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Spatial deficits in ideomotor limb apraxia
A kinematic analysis of aiming movements

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Summary
Ideomotor limb apraxia is a classic neurological disorder manifesting as a breakdown in co-ordinated limb control with spatiotemporal deficits. We employed kinematic analyses of simple aiming movements in left hemisphere-damaged patients with and without limb apraxia and a normal control group to examine preprogramming and response implementation deficits in apraxia. Damage to the frontal and parietal lobes was more common in apraxics, but neither frontal nor parietal damage was associated with different arm movement deficits. Limb apraxia was associated with intact preprogramming but impaired response implementation. The response implementation deficits were characterized by spatial but not temporal deficits, consistent with decoupling of spatial and temporal features of movement in limb apraxia. While the apraxics' accuracy was normal when visual feedback was available, it was impaired when visual feedback of either target location or hand position was unavailable. This finding suggests that ideomotor limb apraxia is associated with disruption of the neural representations for the extrapersonal (spatial location) and intrapersonal (hand position) features of movement. The non-apraxic group's normal kinematic performance demonstrates that the deficits demonstrated in the apraxic group are not simply a reflection of left hemisphere damage *per se*.

Keywords: limb apraxia; stroke; left hemisphere damage; kinematic analysis; motor

Abbreviation: BA = Brodmann area

Introduction
Left hemisphere damage produces a variety of motor disorders including ideomotor limb apraxia, which is a deficit in skilled movement that is not due to primary sensorimotor deficits, aphasia or general intellectual deficits. Clinically, it is examined by asking the patient to perform gestures (e.g. brush teeth, thumb on forehead) to verbal command or to imitation with the hand ipsilateral to lesion. Abnormal postural orientation or spatiotemporal patterning of the arm, hand or fingers is a hallmark of the disorder. Kinematic analyses of apractic gestures (Poizner, 1990; Poizner *et al.*, 1998) have revealed evidence for impairment in space–time relationships (e.g. the normal relationship between velocity and arm position, such that velocity decreases as the arm movement curves, is not seen), joint co-ordination (e.g. greater shoulder movement and less hand movement when turning a key) and spatial trajectories (e.g. more vertical than horizontal movement when carving meat). Although kinematic analyses have been useful in describing movement abnormalities in limb apraxia, the underlying mechanisms of the disturbance continue to be debated. One proposal suggests that ideomotor limb apraxia disrupts the spatiotemporal representation of movement patterns or the retrieval of this information (Heilman *et al.*, 1982). This may explain why patients with apraxia have difficulty using salient spatial or temporal properties of sequential movements to organize them into chunks, thereby simplifying the representation of sequences (Harrington and Haaland, 1992). This proposal is also consistent with the notion that ideomotor limb apraxia is associated with impaired integration of intrapersonal (e.g. hand position) and extrapersonal (e.g. spatial location of the movement goal) features of the movement (Haaland and Flaherty, 1984). This view is supported by the higher incidence of deficits in transitive (i.e. actions involving object use, such as brushing teeth) than intransitive movements (i.e. symbolic actions that do not use objects, such as saluting) because transitive movements require accurate grasping and

movement of an imagined object whereas intransitive movements do not.

Kinematic analyses of simple aiming movements have been useful in identifying mechanisms for movement abnormalities in patients with left or right hemisphere damage (Fisk and Goodall, 1988; Haaland and Harrington, 1989; Winstein and Pohl, 1995) but they have not been used to study limb apraxia. An advantage of this method is that it allows examination of whether apraxics demonstrate kinematic deficits in single arm movements similar to those seen with complex gestures that require multiple arm movements, such as slicing (Poizner *et al*., 1998). Importantly, variables that are thought to affect certain cognitive aspects of movement prior to and during movement can be manipulated more easily in simple aiming tasks to specify better the mechanisms of the disorder. For example, advance planning, in addition to other processes (e.g. attention), is reflected by the reaction time interval preceding movement initiation (Sternberg *et al*., 1978; Kerr, 1978). Systematic variations in RT partially represent the effects of anticipating the forthcoming movement before initiating the response. For instance, RT increases as a function of sequence complexity (e.g. number of elements), not simply the motor initiation requirements of the first response, and this is not due to greater perceptual or attentional processing demands (Sternberg *et al*., 1978; Harrington and Haaland, 1991). Systematic changes in RT are also found with variations in the speed, force and precision of a movement (Fitts, 1954; Keele, 1968). Similarly, RT shortens when response parameters (e.g. direction, hand) are cued prior to an imperative stimulus (Rosenbaum, 1980). These anticipatory effects are widely regarded as evidence for advance planning (Rosenbaum, 1985). It is important to recognize, however, that planning may be ongoing after movement initiation, especially for more complicated responses (Harrington and Haaland, 1992; Pelisson *et al*., 1986).

One investigation of the kinematics of gestures directly compared patients with and without limb apraxia (Hermsdorfer *et al*., 1996). Kinematic features of gestures were more disrupted in patients with left than right hemisphere damage, but the abnormalities were not related to limb apraxia. The latter finding, however, could be due to their definition of apraxia, which was based entirely upon spatial errors in meaningless gestures (i.e. hand orientation, target accuracy), whereas the kinematic analyses focused more on temporal movement characteristics. Despite the inability to distinguish apraxic and non-apraxic patients in the kinematic analyses, examination of individual cases led the authors to speculate that spatiotemporal deficits in apraxia were due to a limited mental representation of the target position.

The high incidence of target errors (e.g. brushing chin rather than teeth) when ideomotor limb apraxics are performing gestures in clinical examinations is consistent with a diminished representation of spatial location. However, these patients demonstrate even greater deficits in hand position and orientation than target location, suggestive of greater abnormality in the intrapersonal aspects of the visuokinaesthetic representation, which translates the examiner's command into an accurate gesture. This pattern of findings predicts that the abolition of hand position or target position information would have a detrimental effect on the performance of patients with limb apraxia, although hand position information should be more influential.

The present study compared patients with and without limb apraxia in order to determine whether deficits in advance planning and movement kinematics were specific to limb apraxia or more general to movement disorders found after left hemisphere damage. The amplitude of the simple aiming movement was cued before the movement began in order to investigate advance planning, which takes less time as movement amplitude increases (Haaland *et al.*, 1993). Visual feedback (i.e. hand position, target location or both) was removed unpredictably after the aiming movement began in order to examine the extent to which patients depended on visual information to guide the movement and, perhaps, modify the action plan. If the kinematics of the movement were abnormally disrupted by the removal of hand information, this would imply an impairment in the visuokinaesthetic representation of the hand. Likewise, if the removal of the target abnormally disrupted movement, this would suggest a deficit in the representation of spatial location.

We predicted that limb apraxic and non-apraxic patients would show normal advance planning because they are able to plan sequential movements ahead (Harrington and Haaland, 1991, 1992). In contrast, we predicted that kinematic measures of movement implementation (e.g. peak velocity, movement time, distance moved, spatial error) would reveal deficits in the apraxic group, especially when hand or target feedback was eliminated due to impairment in their intrapersonal and extrapersonal representations (Haaland and Flaherty, 1984; Hermsdorfer *et al.*, 1996). We examined if these predicted impairments were seen mostly during the initial ballistic movement component, which transports the hand within the vicinity of the target, or the secondary slower velocity movements to hit the target. Finally, we examined the relationship between lesion location, apraxia and disturbances in arm movements to determine whether movement kinematics was differentially impaired in apraxic patients with damage to anterior or posterior areas, as Heilman and colleagues (Heilman *et al.*, 1982) have suggested.

