Kinematic analysis of movement imitation in apraxia

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Summary
Accuracy of the final position and kinematics of movement were analysed during the imitation of meaningless gestures in patients with unilateral brain lesions who performed with the hand ipsilateral to the lesion and in control subjects. Controls imitated the gestures virtually without spatial errors. The kinematics of their movements was characterized by single-peaked and approximately bell-shaped velocity profiles of the transport phase combined with no or only minor corrective movements in the final phase. Patients with right brain damage (RBD) performed as well as control subjects with respect to both spatial accuracy of final hand-positions and kinematic parameters of the movement trajectories. Patients with left brain damage (LBD) committed spatial parapraxias and had a much higher frequency of kinematic abnormalities. However, there was no correlation between kinematic abnormalities and apraxic errors. There were kinematically abnormal movements which reached a correct final position as well as kinematically normal movements leading to apraxic errors. One possible explanation for the combined occurrence of kinematic abnormalities and parapraxias in LBD patients would be that they are independent sequels of left hemisphere lesions. An alternative account is that the associations and dissociations result from an interaction between one common basic deficit and strategies to cope with this deficit. The basic deficit may concern the mental representation of the target position. The LBD patients may react to the absence of an appropriate representation of the target by one of two alternative strategies; they may switch to a strategy of slowed, on-line controlled movements to find the required final position, or they may move their hand smoothly at normal speed to a roughly specified location without taking note of their deficiency. Depending on whether these strategies are successful or not they would lead to the observed associations and dissociations between kinematic abnormalities and spatial parapraxias.

Key words: ideomotor apraxia; motor control; left hemisphere damage; imitation of meaningless gestures; kinematic analysis

Abbreviations: LBD = left brain damage; RBD = right brain damage

Introduction
Apraxic patients commit errors when performing movements with their non-paretic hand. Errors may concern the use of objects and tools as well as movements performed without external objects like the pantomime of object use (transitive gestures) or the demonstration of conventional symbolic gestures (intransitive gestures). Apraxic patients may commit errors even when the correct movements are demonstrated to them and they only have to imitate them (Liepmann, 1908; Geschwind and Damasio, 1985; Poeck, 1986; Heilman and Rothi, 1993). The cardinal feature of apraxia is the occurrence of parapraxias which may affect either the spatial course and final position of single movements or the temporal sequencing of multiple movements into a coherent motor action (Liepmann, 1908; Poeck, 1982).

The riddle of apraxia is posed by the observation that the occurrence of abnormal movements depends on the condition of testing (De Renzi, 1989). The widely accepted definition of apraxia as a 'disorder of skilled movement not caused by weakness, akinesia, deafferentation, abnormal tone or posture, movement disorders (such as tremor or chorea)' (Geschwind...
and Damasio, 1985) implies that there are situations where the left hand is moved with normal skill. Otherwise, the exclusion of other movement disorders could not be verified.

An explanation for the dissociation between normal and abnormal motor control of the left hand could be sought in different demands on the conceptual representation of the action examined (Roy and Square, 1985). Such an explanation can easily account for errors occurring with tool use, pantomime of object use and symbolic gestures; in all of these a correct concept of the intended movement has to be evoked from memory before movement execution can start. This account, however, has problems with impaired imitation of gestures, as in this situation the intended target position is demonstrated by the examiner and the patient is only required to copy the movement. The case against a conceptual source of errors in the imitation of movements seems to be even more convincing if imitation is probed for meaningless and novel movements, the execution of which cannot be helped by evocation of conceptual knowledge. Errors in the imitation of movements have therefore been a main argument for considering apraxia or, at least a variety of it, as a disorder of movement execution (Liepmann, 1908; Morlaas, 1928; Barbieri and De Renzi, 1988; Kimura, 1993).

