

NOTE

ON CROSSED APRAXIA. DESCRIPTION OF A RIGHT-HANDED APRAXIC PATIENT WITH RIGHT SUPPLEMENTARY MOTOR AREA DAMAGE

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ABSTRACT

GP, a right-handed woman, without evidence of familial left-handedness, showed clear-cut bilateral ideo-motor apraxia and oro-facial apraxia after a vascular lesion of the right hemisphere, encroaching upon the fronto-mesial region. She scored normally in most other cognitive tests, including language, but showed signs of callosal disconnection, left anarchic hand and mild unilateral spatial neglect. This cognitive profile points to the possibility of praxis being localized to the right hemisphere in this right-handed patient.

We argue in favour of individual variability of praxis dominance, and maintain that this dominance might be completely right-sided in some subjects. Moreover the anatomical locus of GP's lesion points to the possible role that the frontal lobes (and more specifically the Supplementary Motor Area) play in the genesis of apraxia.

INTRODUCTION

Apraxia is generally defined by default as a disorder in the purposeful execution of movements which cannot be explained by elementary motor or sensory deficits or cognitive impairment (De Renzi, 1989). Usually it is due to a lesion in the parietal lobe of the dominant hemisphere, i.e. the left in right-handers. A possible role of the right hemisphere and of different cortical regions (i.e. pre-motor cortex) in the genesis of apraxic deficits has been only seldom considered (e.g. Foix, 1916) and is still a matter of debate (Faglioni and Basso, 1985; De Renzi, 1989). We report the case of a right-handed patient who, after a right fronto-mesial lesion, presented with bilateral apraxia.

CASE REPORT

GP is a right-handed woman, with five years of formal education, who suffered a subarachnoid haemorrhage due to the rupture of an aneurysm of the right posterior communicating artery. When we first saw her, in 1992, she was 63 years old.

Neurological and Neuroradiological Findings

GP showed left hemiparesis immediately after surgery to clip her aneurysm; her leg was weaker than her arm. When we first saw her (eight years after the accident), she had very mild left hyposthenia noticeable in the lower limb only. No other sign of pyramidal lesion was present: in particular the tendon reflexes were symmetrical and the plantar reflex was bilaterally flexor. There was no ataxia. No cortical disinhibition sign (Tweedy, Reding, Garcia



Fig. 1 – GP's CT scan showing a right mesio-frontal stroke (see text).

et al., 1982) could be elicited, including the grasp reflex (De Renzi and Barbieri, 1992). However, she showed some of the dysexecutive behavioural features of the frontal syndrome. For instance, she punctuated her speech with jokes (Witzelsncht, Oppenheim, 1889) and her behaviour was somewhat disinhibited.

A CT scan (Figure 1), performed at the time we first saw the patient, showed a right mesial frontal hypodensity encroaching upon the Supplementary Motor Area (SMA) which also involved the genu and anterior part of the body of the corpus callosum. In the opinion of the neuroradiologists, the lesion was probably caused by the surgical passage to reach and clip the aneurysm. Another small area of hypodensity, interpreted as the sequela of a previous stroke, was revealed in the right occipital lobe. No abnormalities were detected in the left hemisphere. This CT did not differ from the patient's previous scans. It was not possible to perform a MRI because of the metal clips used to clamp the aneurysm.

A SPECT scan performed with HMPAO-99mTc revealed hypoperfusion in the right frontal lobe, in the anterior portion of the right temporal lobe and, to a lesser extent, in the right occipital lobe. No alteration was detected in the left hemisphere.

The possible lesion to the right SMA was supported by the *Bereitschaftspotential*. Potentials were recorded by averaging 2-s epochs of EEG preceding 100 flexion movements of the elbow of each arm from electrodes at C3', C4', Fz, FPZ (10-20 system) referenced to linked ears. A negative wave (*Bereitschaftspotential*) preceded the movement on both sides in all recordings. However, comparison of the left and right recording revealed that the mean negativity during the last 100 ms. prior to the onset of the movement was significantly lower, at all electrodes, for left movements. This condition has been claimed to be associated with a lesion in the contralateral SMA (Deecke, Lang, Heller et al., 1987).