Methods

Subjects

The study sample consisted of 26 chronic stroke patients with radiological confirmation of a left hemisphere infarct and 24 healthy, right-handed control subjects who were of a similar age and education as the stroke patients. All stroke subjects were right-hand dominant prior to their stroke. The subjects' consent was obtained according to the declaration

Table 1 *Demographic characteristics and cognitive and motor status*[†]

Variable	Control (n = 24)	Non-apraxic (n = 16)	Apraxic (n = 10)
Age (years)	62.0 (10.0)	60.0 (14.0)	67.0 (7.0)
Education (years)	15.0 (2.9)	15.0 (3.0)	14.0 (3.0)
Years post-CVA		4.9 (7.8)	5.7 (5.6)
Lesion volume (ml)[‡‡]		41.3 (36.5)	81.3 (57.3)
Limb apraxia[#]	13.9 (1.1)	13.4 (.8)	8.4 (2.7)[††, ‡‡]
Speech[‡,#]	20.0 (.2)	18.6 (2.5)[§§]	11.6 (6.9)[††, ‡‡]
Comprehension[‡,#]	80.0 (1.6)	75.8 (8.5)	44.2 (24.3)[††, ‡‡]
Repetition[‡,#]	99.0 (1.8)	92.6 (11.2)[§§]	60.9 (32.3)[††, ‡‡]
Block design[§,#]	9.1 (2.3)	8.4 (2.4)	4.7 (2.1)[††, ‡‡]
Grip right[¶,#]	49.8 (7.9)	29.4 (20.1)[§§]	24.4 (23.0)[††]
Grip left[¶]	51.1 (8.1)	51.5 (10.5)	45.2 (9.9)
Tap right[¶,#]	41.7 (7.8)	24.6 (19.6)[§§]	21.8 (21.0)[††]
Tap left[¶]	42.5 (7.4)	41.8 (6.8)	37.9 (14.0)

[†]Values are means with standard deviations in parentheses; [‡]scores are from the Western Aphasia Battery (Kertesz, 1982) and assess spontaneous speech, auditory comprehension and repetition with maximum scores of 20, 80 and 100, respectively; [§]scaled scores from the Block Design Subtest of the Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981); [¶]grip strength and finger-tapping are expressed as a standardized T score; [#]number correct with maximum score of 15; analysis of variance showed significant group differences for limb apraxia [$F(2,47) = 51.7$, $P < 0.001$], spontaneous speech [$F(2,47) = 22.8$, $P < 0.001$], auditory comprehension [$F(2,47) = 34.0$, $P < 0.011$], repetition [$F(2,47) = 21.8$, $P < 0.001$], block design [$F(2,47) = 13.11$, $P < 0.001$], grip strength [$F(2,47) = 7.8$, $P < 0.01$] and finger tapping [$F(2,47) = 9.1$, $P < 0.01$]. [††]t tests corrected for unequal variances showed that the apraxic group was impaired ($P < 0.05$) relative to the control group; [‡‡]t tests corrected for unequal variances showed that the apraxic group was impaired ($P < 0.05$) relative to the non-apraxic group; [§§]t tests corrected for unequal variances showed that the non-apraxic group was impaired ($P < 0.05$) relative to the control group.

of Helsinki, and the study was approved by the Institutional Review Board of the University of New Mexico. Stroke patients were tested a minimum of 6 months after stroke. Subjects were excluded if they had a history of substance abuse, psychiatric diagnoses, peripheral problems that would restrict movement or neurological diagnoses other than stroke. Lesion location was measured using MRI or CT obtained at least 3 months after stroke. Lesions were restricted to the left cerebral hemisphere and did not extend into the cerebellum or brainstem. The stroke group was separated into those with limb apraxia (n = 10) and those who were not apraxic (n = 16), based upon videotape scoring of a 15-item gesture imitation assessment (Haaland and Flaherty, 1984). Errors included abnormalities in hand or arm orientation, shape of the hand (e.g. fist), target position (e.g. brush chin, not teeth) and body-part-as-object errors (e.g. index finger extended to brush teeth). Patients who correctly performed 11 or fewer gestures correctly were considered apraxic while those who performed 12 or more gestures correctly were considered non-apraxic. The cut-off of 11 was 2 standard deviations below the performance of a normal control group (Haaland and Flaherty, 1984). As can be seen in Table 1, there were no significant differences among the three groups in age or education, but group differences were present for all language measures and block design construction. Grip strength and finger-tapping were impaired in both stroke groups in the hand contralateral to the damaged hemisphere but not the ipsilateral hand. The apraxic group's performance was impaired relative to the other two groups on all language measures, block design, and contralateral grip strength and finger-tapping. The non-apraxic group's performance was mildly impaired relative to the control group only on spontaneous speech, repetition, and contralateral grip strength and finger-tapping. Neither stroke group demonstrated impaired grip strength or finger-tapping in their ipsilateral limb. The number of years after stroke was not significantly different between the apraxic and non-apraxic groups.

Lesion reconstruction

Computer reconstructions were based upon MRIs in 19 patients and CT scans in seven patients. MRIs were done on a Siemens or a Picker 1.5 Tesla scanner. CT scan reconstructions were done in seven patients who could not undergo an MRI for medical reasons (e.g. they had a pacemaker). Slice thickness for MRIs and CT scans was 5 mm, and all scans were performed at least 3 months after stroke. Infarcts were traced by a neurologist (R.T.K.) onto axial templates derived from the atlas of DeArmond *et al.* (1989), and then computerized using procedures developed at the VA Medical Center in Martinez, California (Frey *et al.*, 1987; Knight *et al.*, 1988). Figure 1 shows the lesion reconstructions on 11 axial sections for individual apraxic and non-apraxic patients. All but two patients (21 and 22) had infarcts in the distribution of the left middle cerebral artery, typically including cortical and subcortical areas.

