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Compensatory strategies for reaching in stroke

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Summary

A major prerequisite for successful rehabilitation therapy after stroke is the understanding of the mechanisms underlying motor deficits common to these patients. Studies have shown that in stroke patients multijoint pointing movements are characterized by decreased movement speed and increased movement variability, by increased movement segmentation and by spatial and temporal incoordination between adjacent arm joints with respect to healthy subjects. We studied how the damaged nervous system recovers or compensates for deficits in reaching, and correlated reaching deficits with the level of functional impairment. Nine right-hemiparetic subjects and nine healthy subjects participated. All subjects were right-hand dominant. Data from the affected arm of hemiparetic subjects were compared with those from the arm in healthy subjects. Seated subjects made 40 pointing movements with the right arm in a single session. Movements were made from an initial target, for which the arm was positioned alongside the trunk. Then the subject lifted the arm and pointed to the final target, located in front of the subject in the contralateral workspace. Kinematic data from the arm and trunk were recorded with a three-dimensional analysis system. Arm movements in stroke subjects were longer, more segmented, more variable and had larger movement errors. Elbow–shoulder coordination was disrupted and the range of active joint motion was decreased significantly compared with healthy subjects. Some aspects of motor performance (duration, segmentation, accuracy and coordination) were significantly correlated with the level of motor impairment. Despite the fact that stroke subjects encountered all these deficits, even subjects with the most severe motor impairment were able to transport the end-point to the target. All but one subject involved the trunk to accomplish this motor task. In others words, they recruited new degrees of freedom typically not used by healthy subjects. The use of compensatory strategies may be related to the degree of motor impairment: severely to moderately impaired subjects recruited new degrees of freedom to compensate for motor deficits while mildly impaired subjects tended to employ healthy movement patterns. We discuss the possibility that there is a critical level of recovery at which patients switch from a strategy employing new degrees of freedom to one in which motor recovery is produced by improving the management of degrees of freedom characteristic of healthy performance. Our data also suggest that stroke subjects may be able to exploit effectively the redundancy of the motor system.

Keywords: reaching; interjoint coordination; stroke; compensation; redundancy

Abbreviations: DF = degrees of freedom; IRED = infrared light-emitting diode

Introduction

Understanding movement deficits following CNS lesions and the relationships between these deficits and functional ability is fundamental to the development of successful rehabilitation therapies (Lough *et al.*, 1984). Following a stroke, impairment of upper limb function is one of the most common and challenging sequelae, and it limits the patient's autonomy in activities of daily living and may lead to permanent disability (Nakayama *et al.*, 1994). Movement deficits are most evident in the limb contralateral to the side of the stroke and are characterized by weakness of specific muscles (Bourbonnais and Vanden Noven, 1989); abnormal muscle tone (Lance, 1980; Burke, 1988; Wiesendanger, 1990); abnormal postural adjustments (Di Fabio *et al.*, 1986); abnormal movement synergies (Twitchell, 1951; Brunnström, 1970; Bobath, 1990); lack of mobility between structures at the shoulder girdle (Cailliet, 1980; Ryerson and Levit, 1987) and the pelvic girdle (Carr and Shepherd, 1987*a*); incorrect timing of components within a movement pattern (Carr and Shepherd, 1987*a*; Archambault *et al.*, 1999) and loss of interjoint coordination (Levin, 1996*a*). When a stroke patient attempts to move and encounters all these deficits, the natural reaction is to compensate with the available motor strategies. The

occurrence of a pathological synergy (Twitchell, 1951) has been considered a compensatory strategy developed when attempting to move (Carr and Shepherd, 1987*a*, *b*; Bobath, 1990). Hemiplegic limb synergies consist of either a gross extensor movement, called the extensor synergy (shoulder extension and adduction combined with elbow extension, forearm pronation and wrist flexion) or a gross flexor movement, called the flexor synergy [shoulder flexion and abduction combined with elbow flexion, forearm supination and wrist extension (Brunnström, 1970)].

The view that the appearance of gross flexor and extensor synergies precedes the restoration of more advanced motor function following stroke in man is controversial. Some consider that during the early recovery stages the stroke patient should be aided or encouraged to gain control of the basic limb synergies (Twitchell, 1951; Brunnström, 1970). Others favour the opposite opinion, that attempts must be made early to develop normal motor responses (Carr and Shepherd, 1987*a*, *b*; Bobath, 1990). Another example of a compensatory strategy used by stroke patients is the fixation of specific body segments. This strategy may decrease the number of motor elements (degrees of freedom, DFs) the CNS must control to accomplish the motor task (Bernstein, 1967; Vereijken *et al.*, 1992). Fixation patterns (the pelvis on the lumbar spine or the scapula on the thorax) may be a natural response to the inability to maintain balance in posturally threatening situations. A negative consequence may be the lack of limb girdle mobility, which may limit the normal kinematics of upper and lower limb movement. Some therapists (Davies 1985, 1990; Carr and Shepherd, 1987*a*, *b*) believe, however, that the continual practice of fundamentally inappropriate compensatory strategies may be a critical factor limiting recovery following brain damage.

Important in the understanding of voluntary movement production is the concept of redundancy. The musculoskeletal system is considered redundant, since it has potentially a larger number of ways to combine individual joint movements (DFs) than is necessary to achieve the motor task. Alternatively, because of the negative connotation of the word 'redundancy', Latash (1998) has suggested that it be replaced by the word 'abundancy'. In this connotation, the 'abundancy' in the system allows movement to be performed in a variety of ways, permitting the organism to adapt to different environmental conditions. Whichever term is used, the system takes advantage of this feature of the motor apparatus by selecting a desired trajectory and an interjoint coordination among many possible strategies to make goal-directed movements (Kugler *et al.*, 1980; Berkinblit *et al.*, 1986; Mussa-Ivaldi *et al.*, 1988; Kelso *et al.*, 1993; Ma and Feldman, 1995). In healthy subjects, the acquisition of new motor skills may be viewed as a 'process of mastering degrees of freedom' (Bernstein, 1967, p. 127). According to Bernstein, the acquisition of new motor skills would be associated with a gradual decrease in the number of DFs employed and their incorporation into a dynamic, controllable system, represented by 'optimal synergies' (Bernstein, 1967) or 'co-ordinative structures' (Turvey *et al.*, 1978). The idea of 'co-ordinative structures' implies that the nervous system uses available DFs to accomplish the motor task without an explicit classification of DFs into functional units. Each co-ordinative structure is thus designed to accomplish a specific task, and a change in the task results in the reorganization of the co-ordinative structure. From this point of view, synergies are not specifically created by the nervous system but may emerge in a natural, dynamic way from the task-specific co-ordinative strategy. This may be a general strategy used by the healthy CNS to acquire complex motor skills.

In stroke subjects, goal-directed movements are characterized by slowness, spatial and temporal discontinuity and abnormal patterns of muscle activation (Gowland *et al.*, 1992; Trombly, 1992; Levin, 1996*a*). Pointing movements of the arm to different targets on a horizontal planar surface by stroke patients using their affected and non-affected arms were compared in age- and sex-matched healthy subjects by Levin (1996*a*). The motor behaviour of the affected arm was characterized by lower movement amplitudes and prolonged movement times, whereas movement trajectories were more dispersed and segmented spatially. In these movements, the interjoint coordination (between elbow and shoulder joints) of movements made into or out of the typical extensor or flexor synergies was disrupted. Despite these deficits, even subjects with the most severe motor deficits could reach into all parts of the workspace with their affected and non-affected arms. This finding supports those of Fisk and Goodale and of Trombly indicating that the ability to plan movement may be preserved in stroke (Fisk and Goodale, 1988; Trombly, 1992).

