All the hemiparetic subjects had followed the usual rehabilitation procedures associated with their condition. Although sitting balance was not measured directly, all subjects were ambulatory without aids and had no difficulty in maintaining a stable sitting posture during the experiment.

The control group was composed of six healthy individuals (four men and two women), who presented no history of neurological disorders or physical deficits involving the upper limbs or the trunk.

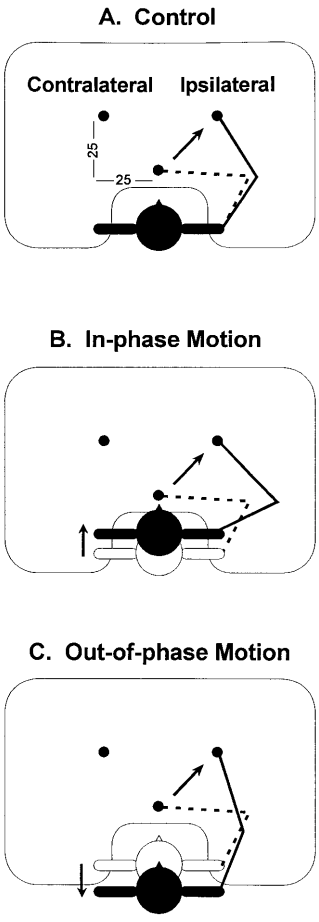

Fig. 1A–C Schematic showing ipsi- and contralateral targets, arm movement without trunk (**A**), in-phase (**B**) and out-of-phase (**C**) arm and trunk movements. *Arrows* show direction of movement

Experimental procedure

Subjects sat in front of a 180 by 120 cm table which was at a height of 80 cm from the floor. Each was seated in a semicircular cut-out section of the table so that when their arm was in the initial target position, the elbow was flexed to 60–90° and their shoulder was in approximately 70° flexion and abduction. The initial target was located 20 cm from the sternum directly in front of the subject. Two final targets (ipsilateral and contralateral) were placed 35 cm away from and at a 45°angle to either side of the initial position (Fig. 1A). All targets were indicated by light-emitting diodes (4 mm^2) embedded in the Plexiglas surface of the table.

Subjects performed pointing movements with and without trunk motion to either the ipsi- or the contralateral target (Fig. 1). Following a preparatory signal, subjects lifted their arm and finger (endpoint) above the initial target. At an auditory "go" signal, subjects reached toward the final target without touching the table. After a short pause (200–500 ms) over the target, they moved their arm back to the initial position. In control trials (Fig. 1A), subjects moved their arm to the target without moving the trunk. In test trials, subjects either moved the trunk forward, in-phase with the endpoint (Fig. 1B), or backward (out-of-phase condition; Fig. 1C). Subjects were instructed to move the arm as accurately and as fast as possible, and to make the movement without correction. For trials with trunk movement, they were instructed to produce a substantial trunk excursion (about 11 cm) together with the arm movement. Trials in which upper body motion was less than 3 cm were not considered. Subjects were instructed to produce the trunk

Table 1 Demographic data and clinical scores for hemiparetic subjects (*M* male, *F* female, *MCA* middle cerebral artery)

Subject	Age (years)	Sex	Months post-stroke	Location and type of lesion	Fugl-Meyer score (max. 66)	Spasticity score (max. 16)
H1	40	M	63	Hemorrhage, temporal lobe and basal nuclei lesions	27	8 (mild)
H2	22	M	78	Hemorrhage, MCA, internal capsule and temporoparietal lesions	29	10 (mod.)
H3	56	F	115	Massive hemorrhage	50	8 (mild)
H4	53	F	32	Embolism, MCA, basal nuclei lesion	55	7 (mild)
H5	42	M	37	Hemorrhage, thalamus, internal capsule and basal nuclei lesions	57	10 (mod.)
H6	67	M	120	Hemorrhage, parietal lobe lesions	61	5 (no)
H7	52	M	34	Hemorrhage, central infracerebral and posterior internal capsule lesions	61	6 (mild)
H8	68	F	44	Hemorrhage, MCA, parietal lobe lesion	62	6 (mild)
$\bar{x}$	50±16		65±36			

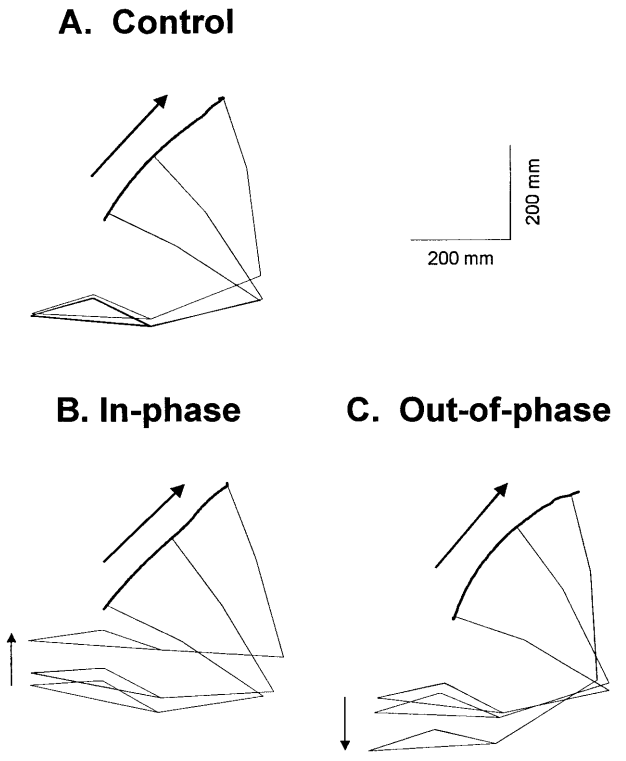

Fig. 2A–C Examples of endpoint trajectories (*thick line*), trunk (*triangles*) and arm positions (*stick figures*) for control (**A**), in-phase (**B**), and out-of-phase (**C**) movements to the ipsilateral target for one hemiparetic subject (H6)

motion by hip flexion/extension while sitting in a chair without sliding or raising the buttocks from it or moving the legs. Indeed, while producing hip flexion/extension, subjects could vary the curvature of the trunk. We measured an integral displacement elicited by the hip and other trunk DFs – the displacement of the sternum marker, which directly estimates the possible perturbing influences of the trunk movement on the arm endpoint. Possible rotations of the trunk about a vertical axis were also controlled by instructing the subjects to lean only the trunk forward/ backward in the sagittal direction. Based on the coordinates of three markers (on the sternal notch, left shoulder, and right shoulder) we observed that healthy subjects basically complied with the instructions. However, some trunk rotation was present in hemiparetic subjects (Fig. 2), which may represent one of the possible strategies to compensate the deficits at the level of the arm joints.