General Neuropsychological Examination

GP was fully oriented and co-operative, although her behaviour during the examination was somewhat disinhibited. Her speech was verbose and punctuated by jokes often inappropriate to the context.

She said that she had always been right-handed and denied consanguinity for left-

TABLE I
CP's Performance in Neuropsychological Tests and Relevant Normative Data

	GP's adjusted scores	Controls' inferential 5th centile	Controls' median score
Control functions			
Selective Attention			
Digit Cancellation test (0-50)	28.2	23.9	40.5
Gottschaldt's Hidden Figures (0-34)	7.75*	16.75	29.50
Intelligence and executive functions			
Verbal Judgements (0-60)	57.5	32	50
Raven Progressive Matrices set A, Ab, B (0-36)	21.5	17.5	31
Weigl Sorting Test (0-15)	9.75	4.25	10.75
Elithorn Perceptual Maze (0-16)	11.5	7.25	14.25
Towers of London (0-18.80)	18.80	9	18
Instrumental functions			
Memory			
Verbal Forward Span (0-inf.)	4.25	2.75	4.50
Spatial Forward Span (0-10)	2.50*	3.50	4.75
Supra-Span Verbal Learning Buschke-Fuld's Selective Reminding Technique (0-180)	159	36	111
Supra-Span Spatial Learning (Corsi's Blocks) (0-29.16)	7.09	6.50	20
Prose Memory (0-150)	76	24	60
Language			
Token Test (0-36)	30.5	26.25	33
Spatial cognition			
Line Orientation Judgement (0-30)	26	17	25
Constructional Apraxia (0-14)	11.25	7.75	12.50
Visual perception			
Discrimination of segments length (0-32)	23	17	26
Face Recognition test (0-54)	40	37	46

Note: All scores have been adjusted for age, years of schooling and sex. The tests mentioned are described in Spinnler and Tognoni (1987) except for Digit Cancellation (Della Sala et al., 1992), Gottschaldt's Hidden Figures (Capitani et al., 1988), Raven Progressive Matrices (Basso et al., 1987), Towers of London (Allamano et al., 1987), Prose Memory (Capitani et al., 1994), Line Orientation Judgement (Benton et al., 1978) and Face Recognition (Benton et al., 1968).

* Pathological score, i.e. below the 5th centile of the normal distribution.

handedness, a datum confirmed by her husband and close relatives. She scored 100% right-handed on the Edinburgh Handedness questionnaire (Oldfield, 1971). She also stated that she had not changed hands for any of the everyday tasks since her operation.

The neuropsychological examination excluded global deterioration, as demonstrated by her performance well above the normality threshold on a screening test for general deterioration, the MODA (Brazzelli, Capitani, Della Sala et al., 1994), as well as on tests of verbal and non-verbal intelligence and on most attentional tasks (see Table I). All GP's scores were adjusted for age, education and, where necessary, gender and they were compared with norms obtained from healthy controls (Spinnler and Tognoni, 1987). She scored below the cut-off (i.e. the inferential 5th centile of the normal population) only on the Gottschaldt's Hidden Figures Test (Capitani, Della Sala, Lucchelli et al., 1988), which is considered to be sensitive to frontal damage (Yacorynksy and Davis, 1945). She also performed consistently below the cut-off on a task testing spatial short term memory span, the Corsi Blocks (Milner, 1971).

Spontaneous language was normal: neither dysprosody nor aphasic errors emerged during informal conversation. GP's performance on formal tests of language abilities (see Table I) as well as on a standard language examination, the Aachener Aphasia Test (De Bleser, Bayer and Luzzatti, 1987; Luzzatti, Willmes, Bisiacchi et al., 1987), allowed us to exclude aphasic deficits. The patient and her relatives denied language difficulty even in the early post stroke period and immediately after surgery; in the medical records there was no mention of any language problem throughout the period of hospitalisation.