Few studies have examined the kinematics of three-dimensional movements made by the affected arm in stroke subjects. Roby-Brami and colleagues showed that prehension movements are characterized by spatiotemporal incoordination between the arm and the trunk (Roby-Brami *et al.*, 1997). Their study indicated that stroke subjects use a new pattern of coordination represented by more trunk recruitment during prehension movements. However, interjoint coordination of the arm was not evaluated in this study.

The goals of the present study were: (i) to characterize motor deficits during three-dimensional reaching movements of the arm in stroke subjects in terms of kinematics and multijoint coordination; (ii) to identify if compensatory strategies are used by patients to achieve the functional goal and, if so, what these strategies are; and (iii) to analyse the correlation between the arm and trunk kinematics and the level of functional impairment measured clinically. Preliminary data have appeared in abstract form (Cirstea *et al.*, 1998).

Methods

Subjects

Nine right-hand dominant healthy subjects and nine right hemiparetic subjects participated in this study. They were

Table 1 *Demographic data and clinical scores for hemiparetic subjects*

Subject	Age (years)/sex	Months after onset	Site/type of stroke	Fugl-Meyer score (66)	Spasticity score	Sensory status	
						Left	Right
1	63/M	10	Frontotemporoparietal subcortical/ischaemic	15	3	(hd) 3.61* (sh) 4.17*	3.22 4.08*
2	40/M	17	Frontoparietal/ haemorrhagic	17	2	(hd) 3.22 (sh) 4.08*	6.10* 4.56*
3	72/M	8	Middle cerebral artery/ ischaemic	31	2	(hd) 4.17* (sh) 4.17*	3.61 3.84
4	57/M	7	Frontotemporoparietal/ ischaemic	35	2	(hd) 2.83† (sh) 2.83†	2.44 3.61*
5	47/F	5	Parietal and subcortical/ ischaemic	41	1+	(hd) 3.22 (sh) 4.08*	2.83† 3.22
6	29/F	11	Parietal and internal capsule/haemorrhagic	52	2	(hd) 2.83† (sh) 3.84*	6.65* 6.65*
7	59/M	4	Thalamic/ haemorrhagic	54	1	(hd) 3.60* (sh) 3.61	4.74* 6.65*
8	74/F	15	Middle cerebral artery/ ischaemic	60	0	(hd) 3.61* (sh) NT	4.17* NT
9	53/M	2	Temporal/ischaemic	62	0	(hd) 4.74* (sh) 4.74*	4.17* 4.17*

M = male, F = female; (sh) = shoulder; (hd) = hand; NT = not tested. *Higher than normal threshold; †lower than normal threshold.

informed of the experimental procedures and gave their written consent, in accordance with the polices of local hospital ethics committees. The stroke group (the experimental group) included three females and six males with a mean (± standard deviation) age of 54 ± 14 years. They had sustained a single left stroke between 3 and 24 months previously (for locations of lesions, see Table 1), leading to right-sided paresis including the arm. All subjects were able to understand simple commands (i.e. there was no receptive aphasia) and to perform a reaching movement with the affected arm [at least stage-3 control of the upper limb according to the Chedoke–McMaster Stroke Assessment Scale (Gowland *et al.*, 1993)]. They had no other neurological, neuromuscular or orthopaedic disorders and no visual attention deficits, as assessed by Bell's test (Gauthier *et al.*, 1989). Explicit exclusion criteria were perceptual, apraxic or major cognitive deficits, shoulder subluxation or pain in the upper limb. In addition, subjects were excluded if they had occipital, cerebellar or brainstem lesions and if there was any indication of possible bilateral brain damage or brain damage of non-vascular aetiology. The healthy subjects (control group) included five females and four males having a mean age of 43 ± 18 years. Exclusion criteria for healthy subjects were a history of a neurological disorder or a physical deficit involving the upper limbs or trunk.

Clinical assessment

Prior to the experiment, the stroke subjects were tested clinically by an experienced physiotherapist to assess physical factors which may have influenced motor performance. Motor function of the upper limb was evaluated with the Fugl-Meyer scale (Fugl-Meyer *et al.*, 1975). This assessment includes an evaluation of muscle tone, range of motion, tendon reflexes and the performance of proximal and distal voluntary movements of the affected arm. A maximum score of 66 corresponds to normal arm function. Two of our subjects (subjects 1 and 2) scored between 15 and 17, indicating a severe motor deficit (gross motor function only), three (subjects 3–5) scored between 31 and 41, indicating a moderate motor deficit (gross and some fine motor function), while the others (subjects 6–9) scored between 52 and 62, indicating a mild motor deficit (Table 1).

Clinical spasticity in the elbow flexors was measured with the modified Ashworth scale (Bohannon and Smith, 1987), assessing the resistance to full-range passive elbow extension. Spasticity scores ranged from 0 to 4, where 0 signifies normal tone and 4 indicates severe spasticity with an almost rigid limb. In our patients, spasticity scores ranged from 0 (no spasticity) to 3 (moderate spasticity) (Table 1).

Sensory discriminative status was measured using Semmes–Weinstein Filaments (Semmes and Weinstein, 1960) to determine the threshold of tactile sensation at two locations on the affected arm, proximally over the anterior deltoid muscle and distally on the palm of the hand. Tactile thresholds higher than 3.61 for the shoulder and 3.22 for the hand area were considered abnormal.

Experimental procedure

All subjects performed the reaching task with their right arm (Fig. 1). Seated subjects were asked to make natural, self-paced pointing movements from an initial target located ipsilaterally on a platform 41.5 cm high and 10 cm lateral to the right hip, to a final target located in the contralateral workspace in front of the subject (~10 cm lateral and 10 cm

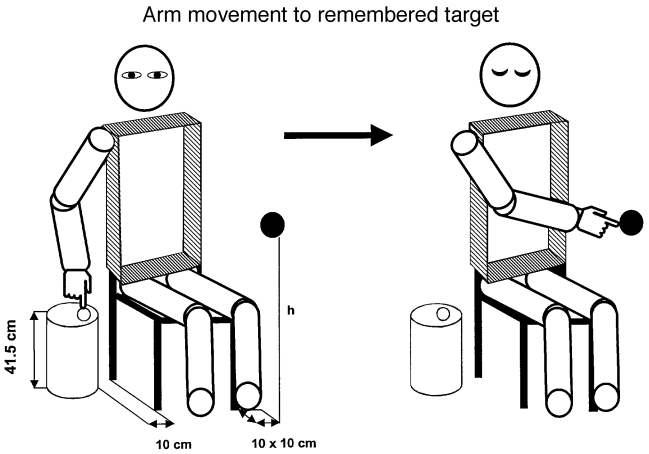

Fig. 1 Schematic diagram of the experimental set-up for reaching movements made in three-dimensional space. The circle located on the cylinder is the initial target and the suspended circle is the final target. Movements were made without vision. h = height of final target.

anterior to the left foot at a height of ~80 cm). The height of the target was modified in accordance with the height (±3 cm) of the sitting subject. The target was placed just beyond the reach of the arm so that there was no contact of the finger with the target.