Blocks of ten trials for each of the four test conditions (ipsi- and contralateral targets, in- and out-of-phase trunk motion) were presented in random order with a rest period of about 2–5 min between the blocks. Each block was preceded by ten control trials, in which subjects pointed to the same target but without trunk motion. As each new task was presented, subjects were allowed to practice it for several trials (usually four to ten) until they felt comfortable.

Data collection

Three-dimensional kinematic data were collected (sampling rate 200 Hz) using an Optotrak Motion Analysis system consisting of infra-red light-emitting diodes (IREDs) and three cameras (model 3010; Northern Digital, London, Ontario). Markers were positioned on the tip of the index finger, wrist (styloid process of ulna), elbow (lateral epicondyle), both shoulders (acromion process) and sternum (sternal notch).

Clinical assessment

Clinical assessments of residual motor function and spasticity were performed by an experienced rehabilitation professional before the start of the experimental session (see Table 1). The hemiparetic subjects' motor performance was rated using the upper limb section of the Fugl-Meyer Functional Assessment (Fugl-Meyer et al. 1975), which measures reflex excitability as well as gross and fine motor skills. The assessment is scored on a total of 66 points, with 66 indicating normal performance. Our subjects' scores ranged from 27 (moderately impaired) to 62 (almost normal). Spasticity in the elbow flexors of the hemiparetic limb was scored on a valid and reliable scale that measures phasic (biceps tendon jerk, wrist clonus) and tonic (resistance to passive, full-range elbow extension) stretch reflex activity (Ashworth 1964; Levin and Hui-Chan 1992). Composite spasticity scores of 1–5, 6–9, 10–12, and 13–16 indicate "no," "mild," "moderate," and "severe" spasticity, respectively. According to this scale, one of our subjects had no, five had mild, and two had moderate spasticity.

Data analysis

Since no specific instruction was given for the return movement, only movements toward the far targets were analyzed. Position data were rotated and translated, using simple geometrical transformations, to a system of coordinates in the plane of the table with the origin at the position of the initial target. Data were then filtered numerically using a 10-Hz high-cutoff frequency. From the position data, endpoint and trunk velocity (three-dimensional and tangential) were calculated by numerical differentiation. Angles of shoulder flexion in the horizontal plane and of elbow extension were computed based on the scalar products of the vectors joining the appropriate IREDs. Averaged two-dimensional endpoint and trunk trajectories for each block of trials were also calculated by normalizing the x and y data with respect to time, using the quickest movement as a template. The mean position and standard deviation at each normalized unit of time were then computed.

Movement onsets and offsets for the endpoint and trunk were determined for each trial using the time at which tangential velocity rose above and fell below, respectively, 5% of its peak value. The endpoint and trunk final positions were determined by averaging the position data for the middle third of the movement between the offset of the movement to the target and the onset of the return phase. From the final position, the endpoint error was calculated and represented in radial coordinates: extent error was defined as the distance between the final endpoint position and the target, and directional error as the difference in angular coordinates between the final endpoint and the target.

Table 2 Synchronization index (S) measured by the difference (Δ) in movement onsets and offsets

	Movement onset		Movement offset	
S	Δ	Sequence	Δ	Sequence
–1	Δ<–20 ms	Trunk starts first	Δ>20 ms	Trunk stops last
0	20>Δ>–20	Simultaneous	20>Δ>–20	Simultaneous
+1	Δ>20	Endpoint starts first	Δ<–20	Endpoint stops last

Movement times were also used to examine the sequencing of arm and trunk recruitment. The difference in time (Δ) between the endpoint and trunk motion at both the onset and offset phases was used to define the index of synchronization (*S*) as indicated in Table 2. A threshold of Δ=±20 ms was used to distinguish between simultaneous and sequential involvement of the endpoint and trunk.

The effects of trunk motion on endpoint trajectory were characterized by the index of deviation from a straight line, which may also be considered as a measure of trajectory shape. This index was calculated as the ratio of the actual three-dimensional length of the path travelled by the endpoint to the length of the straight line joining the initial and final endpoint positions. Thus, for an ideal straight line, the index of deviation is 1, and for a half-circle the index is $\pi/2 \simeq 1.57$. The index of deviation is a measure related to the more commonly used maximal perpendicular distance between the ideal straight line and the actual trajectory. This index was preferred, since in some hemiparetic subjects trajectories could be S-shaped instead of arced, thus intersecting with the ideal straight line.

The number of movement units making up the reach was calculated as the number of peaks in the endpoint tangential velocity profile for each trial.

The effects of trunk motion (control, in-phase and out-of-phase conditions) and target location (ipsi- or contralateral; within-subject factors) on pointing error, synchronization, deviation, peak endpoint velocity, and number of movement units were analyzed statistically for each group using repeated-measures ANOVAs. Comparisons between the healthy and hemiparetic groups were made for endpoint peak velocity, index of deviation, elbow extension, and shoulder flexion using mixed-design repeated-measures ANOVAs. For one hemiparetic subject, movements to the contralateral target were not completed; this subject was therefore not included in the ANOVAs (subject H1). Additionally, correlations between the clinical tests, trajectory curvature, and synchronization were calculated using Pearson's product moment statistics. The level of significance for all tests was set at $P<0.05$.

Results

Kinematics

Figure 2 shows an example of endpoint and trunk movements to the ipsilateral target for a single trial in control and test conditions for one hemiparetic subject (H6). For this, as in other hemiparetic and healthy subjects, both the endpoint and trunk followed relatively smooth trajectories.

In healthy subjects, velocity profiles were bell-shaped (Fig. 3A, right panel) and trajectories for the same test conditions were highly reproducible. Peak velocities of endpoint (Fig. 4A–D) and trunk movement for healthy subjects varied from approximately 700 to 3200 mm/s and from 400 to 700 mm/s, respectively. The ANOVA on endpoint peak velocity showed a significant effect of both target location ($F_{1,5}=12.66$; $P<0.02$) and trunk involvement ($F_{2,10}=6.62$; $P<0.02$). An example of this behaviour can be seen in Fig. 4A, B; however, three healthy subjects showed no difference in endpoint velocity with trunk involvement for movements to the ipsilateral target, as can be seen from the individual means (Fig. 5A). Only one movement unit was observed in all the trials. Movement times ranged from 0.22 to 0.88 s for the endpoint, and 0.44 to 1.06 s for the trunk. Trunk amplitudes varied from 99 to 178 mm for the in-phase condition, and from –95 to –179 mm for the out-of-phase condition.