The patient performed flawlessly on the Albert's test for unilateral visual neglect (Albert, 1973), though she began her cancellation on the right side of the page. The analysis of her errors on the Raven Progressive Matrices (Basso, Capitani and Laiacona, 1987) did not reveal any position preference (Costa, Vaughan, Horwitz et al., 1969). On the other hand (see Halligan and Marshall, 1992) in a line bisection task (Halligan and Marshall, 1989) GP made several errors with a constantly right-sided bias and, when requested to draw a clock from memory (Bisiacchi, Capitani, Luzzatti et al., 1981), she detailed only the right half of the clock-face.

GP was also tested with tasks investigating callosal disconnection. She showed tactile anomia with her left hand (1/10 correct responses with the left *versus* 10/10 with the right). This failure was not due to defective manipulation with her left hand because she subsequently recognised all the objects when allowed to palpate each of them and at the same time see the items. In the absence of visual control, she also had problems in transferring the kinesthetic information of a given posture from one hand to the other (2/10 from left to right and 0/10 from right to left). The severe apraxia of her left hand impeded the execution of writing and drawing tasks with this hand. In conclusion, GP performed normally in most of the neuropsychological domains, including language. However, she showed mild unilateral visuospatial neglect and signs of callosal disconnection. Her motor behaviour and her performance on tests of praxis are described separately (see below).

Motor Behaviour

Although her left hyposthenia was very mild, GP's difficulty to initiate voluntary movement in her left limbs was paramount, and her movements were rather clumsy. We interpret this disability as motor neglect (Critchley, 1953), a phenomenon that has been observed in patients with frontal lesions (Laplante, Talairach, Meininger et al., 1977). Even on verbal command she was unable to initiate and carry out correct movements with either the arm or the leg. On the contrary, unprompted gestures of the left hand, for example during conversation, were florid and spontaneous; automatic "defence" gestures, such as the act of shielding herself from a thrown object, were absolutely normal, and typically executed with her left hand. Moreover, a slight alteration of her upright balance provoked by imperceptible modifications of the position of a posturography board, invariably produced an attempt to regain balance by grasping, with her left hand, any accessible support even if she was told not to do so.

GP also reported that when she grasped an object with her left hand, she found herself unable to release her hold. This appeared to be particularly frequent with the supports she happened to use in *mise-en-train* motions, such as the arm of a chair or the foot of the bed, that she tended to drag along in her walk. This puzzled her husband and the therapists, who could not come to terms with the astonishing divergence between her ability to criticise her own behaviour and her inability to control it.

GP frequently showed involuntary, repetitive movements (i.e. motor perseverations) with her left hand, and at times performed complex actions that were clearly goal directed and well executed but unintended (i.e. 'anarchic hand'). While resting, GP tended to hold the left hand on her lap in order to minimize these unwanted actions. She never doubted that her left wayward hand was a part of her own body, but tended to anthropomorphise it ("see what 'she' does") as if it had a will of its own, which sometimes conflicted with the patient's will. For example, the patient told us that on one occasion, at dinner with friends and relatives, much to her embarrassment and others' dismay, her left hand grabbed the ice-cream that one of the guests was eating, unable to stop herself, at the same time complaining that this was not what she wanted to do. Such behaviour has been dubbed 'alien hand' (Goldberg, Mayer

and Toglia, 1981), or 'anarchic hand' (Della Sala, Marchetti and Spinnler, 1991), and is considered to be a sign of a fronto-mesial lesion (Goldberg, 1985; Della Sala, Marchetti and Spinnler, 1994). This (and not apraxia) was the symptom that most distressed the patient in her everyday life, and she described how she would talk to her "bad" left hand which "would not obey", to try to influence it. No difficulty in daily life was apparent with the right hand. GP's anarchic hand has been described in some detail elsewhere (see Della Sala et al., 1994).