This task was chosen because it involved the coordination of multiple joints and represented a difficult but functional movement. In addition, it represented a movement that ought to be reacquired during recovery from stroke. Prior to recording, subjects practised the movement five times with vision. The movement was then repeated 40 times in a single experimental session. Movements were made without vision to minimize visually guided corrections and to maximize the use of propioceptive feedback. By excluding vision, we tested the ability of subjects to rely predominantly on propioceptive information to make coordinated movements to remembered targets (Adamovich *et al.*, 1998). Subjects had visual feedback of the initial position in all trials. After visualizing the hand in the initial position and the location of the final target, subjects were instructed to close their eyes and move their hand to the final target. Subjects extended their arm to the remembered target as fast as possible in one movement without corrections and maintained the final position until the end of the trial. To avoid fatigue, a rest period (10 s) was allowed between trials. However, a longer rest period of up to 5 min was allowed at the request of the subject. At the end of every fifth trial, subjects in both groups opened their eyes to see the final hand position relative to the target and corrected the error. This procedure minimized the accumulation of error and made movements more consistent over time. An interval of five trials was chosen since feedback given more frequently or more sparsely has been shown to have deleterious effects on movement accuracy (Bilodeau and Bilodeau, 1958; Winstein and Schmidt, 1990).

Data acquisition and analysis

Kinematic data from the arm and trunk were recorded with an Optotrak motion analysis system (Northern Digital, Waterloo, Canada). Infrared light-emitting diodes (IREDs) were positioned on the tip of the index finger, the wrist (head of the ulna), the elbow (lateral epicondyle), the shoulders (ipsilateral and contralateral acromions) and the trunk (top of sternum). Arm and trunk movements were recorded for 2–4 s at a sampling rate of 200 Hz and digitally low-pass filtered at 20 Hz. The kinematic data were analysed in terms of movement times, trajectories, movement errors and interjoint coordination. End-point and trunk tangential velocities were computed from the magnitude of the velocity vector, by numerical differentiation of the x, y and z positional data obtained from the end-point and sternal markers, respectively, and were used to compute movement times. End-point and trunk movement onsets and offsets were defined as the times at which the tangential velocity exceeded or fell below 10% of the peak velocity, respectively. The shape of the end-point trajectory was characterized by the length index—the ratio of the length of the actual path travelled by the end-point in three-dimensional space to the length of the straight line joining the initial and final end-point positions. Thus, this index was 1 for an ideal straight line and $\pi/2 = 1.57$ for a semicircle. This index (rather than the more commonly used maximal perpendicular distance between the ideal straight line and the actual trajectory) was measured because, in some cases, trajectories could be S-shaped instead of arced, thus intersecting with the ideal straight line. Movement accuracy in terms of constant error was computed as the square root of the mean distance (δ) between the final end-point position and the position of the target. To estimate the movement consistency we compared the coefficient of variation (standard deviation/mean) of the end-point velocity, the index of trajectory length and the end-point errors in the two groups of subjects.

The ranges of angular motions were calculated for one DF of the elbow (flexion/extension), two DFs of the shoulder (flexion/extension and horizontal adduction/abduction) and two DFs of the trunk (axial rotation and flexion). For the trunk, axial rotation was defined as the angle of rotation of the vector joining the two shoulder IREDs with respect to a sagittal line projecting on a horizontal plane. The flexion (anterior displacement) of the trunk was measured as the displacement in millimetres of the IRED located on the sternum in the sagittal plane.

To analyse interjoint coordination, joint angles were first computed from the position data as the angles between the corresponding vectors joining adjacent IREDs. Interjoint coordination was estimated qualitatively by constructing angle/angle diagrams for adjacent joint pairs and quantitatively by phase analysis. Phase diagrams (angular velocity versus angle) for elbow flexion/extension and shoulder horizontal adduction/abduction were calculated. From these diagrams, we obtained the phase angle (Li *et al.*,

1999) for each joint at 50-ms intervals throughout the movement, and we calculated the difference over time between phase angles of the shoulder and elbow (phase difference). This analysis produced a staircase-like graph showing that the interjoint coordination varied in terms of the phase difference of the two DFs throughout the movement. We compared the number of stairs or segments in the graphs of the phase difference in the two groups of subjects.

Statistical analysis

Analysis focused on three outcome measures: movement time, end-point errors and trajectories and five other kinematic variables: ranges of angular motion in the elbow (one DF) and shoulder (two DFs) joints, trunk displacement and rotation. Multiple regression (Statistica 4.5, Statsoft, Tulsa, Okla., USA, 1993) was used to analyse the relationships between outcome measures and variables. Spearman rank order correlation (Statistica 4.5) was used to analyse the correlation between the clinical scores and the outcome measures and movement variables in stroke subjects. Depending on the results of tests of homogeneity of variance, we used parametric (Student's t test) or non-parametric statistics (Mann–Whitney U test) to study the differences between groups. The significance level for all statistical comparisons was set at $P < 0.05$.

Results

Most healthy subjects made the pointing movement by initially flexing their elbow and then raising their arm (shoulder flexion), pulling it across the body (shoulder horizontal adduction) and extending the elbow to move the hand towards the final target. These subjects used a minimal amount of trunk movement (mean 37.5 ± 14.1 mm) to accomplish this task. In contrast, stroke subjects used different patterns to reach the target, which depended on their level of arm motor impairment. While most subjects were able to flex the elbow initially and then flex the shoulder, many, instead of horizontally adducting the shoulder and extending the elbow, moved the trunk to bring the hand to the target (110.2 ± 59.7 mm).

Movement trajectories

Examples of mean trajectories in healthy and stroke subjects are shown in Fig. 2. In healthy subjects, the end-point paths were smooth and continuous. In contrast, stroke subject 1, who was severely impaired (Table 1), produced an end-point trajectory characterized by a lack of continuity such that the hand changed direction and reached the target by a series of small sequential movements. Also, the variability of end-point trajectories in this stroke subject was elevated (6.8%) compared with healthy subjects (2.7%). In a subject with moderate motor deficits (subject 4) the end-point trajectories were less segmented and less variable (3.1%) compared with those of subject 1. In contrast, the mildly impaired stroke subject (subject 9) made relatively smooth end-point trajectories with low variability (2.3%). Some differences in movement production in stroke and healthy subjects might have been associated with the differences in movement speed. To test this possibility, we re-evaluated a subset of healthy subjects by instructing them to move at speeds similar to our stroke subjects. At these slower movement speeds (1319.7 ± 284.5 mm/s), the end-point trajectory was similar, in terms of smoothness and variability, to that of healthy subjects making fast movements (Fig. 2). Thus, decreased movement velocity appears not to explain the increase in movement variability and spatial segmentation observed in stroke subjects.

Overall, trajectory length was greater in stroke patients (1.35 ± 0.08) than in healthy subjects (1.27 ± 0.04, t test, $P < 0.02$) and trajectory variability was higher both in terms of standard deviations and coefficients of variability (5.3%) of the index of the trajectory length (Fig. 3).