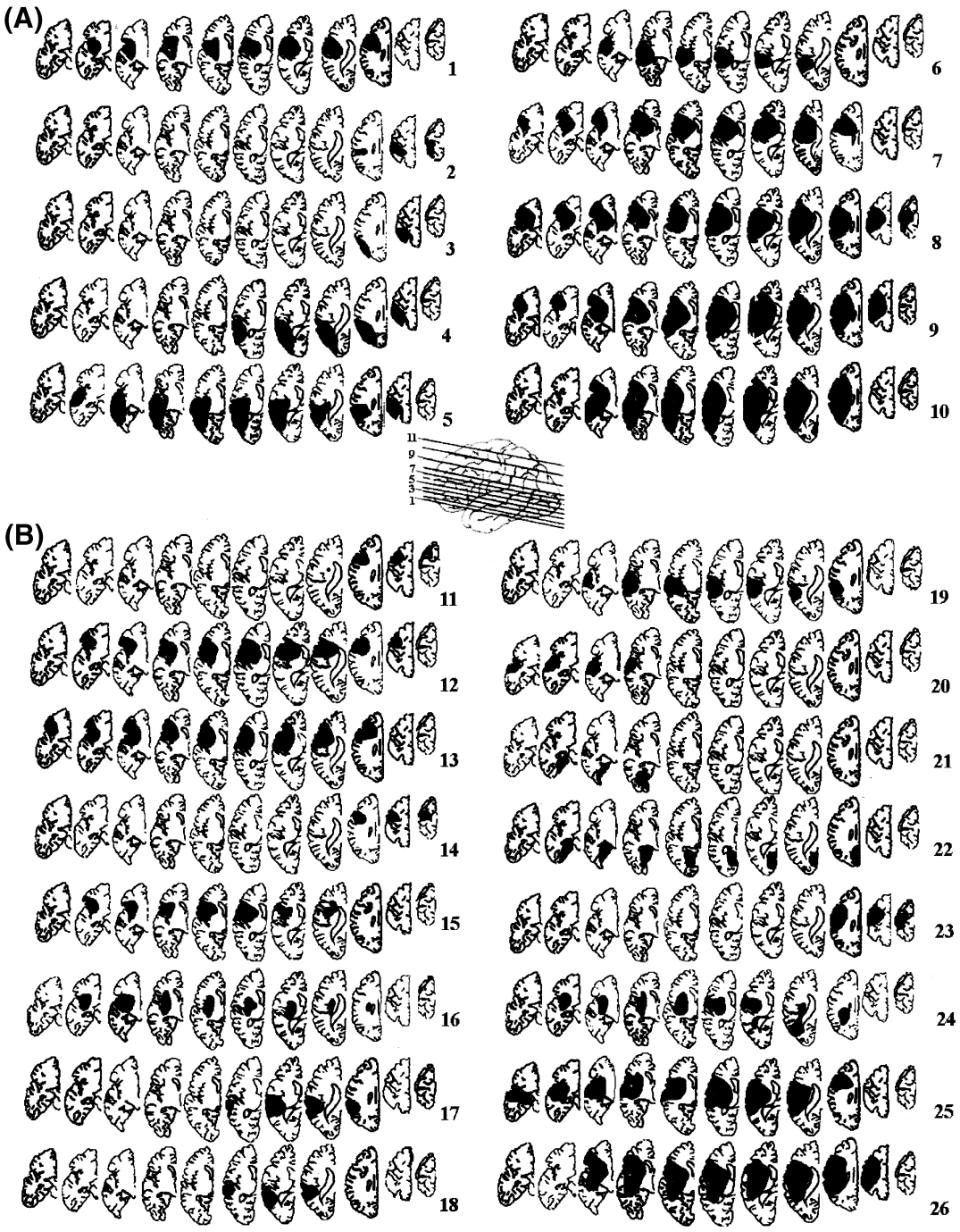

Fig. 1 Infarct location on 11 axial sections in all apraxic (**A**) and non-apraxic (**B**) left hemisphere stroke patients.

Table 1 shows that lesion volume was significantly larger in the apraxic group.

Apparatus

In order to control visual feedback the subject sat in front of a video monitor with the chin placed in a chin rest. The distance from the eyes to the monitor was 813 mm. The subject held a vertical handle hooked to a stylus mounted on a horizontal track which was attached to a digitizing tablet. The target and a dot reflecting the subject's arm position were displayed on the monitor. The spatial and temporal accuracy of the digitizing tablet was 0.007° (0.1 mm) and 0.1 ms, respectively. The digitizing tablet was sampled at a rate of 110 coordinates per second. The display limits of the monitor were 0.04° (0.5625 mm), and there was a one-to-one correspondence between the distance moved on the digitizing tablet and the distance reflected on the monitor.

Although this type of display is not exactly comparable to reaching in the real world, it has been widely used for kinematic research (Meyer *et al.*, 1988; Pratt *et al.*, 1994) and results are similar to those obtained in less artificial conditions.

Procedures

At the beginning of a trial, the start circle and a dot, indicating the subject's arm position, were displayed on the monitor. The subject initiated the trial by moving the left arm to the start circle, located on the far left of the data tablet. When the stylus entered the start circle a 50 ms auditory tone was presented which was followed, after a variable delay of 0.5–1.5 s, by removal of the start circle and presentation of the 5-mm target circle at one of four distances [3° (40 mm), 7° (100 mm), 14° (200 mm) or 20° (300 mm)]. When the target circle was displayed the subject was instructed to move his/her arm inside the target circle as quickly and as accurately as possible.

On visual trials, feedback of the target and arm position was available throughout the trial. During the other conditions visual feedback was removed at the end of the reaction time interval for the duration of the trial. There were four visual feedback conditions: all feedback (target and arm location feedback), target location only, arm position only, and no visual feedback. A trial ended 3 s after the imperative stimulus or when a subject entered the target circle and remained there for 0.5 s, whichever came first. Trials were excluded if the reaction time was $<$150 ms or if the peak velocity of the secondary component was greater than the initial component. The latter criterion was considered a basis for exclusion because it appeared to reflect a false start, as indicated by the fact that the second component on those trials was similar in velocity and duration to the initial components on other trials. Importantly, the number of trials eliminated was extremely small, though it was greater for the two stroke groups [$F(2,47) = 8.11$, $P < 0.001$; control mean (SE) = 0.27 (0.04); non-apraxic mean (SE) = 0.45 (0.09); apraxic mean (SE) = 1.46 (0.55)]. In all subjects, more trials were excluded at the shortest amplitude [$F(3,141) = 15.9$, $P < 0.001$; mean (SE) = 0.9 (0.2), 0.4 (0.1), 0.5 (0.1), 0.4 (0.1) for the 3°, 7°, 14° and 20° amplitudes, respectively].

Design

The four visual feedback conditions and four movement amplitudes were randomly presented in five blocks, each block consisting of three trials at each amplitude and feedback condition (48 trials per block), giving a total of 240 trials or 15 trials per condition. The experimental trials were preceded by practice trials consisting of a random presentation of two trials for each of the 16 conditions to ensure subjects understood the task.

The dependent measures were similar to those used in other kinematic analyses (Meyer *et al.*, 1988; Haaland *et al.*, 1993; Pratt *et al.*, 1994) and were chosen to describe the spatial and temporal characteristics of the movements with a minimum of redundancy. For each subject, a mean was computed for each of the 16 conditions, so that the tables and figures reflect the means across all subjects in each group. Reaction time was defined as the interval between the imperative stimulus (target appearance) and when the velocity of the arm movement exceeded 1.5° per s. Two components of the movement were separated—the initial rapid transport component and the secondary adjustment component—because these components have been shown to be differently impaired after left hemisphere damage (Fisk and Goodale, 1988; Haaland and Harrington, 1989; Hermsdorfer *et al.*, 1996). The initial component began at the end of the reaction time interval and ended when (i) velocity dropped back to noise level (i.e. $\leqslant$1.5° per s); (ii) velocity levelled out to a plateau above noise level (i.e. $<$7.5% change over a 100 ms interval); or (iii) velocity decreased to at least 50% of peak velocity and then increased, indicative of a second acceleration phase and a new movement. The secondary movement began at the end of the initial movement and ended when velocity was $\leqslant$1.5° per s.

