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IMPAIRMENT AND RECOVERY OF IPSILATERAL SENSORY-MOTOR FUNCTION FOLLOWING UNILATERAL CEREBRAL INFARCTION

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SUMMARY

After unilateral cerebral hemisphere stroke, resulting in contralateral arm symptoms but largely sparing higher cerebral function, ipsilateral arm function is generally considered to be unaffected. In this study, 8 subjects with acute unilateral cerebral infarction (confirmed by CT scan) and primarily motor deficits underwent 11 computerized and 6 clinical assessments between 11 days and 12 months poststroke, and were compared with 12 normal subjects. Computerized tests comprised 3 pursuit tracking tasks (preview-random, step and a combination of these), designed to measure different aspects of integrated sensory-motor (S-M) function, and 12 tasks aimed at breaking tracking into various sensory, perceptual and motor components (joint movement sense, visual resolution, object perception, static and dynamic visuospatial perception, range of movement, grip and arm strength, reaction time, speed, static and dynamic steadiness).

The asymptomatic arm was impaired on all but one of the computerized tests throughout the 12-month period, although to a lesser degree than the symptomatic arm. Grip strength was marginally impaired initially. Incomplete neurological recovery was seen in the asymptomatic arm for all functions except strength, speed and steadiness, possibly indicating their resistance to improvement. Clinical assessment detected no asymptomatic arm impairment and only a mild transient deficit of higher mental function.

Our data suggest that (1) all cerebral hemisphere areas involved in S-M functions can exert some degree of bilateral motor control; (2) ipsilateral influence is never greater than contralateral influence, and is usually considerably less; and (3) the proportion of ipsilateral to contralateral control is closely related to the degree of continuous sensory feedback required by the particular task. The mechanism and degree of ipsilateral dysfunction can be explained by a 3-tier cerebral model of S-M integration comprising a lower level of functions with high contralateral specificity (somatosensory and motor), a middle level of non-limb-specific partially lateralized functions (ideomotor praxis and visuospatial perception) and an upper level of global mental activities (intellect, alertness, etc.).

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INTRODUCTION

It is commonly believed that unilateral cerebral hemisphere stroke, resulting in contralateral arm weakness (with or without associated impairment of somatic sensation) but sparing higher cerebral function, does not cause ipsilateral arm dysfunction. There is evidence that this is not correct.

Most studies of ipsilateral limb function in man have used standard psychological tests, particularly those based on the Halstaed-Reitan Neuropsychological Test Battery (Reitan and Davison, 1974). Cerebral lesions investigated have been vascular, neoplastic or traumatic, and have been unilateral as far as could be determined.

Ipsilateral (as well as contralateral) deficits have been demonstrated from lesions in either hemisphere on skilled sensory-motor (S-M) tasks such as pegboard (Vaughan and Costa, 1962; Wyke, 1971; Haaland and Delaney, 1981), static and movement (vertical groove) steadiness (Haaland and Delaney, 1981), maze coordination (Haaland and Delaney, 1981), pursuit rotor (Heap and Wyke, 1972) and on various somatic sensation/perceptual tests (Semmes *et al.*, 1960; Vaughan and Costa, 1962; Boll, 1974). There are two reports of S-M functions being impaired bilaterally from lesions in one but not the other hemisphere. The first is impaired finger tapping from left lesions (Wyke, 1971). We dispute this, however, as analysis of her data indicates significant ($P < 0.05$, one-tail) impairment of tapping with right lesions as well. In addition, Wyke's results conflict with those of Carmon (1971) and Dodrill (1978) who found, and Haaland and Delaney (1981) who did not find, significant ipsilateral impairment of tapping speed with right and left lesions. The second is impaired maze coordination and grooved pegboard performance from left lesions (Haaland *et al.*, 1977). However, subsequent analysis (Haaland and Delaney, 1981), which took account of pronounced heterogeneity of variance across groups, found no difference between the right and left hemisphere groups.

Grip strength is the only S-M function for which no ipsilateral deficit has generally been found (Kimura, 1977; Haaland and Delaney, 1981). Possible exceptions are Dodrill (1978), Finlayson and Reitan (1980) and Hom and Reitan (1982). In Dodrill's brain-damaged subjects, however, the lesions were not necessarily unilateral and in the latter papers the statistical significance of the differences is not stated.

In addition to demonstrating an ipsilateral (and contralateral) component to many S-M functions, several authors have shown significant predominance of certain functions in one or other hemisphere, although such studies have often been contradictory. Greater left hemisphere bilateral control (i.e., left lesions producing greater deficits in both arms than right lesions) has been shown for a wide variety of S-M tests including somatic sensation (Semmes *et al.*, 1960; Vaughan and Costa, 1962), tapping speed (Wyke, 1971), manual sequence (Kimura, 1977), and eye-arm precision, such as on Purdue pegboard (Vaughan and Costa,

1962). Conversely, greater right hemisphere bilateral control has been demonstrated on tactile-perceptual tests (Boll, 1974) and on a wide range of S-M tests from the Halstaed-Reitan battery (Hom and Reitan, 1982), although results of individual tests from the latter were not reported.

As part of a larger study to quantify impairment and recovery of upper limb S-M function following unilateral cerebral infarction, we investigated a wide range of S-M functions in the arm ipsilateral to the cerebral lesion, in patients without major nonmotor (other than possible sensory) deficits. Emphasis was on proximal rather than distal arm function. The development of a battery of 15 computerized tests to measure various aspects of S-M function, especially following brain damage (Jones and Donaldson, 1981, 1986; Jones, 1987), was central to this study.

METHODS

Subjects

Patients were selected from acute stroke admissions to Christchurch hospitals, screening being performed by a neurologist (*see* Table 3). In addition, selective neuropsychological tests were administered (WAIS Picture Completion, Benton Visual Retention, Reading/Demographic and Digit Span), these chosen as representative of the visuospatial and information processing skills employed in performing the S-M tasks used in this study. Patients who proved to have other than mild deficits of higher mental function were excluded.

Eleven patients initially fulfilled the following criteria: (1) unilateral cerebral infarction, (2) CT scan excluding pathologies other than unilateral cerebral hemisphere infarction, (3) symptomatically impaired upper limb function, (4) other than possible somatic sensory impairment, no major nonmotor deficit (i.e., visual, auditory, perceptual, praxic, language, memory, alertness and intellect), (5) premorbid right-handedness, (6) ability to attend over a 12-month assessment period and (7) informed consent. Two patients withdrew at an early stage and a third was excluded by a second major stroke.