In healthy subjects, the end-point and trunk tangential velocity profiles were smooth and bell-shaped. The average peak velocity (2799.1 ± 190.4 mm/s) and the time to peak velocity (0.23 ± 0.03 s) were consistent over trials (Table 2 and Fig. 4A, left panel). Reaches were usually performed by only one movement unit, as evidenced by a single peak in the tangential velocity trace. For the group of stroke subjects, the mean peak velocity was lower (1650.3 ± 519.5 mm/s, U test, $P < 0.05$) and the time to peak velocity was longer (0.42 ± 0.17 s, U test, $P < 0.05$). In the most severely impaired subject (subject 1), the end-point peak velocity was lower (1503.4 ± 183.5 mm/s) than in healthy subjects and the velocity profile was multiphasic (more than four peaks) instead of uniphasic. The presence of multiple peaks in the tangential velocity traces in this subject indicates that movements were produced by repetitive accelerations and decelerations. Movements in the moderately impaired subject (subject 4; Fig. 4B, middle panel) were smoother (two to four peaks) than in the severely impaired subject 1. In contrast, the profile of the end-point tangential velocity in the least impaired subject (subject 9; Fig. 4B, right panel) was bell-shaped (one peak) and higher (2013.3 ± 299.5 mm/s) than in the more severely impaired subjects. In general, over the 40 trials, the number of peaks tended to decrease in all stroke subjects. For the group, the number of peaks, which ranged up to 13 in the first three trials, decreased on average by 47.6 ± 13.0% in the last three of the 40 trials (range 1–8).

To test the possibility that some differences in movement production in stroke subjects may be associated with the differences in movement speed, we analysed the end-point velocity profile in a subset of healthy subjects asked to move at speeds similar to our stroke subjects. At these slower movement speeds (1319.7 ± 284.5 mm/s), the tangential velocity profiles were more segmented (1.3 ± 1.4 peaks) and more variable than those in healthy subjects making faster movements (Fig. 4). Velocity profiles of moderately

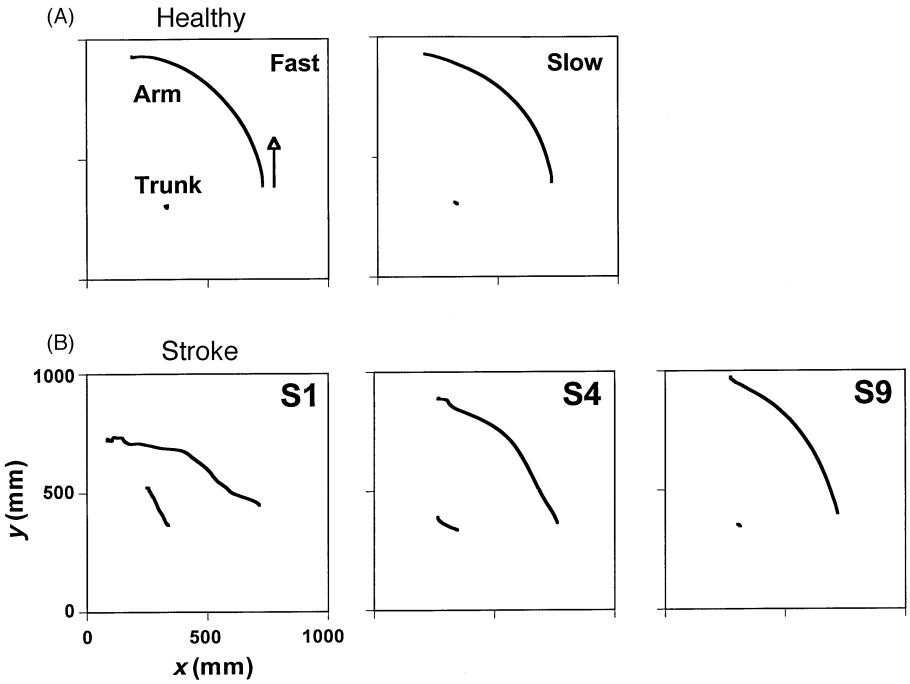

Fig. 2 Averages of end-point (arm) and trunk trajectories (**A**) in one healthy subject making fast and slow movements and (**B**) in three stroke subjects (S1, S4, S9).

impaired subjects were similar in terms of peak velocity but were still more segmented than those of healthy subjects moving slowly. Thus, decreased movement speed may account for some but not all the increase in temporal segmentation observed in stroke subjects.

Movement time was significantly longer in stroke subjects (1.27 ± 0.29 s), usually by a factor of two compared with healthy subjects (0.62 ± 0.05 s, *U* test, $P < 0.05$). Severely impaired subjects had the longest movement times (1.45 ± 0.14 s, subjects 1–6), but even subjects with better functional scores (subjects 7–9) still moved significantly more slowly (0.92 ± 0.08 s) than healthy subjects.

Despite slower velocities, movements in stroke subjects were less precise than in healthy subjects. The average end-point positions for the pointing movements in each subject are illustrated in Fig. 5. The intersection of the three thick lines represents the final target position in three-dimensional space. The final end-point positions in stroke subjects were more widely distributed around the target compared with those of healthy subjects. The degree of movement accuracy was significantly correlated with the severity of clinical symptoms such that the more severely impaired stroke subjects, indicated by subject numbers in Fig. 6, made greater movement errors (see below).

Interjoint coordination

In line with previous studies, our results showed that interjoint coordination was disrupted in stroke subjects. We analysed the interjoint coordination between one DF of the elbow (flexion/extension) and two DFs of the shoulder (horizontal abduction/adduction and flexion/extension). The interjoint coordination in the healthy subjects (Fig. 6A) was characterized by smooth and continuous curves. The movement started with elbow flexion (Fig. 6A, top, arrow) followed by combined movements of elbow flexion with shoulder horizontal adduction (Fig. 6A, top) and elbow with shoulder flexion (Fig. 6A, bottom). The shoulder then moved alone (Fig. 6A, horizontal adduction shown in top panel and flexion in bottom panel) followed by an elbow–shoulder coordination, consisting of combined movements of elbow extension with shoulder horizontal adduction (Fig. 6A, top), and elbow extension with shoulder flexion (Fig. 6A, bottom). Movement ended with a large elbow extension to reach the target.