In contrast, endpoint trajectories in hemiparetic subjects were more variable (Fig. 3B), with endpoint velocity profiles showing multiple acceleration or deceleration

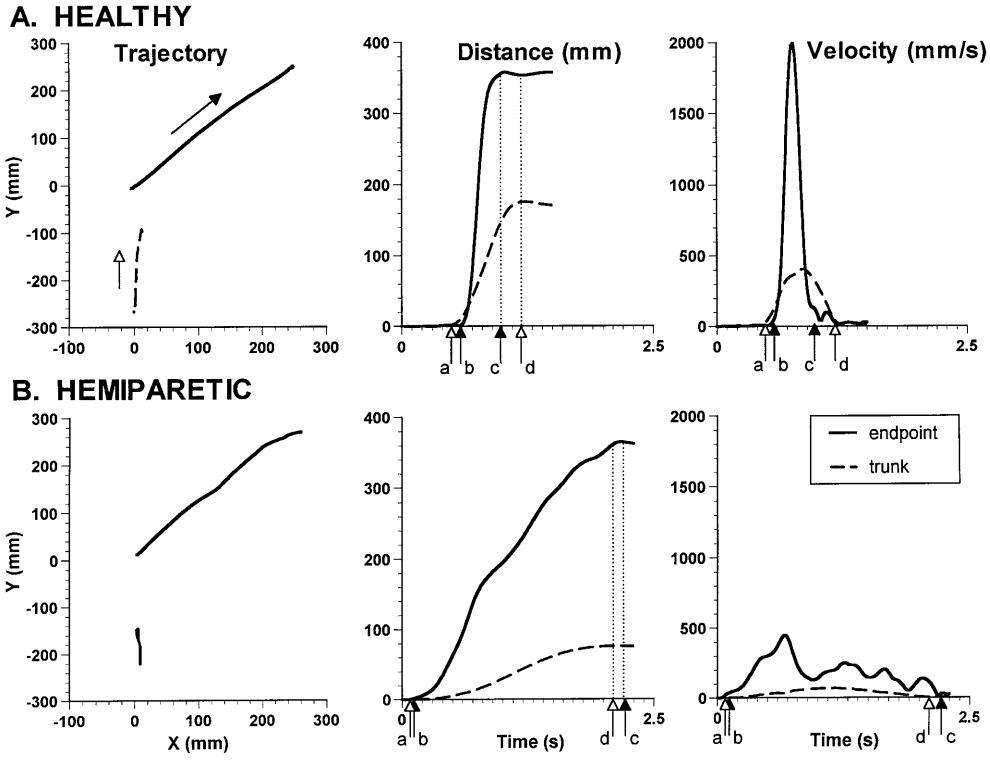

Fig. 3A, B Arm endpoint (*solid lines*) and trunk (*dashed lines*) *x-y* trajectories (*left panels*), the distance traveled by arm endpoint and trunk (*middle panels*) and tangential velocity profiles (*right panels*) for one healthy and one hemiparetic subject (H3; Fugl-Meyer score 50/66). Data are from a single trial (in-phase motion, ipsilateral target). *Arrows* on trajectory graphs indicate movement direction. *Arrows* on the distance and velocity graphs indicate the onsets (*a, b*) and offsets (*c, d*) of the trunk and endpoint motion, respectively

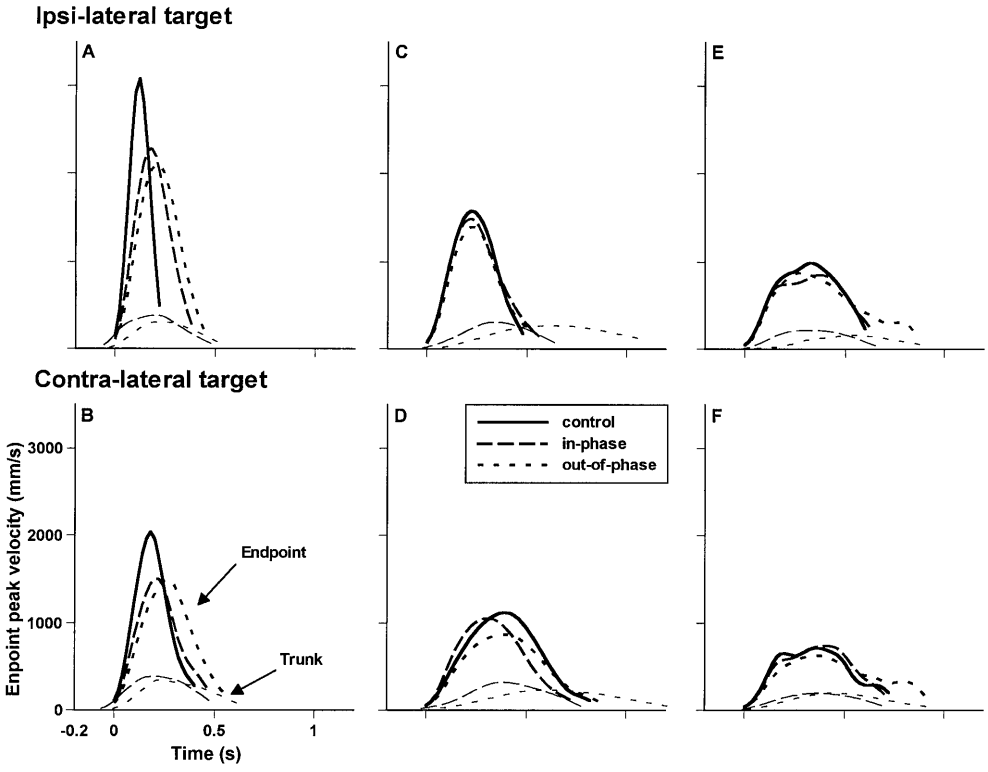

Fig. 4A–F Endpoint velocity for two healthy (**A, B; C, D**) and one hemiparetic (**E, F**) subject calculated from blocks of ten trials in three movement conditions (control, in-phase, out-of-phase)

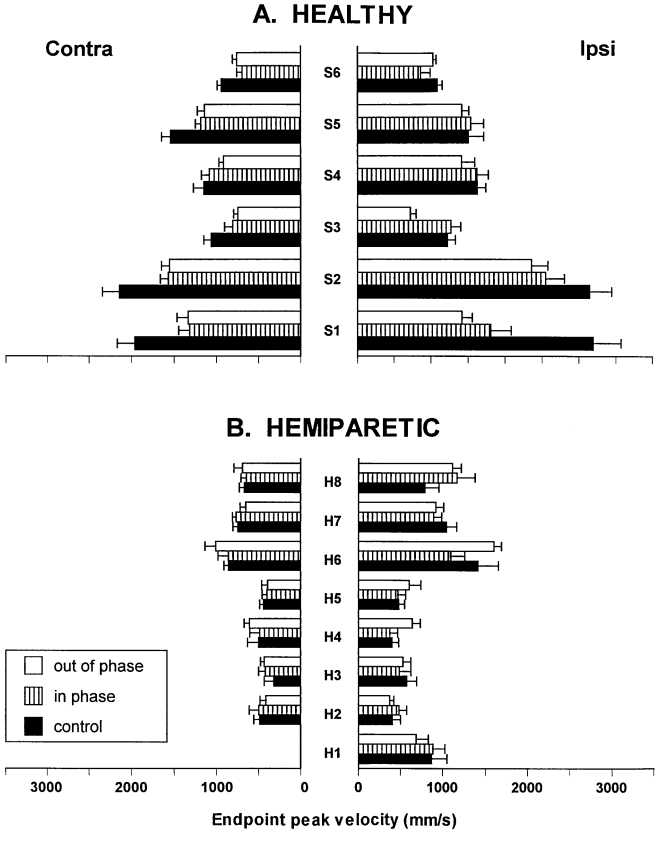

Fig. 5A, B Maximal endpoint velocity averaged across trials for control and test conditions (in-phase and out-of-phase). Hemiparetic subjects are ranked according to their Fugl-Meyer score (see Table 1)