Praxis Examination

On a standard test for ideomotor apraxia (De Renzi, Motti and Nichelli, 1980), GP performed below the cut-off both with her right and left hand (see Table II) in two consecutive sessions. This task is a 24-item imitation test which consists of half symbolic and half non symbolic intransitive movements, half involving independent finger movements and half whole hand/arm movements, half consisting in the holding of a static position and half requiring the execution of motor sequences. The score for each item ranges from 3 to 1 depending on whether the performance is flawless on the first, second or third attempt. A score of zero is given when the performance is unsatisfactory all three times. Therefore the total score ranges from 0 to 72. The cut-off point (i.e. 53) corresponds to the worst score found in a group of 187 controls (De Renzi, Faglioni, Scarpa et al., 1986) and represents the 95% outer tolerance limit of the normal population. A detailed error analysis reveals that GP's apraxia was far more severe with movements or postures of the fingers than of the whole hand or arm (see Table II). On this imitation task, she performed more poorly on sequences than on postures, but no difference emerged between symbolic and non-symbolic gestures.

To investigate GP's praxic abilities with transitive gestures we asked her to mime, first with one hand and then with the other, the use of objects on verbal command, on visual presentation, on imitation, and finally asked her to use real objects. The same ten objects were employed in all four conditions. The score ranged from 0 to 3 for each item: a score of 3 indicated a faultless performance on the first attempt, scores of 2 and 1 were given when it was necessary to repeat the performance once or twice respectively, while a score of zero was given when the patient did not perform satisfactorily even on the third attempt. As earlier tests showed, also in this transitive gestures task, GP's apraxia was more severe in her left hand, i.e. the patient was systematically unable to mime, with this hand, the use of common objects on verbal command (score: 1/30), her performance did not improve with visual presentation (0/30) nor on imitation (0/30). The actual use of real objects improved the score considerably (11/30), although GP's performance remained severely impaired. With her right hand, GP performed at ceiling with real objects, and scored 22/30, 16/30 and 18/30 on verbal command, visual presentation and on imitation respectively. Normal controls

TABLE II
GP's Performance on De Renzi et al.'s (1980) Ideo-motor Apraxia Test

	1st assessment		2nd assessment (interval: 22 months)	
	Right hand	Left hand	Right hand	Left hand
Total score (0-72)	38	10	35	5
Section scores				
Arm-Hand gestures (0-36)	26	8	24	2
Finger gestures (0-36)	12	2	11	3
Significant (0-36)	19	5	19	2
Non significant (0-36)	19	5	16	3
Postures (0-36)	25	10	24	4
Sequences (0-36)	13	0	11	1

Note: The cut-off score (total score) derived from the norms (De Renzi et al., 1986) is 53 (see text). Following De Renzi et al. (1980), each gesture is scored three times to three different dichotomies (section scores).

perform virtually at ceiling on these kinds of task (Duffy and Duffy, 1981; De Renzi, Faglioni and Sorgato, 1982; Mozaz, Pena, Barraquer et al., 1993). When asked to imitate 10 complex positions using both hands (e.g. make a ring with the thumb and index of both hands and link them together) GP scored very poorly (6/30).

All the tasks described above were video-taped and scored independently by two judges: in the case of disagreement, the higher score was given.

Summing up, GP failed all the tasks performed with her left hand. With her right hand she did well in the use of real objects but her performance in the pantomime tasks was far from flawless. In gesture imitation she achieved pathological scores with either hand, the left hand score being worse than the right hand one.

The discrepancy between GP's left and right hand performance cannot be attributed to motor neglect, because errors were not in initiating or producing a given movement, but in its spatial and temporal sequencing. The patient was perfectly aware of her failures, and tried very hard to achieve the required postures, making continuous adjustments to correct the mistaken spatial orientation of the fingers and hand.

Finally, the patient showed signs of oro-facial apraxia, scoring (14/20) below the cut-off (i.e. 16/20) on a standardised test (Spinnler and Tognoni, 1987). GP proved unable to whistle, blow a kiss, chatter her teeth, click her tongue or 'blow a raspberry'. On the other hand she managed to put out her tongue, lick her upper lip, pretend to yawn, puff and clear her throat. The oro-facial apraxia was confirmed on a second more extensive test (Della Sala, Spinnler and Venneri, in preparation) which contains 63 items. GP's age- and education-adjusted score was 77/126, a performance well below the cut-off score (i.e. 113) derived from 186 controls (mean score 120.6).