Imitation of gestures usually requires the patient to move the hand from a starting position to a predetermined target position. Since Woodworth’s seminal studies (1899) manual aiming movements have been a major topic of motor control research. A method used to analyse movement execution which has gained increasing interest during the last decade is the kinematic analysis of the path travelled by the moving limb. It has been shown that unrestrained skilled aiming movements of the arm follow a straight or slightly curved path, and that the corresponding velocity profile has only one maximum which is located near the middle of the time-course (Morasso, 1981; Atkeson and Hollerbach, 1985; Flash and Hogan, 1985). Single-peaked and bell-shaped velocity profiles are generally recognized as the invariant feature of preprogrammed movements. By contrast, irregular and multi-peaked velocity profiles during aiming movements have frequently been observed in patients with cerebral lesions (Jeannerod et al., 1984; Flash et al., 1992; Haggard et al., 1994; Isenberg and Conrad, 1994; Mattingley et al., 1994). In neurologically normal subjects irregular hand velocities are also obvious during development (Konczak et al., 1995) when the target approach requires high precision (Soechting, 1984; Milner and Ijaz, 1990) or when movements are performed with unnaturally low velocities (Milner and Ijaz, 1990). Uncertainty, the belief that the movement to be performed is particularly difficult and extensive attention to movement details can also induce a change from smooth and single-peaked to slowed and irregular movement trajectories indicating a change from preprogrammed to on-line controlled movement execution (Marquardt and Mai, 1994a).

Recently, Poizner and colleagues (Poizner et al., 1990; Clark et al., 1994; Poizner et al., 1995) provided detailed kinematic analyses of apraxic movements. In their first study (Poizner et al., 1990) they investigated the pantomime of object use evoked by verbal command (e.g. ‘erase a blackboard’) in two apraxic patients. In two further studies, the gesture ‘slicing bread’ was analysed in three apraxic patients under different contextual conditions (with and without the object or/and tool present) including the actual object and tool use (Clark et al., 1994; Poizner et al., 1995). Multiple abnormalities in the kinematic features of apraxic movements were found, such as irregular velocity profiles, reduced maximum velocities, decoupling of the normally strong (negative) relationship between instantaneous velocity and curvature of movement path and deficient joint coordination. Kinematic abnormalities during arm movements of apraxic patients have recently also been reported by Platz and Mauritz (1995); when instructed to reproduce triangular arm movements from memory, two apraxic patients yielded acceleration profiles that were more irregular than the corresponding profiles of control subjects.

These results of Poizner et al. and Platz and Mauritz (1995) do not permit a definite conclusion as to the level of gesture production at which kinematic abnormalities arise. In studies of goal directed arm and hand movements (e.g. aiming movements, see above), kinematic features such as irregular velocity profiles have been interpreted as indicators of deficient motor programming. Kinematic abnormalities in apraxic movements may therefore be a direct indication of insufficient programming of the details of movement execution in apraxia. Alternatively, kinematic abnormalities may be an indirect sequel of conceptual errors in the performance of symbolic gestures: the patients may have performed amorphous movements or may have switched to a strategy of slowed on-line controlled movements because they lacked a clear conceptual representation of the intended target positions.

We decided to investigate movement kinematics during the imitation of meaningless gestures in a larger sample of apraxic patients. The imitation of familiar and meaningful gestures could be mediated by existing knowledge about the form and the meaning of the gestures and could therefore be affected by degradation of this knowledge. By contrast, imitation of novel and meaningless gestures appears to involve a direct route from perception to execution of the movement (Rothi et al., 1991; Roy and Hall, 1992). Errors in the imitation of meaningless gestures should, therefore, permit a more direct insight into defective motor execution.

The present study was aimed to assess the relationship between kinematic abnormalities and spatial parapraxies that are typically observed in apraxic imitations of meaningless gestures (Kimura and Archibald, 1974; De Renzi et al., 1980; Barbieri and De Renzi, 1988; Goldenberg, 1995, 1996). It was predicted, that if deficient motor programming is the cause of parapraxic errors, kinematic indications of deficient programming should be detectable in every movement which ends up with a parapraxic error.
Patients and eight RBD patients (range 36-73). The study was approved by the local ethical committee and all patients and controls gave their informed consent to participate.