Measures included movement time (initial and secondary components); distance travelled (initial component); duration of the acceleration phase of the initial movement (i.e. percentage of the initial movement that preceded the peak velocity); peak velocity (initial phase); mean velocity (secondary component); constant error (mean distance from the edge of the target to the location of the hand/stylus at the end of the secondary component); variable error (variability of constant error); and percentage of the trials with a secondary component.

Results

Separate analyses of variance (ANOVAs) with repeated measures tested the between-subject effect of group (control, non-apraxic, apraxic) and the within-subject effects of visual feedback and movement amplitude, for each dependent measure. We focused upon the main effect of group and its interactions with the other factors because our interest was in the differential performance of the three groups. Other main effects and interactions are reported, but are not fully discussed here because the effects of response amplitude and visual feedback in control subjects have been reported elsewhere (Haaland *et al.*, 1993). The ANOVAs and follow-up simple effect analyses were adjusted for multiple tests by using an alpha level of 0.01. Table 2 presents the data for each group, averaged across the different amplitude and feedback conditions.

Reaction time decreased with increasing amplitude [$F(3,141) = 64.43$, $P < 0.001$], but did not differ significantly among the groups. There were no other significant main effects or interactions.

The percentage of trials with a secondary component

Table 2 *Movement measures for the control, non-apraxic and apraxic groups*[†]

Variable	Control (n = 24)	Non-apraxic (n = 16)	Apraxic (n = 10)
Reaction time[‡]	457.00 (10.30)	473.80 (14.10)	512.60 (21.80)
Initial transport			
Percent acceleration[§]	39.90 (0.80)	41.30 (0.01)	44.70 (1.20)
Peak velocity[#]	26.20 (2.10)	23.90 (1.90)	23.40 (2.20)
Movement time[‡]	618.60 (26.90)	678.40 (37.30)	684.80 (40.00)
Distance[††,+]	10.40 (0.10)	10.60 (0.10)	10.40 (0.30)
Secondary component			
Percent secondary[¶]	80.30 (2.00)	79.90 (2.20)	73.40 (4.40)
Mean velocity[#]	2.40 (0.10)	2.30 (0.10)	2.38 (0.20)
Movement time[‡*]	507.40 (14.90)	572.60 (31.70)	715.20 (68.90)
Distance[††]	1.20 (0.10)	1.30 (0.10)	1.70 (0.30)
Constant error[††,+]	–0.04 (0.05)	0.11 (0.09)	–0.01 (0.10)
Variable error[††,+]	0.61 (0.03)	0.68 (0.06)	1.43 (0.25)

[†]Values are means with standard errors in parentheses; [‡]milliseconds; [§]percentage of initial movement which was accelerating; [¶]percentage of movements with a secondary component; [#]degrees per second; [††]degrees; *significant group differences (ANOVA), $P < 0.01$; [+]significant group × condition effects (ANOVA), $P < 0.01$.

increased with movement amplitude [$F(3,141) = 80.3$, $P < 0.001$] and was lowest for the no visual feedback condition and highest for the all visual feedback condition [$F(3,14) = 26.7$, $P < 0.001$]. The significant feedback condition × amplitude interaction [$F(9,423) = 3.04$, $P < 0.003$] showed that there was a smaller difference between the no visual feedback condition and the all visual feedback condition at the longest amplitude (mean differences between the no visual feedback condition and the all visual feedback condition were 14, 21, 19 and 9% for the 3°, 7°, 14° and 20° movements, respectively). No other effects were significant.

Initial transport phase of the movement

Percent acceleration

The percentage of the initial movement devoted to the acceleration phase reflects the relative shape of the velocity profile. It was greatest for the longer amplitude movements [mean (SE) = 37 (0.9), 40 (0.7), 44 (0.8) and 44 (0.8) percent for the 3°, 7°, 14° and 20° movements, respectively; $F(3,141) = 36.4$, $P < 0.001$]. There were no other significant effects.

Peak velocity

The effect of feedback condition was significant [$F(3,141) = 2.5$, $P < 0.001$], but the differences among the conditions were very small and of little practical significance [mean (SE) = 25.4° (1.2), 24.9° (1.3), 24.5° (1.3) and 24.7° (1.2) per s for the all, target, hand and no feedback conditions, respectively). Peak velocity also increased as amplitude increased [$F(3,141) = 275.8$, $P < 0.001$]. No other effects were significant.

Initial movement time

The most notable effect for initial movement time was its large increase with movement amplitude [$F(3,141) = 369.8$, $P < 0.001$]. There was also a significant feedback condition effect [$F(3,141) = 4.2$, $P < 0.01$] which was characterized by longer initial movement times for the all and hand position feedback conditions (i.e. when hand position was always present) than the other feedback conditions [mean (SE) = 660 (20), 647 (21), 659 (20) and 638 (20) ms for the all, target, hand and no feedback conditions, respectively]. The significant condition × amplitude interaction [$F(9,423) = 4.53$, $P < 0.001$] reflected the finding that initial movement time increased with movement amplitude more for the hand and no feedback condition (i.e. when target location was not present) than for the all and target feedback conditions (i.e. when target location was present). The mean difference between the 3° and 20° amplitudes was 430 ms for all feedback, 432 ms for target feedback, 510 ms for hand feedback and 480 ms for no visual feedback. There were no other significant effects.

Distance moved

Each of the three subject groups travelled about 89% of the total distance during the initial movement component. There were significant effects of condition [$F(3,141) = 2.7$, $P < 0.001$] and amplitude [$F(3,141) = 4740.1$, $P < 0.001$] such that the distance moved was greatest in the all and hand position feedback conditions [mean (SE) = 10.8° (0.1), 10.3° (0.1), 10.6° (0.1) and 10.2° (0.1) for the all, target, hand and no feedback conditions, respectively] and at the longest amplitude [mean (SE) = 2.5° (0.1), 6.8° (0.1), 13.6° (0.1) and 19.0° (0.2) for the 3°, 7°, 14° and 20° amplitudes, respectively]. Most importantly, there was a group × condition interaction [$F(6,141) = 3.76$, $P < 0.01$]. Figure 2