phases. However, the trunk velocity profiles were smooth and bell-shaped. Peak velocities were much lower than in healthy subjects: 320 to 1600 mm/s for the endpoint ($F_{1,11}$=12.2; P<0.005) and 50 to 150 mm/s for the trunk ($F_{1,11}$=17.1; P<0.002). Movement times were consequently longer (0.51–3.02 s for the endpoint and 0.71–2.31 s for the trunk). Individual means for endpoint peak velocity in hemiparetic subjects are shown in Fig. 5B. Endpoint peak velocity was significantly affected by target location ($F_{1,6}$=5.96; P<0.05), but not by trunk involvement (Fig. 4C). Hemiparetic subjects in general displayed lower trunk movement amplitudes than healthy subjects for both the in-phase (57–179 mm; $F_{1,11}$=5.5; P<0.04) and out-of-phase (–31 to –134 mm; $F_{1,11}$=8.1; P<0.02) conditions. The number of movement units was always 1 in healthy subjects regardless of the task, but increased to between 5 and 8 in hemiparetic subjects. There was a significant effect of trunk motion on the number of movement units in hemiparetic subjects ($F_{2,12}$=4.8; P<0.03). This occurred for movements to the contralateral ($F_{2,12}$=6.1; P<0.02) but not the ipsilateral target ($F_{2,12}$=1.7; P>0.2). Elbow extension amplitude was lower in the hemiparetic subjects, but the difference was not significant over all the conditions ($F_{1,11}$=4.3; P<0.06). Further analysis showed a significant difference in elbow extension between the groups for movements to the contralateral target ($F_{1,11}$=10.7; P<0.007), but not for the ipsilateral target ($F_{1,11}$=2.7; P>0.1). Shoulder flexion, however, was similar in both groups of subjects. Movement statistics are summarized in Table 3.

The mean final endpoint locations for all movements in each condition for healthy subjects were well con-

Table 3 Endpoint movement time, peak velocity and error, number of endpoint movement units, joint movement amplitude, and extent of trunk motion for contra- and ipsilateral targets and three movement conditions (control, in-phase, and out-of-phase). Means (SD) obtained for the group of healthy (n=6) and hemiparetic (n=8) subjects

	Contralateral			Ipsilateral		
	Control	In-phase	Out-of-phase	Control	In-phase	Out-of-phase
Healthy						
Endpoint movement time**,*** (s)	0.52 (0.13)	0.66 (0.14)	0.67 (0.15)	0.39 (0.12)	0.50 (0.15)	0.55 (0.18)
Endpoint peak velocity**,*** (mm/s)	1422 (421)	1110 (322)	1075 (327)	1963 (951)	1608 (594)	1405 (567)
Extent error*** (mm)	–4 (13)	–3 (10)	–6 (8)	0 (8)	7 (6)	6 (4)
Directional error** (deg)	–0.2 (1.4)	0.2 (0.7)	0.2 (0.9)	–0.2 (0.6)	0.5 (0.5)	–0.9 (0.6)
Movement units (n)	1 (0)	1 (0)	1 (0)	1 (0)	1 (0)	1 (0)
Elbow extension (deg)	118 (16)	96 (15)	131 (16)	97 (10)	79 (14)	119 (20)
Shoulder flexion (deg)	47 (11)	41 (15)	48 (7)	–3 (3)	–22 (7)	7 (7)
Extent of trunk motion (mm)		134.2 (18.2)	–130.4 (30.1)		130.4 (32.3)	–124.6 (23.8)
Hemi						
Endpoint movement time*,** (s)	1.58 (0.94)	1.39 (0.61)	1.51 (0.73)	1.13 (0.54)	1.24 (0.49)	1.27 (0.64)
Endpoint peak velocity*,** (mm/s)	587 (205)	584 (178)	603 (216)	829 (383)	736 (314)	805 (385)
Extent error(mm)	–9 (15)	–6 (11)	–17 (23)	1 (10)	3 (11)	–10 (19)
Directional error (deg)	–1.8 (2.3)	–0.6 (0.8)	–1.5 (3.6)	–0.3 (0.9)	0.5 (1.2)	–1.6 (2.5)
Movement units*,*** (n)	5 (2)	6 (4)	8 (4)	5 (3)	5 (2)	6 (2)
Elbow extension (deg)	110 (13)	97 (10)	126 (16)	105 (10)	87 (14)	126 (20)
Shoulder flexion (deg)	29 (8)	25 (10)	36 (9)	–5 (7)	–16 (7)	7 (8)
Extent of trunk motion*(mm)		93.1 (28.7)	–78.5 (38.3)		117.1 (39.2)	–82.5 (29.6)

* Significant difference between the groups; **significant effect of target location within the group; ***significant effect of trunk involvement within the group