### Apraxia testing

Subjects sat in front of a table with the hand to be tested resting in a comfortable pronated position in front of the body on the table. The examiner faced the patients and demonstrated the gestures ‘like a mirror’, i.e., if the subjects were to use their left hand, the examiner demonstrated with the right hand and vice versa. Subjects were instructed to start the imitation immediately after the examiner had finished the presentation. All gestures required arm movements towards the head with a specific final position of the hand in relation to the head (see Fig. 2). This type of gesture was selected because it proved to be particularly sensitive to defective imitation in apraxia (Goldenberg, 1995, 1996). A total of 23 trials were performed (see Movement registration, below). The spatial correctness of the final position was scored independently by the examiner and a second observer during the examination. Examinations were video-taped, and in very rare cases of disagreement between the two observers (maximum two trials in the most apraxic patients) unanimity was reached after discussion of the video. A spatial parapraxia was noted if the final position of the patient’s movement differed unequivocally from that demonstrated by the examiner.

### Movement registration

The hand movements during the imitation of the meaningless gestures were recorded using an ultrasonic device that continuously calculates the three-dimensional spatial positions of tiny markers attached to moving body parts (CMS 50, Zebris, Isny, Germany). The method is based on the evaluation of the transmission times of ultrasound impulses emitted from the markers and received by three microphones mounted on a stationary frame (size 40×40×5 cm³). The frame was placed 1 m in front of the subject. Two markers were used, both attached to the ulnar aspect of the hand, one to the wrist above the styloid process of the ulna, the other above the metacarpophalangeal joint of the little finger. Spatial coordinates of the two markers were sampled with a frequency of 100 Hz each and a spatial resolution of 0.2 mm.

The examination started with five imitation gestures which were considered for registration of parapraxias but not for kinematic measurement. Then, imitation of six different gestures (Fig. 2) was tested and both spatial parapraxias and movement kinematics were recorded. The six gestures were selected for kinematic analysis because they appeared to be best suited to minimize ultrasound transmission errors during data recording caused, for example, by markers being hidden. Each of these six gestures were repeated a total of three times in order to increase the number of trials available for data analysis. Thus, kinematics of 18 trials were obtained.

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### Table 1 Sex, age, aetiology, type of aphasia and number of spatial parapraxias in the imitation task in 20 LBD patients and eight RBD patients

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Aetiology</th>
<th>Aphasia</th>
<th>Spatial parapraxias</th>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
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<td>Broca</td>
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<tr>
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<td>M</td>
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<td>Broca</td>
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</tr>
<tr>
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<tr>
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<td>Global</td>
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<tr>
<td>20</td>
<td>M</td>
<td>66</td>
<td>Ischaemia</td>
<td>Global</td>
<td>23</td>
</tr>
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</table>

| RBD patients |
| 21        | F   | 52          | Haemorrhage | –   | 0                  |
| 22        | M   | 54          | Ischaemia   | –   | 0                  |
| 23        | M   | 54          | Ischaemia   | –   | 0                  |
| 24        | F   | 44          | Ischaemia   | –   | 0                  |
| 25        | F   | 51          | Ischaemia   | –   | 1                  |
| 26        | M   | 44          | Haemorrhage | –   | 1                  |
| 27        | M   | 66          | Ischaemia   | –   | 1                  |
| 28        | M   | 39          | Haemorrhage | –   | 3                  |

Patients are ordered according to their frequency of apraxic errors with increasing case numbers reflecting increasing numbers of parapraxias.

### Methods

#### Subjects

Twenty-eight patients with unilateral brain lesions participated in the study. Twenty patients had suffered LBD and eight RBD caused by single unilateral cerebrovascular accidents. Clinical and demographic data of the patients are summarized in Table 1 and templates of their lesions are displayed in Fig. 1. All patients were examined 1–3 months after the accident. Patients performed the imitation task with the arm ipsilateral to the lesion. Clinical examination of apraxia had confirmed that all patients understood the instructions necessary to comply with the task (Goldenberg, 1995, 1996).