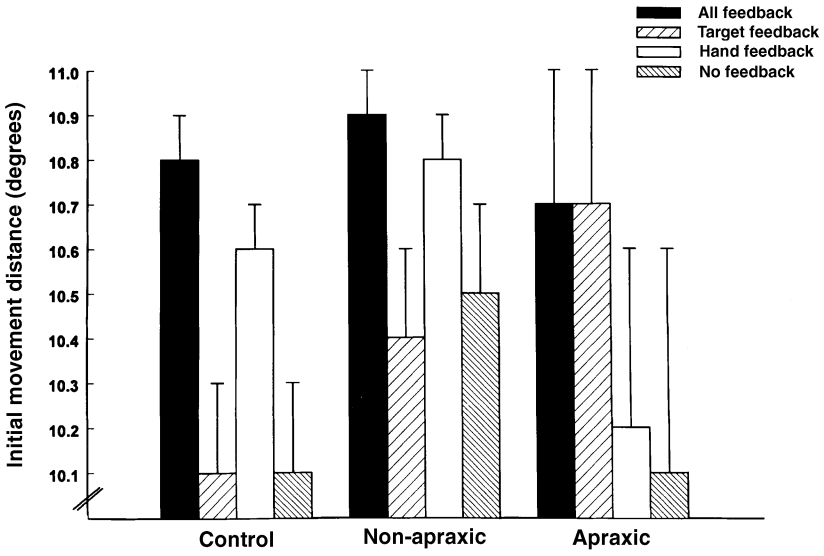

Fig. 2 The mean distance moved in the initial phase of the movement (with standard error bars) is displayed separately for the three groups as a function of feedback condition.

shows that this interaction was due to a significant effect of feedback condition in the control [$F(3,69) = 22.6$, $P < 0.001$] and non-apraxic [$F(3,45) = 6.2$, $P < 0.01$] groups but not in the apraxic group. The control and non-apraxic groups moved further in the initial movement in the all and hand position feedback conditions (i.e. when hand position was available) whereas the distance moved was less in the target and no feedback conditions (i.e. when hand position was not available). In contrast, hand position feedback had no effect on distance moved in the apraxic group. Although Fig. 2 suggests that the removal of target location (i.e. hand position and no feedback conditions) may have been more detrimental to the apraxic group's performance, there was no significant effect of feedback condition in the apraxic group likely due to high variability.

Secondary adjustment phase of the movement

Average velocity

There was a significant effect of feedback condition [$F(3,141) = 21.12$, $P < 0.001$] characterized by lower velocity when all visual feedback was available [mean (SE) = 1.9° (0.1), 2.6° (0.1), 2.2° (0.1) and 2.7° (0.1) per second for the all, target, hand and all feedback conditions, respectively]. The significant effect of amplitude [$F(3,141) = 98.02$, $P < 0.001$] reflected increased velocity with increased amplitude [mean (SE) = 1.6° (0.1), 2.1° (0.1), 2.6° (0.1) and 3.1° (0.1) per s for the 3°, 7°, 14° and 20° movements, respectively]. The condition × amplitude interaction [$F(9,423) = 3.10$, $P < 0.01$] was due to smaller velocity increases as amplitude increased for the all and hand feedback conditions (i.e. when hand position was present). The mean differences in velocity between the 3° and 20° amplitudes were 1.2°, 1.9°, 1.3° and 1.7° per second for the all, target, hand and no feedback conditions, respectively. There were no other significant effects.

Secondary movement time

There was a significant effect of group [$F(2,47) = 9.08$, $P < 0.001$], which was due to longer SMTs in the apraxic group (Table 2). However, this effect was due to the trend for the apraxic group to move further during the secondary movement (Table 2). This explanation is consistent with the absence of significant group differences in the velocity of the secondary component.

The effect of condition [$F(3,141) = 14.36$, $P < 0.001$] was associated with longer secondary movement times in the all feedback condition relative to the other conditions, and the significant amplitude effect [$F(3,141) = 60.26$, $P < 0.001$] was due to longer secondary movement times as amplitude increased. There were no other significant effects.

Constant error

Figure 3 illustrates the group × condition interaction for constant error [$F(6,141) = 4.6$, $P < 0.01$], which was due to the apraxic group's unique pattern of errors across the different feedback conditions. Specifically, follow-up analyses showed that group interacted with condition when the apraxic group was separately compared with the control group [$F(3,96) = 7.0$, $P < 0.01$] and the non-apraxic group [$F(3,72) = 3.7$, $P < 0.05$], but no such interaction was found when the control and non-apraxic groups were compared ($P > 0.20$). Figure 3 clearly shows that constant error was minimal and similar across all three groups when all visual feedback was present. In contrast, when any kind of visual feedback was removed, only the apraxic group showed

impaired spatial accuracy. The apraxics overshot the target when hand feedback was removed and undershot the target when target feedback was removed (i.e. hand position and no feedback conditions). In contrast, constant error did not differ significantly from 0 in any feedback condition for the control and non-apraxic groups.

Variable error

Variable error reflects the spatial consistency of the endpoint of a movement. The pattern of results for variable error was similar to constant error except that there was a significant group effect [$F(2,47) = 17.3$, $P < 0.001$] (Table 2). Figure 4 shows that the group differences varied as a function of condition [$F(6,141) = 3.4$, $P < 0.01$]. Follow-up analyses showed that variable error was greater in the apraxic group than the other two groups for each feedback condition [$F(2,47) = 13.1$, $P < 0.001$; $F(2,47) = 8.6$, $P < 0.001$; $F(2,47) = 12$, $P < 0.001$; and $F(2,47) = 18.8$, $P < 0.001$ for the all, target, hand and no feedback conditions, respectively]. Most importantly, like constant error, the interaction can be attributed to the apraxic group's unique pattern of variability relative to the two other groups. Specifically, there were significant group × condition interactions when the apraxic group was compared with the control group [$F(3,96) = 5.3$, $P < 0.01$] and a trend for an interaction when the apraxic group was compared with the non-apraxic group [$F(3,72) = 2.8$, $P < 0.05$]. However, no such interaction was found when the control and non-apraxic groups were compared with each other ($P > 0.22$). Further, while the control [$F(1,23) = 90.2$, $P < 0.001$] and non-apraxic [$F(1,15) = 61.0$, $P < 0.001$] groups demonstrated less variable error when hand position was available than when target position was available, variable error in the apraxic group was similar when visual feedback of any kind was removed ($P > 0.3$).

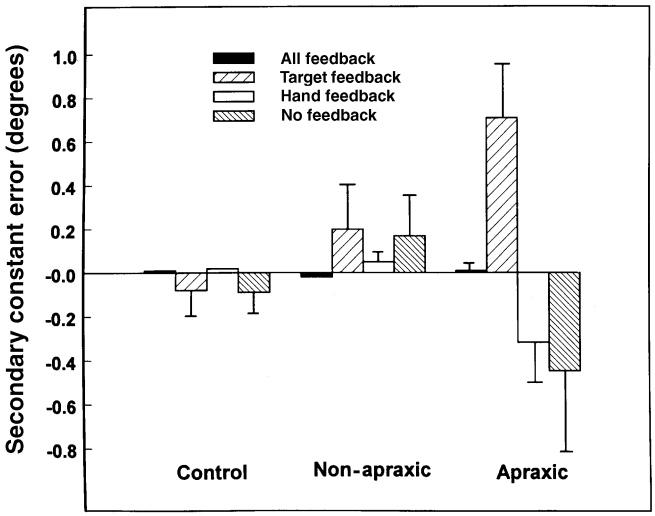

Fig. 3 The mean constant error of the secondary phase of the movement (with standard error bars) is displayed separately for the three groups as a function of feedback condition. Positive constant error reflects target overshoot and negative constant error reflects target undershoot.