DISCUSSION

The patient (GP) whom we describe in this paper is, to the best of our knowledge, the first reported case of a right-handed patient showing bilateral apraxia following damage to the right mesio-frontal area encroaching upon the Supplementary Motor Area (SMA). Besides bilateral limb apraxia, GP presented with oro-facial apraxia, signs of callosal disconnection, anarchic hand (Goldberg et al., 1981; Della Sala et al., 1991, 1994), and mild left neglect, both in the visuo-spatial and motor domain, still present eight years after brain lesion. No other cognitive deficits emerged in the extensive neuropsychological investigation.

Hemispheric Dominance in Apraxia

GP showed 'crossed apraxia' but not crossed aphasia. This confirms a possible dissociation between praxis and language (Selnes, Rubens, Risse et al., 1982; Junqué, Litvan and Venderell, 1986; Della Sala, Basso, Laiacona et al., 1992; Papagno, Della Sala and Basso, 1993). Usually the dissociation between these two functions in 'crossed' patients is traced back to the migration of language, but not praxis, to the right hemisphere (Brown and Wilson, 1973; Heilman, Coyle, Gonyea et al., 1973; Alexander, Fischette and Fisher, 1989; reviews in Joannette, Puel, Nespoulous et al., 1982; Castro-Caldas, Confraria and Poppe, 1987). Instead, GP's cognitive profile and anatomical lesion suggest the possibility of a right hemisphere dominance for praxis and a left for language.

The complete left dominance for praxis in right handers has rarely been questioned (for a recent review see De Renzi, 1989). In fact it is generally assumed that manual preference and praxis control are undertaken by the same cerebral structures. Therefore, at least in right handers, the organisation of voluntary movement control is thought to be invariably managed by the hemisphere contralateral to the dominant hand (Liepmann, 1920; Goldstein, 1948; Brown and Wilson, 1973; Geschwind, 1975; Margolin, 1980; Jason, 1983a, 1983b; Castro-Caldas et al., 1987; Alexander et al., 1989). Praxis is said to be related to manual preference more strongly than is language (Brown and Code, 1987; Alexander et al., 1989; Selnes, Pestronk, Hart et al., 1991). This view is supported by the relative frequency of left-handed patients with a lesion in the right hemisphere who show apraxia but not aphasia (e.g. Heilman et al., 1973; Margolin, 1980). Accordingly, several right-handed patients showing apraxia

without aphasia after a left hemisphere lesion have been reported (Selnes et al., 1991; Heilman, Gonyea and Geschwind, 1974; Junqué et al., 1986). Cases have also been reported of right handed patients with a lesion in the corpus callosum whose performance pointed to a left dominance for praxis and a right dominance for language (Tanaka, Iwasa and Yoshida, 1990).

In his review on crossed aphasia, Donoso (1984) excluded the possibility that crossed aphasics could also show apraxia (p. 137). Similarly, Junqué et al. (1986) maintained that praxis is the only cognitive function that does not "cross" to the other hemisphere: in support of this claim they reported the case of a right-handed patient who, following a lesion in the left hemisphere, presented with a typical right hemisphere cognitive profile and, at the same time, became apraxic but not aphasic. This patient, therefore, had all the right (impaired) and left (unimpaired) cognitive functions crossed, except for praxis. A very similar cognitive profile was shown by two out of the four patients reported by Fisher, Alexander, Gabriel et al. (1991).

Notwithstanding the bulk of evidence quoted above, which testifies to a strict connection between praxic dominance and manual preference, some data exist which call into question the rule of a rigid association between apraxia and left hemisphere lesion in right-handers. In fact, cases can be gleaned from the literature of crossed aphasics who also showed apraxia (Fernandez-Martin, Martinez-Lage, Madoz et al., 1968; Assal, Perentes and Deruaz, 1981; Henderson, 1983; Basso, Capitani, Laiacona et al., 1985, cases no. 3 and 5; Heilman, Gonzales-Rothi, Feinberg et al., 1986; Rapcsak, Gonzales-Rothi and Heilman, 1987; Reinvang, 1987; Berthier, Starkstein and Leiguarda, 1987; Perani, Papagno, Cappa et al., 1988, case no. 2). Castro-Caldas et al. (1987) countered that a certain degree of familiarity for left-handedness was present in some of these cases (e.g. Assal et al., 1981; Henderson 1983). They reported an association between aphasia and ideo-motor apraxia in 17% of the 66 cases of crosses aphasics they reviewed. Comparing this percentage with the much higher one (40%) reported in groups of left-hemisphere damaged aphasic patients (Kertesz, Ferro, Shewan et al., 1984), Castro-Caldas et al. (1987) maintained that the yoke between the mechanisms which subservise motor dominance and those which subservise limb praxis is stronger than that between praxis and language.