In contrast, the interjoint coordination in stroke subjects was disrupted. The number of segments in the phase difference for the whole group was greater (6.5 ± 2.2 segments) than that in healthy subjects (1.1 ± 0.3, *U* test, $P < 0.05$) and elbow and shoulder excursions were smaller (Table 2). Examples of interjoint coordination in subjects with severe (subject 1), moderate (subject 4) and mild (subject 9) impairments are shown in Fig. 6B. The subjects with severe and moderate motor deficits could not produce smooth coordinations between the elbow and the shoulder movements. Data on phase differences for individual subjects are shown in Table 3. This analysis was sensitive to the clinical severity, mildly affected subjects having a smoother interjoint coordination (fewer phase segments) than more severely affected patients. For example, subject 1 had the

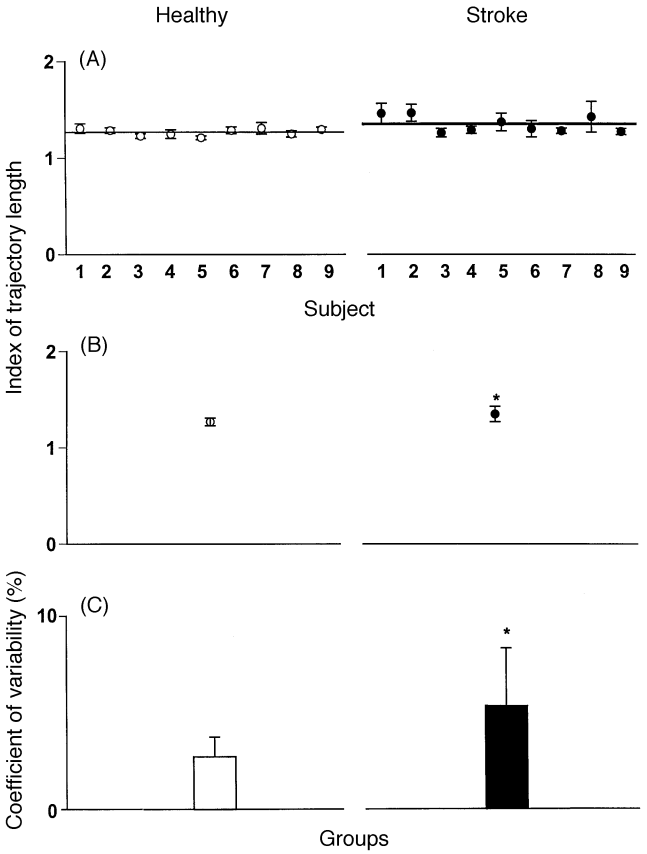

Fig. 3 Average and standard deviation of the index of trajectory length in (**A**) healthy subjects (open symbols) and stroke subjects (filled symbols); the two horizontal lines represent the average index for all healthy (thin line) and stroke (thick line) subjects. (**B**) Average and standard deviation of the index of trajectory length for each group of subjects. (**C**) Coefficient of variation of the index of trajectory length in healthy and stroke groups. Asterisks indicate significant differences between the groups of subjects ($P < 0.05$).

most difficulty in making this movement and was unable to coordinate elbow and shoulder movements: a small elbow flexion was followed by a small shoulder abduction, followed by a small elbow flexion–shoulder adduction coordination, followed by shoulder adduction movement alone, and so on. An important feature of movement in stroke subjects was the lack of elbow extension at the end of movement. Subject 1 also had the greatest amount of temporal and spatial segmentation of movement. Subjects with moderate motor deficits had interjoint coordination patterns at the beginning of the movement typical of healthy subjects (subject 4). However, the middle and later parts of the movement were marked by large decreases in the range of arm-joint excursions compared with healthy subjects (U test, $P < 0.01$) and the end of movement was characterized by a lack of elbow extension (Fig. 6B, subjects 1 and 4). On the other hand, subjects with mild motor deficits had interjoint coordination patterns and ranges of joint excursions similar to those of healthy subjects (Fig. 6B, subject 9). In general, for the group, the ranges of active motion for the elbow and for the two DFs of the shoulder were decreased in stroke compared with healthy subjects (Table 2). This decrease was most marked for subjects with severe and moderate clinical impairment (subjects 1–6: elbow extension, 4.8 ± 4.7°; shoulder horizontal adduction, 92.6 ± 23.8°; shoulder flexion, 48.7 ± 14.3°), whereas more mildly affected subjects could have a normal range of movement (subjects 7–9: 27.6 ± 13.9° for elbow; 123.3 ± 26.8° and 81.7 ± 7.15° for shoulder horizontal adduction and flexion, respectively).

Trunk involvement

Despite the fact that the stroke subjects generally used a smaller range of motion and an abnormal coupling between the joints, all were still able to move the end-point to the target. The decrease in the active range of elbow and shoulder movements in severely and moderately affected stroke subjects (subjects 1–6) occurred together with increased involvement of the trunk (Fig. 2). The mean values of the trunk displacement were 110.2 ± 59.7 mm in all stroke patients compared with 37.5 ± 14.1 mm in all healthy subjects (U test, $P < 0.001$; Table 2). Differences observed for arm-joint excursions among the severely, moderately and mildly affected subjects were also observed for the involvement of the trunk. Subjects with moderate and severe clinical impairment (subjects 1–6) used more trunk recruitment (139.5 ± 47.9 mm) than those with mild impairment (subjects 7–9, 51.7 ± 28.52 mm). The increased involvement of the trunk was significantly correlated with the decrease in elbow extension ($r = 0.68$) and the decrease in shoulder flexion ($r = 0.89$), whereas as a group the correlation with shoulder horizontal adduction was not significant ($r = 0.51$). Trunk rotation was significantly increased (24.3 ± 8.5°) for the whole group of stroke subjects compared with healthy subjects (12.7 ± 4.7°, U test, $P < 0.05$; Table 2).

Clinical correlation

Clinical motor function (Fugl-Meyer scale) and spasticity scores were correlated with outcome measures and movement variables. The level of motor function was significantly correlated with three kinematic measures: elbow extension ($r = 0.81$), shoulder flexion ($r = 0.89$) and the displacement of the trunk ($r = –0.86$). In addition to these three measurements, significant correlations were also found between the degree of clinical spasticity and movement time ($r = 0.70$) and trunk displacement ($r = 0.88$). This suggests that the deficits in the joint excursions, that occurred together with the recruitment of the trunk, may be directly related to the degree of motor impairment. There were no correlations between the threshold of tactile sensation at the hand or shoulder and any of the measures of kinematic outcomes or variables.

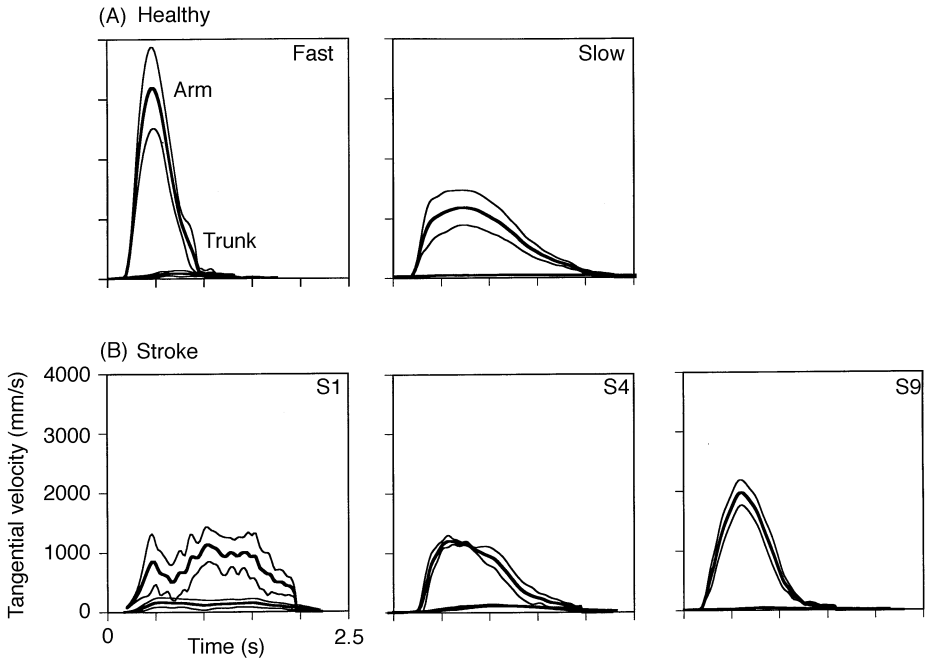

Fig. 4 Average (thick lines) and standard deviation (thin lines) of tangential velocities of the arm and trunk in one healthy subject (**A**) and three stroke subjects with different degrees of impairment (**B**). These data show differences in peak velocity, total movement time and temporal segmentation in the different subjects.