Lesion location

Lesions from each patient were superimposed on axial sections. Figure 5 displays the overlapping images across the six axial sections that reflected the greatest amount of lesion overlap among the patients. Apraxic patients were separated into anterior, posterior and anterior–posterior groups to determine whether lesion location was associated with particular deficits in arm movement. The anterior group included patients whose lesions were mainly anterior to the central sulcus, but also could involve damage to the sensorimotor cortex (BA 3, 1 and 2) or temporal lobes. One subject in this group (Patient 13) had slight damage to the

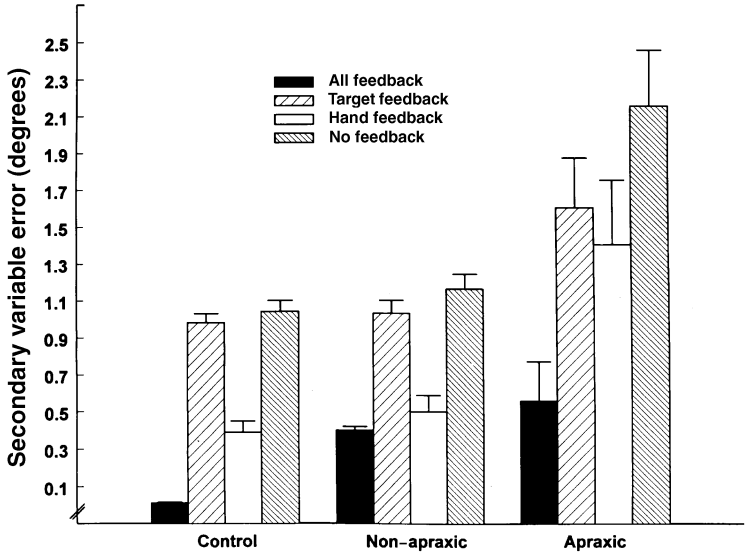

Fig. 4 The mean variable error of the secondary phase of the movement with standard error bars is displayed separately for the three groups as a function of feedback condition.

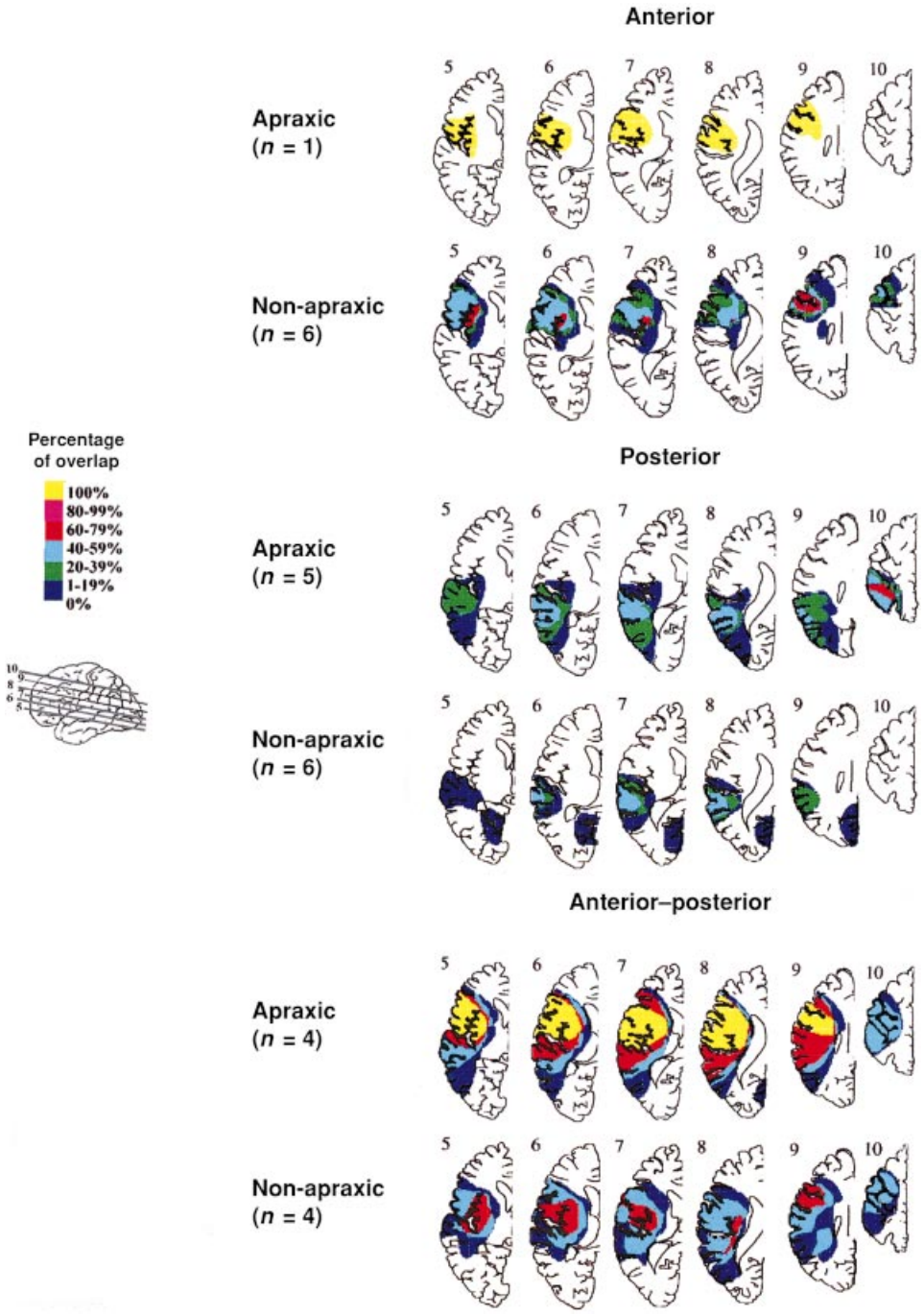

Fig. 5 Overlapping infarct locations on six axial sections (slices 5–10) in apraxic and non-apraxic left hemisphere stroke patients separately displayed for those with primarily anterior, posterior, or anterior and posterior damage. Figure 1 displays the individual scans for each of the patients included in the six groups. The following patients are included in each group: anterior apraxic (patient 1) and non-apraxic (patients 11–16), posterior apraxic (patients 2–6) and non-apraxic (patients 17–22) and anterior–posterior apraxics (patients 7–10) and non-apraxic (patients 23–26).

supramarginal gyrus (BA 40) on axial section 7 only (see Fig. 1). This patient was included in the anterior group because the posterior involvement was so small, and this patient's lesion was more similar to the anterior than the anterior–posterior group. The posterior group included patients whose lesions were mainly posterior to the central sulcus, but could also involve damage to the primary motor cortex (BA 4) or temporal cortex. Figure 5 shows that there were similar numbers of apraxic and non-apraxic patients with posterior or both anterior and posterior damage, but there was only one apraxic patient (Patient 1) with an anterior lesion. When the areas of damage were compared between

Table 3 *Selected movement measures for the apraxics with different lesion locations*[†]