In the early literature there are reports of right-handed patients showing apraxia after a lesion of the right hemisphere (review in Hécaen and Gimeno-Alava, 1960). However, the clinical description of these cases is far from satisfactory according to Faglioni and Basso (1985) who maintain that the case Gl. described by von Monakow (1914, case XI) and subsequently by Brun (1921, case n. 7) is the only convincing right-handed patient showing bilateral apraxia, more severe in the left hand, following a massive right posterior haemorrhage. However, post-mortem this patient also showed a softening in the left occipital lobe and a cyst within the lateral margins of the left occipito-temporal gyrus, which was large enough to encroach upon the optic tract. Therefore, although occipital lesions do not usually result in apraxia, the possible role of the left hemisphere in the genesis of Gl.'s apraxia cannot be ruled out, as von Monakow himself acknowledged¹.

Recently, Leiguarda, Starkstein and Berthier (1989) reported the case (no. 3) of a right-handed patient who, after a lesion of the corpus callosum, presented with a clinical picture compatible with right dominance for language and praxis. He was apraxic with his right hand and showed aphasic agraphia with the same hand. This case is very similar to the famous *Regierungsrat* promulgated by Liepmann (1900), a right-handed patient who showed apraxia only with his right hand following a lesion that almost entirely destroyed his corpus callosum, thus pointing to a right hemisphere involvement in praxic control.

Heilman et al. (1986) described a patient who showed unilateral (left) apraxia after a lesion of the right superior and inferior parietal lobe. The patient was not strongly right-handed and the authors proposed that she might have bilateral representation of space-time engrams, so that each hemisphere programmed the skilled movements of the opposite hand.

¹ "Gleichwohl ist m. ER. die Moeglichkeit keineswegs ausgeschlossen, dass in diesem Falle der linksseitige Herd, welcher sich weiter nach vorn tief in das Mark des Gyr. occipitotemporalis erstreckte, bei dem Zustandekommen der bilateralen Apraxie eine nicht unwesentliche Rolle gespielt hat" (p. 531). [I believe that the possible role of the left-sided lesion, which extended ventrally into the white matter of the occipito-temporal gyrus, in the genesis of the patient's bilateral apraxia, cannot be ruled out].

In addition to these case reports, other arguments have been put forward to support the notion that the right hemisphere is not extraneous to the process of organising and programming voluntary gestures in right-handers. Lange (1936), for instance, maintained that clinical experience suggests that the most severe cases of apraxia are observed in patients with bilateral lesions. This is certainly true in slowly progressive apraxia of a degenerative nature (De Renzi, 1986, 1992; Rapcsak, Ochipa, Anderson et al., 1995; Habib, Alicherif, Balzamo et al., 1995; Della Sala and Spinnler, 1996). However, systematic group studies have not confirmed that apraxia associated with a focal bilateral lesion is more severe (Basso, Capitani, Della Sala et al., 1987a) or long-lasting (Basso, Capitani, Della Sala et al., 1987b) than that shown by unilaterally lesioned patients, once age, formal education, interval from stroke, and initial severity are controlled. Even patients with Alzheimer's Disease do not present apraxia as a constant feature and, at least in the early stages of the disease, the evolution of ideo-motor apraxia is rather slow (Della Sala, Lucchelli and Spinnler, 1987). Bi-hemispheric representation of praxis has also been proposed as an explanation for persistent aphasia coupled with a good recovery from apraxia (Kertesz et al., 1984). Interestingly, a similar argument, albeit in the opposite sense, was used by Selnes et al. (1982) who described a patient presenting with severe apraxia coupled with a mild, recovering aphasia and postulated a bilateral representation of language but unilateral (left) representation of praxis.