Table 2 *Mean (± standard deviation) values of all outcome measures and variables in healthy and stroke subjects*

Parameter	Healthy	Stroke
Movement time (s)	0.620 ± 0.05	1.270 ± 0.29*
Constant errors (mm)	64.2 ± 14.5	113.5 ± 50.9*
Coefficient of variation (%)	31.7	34.6
Trajectory length	1.27 ± 0.04	1.35 ± 0.08*
Coefficient of variation (%)	2.7	5.3*
Peak velocity (mm/s)	2799.1 ± 190.4	1650.3 ± 519.5*
Coefficient of variation (%)	11.5	13.9*
Temporal segmentation (no. of peaks)	1.2 ± 0.3	3.3 ± 0.9*
Elbow extension (°)	34.4 ± 13.3	12.4 ± 13.8*
Shoulder horizontal adduction (°)	128.3 ± 8.0	105.2 ± 27.7*
Shoulder flexion (°)	86.5 ± 8.1	60.3 ± 20.3*
Trunk displacement (mm)	37.5 ± 14.1	110.2 ± 59.7*
Trunk rotation (°)	12.7 ± 4.7	24.3 ± 8.5*

*$P < 0.05$.

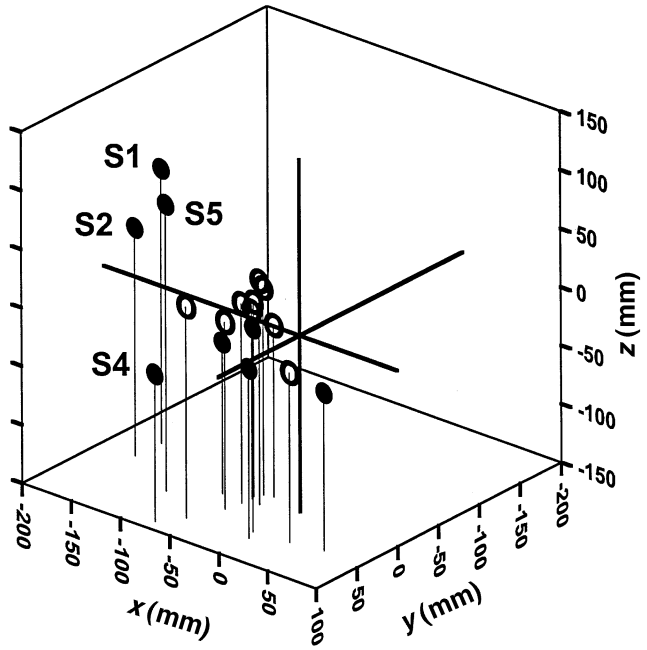

Fig. 5 Average final end-point positions for movements made in healthy subjects (open circles) and stroke patients (filled circles) shown in a three-dimensional system of coordinates. Vertical lines show the projections of the points representing the final positions of the fingertip on the horizontal plane (x, y). The intersection of the three thick lines is the position of the final target.

Relationship between multiple kinematic variables

Since significant correlations were found between several kinematic variables, multiple regression analysis was used to describe the relative contributions of each of the five kinematic variables to the three outcome measures for each group. In the healthy subjects, multiple regression analysis revealed that the first outcome, movement error, was related ($R = 0.425$, $P < 0.001$) to two variables: trunk rotation ($\beta = -0.35$) and trunk displacement ($\beta = 0.47$). Correlations were positive between trunk displacement and movement error (ranging from 0.50 to 0.66) in five out of nine healthy

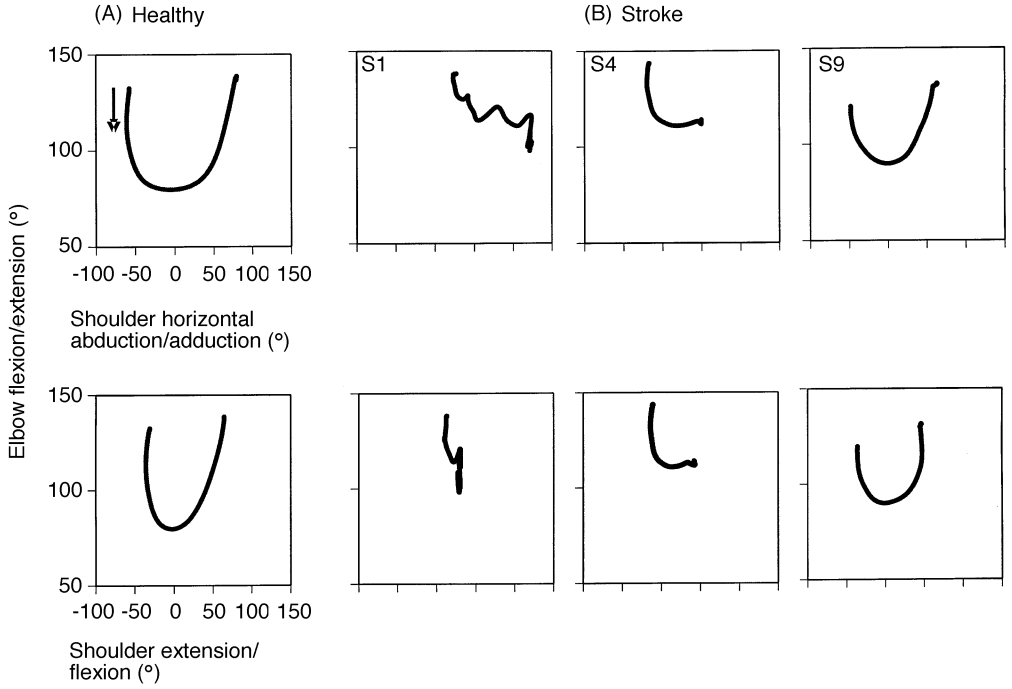

Fig. 6 Interjoint coordination for (**A**) one healthy subject and (**B**) three stroke subjects with severe (S1), moderate (S4) and mild (S9) clinical deficits. Elbow flexion and extension movements are paired with shoulder horizontal abduction and adduction movements in the upper row and with shoulder extension and flexion movements in the lower row. In each graph, elbow flexion is downward, shoulder horizontal adduction is rightward (upper row) and shoulder flexion is rightward (lower row). The arrow in the upper left panel indicates the direction of movement.