The 8 stroke subjects who completed the study are outlined in Table 1. Patients fell into two broad categories: mild to moderate upper limb weakness (n = 5) and severe upper limb weakness (n = 3). One densely hemiplegic subject (Case 8) had a degree of constructional dyspraxia. Corrected

TABLE 1. STROKE GROUP (ACUTE UNILATERAL CEREBRAL INFARCTION)

			CT scan (infarct)				
Case	*Sex*	*Age (yrs)*	*Site*	*Diameter (approx.)*	*S-arm*	*Paresis (S-arm)*	*Sensory deficit (S-arm)*
1	M	68	R external capsule + BG	1 cm	L	Mild	Nil
2	F	76	L frontoparietal (cortical + subcortical)	4 cm	R	Mild	Mild
3	F	61	Normal (presumed L subcortical lacune)	—	R	Moderate	Nil
4	M	61	R temporoparietal (cortical + subcortical; IC spared)	4 cm	L	Mild	Severe
5	F	73	L frontoparietal (subcortical)	1 cm	R	Mild	Nil
6	M	57	R frontal (subcortical) + IC + BG	3 cm	L	Severe	Moderate
7	M	62	R frontotemporal (subcortical) + IC + BG	4 cm	L	Severe	Severe
8	M	53	R frontotemporal (subcortical) + IC + BG	4 cm	L	Severe	Severe

IC = internal capsule; BG = basal ganglia; S-arm = symptomatic arm; R = right; L = left.

visual acuity was 6/9 or better in one eye (only one patient was not 6/6 or better with both eyes together) and no patient had a field defect or diplopia.

There were two normal control groups, corresponding to single and multiple sessions on the test battery. The single session group contained 36 subjects divided evenly into sex, and six age subgroups, with a subset of 12 of these making up the second group who undertook a further 10 test sessions. All subjects were right handed, had corrected visual acuity of 6/9 or better in one eye and were drawn from a wide range of employments and backgrounds.

TABLE 2. STROKE, STROKE-MATCHED NORMAL (1 SESSION) AND MULTIPLE SESSION NORMAL GROUPS

		Normal	
	Stroke	*Stroke-matched** (*1 session*)	*Multiple session* (*11 sessions*)
n	8	8	12
Age (yrs)			
Mean	63.9	63.5	44.7
SD	7.9	8.2	18.2
Range	53-76	47-72	22-72
Male:female	5:3	5:3	6:6
Driving licence	5	5	11

* Four subjects in the stroke-matched normal group were also in the multiple session normal group.

Most analyses of our data involve comparison of the patient group with the multiple session normal group. As indicated in Table 2, the composition of these two groups differ in age, sex and driving status (i.e., licence holder). To evaluate whether these factors might account for differences in performance, a subgroup of 8 of the 36 subjects in the single session group was formed to match as closely as possible these factors in the stroke group (Table 2). This could then be compared with the stroke and multiple session normal groups (although only on first session) to establish whether these biographical differences had any influence on performance. The resultant stroke-matched normal group and the stroke group were nearly identical in mean age, sex, dominance and driving status, although match was not done on a paired subject basis.

Apparatus and tests

Quantitative and clinical assessments were applied serially and in parallel during the study.

Quantitative S-M test battery assessment (QSMB)

System hardware was based around a PDP-11/34 computer with a VT11 graphics screen (279 mm × 228 mm) for displaying test stimuli (eye-screen distance 132 cm). A steering wheel (395 mm diameter, and minimal 1.0 N friction at perimeter) was used to measure the subject's motor output except for grip strength. S-M integrated and component tests (*see* below and fig. 1) were generated and analysed by two programs (TRACK and SMC, respectively), each of which ran within 64 kbytes of memory under the RT11 operating system. Software was written in FORTRAN IV, except for display of moving stimuli in the dynamic perception task and three tracking tasks, for which the faster MACRO assembly language was necessary.

Integrated function tests. As details are given elsewhere (Jones and Donaldson, 1986), only a summary of the 3 tracking tasks is provided. Each task lasted 120 s and subjects were instructed to

maintain an arrow point on the input target signal throughout the test. Rotation of the wheel moved the arrow horizontally.

1. Random tracking task (preview). The input target signal was a random waveform of 0.21 Hz bandwidth. This descended from the top of the screen giving an 8.0 s preview time before reaching the point of an arrow. The task required smooth movements over a 175° range of the steering wheel. Of several performance parameters obtained, the mean absolute error is the only one presented in this study.

2. Step tracking task (nonpreview). This task comprised 32 abrupt steps alternating between displacement from and return to centre screen. Spatial unpredictability was present in the outward steps through four randomly distributed amplitude/direction movements: large steps (90° on wheel) and small steps (22.5° on wheel), both to right and left of centre. Four randomly distributed durations between steps (2.8, 3.4, 4.0, 4.6 s) and lack of preview ensured temporal unpredictability. Stimulus unpredictability and ballistic response place the step task at the opposite end of the S-M spectrum to the random task. Mean absolute error is again the only score presented in this study.

3. Combination tracking task. In combination tracking the stimulus alternately cycled between the random and step tracking modes over 11 s cycles. Thus, while tracking the random input, the preview signal was abruptly and unpredictably replaced by a stationary vertical line at a distance horizontally displaced from the random tracking signal. The reverse applied at the end of the step tracking mode with the reappearance of the random tracking random signal at another place on the screen. Combination tracking allows determination of the effect of repeated translation between different tracking modes on performance.

Component function tests. Each component function test was designed to isolate and quantify various elements of integrated function measured during tracking. Consequently, there was close resemblance between the component and integrated function tests and validity of comparisons between them was maximized. In those tests allowing several attempts, best rather than average scores were used as best score is considered a more accurate measure of maximal ability by minimizing the effect of inconsistency. Tests moved progressively through visual (sensory/perceptual), proprioceptive and motor component functions.

1. Visual acuity. Corrected visual acuity for each eye measured on the Snellen chart at 6 m.

2. Visual resolution. Visual resolution was measured by ability to identify the position of a dot with respect to a vertical line on the graphics screen. Dot-line separations were in multiples of 0.27 mm.