When formally tested, a certain degree of apraxia has indeed been reported in a number of right-brain damaged patients. De Renzi et al. (1980), for instance, found that 20% of their sample of 80 right-brain damaged patients scored below the cut-off point (62/72) on an ideo-motor praxis task in which the patients were asked to imitate gestures. However, if one uses, as later suggested by the same authors (De Renzi et al., 1982), a different, stricter, cut-off (SV72, i.e. the worst performance of normal controls) only three out of the 80 right-brain damaged patients would have been considered pathological, and their scores were not so bad either (45 is the worst of the three). These investigations were group studies and information on each single case is too scanty to allow any speculation on the possible differing characteristics of the outliers.

Haaland and Flaherty (1984) tested 41 left-brain damaged and 18 right-brain damaged patients with an imitation task made up of 15 gestures: 5 transitive (pretended object use), 5 intransitive but symbolic, and 5 intransitive and non symbolic. The left-brain damaged group differed significantly from the right only in the number of errors they made in the transitive gesture task. No difference emerged between the two groups in the performance of intransitive gestures, whether symbolic or non symbolic. According to these authors the known link between apraxia and left hemisphere is a bias due to the usual modality of testing apraxia: i.e. pantomimes of object use (transitive gestures only). It should be remarked, however, that most of Haaland and Flaherty's patients had perfect scores (ceiling effects) and that their findings are at variance with those of De Renzi et al. (1980). However, the hypothesis of a bilateral representation of intransitive gestures has been reiterated by Rapcsak, Ochipa, Beeson et al. (1993) who reported the case of a patient with a massive left hemisphere lesion who performed very poorly on transitive gestures regardless of the stimulus (verbal, visual, imitation), but was much less impaired when tested with intransitive, symbolic gestures.

GP also showed oro-facial apraxia. Apraxia of the oro-facial muscle system is maintained to be linked more to language deficits than to apraxia of the limbs (Brown and Code, 1987; Alexander et al., 1989). This argument is usually based on the evidence that oral apraxia is more common (about 50%) than limb apraxia (17%) in crossed aphasic patients (Castro-Caldas et al., 1987). This implies that oro-facial praxis is liable to migrate from one hemisphere to the other together with language. Contrary to this assumption, GP's cognitive profile points to a common dominance for oro-facial and limb praxis, both being dissociated from language. The presence of crossed oro-facial apraxia without aphasia has already been reported by Kramer, Delis and Nakada (1985) in a right-handed patient with a vascular lesion in the right postcentral and anterior parietal areas.

A Hypothesis for Crossed Apraxia

De Renzi (1989) maintained that "while the dominance of the left hemisphere in movement planning is likely to be the rule, the degree of its intensity is variable from subject

to subject and there can be cases of equipotentiality" (p. 248). GP's cognitive profile suggests the hypothesis that the continuum of such intersubject variability may have at one end a few subjects showing a complete crossed dominance for praxis. In such individuals a right-sided lesion produces a bilateral apraxia.

The Role of the Premotor Frontal Cortex

The role of the pre-motor areas in the genesis of apraxia was emphasised by the early authors (Liepmann, 1905; Kleist, 1906; Von Monakow, 1914; Lange, 1936) and has been more recently reappraised (Geschwind, 1965; Watson, Fleet, Gonzales-Rothi et al., 1986). However, the clinical evidence supporting this view is scanty and rather contradictory. Faglioni and Basso (1985) gathered 16 cases from the literature, nine of which are far from convincing because of the presence of other lesions that could account for the symptom. In four further cases the lesion extended well beyond the pre-motor areas. In the remaining three cases originally reported by Brun (1921, case no. 10) and Morlaas (1928, cases no. 15 and 16) the lesion was said to lie in the left pre-motor dorsal areas. The mesial aspects of the pre-frontal cortex were never encroached upon.