Table 3 *Constant and variable errors of end-point at final position (millimetres) in healthy and stroke subjects and number of segments in the phase difference analysis in stroke subjects*

Subject	Healthy (error) (mm)	Stroke (error) (mm)	Stroke (no. segments)
1	57.6 ± 22.9	178.2 ± 80.8	9.1 ± 1.1
2	64.4 ± 8.5	136.5 ± 91.3	9.6 ± 2.9
3	62.4 ± 20.6	73.5 ± 29.7	6.7 ± 1.2
4	62.9 ± 21.8	149.3 ± 11.9	6.3 ± 0.8
5	56.7 ± 15.8	190.3 ± 27.0	5.7 ± 1.2
6	52.2 ± 12.9	91.7 ± 25.9	6.7 ± 1.6
7	65.6 ± 18.5	75.1 ± 38.9	5.1 ± 0.9
8	54.9 ± 31.7	49.8 ± 20.3	5.0 ± 1.1
9	101.0 ± 26.8	77.9 ± 12.8	4.5 ± 0.7
Mean ± SD	64.2 ± 19.9	113.6 ± 37.6	6.5 ± 2.2

The mean number of segments in healthy subjects was 1.1 ± 0.3.

subjects. This suggests that the more the trunk moved the larger was the error (Fig. 7). Movement time was related only to trunk displacement ($\beta = -0.51$, $R = 0.378$, $P < 0.001$). The third outcome, the length index of the trajectory, was related to four variables ($R = 0.378$, $P < 0.001$): elbow extension ($\beta = -0.48$), shoulder flexion ($\beta = 0.41$), trunk rotation ($\beta = -0.48$) and trunk displacement ($\beta = 0.60$).

In the hemiparetic group a different pattern emerged. Significant relationships for the group were found only between one outcome, movement error ($R = 0.568$, $P < 0.001$), and two variables, shoulder flexion ($\beta = -0.84$) and shoulder horizontal adduction ($\beta = 0.46$). Surprisingly, there was no significant relationship between the movement error and the amount of trunk displacement when data from the whole group of stroke subjects were pooled. However, at least three out of the five more severely affected subjects (subjects 1, 3 and 5) but only one out of the four mildly affected stroke subjects (subject 8) had significant correlations between the movement error and the amount of trunk displacement (Fig. 7). These correlations ranged from –0.47 to –0.78. It should be noted that the sign of the correlation in stroke subjects was opposite to that in healthy subjects, implying that the more the trunk moved, the smaller was the movement error in these subjects.

Figure 8 illustrates the relationship between the movement errors and shoulder flexion (top) and horizontal adduction (bottom) for all healthy (open symbols) and stroke subjects (closed symbols). Data from individual subjects 1, 2, 3 and 9 are highlighted by rectangles. Data from the other stroke subjects are represented by closed symbols scattered throughout the plot. In contrast, all data from healthy subjects are enclosed by a single rectangle. Data from individual subjects show clear patterns in the coordinate plane, data

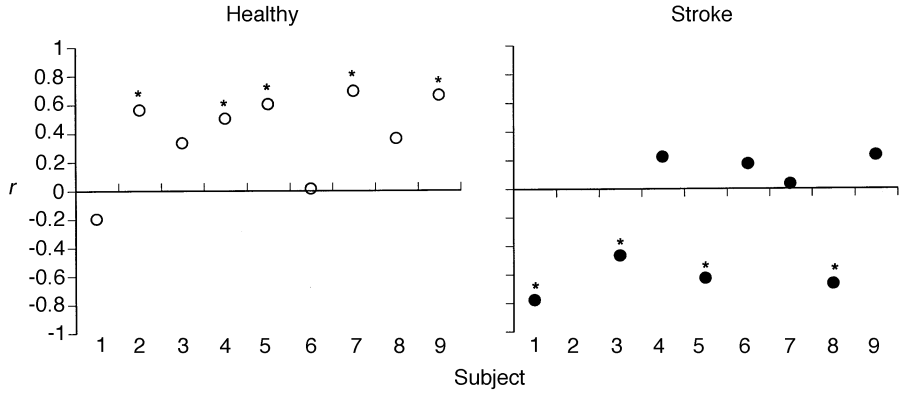

Fig. 7 Correlation between the trunk displacement and movement errors (r) in healthy subjects (open circles) and stroke patients (filled circles). Asterisks indicate significant correlation between the two variables. Data from stroke subject 2 were not analysed. Note that the sign of the correlation in some stroke subjects is opposite to that in healthy subjects.

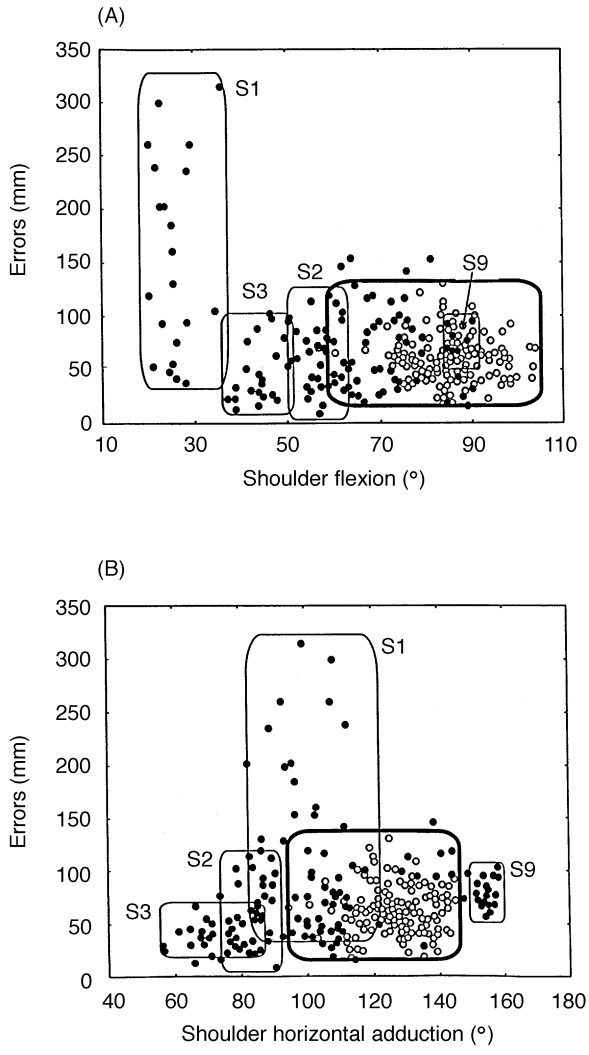

Fig. 8 Relationship between the constant errors and (**A**) shoulder flexion and (**B**) shoulder horizontal adduction in healthy (open circles) and stroke (filled circles) subjects. The thin rectangles delimit the data for each stroke subject (S1–S3 and S9) and the thick rectangle delimits the data for all healthy subjects. For clarity, data from other stroke subjects are not highlighted.

from stroke subjects being more dispersed than those from healthy subjects. It may also be seen that the data from the stroke subjects are distributed in the plane according to their level of clinical impairment. For example, the most impaired subject (subject 1) had the least shoulder flexion movement, the greatest trunk displacement and the greatest movement errors. In general, the points for the more mildly impaired stroke subjects (for example, subject 9) were in the area of the healthy subjects while those for the more severely impaired subjects were further from the healthy group.

Discussion

Movements in stroke subjects were described in terms of trajectory, velocity, accuracy and interjoint correlation. Movement trajectories were characterized by a greater segmentation and a larger degree of dispersion than in healthy subjects (Figs 2–4). Movements were also slower (decreased peak velocities) and velocity profiles were more segmented and more variable than those in healthy subjects (Table 2 and Fig. 4). The decrease in movement precision was another indication that performance in stroke subjects was different from that in healthy subjects (Table 3 and Fig. 5). The movement patterns in stroke subjects were significantly correlated with the level of arm motor impairment, such that more severely impaired subjects had greater segmentation, higher variability and lower accuracy compared with moderately and mildly impaired subjects.