Twenty age-matched subjects served as a control group. All were right-handed by self-report with no previous histories of neurological or psychiatric illness. The control group was split into two subgroups of 10 subjects each, one performing the imitation task with the right hand (two female, eight male, mean age 49.7 years, range 37–79) and the other with the left (five female, five male, mean age 52.6 years, range 36–73). The study was approved by the local ethical
Fig. 1 Horizontal sections of the left brain in 20 LBD patients showing the location of brain lesions. The case numbers refer to Table 1; patients are ordered by increasing frequency of parapraxias.
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Fig. 2 Performance of a 56-year-old, male control subject during the imitation of meaningless gestures. Results from the first presentation of the six gestures are shown. The subject performed with the left arm. The drawings sketch the final positions which were always correct. Movement trajectories of two markers (solid line = wrist; broken line = MCP joint of the little finger) in the frontal plane (seen from the subjects' rear) and the tangential velocity profile of the wrist marker for each gesture are also shown.

while 23 trials were considered for the assessment of parapraxias.

Data analysis
The positional data of the two markers were analysed interactively using a specially designed software ('3DA'; Marquardt et al., 1994). Raw data were smoothed and time derivatives were calculated by means of kernel estimates (cut-off frequency 12 Hz) which provide a non-parametric estimation of regression functions by moving weighted averages (Marquardt and Mai, 1994b). Analysis of the movement kinematics concentrated on the marker at the subjects' wrist. The wrist was selected because studies of unrestrained aiming movements demonstrated that the qualitative features of wrist kinematics remain invariant with changing movement direction and amplitude while joint kinematics may differ substantially (Morasso, 1981; Flash and Hogan, 1985). From the markers' spatial coordinates the three-dimensional trajectories, the tangential velocity along the path and the corresponding accelerations were determined.

For the wrist movement of each gesture two phases were distinguished: a transport phase that brought the hand into the vicinity of the final position and an adjustment phase that ended when the hand was held stationary. The transition from transport phase to adjustment phase was determined interactively according to the following criteria: (i) a local velocity minimum (or alternatively, a local acceleration maximum if it indicates a flatting of the velocity profile without an inversion of velocity) was detected during the deceleration of the movement when the velocity was <15% of its peak value; (ii) the hand had been raised to an altitude corresponding roughly to the subject's head. Similar methods to distinguish movement phases have been used by other authors (cf. Meyer et al., 1988). The start of the transport phase and the end of the adjustment phase were determined interactively from the velocity and acceleration curves as the time points where the signals departed from or respectively, returned to their baseline. In all subjects, velocity stabilized around zero for several hundred milliseconds before the hand was returned to the table.

Several kinematic parameters were determined to characterize the wrist transport phase: the duration of the transport movement, the peak tangential velocity, the percentage time to peak velocity (ratio of the time to peak velocity to the movement duration), the amplitude of the movement
It is apparent from Fig. 1 that the number of parapraxias found in patients with parietal, frontal, temporal, subcortical increased with increasing size of the lesion. Apraxia was classified as non-apraxic (Cases 1-7) and the 13 LBD patients (see Table 1). Taking the highest incidence of parapraxias in the RBD group as a cut-off to define apraxia, the seven LBD patients with zero to three apraxic errors would have been classified as non-apraxic. By contrast, spatial parapraxias were frequently observed in LBD patients. Some parapraxias resulted in gestures grossly different from the examiner’s demonstrated gestures. For example, instead of placing the fingertips under the chin with the ulnar side of the hand oriented anteriorly (gesture 4 in Fig. 1), the patients moved their hand to the mouth with the palmar fingers covering the lips. In minor parapraxias, the patient’s response resembled the demonstrated gestures but deviated in significant details. For example, in gesture 4, the hand was moved toward the chin, and was oriented correctly, but the fingers were in front of the chin with the tips touching the lips rather than the underside of the chin. The number of spatial parapraxias in LBD patients ranged from zero to the maximum of 23 (see Table 1). Taking the highest incidence of parapraxias in the RBD group as a cut-off to define apraxia, the seven LBD patients with zero to three apraxic errors would have been classified as non-apraxic (Cases 1–7) and the 13 LBD patients with more than three apraxic errors as apraxic (Cases 8–20). It is apparent from Fig. 1 that the number of parapraxias increased with increasing size of the lesion. Apraxia was found in patients with parietal, frontal, temporal, subcortical and basal ganglia lesions. However, none of the areas was specifically related to apraxia (Fig. 1). Table 1 shows that five out of a total of six LBD patients with severe global aphasia were among those seven patients with the highest frequency of parapraxias. Therefore, LBD patients with a high frequency of apraxic errors had on the average more severe aphasia and larger lesions than LBD patients with few apraxic errors (Kertesz and Ferro, 1984; Alexander et al., 1992).