3. Arrow perception. Perception and comprehension of the components of an arrow identical to that used in tracking tasks with particular emphasis on arrow point.

4. Static perception. Perception of position of the arrow point with respect to a static vertical line in 4 trials and a static sinewave in 16 trials.

5. Dynamic perception. Determination of whether an arrow point stayed perfectly on a random input descending the screen with 8 s preview time. The duration of 20 trials decreased from 10 to 2 s and various error offsets were simulated.

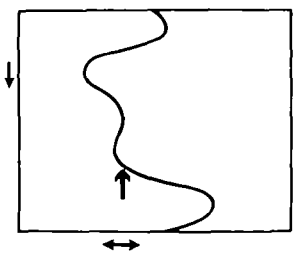
6. Joint movement sense. Determination of the direction of small manually applied and mechanically limited movements to the rim of a steering wheel while the subject was blindfolded, with his hand grasping the top of the wheel. Displacement stimuli ranged from 32.0 mm down to 0.5 mm.

7. Grip strength. Best of three attempts on a TEC dynamometer with the arm extended from the side.

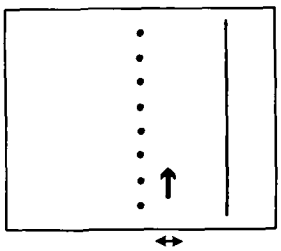
8. Range of movement. Comfortable active range of movement on a steering wheel while maintaining a firm grip.

9. Arm strength. Average of greatest force achieved on 2 attempts at each of 4 hand position-direction conditions on a steering wheel (0° to right, 0° to left, +90° to left, −90° to right; where 0° is the top of the wheel when the arrow is centre screen).

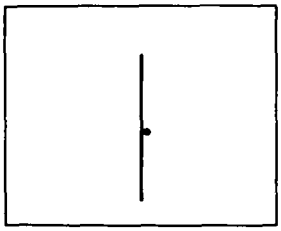
10. Ballistic movement. Fastest possible arm movement in response to a random nontarget stimulus



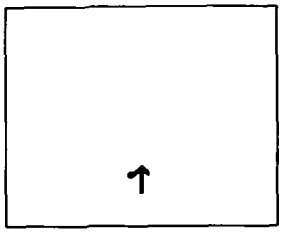
Random tracking (preview): keep point of arrow on or as close to descending random waveform



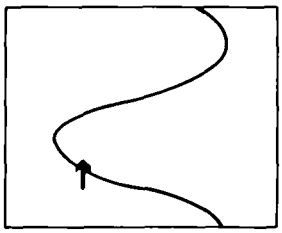
Step tracking (nonpreview): keep point of arrow on jumping line; place emphasis on both speed and accuracy



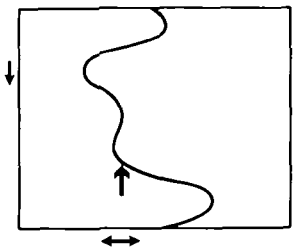
Visual resolution: determine whether dot is to the left of, right of, or on vertical line



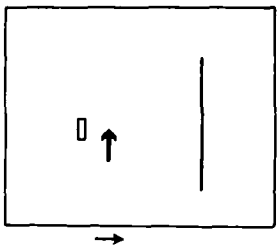
Arrow perception: determine whether flashing dot is on point of arrow or not (3 on, 10 off)



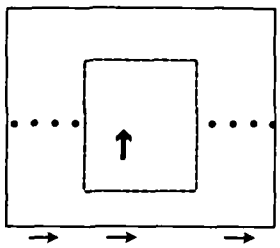
Static perception: determine whether point of arrow is to the left of, right of, or on static waveform (5 on, 15 off)



Dynamic perception: determine whether point of arrow stays on descending random input (6 on, 14 off)



Ballistic movement: move arrow out of box and across line as fast as possible when dotted line goes solid (gives reaction time and speed)



Steady movement: move arrow through box region as smoothly as possible at a speed similar to pacing dots

FIG. 1. Further description of some of QSMB tests.

(no accuracy required). This required moving the arrow out of a box and across a pass-line equivalent to 90° of movement on a steering wheel in response to a random 3–7 s latency stimulus. The best reaction time and speed over 8 attempts were recorded.

11. Static steadiness. Steadiness with the arm extended and the hand grasping a steering wheel at +90° or −90° (= maximum gravity positions) and measured as average deviation of velocity from zero over 7 s duration.

12. Steady movement. Steadiness of attempted constant-speed nonpursuit movement on the steering wheel over a range of 116°. The best of 8 attempts within speed range of 17.7 to 34.7°/s was recorded.

Clinical S-M examination

Comparison of QSMB with conventional neurological examination was important in determining the former's sensitivity, reliability and validity. Published ordinal neurological evaluations were considered unsuitable and a standardized 'Clinical examination of upper limb S-M system' was designed (Table 3). Neurological signs and functions in the examination are in the categories of: higher mental functions, range of limb movement, painful movement, involuntary movement, strength, tone, coordination, reflexes and somatic sensation. Items were rated on a 4-level ordinal impairment scale except for strength and coordination categories in which intermediate values, and hence 7-level items, were considered appropriate. Conversely, only a 'normal' or 'abnormal' 2-level score was meaningful for sensory inattention.

Two aggregate scores were derived for each arm from clinical examination. (1) Clinical Deficit (CD). CD is the sum of all scores per arm (including nonlateralized function) with unity weightings. This cumulative index includes diagnostic signs which may have no direct functional implication. (2) Functional Deficit (FD). FD is the sum of all functionally weighted scores per arm. Weighting factors were empirically assigned according to perceived importance of items to general upper-limb performance, that is, on a wide range of unspecified eye-arm tasks.

Procedure

The stroke and multiple session groups underwent 11 sessions over 1 yr. Spacing was approximately exponential with patients starting on the eleventh day poststroke. Clinical and component tests were applied on alternate sessions. Subjects started tests on the first session with their 'preferred arm'. For all normal subjects this was their right arm whereas for patients it was their asymptomatic arm. The starting arm was alternated in subsequent sessions. All clinical examinations were carried out by one neurologist and without reference to previous test scores.