We cannot rule out the possibility that muscle fatigue due to the repetitive trials affected movement patterns in our stroke patients, as evidenced by multiple peaks in the tangential velocity profiles of the endpoint. However, fatigue seems to be an unlikely explanation since the increased number of peaks in stroke patients' movements occured even in the early movement trials (Fig. 4 shows the eighth trial of the sequence for subject 1). In addition, our data indicate that with repeated practise the number of peaks decreases rather than increases.

The differences between the movement patterns in stroke and healthy subjects may be due to disruptions in interjoint coordination (Fig. 6), reflecting a deficit at the central level of motor control. Indeed, all of our subjects had lesions located in the left hemisphere, which is thought to play a greater part than the right hemisphere in the production of fine temporal resolutions needed for rapid complex movements (Haaland and Harrington, 1989). Other functions ascribed to the left hemisphere include greater roles in the integration of sensory information with movement control and in the selection of an appropriate motor programme (Kimura and Archibald, 1974; Kimura, 1982; Goodale, 1988). The limitations in active range of motion in the elbow and shoulder in stroke subjects (Table 2) may partly explain the differences between the movement patterns in stroke patients and healthy subjects. Limitations in active range may have been caused by weakness of muscles directly involved in the movement (Bourbonnais and Vanden Noven, 1989) and spasticity (Bobath, 1990). However, since all stroke subjects had full passive range of motion of the elbow and shoulder, it is unlikely that articular rigidity due to contractures could have been responsible for the joint restrictions we observed. In addition, it has been suggested that a decrease in the ability to regulate stretch reflex thresholds and to coordinate changes in thresholds for a group of muscles may also cause restrictions in the active range of motion and affect the stability of posture (Levin and Feldman, 1994; Levin and Dimov, 1997). It is possible, then, that the configuration of the arm as the end-point neared the target (shoulder horizontal adduction combined with shoulder flexion and elbow extension) may have been an unstable one for some subjects. The avoidance of this posture may have led to the recruitment of additional DFs of the trunk in order to accomplish the motor task (Fig. 2). It is most likely, however, that the recruitment of trunk DFs was elicited by the necessity of overcoming the limitations in the range of motions of arm DFs. Indeed, the amount of trunk recruitment was directly correlated with the limitations of range in these arm DFs in our subjects.

One important distinction characterizing the movement in stroke subjects was the non-typical (for healthy subjects) use of the trunk for reaching a target placed well within the range of the arm's reach (Fig. 2). The use of the trunk is considered a compensatory strategy, and it was significantly correlated with the level of motor impairment in our subjects. This finding suggests that, during recovery from stroke, the nervous system may retain the ability to exploit the redundancy of the motor system by substituting lost elements of the motor pattern (the lack of full range of elbow extension with shoulder adduction) with new elements (trunk DFs) to achieve the functional goal. This adaptation to CNS damage may be due to neural plasticity (Bach-y-Rita, 1981). Some mechanisms of plasticity include: (i) the reversibility of local ischaemic damage to neurons (Fisher, 1992); (ii) the resorption of oedema (Fisher, 1992); (iii) the reorganization of the adjacent cortex or other cortical motor areas of the involved or contralateral hemisphere (Fries *et al.*, 1990; Chollet *et al.*, 1991; Weiller *et al.*, 1992); (iv) the additional involvement of subcortical structures (Weiller *et al.*, 1993; Donoghue, 1995); (v) neuronal sprouting (Raisman and Field, 1973); (vi) the activation of latent synapses (Wall, 1980); and (vii) synaptic plasticity (Eccles, 1979; Tsukahara, 1981). Evidence suggests that, following a period of rapid sensorimotor recovery in the first 3 months after stroke, improvement occurs more gradually for a period of up to 2 years and perhaps longer (Katz *et al.*, 1966; Andrews *et al.*, 1981; Skilbeck *et al.*, 1983). As our subjects were not in the acute phase of recovery, it seems unlikely that mechanisms (i) and (ii) would be in play. However, any of the longer-term mechanisms may have contributed to motor recovery or to the appearance of compensatory trunk involvement in our subjects.

Our findings suggest that the use of a compensatory trunk strategy was significantly correlated with the deficit in motor function and the degree of spasticity in our subjects. In general, patients with mild clinical impairment (subjects 7–9) used an amount of trunk recruitment (51.7 $\pm$ 28.6 mm) similar to that used by healthy subjects (37.5 $\pm$ 14.1 mm), whereas those with moderate to severe impairment (subjects 1–6) used significantly more trunk displacement (139.5 $\pm$ 47.9 mm). The negative correlations between the trunk recruitment and movement errors seen in severely and moderately affected patients suggest that the increase in trunk involvement may contribute to a reduction in movement errors. This may occur since, without trunk recruitment, movement errors would be greater because of the inability of the subject to bring the end-point near the target. In addition, the increased trunk involvement coincided with the subjects' impaired ability to extend the elbow, flex the shoulder, horizontally adduct the shoulder, or to carry out all three movements.

This suggests that there may be a critical level of clinical severity related to the ability of the subject to reach with or without compensatory trunk involvement. This critical level may be defined by a bimodal distribution of the range of active elbow extension, but studies in larger patient populations are necessary for the development of this concept. The notion of a critical level being related to the use of compensatory strategies may have prognostic value. Patients falling below this level (with less elbow extension, shoulder flexion and adduction) may tend to compensate for this loss of ability by increasing their trunk involvement. Whether or not therapies limiting compensatory trunk involvement (for example) can improve active joint motion in these patients is unknown. On the other hand, patients above this level, although they may also have the tendency to use a trunk-compensatory strategy, may retain the ability to exploit their ranges of arm-joint motion. It is possible that these patients will be those most likely to improve and retain lost ranges of joint motion with appropriate therapy, but this requires further study.

One possible explanation for the appearance of a trunk-compensatory strategy for reaching is that control of the trunk may be bilaterally organized and therefore less affected

than that of the arm by the unilateral hemispheric lesion (Gowers, 1893; Willoughby and Anderson, 1984). Another possibility is that, in attempting to stabilize the arm in an unstable position, the subject may fix the shoulder girdle to the trunk, thus reducing the number of DFs associated with the arm. This could be supported by our finding of reduced variability in elbow and shoulder movements in the more severe subjects. The additional trunk recruitment may substitute for the lack of isolated shoulder and elbow movement and give the system additional stability (Carr and Shepherd, 1989).

Latash and Anson have suggested that movement patterns different from those typically observed in persons without impairments should be considered adaptive and therefore should not be corrected (Latash and Anson, 1996). The results of the present study are in contradiction with this point of view, suggesting that the use of fundamentally inappropriate compensatory strategies may limit recovery after stroke [i.e. return of elbow extension (Levin, 1996*b*)]. In our subjects, the compensatory strategy used by the severely and moderately impaired subjects may be considered maladaptive, because such patterns often reinforce distorted positions of the joints, produce excessive muscle shortening and may lead to orthopaedic problems.

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