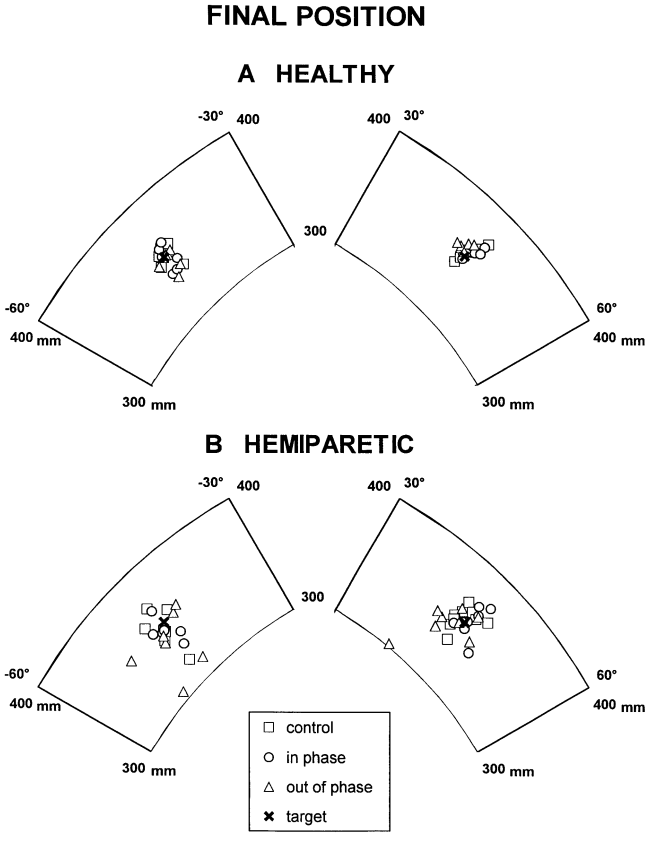

Fig. 6A, B Mean locations of the arm endpoint for healthy (**A**) and hemiparetic (**B**) subjects calculated from blocks of ten trials in three movement conditions (control, in-phase, out-of-phase)

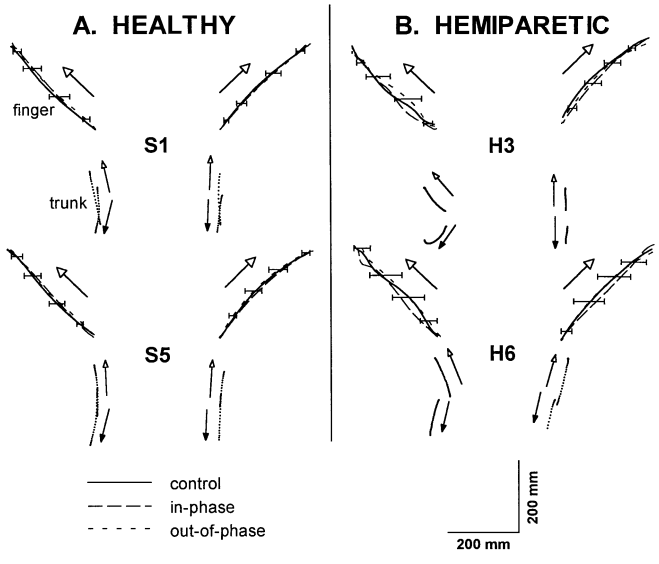

Fig. 7A, B Typical averaged trajectories of the endpoint and trunk in the three movement conditions to the contralateral and ipsilateral targets for two healthy (*S1, S5*) and two hemiparetic subjects (*H3, H6*; Fugl-Meyer score 50/66 and 61/66, respectively). For each subject and target, only standard deviations in the *x* direction from the three movement conditions are displayed (*error bars*). Standard deviations in the *y* direction were of similar magnitude as in the *x* direction

strained and tightly distributed around the target, while those for hemiparetic subjects had a slightly larger distribution (Fig. 6). A few subjects from both groups had a tendency to lean sideways with their trunk toward the target. However, the amplitude of trunk motion in the forward or backward direction was still substantial. For

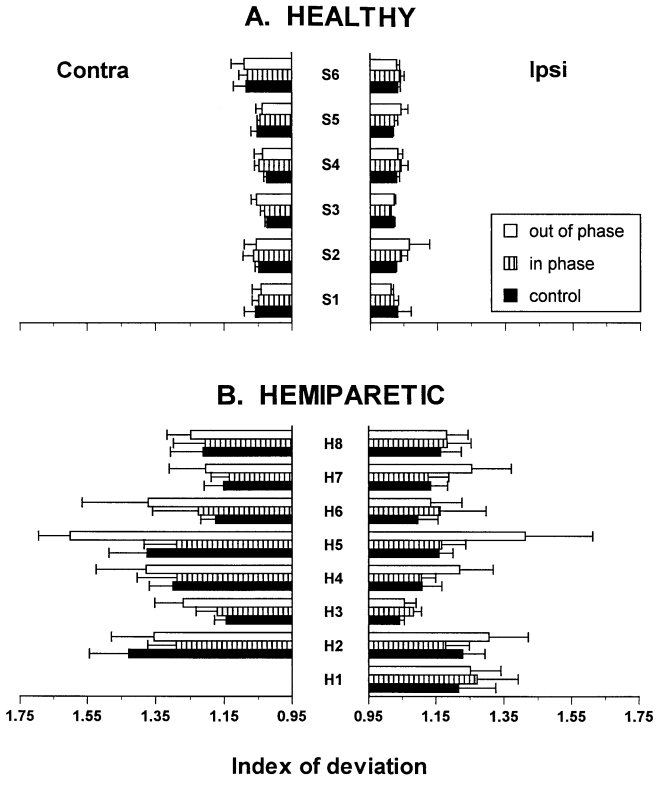

Fig. 8A, B Indices of deviation averaged across trials for control and test conditions (in-phase and out-of-phase). Hemiparetic subjects are ranked according to Fugl-Meyer score

the hemiparetic subjects (Fig. 6B), there was no significant effect of trunk motion or target location on endpoint extent or directional error. For healthy subjects (Fig. 6A), ANOVAs showed significant effects of target location on extent error ($F_{1,5}$=21.7, P<0.01), and of trunk motion on directional error ($F_{2,10}$=16.8, P<0.001), but these were very small (less than 1 cm and 1°, respectively). In this group of subjects, there was a tendency to overshoot the ipsilateral target and undershoot the contralateral target.

The mean endpoint trajectories obtained with and without trunk motion were not different for healthy and hemiparetic groups (Fig. 7). However, the variability of trajectories was clearly higher in hemiparetic subjects.

Index of deviation

For the healthy subjects (Fig. 8A), two-factor ANOVAs revealed a significant effect of target location on the index of deviation ($F_{1,5}$=23.5; P<0.01) but not of movement condition. Indeed, the index of deviation was substantially lower for movements to the ipsilateral target, compared with the contralateral target. For the hemiparetic subjects (Fig. 8B), movements in the out-of-phase condition and movements to the contralateral target were generally more curved. Both target location ($F_{1,5}$=9.6; P<0.02) and trunk involvement ($F_{2,12}$=8.0; P<0.01) had a significant effect on deviation. The difference in trajectory straightness for the healthy and hemiparetic subjects was also highly significant ($F_{1,11}$=43.2; P<0.001).

For the healthy subjects, the index of deviation was very close to unity, indicating a fairly linear trajectory (overall mean 1.04±0.02). This seemed to be common for all subjects within this group, as shown by the low standard deviation. For the group of hemiparetic subjects, values were higher and more variable (overall mean 1.22±0.11).

Synchronization index

The most striking differences between individuals and groups was in the sequencing of endpoint and trunk movement. Bar graphs in Figs. 9 (healthy subjects) and 10 (hemiparetic subjects) depict the individual synchronization indices as characterized by the index of synchronization (S).