Analysis

Statistical analysis was by BMDP Statistical Software (Dixon, 1981). Nonparametric statistics have been applied throughout because several variables are inherently nonquantitative (e.g., clinical S-M examination, visuoperceptual tests) and many quantitative variables have skewed distributions as well as different variances between normal and patient groups.

Two sources of confounding bias. If present, impairment in the stroke group should be at its lowest level and most difficult to demonstrate in the asymptomatic arm at 12 months. Hence it will also be the most vulnerable to any confounding effect from nonmatching between subject groups or in experimental design. Apart from the stroke, there were two important differences between stroke and multiple session control groups which could be responsible for small differences in performance.

1. Nonmatching of age, sex and driving status. Multiple session normal subjects were in part optimally chosen for study of the effects of age and sex on normal performance (Jones *et al.*, 1986). This, together with random presentation of suitable stroke patients, meant that the two groups differed in age, sex and driving status (Table 2).

2. Nonmatching of starting-hand. As outlined above, all subjects started tests on the first session

TABLE 3. CLINICAL EXAMINATION OF UPPER LIMB SENSORY-MOTOR SYSTEM

Higher mental function[a]
- Alertness (2)*
- Orientation (2)*
- Intellect (2)
- Motivation (2)*
- Cooperation (2)*
- Emotional stability (2)*
- Language comprehension (2)*
- R/L discrimination (2)
- Visuospatial orientation (2)*
- Praxis—constructional (2)
- Visual inattention (2)

*Range of movement**
- Shoulder (1)*
- Elbow (1)*
- Forearm (1)*
- Wrist (1)*
- Digits (1)*

*Painful movement**
- Shoulder (1)*
- Elbow (1)*
- Forearm (1)*
- Wrist (1)*
- Digits (1)*

*Involuntary movement**
- Tremor (3)*
- Other (3)*

Hypertonia
- Shoulder (1)
- Elbow (1)
- Forearm (1)
- Wrist (1)

Muscle weakness[b]
- Shoulder—flexion (1)
- —extension (1)
- —abduction (1)
- —adduction (1)
- Elbow—flexion (1)
- —extension (1)
- Forearm—pronation (1)
- —supination (1)
- Wrist—flexion (1)
- —extension
- Digits—flexion (1)
- —extension (1)
- —abduction (1)
- —adduction (1)

Coordination[b, c]
- Rapid alternating movement (3)
- Finger-nose-finger (3)
- Finger agility (3)

Reflexes (increased or reduced)
- Biceps (0)
- Triceps (0)
- Supinator (0)
- Finger (0)

Somatic sensation
- Touch (3)
- Pain (0)
- Vibration (0)
- Position (3)
- 2-point discrimination (0)
- Sensory inattention (3)

All items rated on a 4-point ordinal impairment scale (0 = normal, 1 = mild, 2 = moderate, 3 = severe) giving maximum unweighted score of '3' for all items, except sensory inattention which is '1'. Clinical deficit = CD = sum of unweighted scores (per arm). Functional deficit = FD = sum of weighted scores (per arm). FD weightings are in parentheses after each item.

[a] Scores of nonlateralized functions added to scores of both arms. [b] Intermediate levels permitted (7-point ordinal scale). [c] Items untestable due to weakness (e.g., coordination) were given maximum impairment score of '3'. * Items had '0' (normal) score for all subjects.

with their 'preferred arm'. Thus although all patients were premorbidly right handed, 3 started with their left arm. As the limb tested second benefits by the experience of the first, functions for which the dominant right arm is normally superior are inherently disadvantaged in the asymtomatic arm of the stroke group, and advantaged in the symptomatic arm, with respect to the normal subject group. It might be considered that this confounding effect could be eliminated by always comparing right with right, and left with left, but this would introduce a more serious practice order effect. Another possibility might have been for patients to start with right arm irrespective of paresis. This

TABLE 4. INFERIORITY BIASES OF STROKE GROUP WITH RESPECT TO MULTIPLE SESSION NORMAL GROUP

	Inferiority biases[a]		
Test	*Age/sex/ driving*[b]	*Starting hand*[c]	*Total*
Grip strength (kg)	−2.0	0.7	−1.3
Arm strength (N)	−6.5	1.9	−4.6
Reaction time (ms)	−3.1	−1.6	−4.7
Speed (deg/s)	80	10	90
Steadiness (mdeg/s)	30	2	32
Steady movement (deg/s)	−0.08	0.01	−0.07
Random (bit)	3.34	−0.06	3.28
Step (bit)	1.34	−0.03	1.31
Combination (bit)	2.55	0.93	3.48

[a] Inferiority biases are positive if the stroke group was determined to be premorbidly inferior to multiple session normal group, irrespective of whether function is measured as performance or error. Biases are in absolute units of the test functions.

[b] Age/sex/driving status biases for component functions (grip strength, arm strength, reaction time, speed, steadiness, steady movement) are equal to absolute difference between multiple session and stroke-matched normal groups on session 1. The same biases for integrated functions (Random, Step, Combination) are equal to the same differences but reduced in proportion to decreases in error scores of multiple session normal group over the 12-month trial. This reduced bias takes into account greater independence of percentage than absolute performance increments from performance level (Jones, 1987) (*see also* Jones and Donaldson (1981) for illustration of decrease in effect of age on tracking with subsequent sessions).

[c] Starting-hand inferiority bias for asymptomatic arm = 0.375 × R/L differential, where 'R/L differential' is the trial average absolute differential between right and left arms for multiple session normal group (differential is positive if R is superior to L) and '0.375' corresponds to 3 of 8 stroke subjects who chose their premorbid nondominant left arm to be the 'preferred' starting arm.

would unfairly disadvantage patients with severe right-sided paresis, whose left arm would receive little or no benefit from the prior experience of the right.

Adjustments to motor test scores of stroke patients were made to offset artificially the two sources of nonmatching bias. The biases and their derivation are given in Table 4.

RESULTS

Impairment

Both raw and 'match-adjusted' ipsilateral deficits of the stroke group at 11 days and 12 months poststroke are given in Table 5. Raw scores for the contralateral arm are presented to illustrate the much greater deficits in that limb.