	Anterior (n = 1)	Posterior (n = 5)	Anterior–posterior (n = 4)
Apraxia severity[‡]	10 (0)	10.0 (0.6)	6.0 (1.2)
Initial distance[§]			
All feedback	12.6 (0)	10.3 (0.50)	10.7 (0.2)
Target	11.7 (0)	10.2 (0.50)	11.0 (0.1)
Hand	11.6 (0)	10.3 (0.50)	9.6 (0.8)
None	11.5 (0)	10.0 (0.50)	9.8 (1.0)
Secondary CE[§]			
All feedback	0 (0)	0.1 (0.03)	0 (0.0)
Target	0.2 (0)	0.7 (0.30)	0.9 (0.6)
Hand	–0.1 (0)	–0.1 (0.20)	–0.7 (0.4)
None	–0.4 (0)	–0.1 (0.30)	–1.1 (0.8)
Secondary VE[§]			
All feedback	0.4 (0)	0.8 (0.40)	0.3(0.2)
Target	0.9 (0)	1.9 (0.50)	1.5 (0.30)
Hand	0.5 (0)	1.4 (0.50)	1.6 (0.70)
None	1.5 (0)	2.1 (0.50)	2.4 (0.50)

[†]Values are means with standard errors in parentheses; [‡]numbers of correct items on the 15-item battery (Haaland and Flaherty, 1984); [§]degrees.

the anterior and anterior–posterior apraxics and non-apraxics, parts of the middle and inferior frontal gyri, in the vicinity of BA 6, 8 and 46, were particularly critical. More specifically, these gyri were involved in 100% of the anterior–posterior apraxics and in the single anterior apraxic patient. However, these areas were also damaged in 50% of the anterior non-apraxics and 50% of the anterior–posterior non-apraxics, suggesting that damage to these areas does not always produce apraxia. In order to identify the more critical posterior areas, the areas of damage in the posterior and anterior–posterior apraxics and non-apraxics were compared. The lower superior parietal lobe and upper inferior parietal lobe in the vicinity of BA 7, 39 and 40 appeared to be most critical with 80% of the posterior apraxics, 50% of the anterior–posterior apraxics, none of the posterior non-apraxics and 25% of the anterior–posterior non-apraxics having damage in these areas.

We examined the arm-reaching performance across these groups to determine whether the pattern of performance varied as a function of intrahemispheric lesion location (Table 3). Given the small number of patients in each group, statistical analyses were not possible, but the pattern of findings appears to be similar across the three groups and similar to the overall findings in the apraxic group. Feedback condition affected the initial distance very little, and secondary constant error and variable error were lowest when visual feedback was available relative to the three other conditions.

Discussion

Preprogramming

The time to plan and initiate simple aiming movements was not impaired in the apraxic and non-apraxic groups. Patients with left hemisphere damage were able to use amplitude information normally to develop a plan of action. The increased reaction time for shorter amplitude movements in all groups suggests that subjects capitalized on the fact that longer-amplitude movements allow the continued development of the motor programme after the initiation of the movement (Meyer *et al.*, 1988; Harrington and Haaland, 1992). The shape of the initial component's velocity profile probably reflects a combination of factors, including preprogramming and response modification based upon the specific task requirements and constraints on the movement (Pelisson *et al.*, 1986; Marteniuk *et al.*, 1987). The importance of task constraints is supported in this study by the finding that the proportion of the initial component in the acceleration phase and the peak velocity of the initial component was greater as response amplitude increased. However, the shape of the initial component's velocity profile, as evidenced by the same variables (i.e. proportion of time in the acceleration phase and peak velocity of the initial component), was comparable across groups even as feedback condition varied. Therefore, all three groups organized and implemented the transport or ballistic phase of the movement similarly.

These results are in agreement with previous findings showing that apraxics and non-apraxics normally preprogrammed repetitions of hand postures (Harrington and Haaland, 1992). Simple aiming is like repeating a hand movement in that both place relatively minimal demands on advance planning. Conversely, we have shown that planning and implementing sequences of different hand movements, which depend on developing and retrieving different motor programmes for each movement in the sequence, are specifically impaired in ideomotor limb apraxia (Harrington and Haaland, 1992). This suggests the possibility that advance planning deficits in apraxia might emerge if sequences of different simple aiming movements (e.g. different amplitude and/or target diameters) are studied.

Movement implementation

In contrast to the similarities across the groups in advance planning, the implementation of the movement in the apraxic group was especially dependent on the availability of visual feedback. This is the first study to show that a disruption in movement kinematics is specific to ideomotor limb apraxia rather than associated with left hemisphere damage *per se*. Importantly, kinematic abnormalities in apraxia were uncovered in spatial but not temporal aspects of movement. The longer SMT in the apraxic group was an artefact of the longer distance travelled by this group, which was supported by the lack of a significant group difference in velocity, which controls for variations in distance moved. This finding further supports our conclusion that the spatial, but not the temporal, measures differentiate the apraxic group from the non-apraxic and control groups. Case studies of apraxia have shown temporal and spatial abnormalities during the performance of gestures (Poizner *et al.*, 1998). This finding suggests that when reaching movements contain more degrees of freedom, deficits in both aspects of movement would be expected in apraxia, because of the greater need to integrate spatial and temporal information when the movement requires the sequencing of a series of movements (e.g. brushing teeth). Performance of a movement depends on the context in which it occurs, even when biomechanical factors are controlled. Specifically, planning and on-line control depend on temporal and spatial features of each element in the sequence and the relationship of these features across the elements, which increases the spatiotemporal integration requirements (Restle, 1973; Povel and Collard, 1982).

Our findings contrast with those of Hermsdorfer and colleagues (Hermsdorfer *et al.*, 1996), in which abnormalities in the kinematics of gestures were similar in apraxic and non-apraxic patients with left hemisphere damage. The focus of their kinematic analyses, however, was on the shape of the trajectory and the duration of the secondary movement, also measures which did not discriminate apraxic and non-apraxic patients in our study. Although we did not uncover impairments in these aspects of movement in either left hemisphere-damaged group, which conflicts with their findings, simple aiming movements are less complex than gestures and therefore may not be as sensitive to potential disturbances in temporal processing. However, Hermsdorfer and colleagues (Hermsdorfer *et al.*, 1996) did not examine spatial accuracy in their kinematic analyses and, contrary to their results, spatial accuracy was the only measure in our study that discriminated apraxic from non-apraxic patients. Moreover, we found distortions in spatial accuracy in apraxia only when visual feedback was removed, implying a greater dependency on hand position and target location information than was found for controls or left hemisphere-damaged non-apraxic patients.

During the initial transport movement, the performance of controls and non-apraxic patients was most altered by the removal of hand position information, which reduced their movement distance. This is consistent with the work of some (Prablanc *et al.*, 1979; Carlton, 1981) but not others who have found that the removal of target location information has a more detrimental effect on performance (Whiting and Cockerill, 1974; Eliott, 1988). All of these studies examined the error at the end of the movement, and in our study the normal control group and non-apraxic group demonstrated no significant constant error at the end of the movement regardless of visual feedback condition. However, as seen in Fig. 4, variable error was less when visual feedback of arm position was available than when visual feedback of target location was available, supporting the greater importance of arm position information. However, in the secondary adjustment phase both groups reached the target destination with a high degree of precision, regardless of the availability of different kinds of visual information. This pattern of results indicates that the representation of spatial location and its integration with hand position information was highly accurate and sustained throughout the trial. Furthermore, while the removal of hand information altered the trajectory, controls and non-apraxic patients compensated without sacrificing end-point accuracy.