For the in-phase condition, all healthy subjects initiated the movement with the trunk first (S=–1; Fig. 9A). During out-of-phase motion, two distinct patterns were seen: three subjects (subjects 1, 2, 6) started the movement with their trunk (S=–1), while three others (subjects 3, 4, 5) started with their endpoint (S=1). Furthermore, there was a strong similarity in synchronization patterns for movements to both targets. In other words, different subjects could use different sequences of endpoint and trunk movements, but once a sequence was selected by a subject, the pattern was basically reproduced in other trials whether the subject produced movements to the ipsi- or to the contralateral target (compare solid and dashed bars in Fig. 9). For all subjects within this group, movement finished with the trunk stopping last in all conditions, except for subject 6's movement to the contralateral target (S=–1; Fig. 9B).

The hemiparetic group showed much less stereotyped behavior. Two subjects (H6 and H8) tended to move their endpoint first in most task conditions, two (subjects H1 and H4) started with the trunk in most conditions, while the other four had no specific sequencing pattern. In many cases, the averaged index of synchronization was close to zero, indicating that there were some trials where the motion was initiated by the trunk and others where the endpoint was first. There was a tendency for the group of hemiparetic subjects to finish the movement with the trunk last, especially in the out-of-phase condition, but this tendency was not as pronounced as in the healthy subjects. In addition, the similarity in synchronization for movements to both targets observed in healthy subjects was not seen in the hemiparetic group. To determine the presence of a pattern in the index of synchronization for each hemiparetic subject and within each block of ten trials, a nonparametric procedure termed a one-sample runs test (Zar 1974) was done. A run is defined as a time sequence of like values bounded on either side by unlike or no values. The number of runs is compared with the mean one would obtain if the values were to appear in random order. The result was that none of the subjects showed a pattern in the index of synchronization that significantly differed from a random one.

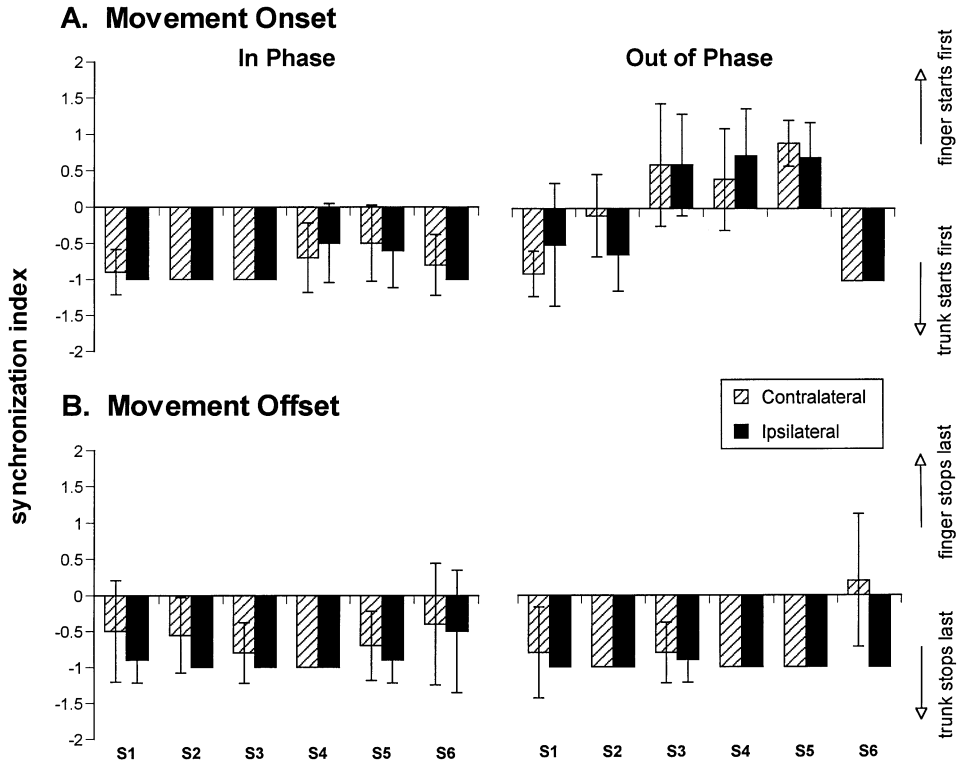

Fig. 9A, B Indices of synchronization (±SD) of endpoint and trunk motion onsets (**A**) and offsets (**B**) for healthy subjects

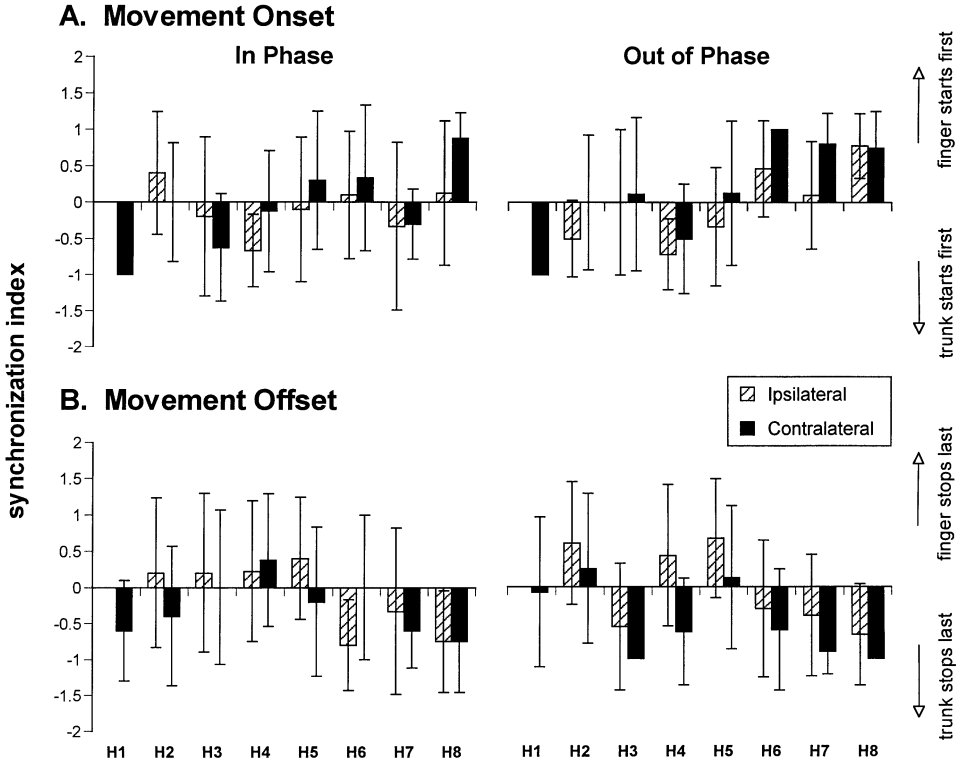

Fig. 10A, B Synchronization indices (±SD) for hemiparetic subjects, ranked according to Fugl-Meyer score. Note a higher variability compared with healthy subjects (Fig. 9), in the patterns of sequencing of the initiation and termination of endpoint and trunk movements