Static and dynamic perception were impaired in the stroke group at 11 days, even although visual resolution was superior to that of the normal group. Arrow perception was not impaired, which may reflect the simplicity of the test. Direct comparison between stroke and stroke-matched normal groups was also possible

TABLE 5. IMPAIRMENT: DIFFERENCES IN PERFORMANCE BETWEEN STROKE AND NORMAL GROUPS

	Session 1: 11 days poststroke			*Session 11: 12 months poststroke*		
	Symptomatic arm[a]	*Asymptomatic arm*[b]		*Symptomatic arm*[a]	*Asymptomatic arm*[b]	
Test	*Measured*	*Measured*	*Adjusted*[c]	*Measured*	*Measured*	*Adjusted*[c]
Visual resolution (bit)		−0.33§	−0.5 §			
Arrow perception (incorr.)		0.50	0.25			
Static perception (incorr.)		2.92*	1.50			
Dynamic perception (incorr.)		3.54**	2.62*			
Joint movement sense (mm)	2.94***	0	0	1.8*	0	0
Grip strength (kg)	26.2***	5.5	6.8§	16.2**	1.7	3.1
	(66%)	(13.2%)	(16.5%)	(41%)	(4.2%)	(7.6%)
Arm strength (N)	63.6***	20.9§	25.5§	51.0**	15.9	20.5§
	(63%)	(20%)	(25%)	(45%)	(13.3%)	(17.2%)
Reaction time (ms)	234***	87.9**	92.7**	135.3*	36.4§	41.2*
	(100%)	(36%)	(38%)	(56%)	(14.6%)	(16.5%)
Speed (deg/s)	628***	285***	195**	421***	242**	152§
	(63%)	(28%)	(19.5%)	(42%)	(23%)	(14.5%)
Steadiness (mdeg/s)	1265***	9	−12	809**	29**	−3[d]
Steady movement (deg/s)	9.18***	2.34***	2.41***	5.93***	0.79**	0.87**
	(334%)	(96%)	(99%)	(242%)	(31%)	(34%)
Random tracking (bit)	51.9***	17.2***	13.9**	28.0***	7.05**	3.77§
	(350%)	(96%)	(78%)	(323%)	(77%)	(41%)
Step tracking (bit)	34.2***	15.4***	14.1**	26.6**	7.46**	6.15**
	(98%)	(41%)	(38%)	(89%)	(25%)	(21%)
Combination tracking (bit)	65.0***	26.6***	23.1***	55.4**	9.36**	5.88*
	(134%)	(63%)	(55%)	(163%)	(30%)	(18.9%)
Clinical deficit 'CD'	45.1***	0.75**		33.2***	0.25	
Functional deficit 'FD'	57.7***	1.50**		37.8***	0.50	

All the above figures are differences between, not absolute values for, the stroke and normal groups. Values are positive if stroke group performance is inferior to normal group, irrespective of whether function is measured as performance or error. Percentage differences (in parentheses) are with respect to normal means and have been omitted for nonsensical 'error' scores due to zero (CD, FD) or near zero (joint movement sense, steadiness) 'ceiling' scores for normals. Bilateral nonmotor measures are listed under the asymptomatic arm, to emphasize that both arms were equally affected. Visuospatial functions were not reassessed at the final session. Significance of absolute differences by Mann-Whitney test.

[a] 'Symptomatic arm difference' = symptomatic arm (stroke)—left arm (normal). [b] 'Asymptomatic arm difference' = asymptomatic arm (stroke)—right arm (normal). [c] Stroke impairment scores are adjusted for nonmatching by adding or subtracting the inferiority bias depending on whether performance or error score respectively (*see* Table 4). The 'adjusted' visuospatial scores are from a direct comparison of stroke and stroke-matched normal groups. [d] Loss of significance (** → n.s.) on adjustment of final session steadiness scores is considered to be due to inappropriateness of applying a constant absolute adjustment to data with a marked ceiling effect followed by nonparametric analysis, rather than reflecting lack of impairment (as 4 stroke subjects had perfect '0' scores, even a minuscule adjustment would make them appear superior to all normal subjects). § $P < 0.1$. * $P < 0.05$. ** $P < 0.01$. *** $P < 0.001$, all one-tailed.

on session 1, and resulted in loss of and reduction in significance for static perception and dynamic perception, respectively. This confirms that any visuo-spatial impairment in the stroke group was mild.

At the first session, the symptomatic arm in the stroke group was inferior to controls on all quantitative tests and clinical deficit scores. With the exception of joint movement sense, the same was true for the so-called 'nonaffected' arm. Ipsilateral deficits of grip and arm strength were, however, of marginal significance ($P < 0.1$). For all quantitative tests, ipsilateral arm deficits were much less than contralateral ones.

Steadiness was an apparent anomaly as difference and significance between the groups increased over 12 months. While worsening in the nonparetic arm cannot be discounted, it seems more likely that this result is in error and that steadiness was impaired at 11 days.

At 12 months, the symptomatic arm was also inferior to controls on all lateralized quantitative tests scores. Similarly, arm strength, reaction time, speed, steadiness, steady movement and all 3 tracking tasks, remained impaired in the asymptomatic arm, although arm strength, speed and random tracking were only marginally affected. Grip strength, however, was normal ipsilaterally. Clinical scores (CD and FD) for the symptomatic arm were still inferior overall in the stroke group at 1 yr. However, 3 patients had normal function (FD = 0), 2 had mild deficits (FD < 25) and 3 remained densely hemiparetic (FD > 81). There was no deficit in clinical scores in the asymptomatic arm.

Comparison of quantitative and clinical impairment scores

Probability values obtained for CD and FD were identical on first and final sessions. As FD is a weighted subset of CD, its sensitivity to impairment cannot be improved near the zero impairment level. If CD is zero, so must FD. Hence neither CD nor FD is superior to the other in detection of impairment (differences in their magnitudes are not relevant as they are only incomparable ranking measures).

At the first session, 4 stroke subjects had nonzero CD scores on the asymptomatic arm. These were derived solely from mild deficits in higher mental function: 2 had nonspecific mental slowing, 1 had right/left discriminatory impairment, 1 had subtle left visual inattention and 2 had constructional dyspraxia. All other clinical tests of asymptomatic arm function were normal. Thus impaired asymptomatic arm performance on all quantitative motor tests indicates that they are more sensitive than CD to these types of deficits, or that impaired asymptomatic arm performance results from abnormalities in higher mental function. That the former is at least partly true is suggested by inability of clinical assessment of strength, speed and coordination to detect the absolute impairment demonstrated on related quantitative motor tests (strength, reaction time, speed and steady movement) in the asymptomatic arm of individual patients or the overall group.