These findings contrasted with those of the apraxic patients, who tended to reduce the distance of the initial movement mostly when target location was unavailable. There was a large amount of variation within the apraxic group, however, so that the spatial aspect of the transport movement was not consistently disrupted, regardless of the kind of visual feedback. Conversely, the removal of both kinds of visual feedback during the secondary adjustment phase produced significant spatial error and disrupted the consistency of hitting the target location.

It appears, therefore, that all subjects depend on visual information to monitor and adjust reaching movements, although hand position feedback clearly plays a greater role during the initial transport movement in maintaining spatial accuracy in controls and non-apraxic patients than in apraxic patients. Apraxic patients, however, continue to rely on visual feedback during the secondary adjustment phase and, importantly, never achieve normal end-point accuracy when visual feedback of hand position and/or target location feedback is unavailable. It was also notable that, despite impairments in spatial accuracy, the temporal properties (e.g. velocity, movement time) of the entire aiming movement remained normal. This finding is also compatible with a decoupling of temporal and spatial representations, which has been described elegantly in case studies of ideomotor limb apraxia using three-dimensional kinematic analyses of gestures (Poizner *et al.*, 1997).

Mechanisms underlying kinematic abnormalities in apraxia

One possible explanation for kinematic abnormalities is that apraxia disrupts the time course for using visual feedback,

perhaps because patients are simply slower. This explanation, however, would predict that temporal aspects of movement (reaction time, movement time, velocity) should be slowed as a function of visual feedback, which was not found. Visual feedback also altered velocity and movement time of the initial movement in all three groups, indicating that early in the transport phase of the movement apraxic patients were able to process visual information. Moreover, spatial accuracy in the initial and secondary phases of the movement was normal in apraxic patients when complete visual feedback was available, showing that they can use this information as rapidly as controls to guide a simple movement.

An alternative proposal is that apraxia disrupts the neural representations that control the spatial characteristics of gestures as well as other less complex movements. This hypothesis is supported by the greater dependence of apraxic patients on both kinds of feedback. The spatial abnormalities found when target location was eliminated suggests that the representation of extrapersonal space is diminished in apraxia. Likewise, the reliance on hand position feedback to sustain spatial accuracy may imply a disruption in the representation of intrapersonal spatial properties (e.g. hand position, location, orientation) of hand positions. This proposal is consistent with clinical assessments of the disorder in which target location and hand position errors are commonly reported (Haaland and Flaherty, 1984; Rothi *et al.*, 1988).

The above proposal is consistent with the speculation of Heilman and colleagues (Heilman *et al.*, 1982) that damage to the parietal lobes directly disrupts visuokinaesthetic engrams for movement. This is also compatible with our lesion reconstruction findings in which limb apraxia was especially associated with damage to the superior parietal (area 7) cortex, although the superior aspect of the inferior parietal cortex (areas 39 and 40) also is frequently damaged in these patients. Area 7 has been associated with processing spatial location (Haxby *et al.*, 1993). However, in primates muscimol injections in the intraparietal sulcus produce problems positioning fingers or adjusting the orientation of the hand (Gallese *et al.*, 1994), similar to apraxic patients. Recently, we found increased activation in the superior parietal cortex when increasing the number of different fingers in a sequencing task (D. L. Harrington, S. M. Rao, K. Y. Haaland, J. A. Bobholz and A. Mayer, unpublished results), further endorsing the view that this area is crucial for specifying spatial and proprioceptive representations of effectors. Importantly, representations of effectors may be bound by the intended goal of an action, which in the present study involved hitting a target in a particular spatial location. Thus, the superior parietal cortex may be involved in the integration of extrapersonal spatial information with intrapersonal spatiotemporal properties of effectors.

Heilman and colleagues (Heilman *et al.*, 1982) also suggested that ideomotor limb apraxia in patients with anterior damage was due to a disruption in the retrieval of visuokinaesthetic engrams for movement. In our study, damage to the middle and inferior frontal gyrus was also associated with limb apraxia. The middle frontal gyrus receives projections from the parietal cortex (Selemon and Goldman-Rakic, 1987), which places it in a strategic position for accessing visuokinaesthetic engrams or motor representations presumably located in the parietal lobe. The middle frontal gyrus has been associated with working memory (Goldman-Rakic, 1988), which could be important for imitating an examiner's gestures, but the precise functions of these systems is not well understood. The assessment of apraxia also probably involves multiple sensory and cognitive processes, including retrieval. Our analyses, however, failed to uncover different patterns of kinematic abnormalities in patients with anterior, posterior or anterior–posterior damage. Although this might argue that both areas are involved in similar forms of processing, our simple aiming task was not designed to distinguish retrieval or working mechanisms from other forms of processing.

The apraxic group's deficits when visual feedback of target location or hand position was removed could also be explained by impairment in proprioception. For example, target location information can be obtained visually or through feedback of head and eye position, and hand location information can be obtained from arm position feedback. Although these possibilities were not directly investigated in the present study, proprioceptive deficits have not been reported in the arm or index finger ipsilateral to a unilateral lesion (Haaland and Harrington, 1989), making that explanation less likely.

Lesion location and limb apraxia

While the neuroanatomical correlates of ideomotor limb apraxia are not the focus of this study, our method of overlapping lesions has promise in better identifying the relative importance of different neural areas in limb apraxia. The current results suggest that areas within the frontal and parietal lobes are important, contrary to some authors, who have emphasized subcortical pathways connecting the frontal and parietal lobes or only the parietal lobe (for review, see Faglioni and Basso, 1985). Others have suggested that limb apraxia is such a complex behaviour that damage in multiple areas can produce the syndrome (Basso *et al.*, 1980; Alexander *et al.*, 1992).

Implications

The findings from this study suggest that ideomotor limb apraxia is associated with disruption of the neural representation for extrapersonal and intrapersonal features of goal-directed movement. This impairment is seen even though visual feedback is available immediately before the movement begins. Still, the limb-apraxic patient can use visual feedback of target location and arm position to perform as accurately as non-apraxics with left hemisphere damage or normals. This suggests that a visuomotor disconnection cannot explain these deficits, despite the apraxics' high incidence of damage to area 7, which has been associated with optic ataxia.

While our emphasis upon the importance of parietal and frontal areas is consistent with early work in apraxia (Heilman *et al.*, 1982), definite conclusions cannot be made about whether the representations for extrapersonal and intrapersonal features of goal-directed movement are mediated by multiple parietofrontal circuits or a single parietofrontal circuit, as has been suggested by neuroimaging and neuroanatomical data (Rizzolatti *et al.*, 1998 and D. L. Harrington, S. M. Rao, K. Y. Haaland, J. A. Bobholz and A. Mayer, unpublished results). Our study also did not demonstrate different roles for frontal and parietal areas using this relatively simple task. Both of these issues deserve further study in order to elucidate the neural substrates of limb apraxia.

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