Fatigue

Subjects were given 2–5 min to rest after each block of ten trials. To determine whether fatigue had an effect on task performance, we correlated the trial number with parameters that were not affected by task condition (extent and direction error). For one hemiparetic subject, there was a weak correlation between trial number and both extent (r^2=0.65) and directional error (r^2=0.66), while no correlation was observed for any of the other

seven hemiparetic subjects, nor for any of the healthy subjects. Moreover, trials within each block were consistent for both groups of subjects. Indeed, for all the variables analyzed, none of the repeated-measures ANOVAs revealed an effect of trial number. Subjects were therefore not affected by fatigue.

Correlation with clinical scores

In general, none of the movement indices were significantly correlated with the clinical scores. However, this may be due to the small number of subjects included in the study and the rather large homogeneity in the group's clinical scores, especially for subjects H3–H8 (see Table 1). An interesting trend was the moderate correlation between the index of curvature and the Fugl-Meyer score for the control condition (r^2 of –0.65 for the contralateral and –0.63 for the ipsilateral target).

Discussion

Basic results

This study examined the influence of additional DFs associated with trunk movement on the endpoint trajectory of the hand during a pointing task in healthy and hemiparetic subjects. In healthy subjects, endpoint peak velocity and endpoint error were affected by target location and trunk involvement, while the index of deviation was affected by target location only. Velocities remained monophasic for both targets, regardless of trunk involvement. In the hemiparetic group, endpoint peak velocity was affected by target location only, while precision was unaffected by movement condition. Trunk involvement and target location both had an effect on the index of deviation. Trunk involvement also increased the number of endpoint movement units for the contralateral target. Performance in hemiparetic subjects was generally slower and more variable than in healthy subjects. Compared with the healthy group, the endpoint peak velocity and amplitude of trunk motion were lower and the index of deviation greater, while the endpoint precision was relatively unaffected. Elbow extension was similar in both groups, while hemiparetic subjects used less shoulder flexion.

Even though there were significant task- and target-related differences in endpoint precision for the healthy subjects, these were probably due to differences in the visual appraisal of the target. Indeed, subjects were required to point over the target, without touching the table. Moreover, the healthy subjects were very precise, generally aiming within 1 cm and 1° of the target. These differences were not seen in the hemiparetic group, probably owing to the higher variability in extent and directional error between the subjects.

Healthy subjects displayed a significant effect of trunk involvement on endpoint peak velocity, which may suggest that the subjects performed, in a sense, a different movement when the trunk was involved. However, this does not imply the absence of a compensatory arm-trunk coordination, for the following reasons:

1. Not all healthy subjects changed the endpoint peak velocity with trunk recruitment (see Figs. 4C,D and 5A).
2. If there had been no compensation, the endpoint velocity would increase with in-phase trunk movements, and decrease with out-of-phase trunk movements. This was not the case in this study.
3. The shape of the endpoint trajectory was not affected by trunk recruitment in this study (see also Ma and Feldman 1995).
4. Although pointing errors depended on movement condition, they were substantially lower than those that would have occurred if the trunk influence on the endpoint were not minimized by compensatory movements in the arm joints.
5. Although instructed to move as fast as possible, subjects could vary the movement speed from block to block.

These data imply that movement velocity is controlled by the nervous system independently of the interjoint coordination required by the motor task. In the equilibrium-point hypothesis, for example, movement velocity is controlled by changing the rate of shifts in the equilibrium state of the system (Feldman and Levin 1995). Subjects may simply slow the movement depending, for example, on the instructions or on task difficulty (Fitts 1954). This assumption is consistent with the observation that those healthy subjects who moved more slowly were able to preserve the same peak velocity whether or not the trunk was involved (Fig. 5A). All the hemiparetic subjects, moving slower than the healthy subjects, also preserved the peak velocity regardless of trunk involvement (Fig. 5B). We conclude that velocity is an integral behavioral factor, not a specific outcome of the arm-trunk coordination. Subjects may move faster or slower with the same efficiency in reaching the goal.

In hemiparetic subjects, the shape of the trajectory measured by the index of deviation was significantly affected by trunk motion. This can be explained by taking into account that out-of-phase and contralateral movements required substantial elbow extension, which most hemiparetic subjects may have been unable to provide because of limitations in the active range of motion (Fugl-Meyer et al. 1975) and arm stability in this range (Levin and Dimov 1997; Levin et al. 1997). Indeed, analyses showed that our subjects used less elbow extension for movements to the contralateral target. Thus, requiring a hemiparetic subject to produce more elbow excursion may have caused a destabilizing effect on the endpoint trajectory.

Despite the general similarity in the averaged trajectory shapes across conditions, the timing of the onsets and offsets of movements of the endpoint and trunk was radically different in the two groups of subjects. All

healthy subjects showed a preferred sequence of arm and trunk recruitment and derecruitment. Although different healthy subjects could use different sequences in out-of-phase conditions (trunk leading in three subjects and endpoint leading in the others), they reproduced the same sequence in repeated trials to the same or different targets (Fig. 9).

In contrast, hemiparetic subjects had difficulty in stabilizing the pattern of endpoint and trunk recruitment and derecruitment in all conditions, not only in movements to different targets but even in repeated movements to the same target, as evidenced by the comparison of indices of synchronization for healthy and hemiparetic subjects (Figs. 9, 10). Furthermore, the one-sample runs test showed the absence of a consistent pattern in the indices of synchronization within each block of ten trials. The quantification in terms of the index of synchronization has revealed problems in motor performance even in those hemiparetic subjects who, according to clinical scores, are most similar to healthy subjects. The synchronization index is thus a more sensitive indicator of motor pathology than clinical scores. Another indication that the performance of hemiparetic subjects was different from that of healthy subjects is the finding of a large variability in the endpoint trajectories. These deficits in arm-trunk synchronization during active trunk recruitment cannot be attributed to problems of postural stability, even if this was not assessed directly in our group of hemiparetic subjects. Indeed, it can be argued that all our subjects had no clinically apparent trunk stability problems and that even mildly affected subjects had difficulty in stabilizing the coordination pattern. Furthermore, in most hemiparetic and in all healthy subjects, trunk movement trajectories were smooth and characterized by a bell-shaped velocity profile, indicating good trunk control. In contrast, the endpoint trajectories were segmented, with multiple velocity peaks (Fig. 3B). This finding, not observed in healthy subjects, implies that the arm-trunk coordination was substantially disrupted, forcing the subjects to diminish movement speed and correct deflections of the endpoint trajectory from the desired one in several phases of movement ("step-by-step" strategy), leading to an increase in movement duration. Hemiparetic subjects could also make trunk rotations to compensate for the arm deficits in the production of pointing movements (Fig. 2). Based on the observation that the number of movement units increased with trunk involvement, one may assume that the hemiparetic subjects produced discrete corrections of the endpoint deflections elicited by the trunk movement, rather than preventing them by continuous compensatory arm movements. In other words, being unable to use a compensatory synergy, the hemiparetic subjects nevertheless conserve the ability to correct accumulating movement errors by using discrete arm movements.