Twelve months later, only 1 patient had a nonzero asymptomatic arm CD (Case

8 had a degree of constructional dyspraxia) and, in contrast to QSMB functions, the overall CD was nonsignificant. Thus although all motor tests in QSMB, except grip strength, remained significantly impaired (arm strength, speed and random tracking only marginally so), clinical examination did not reveal any significant deficit of arm function, either directly (via lateralized arm scores) or indirectly (via higher mental function scores) in the overall group.

Effect of nonmatching biases

Elimination of nonmatching on session 1 resulted in only minor changes (Table 5). Most notable were decreased visuospatial deficit and marginal impairment of grip strength in the asymptomatic arm (16.5%, $P < 0.1$).

Except for steadiness (*see* Note 'd' in Table 5), all asymptomatic arm functions impaired at 12 months (i.e., reaction time, speed, steady movement and random, step, combination tracking) retained significance to at least the $P < 0.1$ level after

TABLE 6. NEUROLOGICAL RECOVERY: DIFFERENCES IN AVERAGE INCREMENTS BETWEEN STROKE AND NORMAL GROUPS

Test	*Asymptomatic arm*
Grip strength (kg)	0.24
	(45%)
Arm strength (N)	−0.04
	(−0.7%)
Reaction time (ms)	8.0*
	(195%)
Speed (deg/s)	6.6
	(25%)
Steadiness (mdeg/s)	2
	(100%)
Steady movement (deg/s)	0.30**
	(223%)
Random (%)	1.30*
	(21%)
Step (%)	1.20*
	(48%)
Combination (%)	2.05***
	(68%)
Clinical deficit (CD)	0.10**
Functional deficit (FD)	0.20**

'Average increment' is the average performance increment per session over the 12-month trial. It is calculated from record scores and is an absolute value, except for tracking in which percentage increments are more appropriate (Jones, 1987). Record, rather than raw, scores have been chosen as they give better separation of neurological recovery from practice (Jones, 1987). Differences for asymptomatic arm are relative to right arm of normal group. Percentage differences (in parentheses) are with respect to normal mean; these have been omitted where nonsensical due to zero scores for normals. In contrast to impairment data, increments and probabilities remain unaffected by adjustments for nonmatching. Significance of absolute differences by Mann-Whitney test. * $P < 0.05$. ** $P < 0.01$. *** $P < 0.001$, all one-tailed.

adjustment. Differences increased slightly on grip strength, arm strength and reaction time, but grip strength remained nonsignificant and arm strength became only marginal ($P = 0.09$). Conversely, the significance of impairment was reduced slightly for combination tracking and markedly for speed and random tracking.

In summary, adjustment for age, sex, driving experience and starting-hand differences between stroke and multiple session normal groups did not reveal or eliminate asymptomatic arm impairment at 12 months, although there were increases and decreases in magnitude and level of significance of several functions.

Neurological recovery

Significant improvements in performance above that seen with normal learning, implying neurological recovery, occurred over 12 months in the asymptomatic arm of the stroke group on reaction time, steady movement, random tracking, step tracking, combination tracking, CD and FD (Table 6). Although grip strength, arm strength, speed and steadiness were impaired initially, they did not show significant recovery. It is not clear whether these tests are less sensitive or whether such functions are resistant to recovery. CD and FD have the same sensitivity to recovery, as the difference between these scores (Table 6) is entirely due to a nonvarying scaling factor of '2' (Table 3).

DISCUSSION

Impairment of ipsilateral and bilateral sensory-motor function following unilateral cerebral lesions

Clinical examination initially found mild higher mental function involvement (4 of the 8 stroke subjects), although no direct asymptomatic arm impairment, but by 12 months no significant higher cerebral or ipsilateral deficit remained overall. Conversely, all QSMB tests of asymptomatic arm function, except for grip strength, showed significant impairment throughout the 12 month period.

Major reasons for not detecting ipsilateral deficits in clinical practice are their relative subtlety, failure to use sensitive tests (Brodal, 1973), use of the asymptomatic arm as the reference for impairment in the symptomatic arm and the emphasis placed on the symptomatic arm in rehabilitation.

Subject numbers are too small to allow meaningful comparison between the two hemispheres and the site and severity of lesions in either hemisphere were not comparable (*see* Table 1).

This study and its findings relate to current knowledge on ipsilateral and bilateral aspects of S-M control as follows.

1. Except for grip strength and steadiness, all QSMB functions were physically and conceptually different from those in other studies. Emphasis was on arm (proximal) rather than hand (distal) function. The new tests are arm strength, arm

reaction time, ballistic arm speed, steady movement, smooth preview tracking (full planning possible), step tracking (spatially and temporally unpredictable stimuli), combination of preview and step tracking, and joint movement sense.

2. Grip strength was marginally impaired ipsilaterally, if only for a week or two poststroke. This concurs with Glees and Cole (1952) who found ipsilateral grip strength deficits, following experimental lesions in motor cortex of monkeys, recovered to preoperative levels within 2 wks. Several studies in man (Kimura, 1977; Haaland and Delaney, 1981) have also found no grip strength deficit after several months, but others (Dodrill, 1978; Finlayson and Reitan, 1980; Hom and Reitan, 1982) have suggested possible long-term ipsilateral impairment.

3. Ipsilateral arm strength was marginally impaired throughout the 12 month poststroke period and this has not been previously noted.

4. Joint movement sense was not impaired ipsilaterally. This conflicts with other studies which found somatic sensation significantly impaired ipsilaterally (Semmes *et al.*, 1960; Vaughan and Costa, 1962). However, neither of these studies measured joint movement sense but concentrated on pressure threshold and two-point discrimination.

5. Ipsilateral reaction time, speed, steadiness, steady movement, random tracking, step tracking and combination tracking were impaired throughout the 12 month poststroke follow-up period.

6. On all QSMB and clinical tests, contralateral function was more impaired than ipsilateral function, as expected from the contralateral predominance of the somatosensory and motor pathway projections.