**Results**

Analysis of results will proceed in four steps. (i) We will consider the number of spatial parapraxias; (ii) we will describe normal and abnormal movement kinematics in order to derive parameters for their statistical analysis; (iii) we will statistically compare movement parameters between the groups (controls, LBD, and RBD patients); (iv) we will investigate the relationships between kinematic abnormalities and parapraxias.

**Spatial parapraxias**

Out of 20 controls, 18 reached the correct final position in all of the 23 trials and two made one spatial error. Seven out of eight RBD patients performed in the range of controls; one was slightly worse. By contrast, spatial parapraxias were frequently observed in LBD patients. Some parapraxias resulted in gestures grossly different from the examiner’s demonstrated gestures. For example, instead of placing the fingertips under the chin with the ulnar side of the hand oriented anteriorly (gesture 4 in Fig. 1), the patients moved their hand to the mouth with the palmar fingers covering the lips. In minor parapraxias, the patient’s response resembled the demonstrated gestures but deviated in significant details. For example, in gesture 4, the hand was moved toward the chin, and was oriented correctly, but the fingers were in front of the chin with the tips touching the lips rather than the underside of the chin. The number of spatial parapraxias in LBD patients ranged from zero to the maximum of 23 (see Table 1). Taking the highest incidence of parapraxias in the RBD group as a cut-off to define apraxia, the seven LBD patients with zero to three apraxic errors would have been classified as non-apraxic (Cases 1–7) and the 13 LBD patients with more than three apraxic errors as apraxic (Cases 8–20). It is apparent from Fig. 1 that the number of parapraxias increased with increasing size of the lesion. Apraxia was found in patients with parietal, frontal, temporal, subcortical and basal ganglia lesions. However, none of the areas was specifically related to apraxia (Fig. 1). Table 1 shows that five out of a total of six LBD patients with severe global aphasia were among those seven patients with the highest frequency of parapraxias. Therefore, LBD patients with a high frequency of apraxic errors had on the average more severe aphasia and larger lesions than LBD patients with few apraxic errors (Kertesz and Ferro, 1984; Alexander et al., 1992).

**Description of movement kinematics**

The typical performance of a control subject is shown in Fig. 2. The trajectories of the hand movements were smooth without abrupt changes in direction. According to the different final positions required, the trajectories ended at different heights. In all final positions the two markers were finally located on either an approximately vertical (gestures 1, 4, 5 and 6) or horizontal axis (gestures 2 and 3). The tangential velocity profile of the wrist had a single peak and was bell-shaped. The velocity minimum which terminated the transport phase occurred only when the hand was already very close to its final position and was followed by only a short phase of slow adjustment.

The peak velocity of the transport phase varied between movements. Generally, it increased with increasing amplitude of the movement. The scaling of movement velocity with movement amplitude was significant for each single control subject with correlation coefficients between $r = 0.71$ and $r = 0.97$. The mean peak velocity of all movements varied between control subjects from 705 to 1493 mm s$^{-1}$ (mean 1050 mm s$^{-1}$). The mean percentage time to peak velocity ranged from 35.3 to 48.5% (mean 41.1%), indicating a tendency of the controls to spend more time in deceleration than in acceleration of the transport movement. Individual means of movement duration ranged from 641 to 1162 ms (mean 836 ms) and thus exhibited a variability comparable to that of peak velocity.

While peak velocity and movement duration varied, the number of velocity peaks remained virtually invariant. All except seven (2.1%) transport movements of the control subjects had only one velocity peak (see Fig. 3A). A multi-peaked movement never occurred more than once in controls. In controls, the individual means of the path length travelled by the wrist during the adjustment phase ranged from 3.3 to 23.7 mm (mean 11.8). As obvious from Fig. 3B, the pooled path lengths were not normally distributed due to the boundary effect of zero length. Thus, most transport movements of control subjects reached the final position either without any, or with only minor, adjustment.