Synchronization of motion in healthy subjects

Several conclusions may be drawn from the observation in this group of healthy subjects that trunk motion does not affect the endpoint trajectory, and that the sequence of arm and trunk movements is selected individually by each subject. The fact that the trunk movement is compensated for indicates the importance of the endpoint trajectory in movement planning (Gordon et al 1994; Haggard et al 1995; Saling et al. 1996). Our data support the hypothesis that the nervous system makes use of the redundancy in the number of DFs and modifies the elbow and shoulder joint angles to preserve the endpoint trajectory despite the addition of trunk motion (Ma and Feldman 1995; Saling et al. 1996; Stelmach and Wang 1997). Additional support for the existence of compensatory arm-trunk coordination comes from the fact that the trunk starts to move before the onset and continues to move after the offset of the arm endpoint motion. Taken together, these observations suggest that movement in healthy subjects results from the superposition of two synergies: a reaching synergy using the elbow and shoulder joints to shift the arm endpoint to the target, and a compensatory synergy continuously coordinating trunk and arm joints to prevent undesired shifts of the arm endpoint (Ma and Feldman 1995). In the absence of this form of compensation, the trunk movement would directly affect endpoint position, resulting in movement error. Postural stabilisation during pointing movements or indeed during tasks involving standing or bending of the trunk may involve additional synergies preventing displacement of the center of body mass (Gurfinkel et al. 1971; Bouisset and Zattara 1987). One may suggest that these synergies might be combined with synergies subserving other functional tasks. Superposition of simple synergies to produce more complex motion has also been suggested as a possible mechanism for trajectory modifications made in movement to double-step targets (Flash and Henis 1991; Flanagan et al. 1993).

One important distinction between healthy and hemiparetic subjects in the present study is that the former, after having selected a sequence of trunk and endpoint movements, preserved it in subsequent trials to either the same or different targets. Stereotyped sequencing of movement synergies favors improvement in performance from trial to trial in terms of velocity profile, as well as smoothness and precision of trajectories in all movement conditions. Indeed, studies have shown that with practice a motor task becomes faster, less variable, and more stereotyped (Dugas and Marteniuk 1989; Schneider et al. 1989; Corcos et al. 1993).

Synchronization of motion in hemiparetic subjects

Despite small differences for movement to the contralateral target, endpoint trajectories for the hemiparetic subjects were similar whether or not the trunk was involved. In fact, in this group of subjects, endpoint precision and

mean trajectories were fairly close to those of healthy subjects. On the other hand, parameters such as movement time, variability of trajectories, and especially sequencing of the trunk and endpoint movements were significantly different from those seen in healthy subjects. This would indicate that even though hemiparetic subjects do compensate for the effects of trunk motion they do not always use a strategy involving the superposition of simple synergies. Specifically, the great variability in the sequencing of arm and trunk recruitment and derecruitment shows that the hemiparetic subjects vary their interjoint coordination from trial to trial. In a study of reaching and grasping in hemiparetic subjects, Roby-Brami et al. (1997) observed different patterns of arm and trunk movement and greater variability in segment involvement than in healthy controls. These authors suggested that the use of different intersegmental strategies to achieve the functional goal might be an adaptive behavior to compensate for lost motor function. Based on our results, this behavior may be achieved by discrete corrections of the accumulating movement error, rather than by preventing this error using continuous arm movements. The latter would occur if the compensatory synergy were intact.

Our results concur with those of Roby-Brami et al. (1997), suggesting that the increase in variability and the lack of stereotypic behavior in stroke subjects, even in those with mild sensorimotor symptoms, may be caused by a deficit in short-term motor learning. In other words, they are unable to stabilize movement patterns during a relatively long period of movement repetition (i.e., at least 80 trials). Indeed, no change was seen for endpoint error within the whole experiment, nor in any of the other variables observed within each block of ten trials. In effect then, each trial is like a new movement. Again, this variability in the temporal or spatial aspects of movement production is possible due to the presence of redundancy in the number of DFs. The "cost" of such performance is a decrease in both velocity and trajectory smoothness, the former having already been suggested by Bouisset and Zattara (1990) for parkinsonian patients.

Pointing trajectories in hemiparetic subjects, while relatively accurate in the presence of visual feedback, were more variable and took longer to perform, when compared with those of healthy subjects (Trombly 1992). Movements have also been characterized by multiple acceleration and deceleration phases, or segmentation (Levin 1996). Trombly (1992) associated the slowness and segmentation of pointing movements in stroke patients with peripheral factors, while Levin (1996) attributed these more specifically to deficits in interjoint coordination of arm segments. Among other deficits observed in hemiparetic patients relevant to stabilizing endpoint trajectories during reaching movements are muscle weakness (Colebatch et al. 1986), increased agonist/antagonist cocontraction with prolonged agonist activity during movement (Hammond et al. 1988; Gowland et al. 1992), and difficulty in stabilizing the elbow position in different parts of the angular range (Levin and Dimov 1997). Although factors such as muscle weakness and altered reflex properties may conceivably play a role in disrupting trajectory curvature, the variability in arm-trunk synchronization revealed in our study implies deficits at the level of central planning of movement: hemiparetic subjects cannot plan the compensation of trunk movement beforehand and are forced to produce it in a segmented, discrete way in the course of the task.

Since the localization of lesions in our patients varied considerably, deficits in trajectory curvature and movement synchronization cannot be associated with dysfunction in a specific brain area. On the other hand, all patients had lesions localized in the left hemisphere, which is thought to play a greater role than the right in sensorimotor integration and selection of an appropriate motor program (Haaland and Harrington 1989). Indeed, it has been shown that subjects with left-sided stroke have more impairments than subjects with right-sided lesions in movement reversals during rapid cyclic tasks involving finger tapping (Giuliani et al. 1997). This could be related to the variability in movement synchronization observed in our study. To test this possibility, pointing movements of the unaffected and affected arm of subjects with left-sided stroke could be compared. One can predict that the synchronization deficits observed in the affected arm should also be seen in the unaffected arm if these deficits are associated with damage to the left hemisphere.

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