7. Significant but incomplete recovery was demonstrated ipsilaterally for reaction time, steady movement and all three forms of tracking over the 12 month poststroke period. Lack of recovery in strength, speed and steadiness may indicate less reversibility after stroke. No other studies have measured degree of recovery of ipsilaterally impaired S-M function.

8. The only deficit detectable on clinical examination, which could affect ipsilateral function, was a mild nonlateralized (i.e., not arm specific) disturbance of higher mental function and at 12 months this had resolved (except for 1 case with a degree of constructional apraxia). There has been no previous clinical and quantitative comparison on ipsilateral function.

9. These patients had unilateral cerebral hemisphere strokes resulting in contralateral arm weakness but with higher cerebral functions largely spared. Other studies have placed little restriction on subjects other than including predominantly unilateral cerebral disease (stroke, tumour, trauma).

This study differs from previous reports of nonpraxic motor function in the ipsilateral arm following cerebral hemisphere lesions. The majority of these studies have not verified lesions by CT and in none have all patients had CT scans. In only two studies (Semmes *et al.*, 1960; Haaland *et al.*, 1977) has there been a single pathology and each (trauma and tumour, respectively) could have had possible

effect on both hemispheres. In fact, in all studies but one (Semmes *et al.*, 1960) tumours have been included. Many tumours produce bilateral effects through a variety of mechanisms. These include compromise of the opposite hemisphere through midline shift, ventricular obstruction and dilatation and malignant infiltration across the corpus callosum. Although it is not possible totally to exclude minor lesions in the opposite hemisphere by imaging techniques, the present study is the first to examine patients with a single pathology and to screen for subclinical contralateral hemisphere lesions or compression by CT scan in every patient.

This study is also the first to screen subjects for the presence of higher mental function deficits that may have an influence on the performance of sensory-motor tasks. Ideally, the patients should have had no deficit of higher mental function. If, however, testing is comprehensive enough, it is likely some such abnormality can be found in virtually every patient with cerebral infarction. Thus screening in the present study was to exclude subjects with other than mild deficit of higher mental function. The small number of patients ($n = 8$) was selected after evaluating all stroke admissions to two large general hospitals over an 8 month period. We believe demonstrated deficits in ipsilateral upper-limb S-M performance were not due to the mild abnormalities in higher mental function as these seem unlikely to affect such simple motor activities as strength, movement speed, steadiness and steady movement, nor produce a deficit in reaction time and tracking of the extent seen. In addition, these abnormalities in S-M performance persisted for 12 months after the stroke when signs of impaired higher mental function had resolved in all but 1 patient.

Mechanisms of ipsilateral and bilateral control of sensory-motor function

Ipsilateral deficits after unilateral cerebral lesions could be due to direct ipsilateral input to lower motor neurons or involve the contralateral hemisphere with its crossed corticospinal output. Interhemispheric connections are well established but what evidence is there for direct ipsilateral pathways?

Although most corticospinal fibres decussate in the medulla to form the contralateral lateral corticospinal tract, 25% remain uncrossed (Nyberg-Hansen and Rinvik, 1963; Yakovlev and Rakic, 1966; Brodal, 1981). Of these, 15% continue as the ventral corticospinal tract (Nyberg-Hansen and Rinvik, 1963) which mainly decussates in the spinal cord to innervate the contralateral side (Noback, 1967). The remaining 10% of uncrossed fibres stay in the ipsilateral lateral corticospinal tract (Glees and Cole, 1952; Nyberg-Hansen and Rinvik, 1963; Brodal, 1981) and might contribute ipsilateral input if they supply the arm. The same applies to corticospinal fibres which decussate at the medulla but recross in the spinal cord, as has been demonstrated for the lower limbs (Nathan and Smith, 1973). The possibility of multiple decussations within the cord has also been shown by the effects of two unilateral cordotomies in cats (Jane *et al.*, 1964) and man (Nathan and Smith, 1973). Nathan and Smith concluded that the

lateral corticospinal tract carried the majority of ipsilateral fibres, as recovery of the limb on the side of the first cordotomy occurred even when alternative corticofugal pathways, such as rubrospinal and reticulospinal tracts, were severed bilaterally.

Support for ipsilateral control also comes from studies of suprapyramidal lesions. After destroying 83% of corticospinal fibres in one cerebral peduncle, Bucy *et al.* (1964) observed effectively full recovery from contralateral hemiplegia. It is possible, however, that such recovery was due to intact pathways remaining on the lesioned side. After the second operation in staggered unilateral sections of corticospinal tracts in both cerebral peduncles in monkeys, the deficit due to the initial surgery was increased, demonstrating bilateral influence of each corticospinal tract (Bucy *et al.*, 1966).

Further evidence for ipsilateral cortico-limb pathways has been gained from split-brain studies in which contralateral hemisphere effect can be discounted. Following complete section of neocortical commissures, both hemispheres are able to produce ipsilateral and contralateral movement (Gazzaniga *et al.*, 1967; Brinkman and Kuypers, 1973). Ipsilateral control appears strongest for axial and proximal limb musculature and weakest for individual finger movements, especially of the right hand (Gazzaniga *et al.*, 1967). These observations are in keeping with Brodal's (1973) contention that the corticospinal supply to the hand is almost entirely contralateral, as appears to be the case in the monkey (Kuypers and Brinkman, 1970).

An important distinction between studies of ipsilateral function using neocortical commissurotomies and those involving unilateral lesions is that the contralateral system is intact only in the former. This, together with the minor disturbance of ipsilateral movement in split-brain subjects, suggests a third possible mechanism for ipsilateral control, namely modulation of the contralateral system via subcommissural pathways from the other (ipsilateral to limb) hemisphere.

Two apparent paradoxes remain largely unexplained. If ipsilateral pathways produce such substantial recovery in contralateral limbs following unilateral spinal cord or cerebral peduncle lesions, why are ipsilateral deficits so subtle after unilateral cerebral lesions? Studies of subhemispheric unilateral lesions, including the pyramidal tract in the medulla in monkey (Tower, 1940) and man (Ropper *et al.*, 1979), have not observed ipsilateral deficits. Secondly, why do ipsilateral pathways not facilitate similar recovery following internal capsule lesions in which contralateral limb deficits may be severe and irreversible? The first paradox may be explained by suppression of ipsilateral pathways when the contralateral system is intact, as has been suggested in split-brain studies (Gazzaniga *et al.*, 1967). In the second paradox, severity of deficit following capsular lesions probably reflects disruption to other corticofugal tracts, which are more intermingled with corticospinal fibres than at the level of the pyramids (Brodal, 1981). This cannot, however, explain the failure of ipsilateral pathways largely to prevent hemiparesis from such lesions.