Figure 4 provides selected examples of movement kinematics during the imitation of gesture 1 by LBD patients. The movements presented in the upper row show abnormalities of the transport movement. In contrast to the smoothly curved trajectories of the control subject (Fig. 2), the trajectories of the LBD patients show repeated changes in the direction of movement. The corresponding velocity profiles have
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Fig. 3 Frequency of the number of velocity peaks during the transport phase (A) and the path length travelled by the wrist during the adjustment phase (B) for the pooled movements of 20 control subjects. The broken line indicates the cut-off defined to categorize movement kinematics (see below).

multiple velocity peaks. Mainly in the example displayed on the left (Case 5), there was a profound slowing of the movement. Velocity profiles like these suggest an awkward and hesitant transport phase. By contrast, in the two examples in the lower row the velocity profiles of the transport phases were smooth and single-peaked, but the adjustment phase was prolonged. In the example displayed at the left (Case 12) the end of the transport phase was followed by low velocity adjustments of the hand which eventually led to a late corrective movement. No discrete corrective movement was apparent in the other example (Case 9) but the length of the path travelled during adjustment clearly exceeded the values found in control subjects.

In order to characterize the kinematics of the patients’ movements, the transport and the adjustment phase of movements were classified on the basis of the performance of control subjects. As peak velocity and duration of transport varied substantially within and between the control subjects, the transport phase was categorized solely on whether it was single-peaked or not (Fig. 3A). In order to categorize the path length of the adjustment phase, the 95% percentile of the non-Gaussian distribution of the control values was taken as the cut-off. It was 30.0 mm (Fig. 3B). Higher values were considered to indicate prolonged adjustments.

Comparison of kinematic parameters between groups

Figure 5 summarizes the results from the analysis of movement kinematics in the control subjects and the patients.

None of the parameters used to characterize the transport and adjustment phase yielded a statistically significant difference between the two control groups who performed with the left and right arm (Student’s t test and Mann-Whitney U test, P > 0.1). On average, controls performed 92.7% (SD 7.6%) of the gestures with only one velocity peak in the transport phase and <30 mm of final adjustment. The mean frequency of kinematically equally perfect movements in RBD patients was 86.2% (SD 14.0%). By contrast, only 42.5% (SD 32.5%) of the movements of LBD patients had perfect kinematics. In Fig. 5, kinematically abnormal movements which showed a prolonged adjustment phase but had a normal transport phase were distinguished from movements that deteriorated earlier during the initial transport yielding an irregular, multi-peaked velocity profile. Within the LBD group both patterns revealed similar frequencies (abnormalities restricted to adjustment: mean 30.2%, SD 26.3%; multi-peaked transport: mean 27.2%, SD 23.6%) which were much higher than the corresponding frequencies in controls and RBD patients (Fig. 5).

Separate group comparisons were performed for the
Fig. 5 Frequencies of different kinematic movement characteristics during the imitation of meaningless gestures in control subjects, in patients with LBD and in patients RBD. Open bars = single-peaked velocity profile during hand transport and adjustment path lengths below cut-off; stippled bars = transport single-peaked, but adjustment prolonged; filled bars = transport multi-peaked, irrespective of the adjustment. Within each group subjects are ordered by increasing number of spatial parapraxias (see Table 1). In each subject a total of 18 movements was recorded; because not all movements could be analysed (see Methods) percentages, rather than absolute numbers are indicated on the vertical axis.

Relationship between movement kinematics and final position

In Fig. 6 the number of spatial parapraxias is set in relation to the kinematics of movement execution in the LBD patients. Taking both measures to define a cut-off, only two out of the 20 LBD patients (10%) performed like controls, and a total of four LBD patients (20%) performed in the range of RBD patients. The LBD patients differed from both other groups having higher frequencies of both spatial parapraxias and kinematic abnormalities. This might be due to a coupling of kinematic deteriorations and apraxic errors in LBD patients; however, this was not true for the within-group analysis of LBD patients (Fig. 6). There was no significant correlation between the number of parapraxias and percentage of kinematically normal movements within the LBD group ($r = 0.15, P > 0.2$).
individual LBD patients. About one-third (32.2%) of the movements were perfect with respect to both the execution and the final position. Degraded kinematics were associated with correct final positions in 35.9% of the movements and with apraxic errors in 22.0% of them, whereas 9.8% of movements were kinematically normal but ended in an apraxic error. The latter dissociation was the prevalent type of performance in two patients with severe apraxia (Cases 18 and 20, see Fig. 7 and Table 1) and it was found in a significant portion of movements (16.7%) in two further apraxic patients (Cases 15 and 16).

Fig. 7 Movement kinematics and accuracy of final position of the gestures performed by LBD patients. The bars denote the different combination of both performance aspects (see inset; minus = deteriorated kinematics and wrong final position; plus = normal kinematics and correct final position). Patients are ordered by increasing number of parapraxias (see Table 1).

The transport phases of those gestures of Case 18 which showed a dissociation of normal kinematics from apraxic errors were submitted to a closer analysis. Their mean movement duration was 791 ms and the mean peak velocity 1094 mm s⁻¹. The percentage of time to peak velocity was on average 41.6% of total movement duration. The peak velocity correlated strongly with the amplitude of the movement (r = 0.83). Thus, in Case 18, the movements which led to spatial parapraxias were perfect with respect to all of the kinematic measures derived from the control subjects. Case 20 performed somewhat slower than Case 18, but the mean movement duration (996 ms) and mean peak velocity (837 mm s⁻¹) of the 14 movements were clearly within the normal range. The percentage of time to peak velocity was 53.9%, indicating that he spent relatively less time on deceleration than control subjects. The correlation between peak velocity and movement amplitude in Case 20 was positive, but low and not statistically significant (r = 0.28, P > 0.1). As in Case 15 (see above), the latter finding has to be interpreted with care, because the wrong final positions were all located close together, resulting in low variations of movement amplitude (range only 177 mm).

Discussion

The kinematic analysis yielded evidence that control subjects are able to preprogram motor execution in the imitation of meaningless gestures. The transport phases of their movements were single-peaked and adjustments or corrective movements were absent or very small. Single-peaked and approximately bell-shaped velocity profiles are considered as an invariant characteristic of skilled preprogrammed movements (Morasso, 1981; Flash and Hogan, 1985; Atkeson and Hollerbach, 1985; Kaminski and Gentile, 1989; Virji-Babul et al., 1994). The peak velocity of the movements had a strong positive correlation with the movement amplitude. The scaling of velocity with amplitude has been attributed to a planning strategy which simplifies movement control (Freund and Büdingen, 1978; Milner, 1986; Gordon and Ghez, 1987).

Multi-peaked transport movements and prolonged adjustments were rare in control subjects and RBD patients, but occurred frequently in LBD patients. This observation would be consistent with the hypothesis that motor programming is defective in LBD patients. However, the lack of a correlation between kinematic abnormalities and spatial parapraxias speaks against the assumption that deficient preprogramming is the cause of apraxic errors. Degraded kinematics were associated with correct final positions even more frequently than with apraxic errors. Furthermore, there were apraxic patients in whom kinematic analysis did not show evidence of insufficient motor programming: in four patients, kinematic abnormalities were largely confined to a prolongation of the adjustment phase, whereas the transport phase showed the profile of a normally preprogrammed movement; in four other patients movements that ended in apraxic errors showed completely normal kinetic profiles.

There are two possible ways to account for the greater frequency of kinematic abnormalities in apraxic patients as well as for the dissociations between kinematic abnormalities and apraxic errors. One possible explanation would be that insufficient motor preprogramming of target directed movements and errors in the determination of the correct target position are two independent sequels of left hemisphere lesions. One of them leads to kinematic abnormalities, and the other to parapraxias. As both disturbances are sequels of left hemisphere lesions, they occur in the same group of patients, but as they are in principle independent they may dissociate in individual patients.