Our results suggest three conclusions regarding the bilateral control of S-M functions.

First, all cerebral hemisphere areas involved in S-M functions can exert some degree of bilateral motor control. Of the wide range of S-M functions tested in this study, all were impaired bilaterally by unilateral lesions, although only marginally so for grip and arm strength. Substantial support for this conclusion has also been provided by several other studies (Vaughan and Costa, 1962; Carmon, 1971; Wyke, 1971; Heap and Wyke, 1972; Dodrill, 1978; Haaland and Delaney, 1981).

Secondly, ipsilateral influence is never greater than contralateral influence, and in many cases is considerably less. There were no exceptions to this in our study, although potential exceptions exist. For example, it has been proposed that visuokinetic engrams from left parietal lobe travel intracerebrally to left premotor cortex (for right arm control) and then intercerebrally to right premotor cortex (for left arm control) (Heilman *et al.*, 1982). Thus frontocallosal lesions may produce ideomotor praxic deficits which are greater, or solely, in the ipsilateral left arm (Geschwind and Kaplan, 1962; Watson *et al.*, 1986; Watson and Heilman, 1983; Graff-Radford *et al.*, 1987). In none of the above cases, however, has the lesion been confirmed as being strictly unilateral, as midline structures were affected.

Thirdly, the proportion of ipsilateral to contralateral cerebral control is closely related to the degree of continuous sensory feedback (visual, proprioceptive, tactile) required by the particular task (Haaland and Delaney, 1981). Thus as the complexity of a S-M task increases so does dependence on bilateral cerebral hemisphere function. Of functions tested, grip and arm strength were least impaired ipsilaterally in keeping with strength tests requiring minimal S-M skill and depending minimally on ipsilateral cerebral involvement. Speed is the only other QSMB test not requiring continuous S-M feedback. This task is of relatively low complexity and ipsilateral impairment was marginal. All QSMB tests requiring continuous sensory feedback, that is, reaction time, steadiness, steady movement and the 3 tracking tasks, were significantly impaired ipsilaterally. Furthermore, the degree of impairment tended to reflect complexity of sensory and motor requirements. Steadiness and steady movement can be performed with eyes closed. Thus visual input is not essential but the tasks need continuous proprioceptive feedback. Reaction time utilizes most elements of the S-M system, ensuring it of a bilateral influence. Simplicity and brevity, however, minimize the integration involved, resulting in mild ipsilateral impairment (16% at 12 months). In keeping with the complexity of tracking, ipsilateral impairment on the 3 tracking tasks was greater than for other QSMB tasks ranging from 19% to 41% at 12 months.

Although a gross simplification, we suggest that the present pattern of severe contralateral and milder ipsilateral deficits after unilateral cerebral hemisphere stroke can be explained by a 3-tier cerebral model of S-M integration (fig. 2). 'Lower' level cerebral functions have effects which are strongly lateralized to the

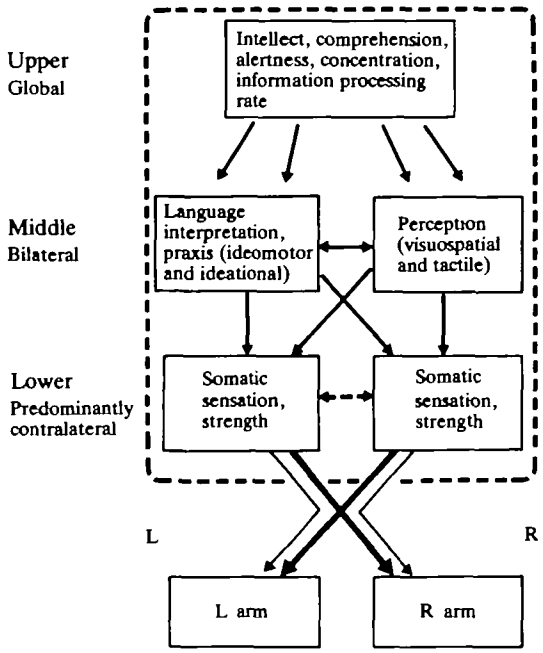


FIG. 2. Proposed 3-tier cerebral model of sensory-motor integration (*see* text for details).

contralateral limbs and have well-defined cortical representation, for example, somatic sensation (postcentral gyrus) and strength (precentral gyrus). In contrast, 'middle' level cerebral functions have effects which are bilateral and influence the lower level in both hemispheres nearly equally. The site of these functions is not well defined but lies principally within the parietal lobes and is partially lateralized. The left hemisphere appears dominant for language interpretation (Lezak, 1976; Walsh, 1978; Brodal, 1981), ideational praxis (Heilman, 1973) and ideomotor praxis (Heilman *et al.*, 1982; Kertesz and Ferro, 1984; Watson *et al.*, 1986; Graff-Radford *et al.*, 1987). The right hemisphere is dominant for visuospatial perception (Lezak, 1976; Walsh, 1978) and tactile perception (Boll, 1974). Finally, 'upper' level cerebral functions have contralateral and ipsilateral effects which are essentially equal. These functions are diffusely sited and include intellect, comprehension, alertness, concentration, and information processing rate. They may be affected by lesions at various cerebral sites, particularly if diffuse, bilateral, or involving the ascending reticular activating system. Their influence is global and impairment can degrade performance of functions at any lower level.

Most ipsilateral arm deficits can be explained by the degree to which various components of the S-M system (as depicted in fig. 2) are required to perform particular tasks. Overriding factors are the predominance of contralateral projections for somatosensory and motor functions at the lower level, and multiplicity of intra- and interhemispheric connections at higher levels.

Overall, evidence from this and other studies indicates unequivocal impairment of ipsilateral arm function on a wide range of simple and complex sensory-motor tasks following unilateral cerebral lesions. The same principles may also apply to lower limb function. Although some recovery occurs, certain deficits may persist, even after several years. These are unlikely to be detected by standard clinical examination. Awareness of impaired ipsilateral function may be important in rehabilitation.

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