A specialized role of the left hemisphere in motor programming has been suggested by studies investigating tasks such as pointing to an external target or repetitive aiming between two targets (Fisk and Goodale, 1988; Haaland and Harrington, 1989; Haaland and Harrington, 1994; Weinstein and Pool, 1995). They revealed specific kinematic abnormalities in both nonapraxic and apraxic LBD patients. These abnormalities, however, appear to be more discrete than the gross aberrations of the kinematic profiles that we...
found in some of the patients (e.g. Fig. 4, LBD 5). Moreover, apraxic patients in whom the majority of movements showed signs of deficient preprogramming executed some imitations with kinematic characteristics that were comparable to those of control subjects (Fig. 5). Given the similarity of the gestures tested, a general and stable impairment of the capacity to preprogramme movements seems unlikely in these patients.

If the co-occurrence of parapraxias and kinematic abnormalities were due to simultaneous lesions in two different left hemisphere regions one would expect the CT scans to indicate two distinct locations of lesions to be associated with each of these symptoms. Consideration of the CT scans (Fig. 1) does not meet this expectation. Patients who often showed a dissociation of abnormal kinematics from correct final position (Cases 5, 7, 8 and 9 in Fig. 7) displayed lesions in the temporal lobe, parietal lobe, subcortical white matter and basal ganglia and no common lesion area was evident (Fig. 1). All of these areas were affected in Case 20 who had a particularly large lesion (Fig. 1); this patient, however, showed a clear dissociation of normal kinematics from frequent apraxic errors. In accord with previous studies of lesion location in apraxia (Basso et al., 1980, 1985; Kertesz and Ferro, 1984) parapraxias were found after frontal, parietal and subcortical lesions.

An alternative explanation could start from the assumption that left hemisphere lesions affect only the mental representation of the requested target position. Some LBD patients react to this insecurity by switching to a strategy of hesitant and on-line controlled movements. This strategy leads to multi-peaked transport phases. Other patients programme and execute a movement into the vicinity of the target. Having arrived there, they try to find the exact target position with further slow corrective movements. The resulting movement has the kinematic characteristics of adequately preprogrammed transport phase followed by prolonged adjustments and corrective movements (see Figs 4 and 5). Both of these strategies can be successful and lead to a dissociation of abnormal kinematics from correct end-position. Finally there are patients who are not aware of the deficiency of their mental representation of the target position. They perform kinematically normal movement to inaccurate or wrong target positions and thus produce a dissociation of normal kinematics from apraxic errors. The anatomy of the lesions gives support to the idea that kinematic abnormalities resulted from a choice of strategies for coping with apraxia rather than being a genuine expression of apraxia itself. The two patients who made kinematically normal movements to wrong target positions were the only ones in whom the lesions encroached upon prefrontal cortex (Fig. 1). The frontal extensions of the lesions in all other patients were confined to premotor cortex. While premotor cortex subserves programming and recognition of motor action (Freund and Hummelsheim, 1985; Fadiga et al., 1995; Jeannerod et al., 1995), prefrontal cortex is generally recognized as playing a major role in self-control of cognition and action, and in selection of strategies for problem solving and trouble shooting (Damasio, 1979; Luria, 1980; Norman and Shallice, 1986). Prefrontal lesions may reduce awareness of deficits as well as the ability to apply compensatory strategies for overcoming them (Stuss, 1991).

In conclusion, kinematic abnormalities in apraxic movements are not restricted to meaningful gestures (Poizner et al., 1990; Clark et al., 1994; Poizner et al., 1995), but are also apparent when unfamiliar meaningless gestures are imitated by apraxic patients. Kinematic abnormalities, however, are not correlated with parapraxic errors. Instead of hypothesizing two distinct disturbances, one responsible for kinematic deteriorations and another for apraxic errors, we suggest that the basic deficit concerned the representation of the target. When confronted with such a deficiency, the patients may or may not choose to compensate their inadequate representation of the target position. If they try to compensate, their movement kinematics will deteriorate but they may nevertheless eventually succeed in reaching the correct target position. If they do not succeed, apraxic errors in addition to kinematically degraded movements result. Finally, if they do not try to compensate for the loss of an adequate representation they can execute movements as well as controls, but whether or not they hit the intended target position becomes a matter of chance.

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References


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