More recently, Watson et al. (1986) described two patients with a lesion in the left Supplementary Motor Area (SMA), who showed bilateral apraxia limited to the use of common objects, in the absence of oro-facial apraxia. Also Masdeu, Schoene and Funkenstein (1978), describing the language deficit (transcortical motor aphasia) of a right hemiparetic patient with a left SMA lesion, mentioned incidentally that the patient was unable to voluntarily execute simple movements with his left hand (and with the oro-facial muscles). Moreover, cerebral blood flow studies support the hypothesis of a SMA role in the genesis of motor activities (Roland, Larsen, Lassen et al., 1980).

These few positive cases are, however, counterbalanced by the numerous negative cases, reported in the literature. No overtly apraxic patient was found by Freund and Hummelshein (1985) in 13 patients with a unilateral lesion in the prefrontal areas, nor by Bogousslavsky and Regli (1990) in 27 patients with a vascular lesion in the territory of the anterior cerebral artery though one patient had left apraxia due to a callosal lesion. Moreover, patients affected by the so-called 'anarchic (alien) hand' syndrome following a lesion in the SMA of either side usually do not show bilateral apraxia (see review in Della Sala et al., 1994). The only exceptions are Watson et al.'s case no. 1 and the case that we report in this paper.

In De Renzi, Faglioni, Lodesani et al.'s (1983) study, five out of 13 frontal patients performed below the cut-off score in apraxia tests. However, their impairment was mild enough to allow the authors to conclude that the role of the pre-rolandic areas in motor planning is marginal. Similarly, Kolb and Milner (1981) tested groups of parietal and frontal patients with a motor sequence imitation task. They found apraxia in both groups, although in the frontal patients it was less severe and without the left hemisphere bias. The authors concluded that while their findings confirmed the left parietal dominance in the organisation of voluntary movements, they pointed to a less clear-cut asymmetry in the frontal lobe, at least for the imitation task used. Watson et al. (1986) agreed with this interpretation and, considering that the SMA influences the movements of the ipsilateral as well as the contralateral hand, suggested that the left dominance could be, at this level, less absolute, and, in a fleeting comment, did not exclude the possibility of finding apraxia after a lesion of the right SMA. However, this is in striking contrast with the absence of reports of right-handed patients who became apraxic after a right pre-frontal lesion. A direct consequence of the hypothesised contralateral redundancy is that apraxia following a frontal lesion would be less frequent and of shorter duration than that due to a posterior lesion. GP's apraxia, still quite severe eight years post onset, militates against the hypothesis that frontal apraxia is necessarily a transient symptom.

To the best of our knowledge, Watson et al. (1986) reported the only two cases of bilateral apraxia following a lesion in the left SMA. Only one of them (case n. 1) was assessed extensively. He differed in many respects from GP. First, he had great difficulty in miming transitive gestures on verbal command, particularly with the left hand, and did not show, as GP, bilateral (though more marked on the left) apraxia for symbolic and non symbolic intransitive gestures on imitation. His performance improved with the use of real

objects and became normal with the right hand. Moreover, execution of intransitive gestures was normal and he did not show oro-facial apraxia. GP also showed oro-facial apraxia. The more apraxic hand of Watson et al.'s patient was the left; this fact, together with the information that 13 months after onset apraxia was still present but only on the left (Verfaeille and Heilman, 1987), leaves us with the suspicion that his apraxia might be due to callosal lesion.

Watson et al. (1986) proposed that, taking for granted the role of the inferior parietal lobe as the locus of the spatial and temporal representation of transitive learned gestures (Heilman, Rothi and Valenstein, 1982), the SMA could be held responsible for the translation of these representations into motor programs. Our findings do not rule out this hypothesis in its general terms, but do not either support the idea that the role of the SMA is limited to the programming of transitive gestures. GP showed deficits in the execution of all kinds of gestures mainly but not exclusively on the left. This seems to suggest that the SMA is called into play in programming gestures of the two hands. It is perhaps foreseeable that each SMA, independently from the type of gesture, controls both upper limbs, the contralateral link being stronger. This hypothesis is in line with multifarious data indicating the involvement of both SMAs in the organisation of complex movements (Orgogozo, Larsen, Roland et al., 1979; Roland et al., 1980). Rapcsak et al. (1993) maintain that the right hemisphere may play some controlling role in left-hand automatized movements, but to a much lesser degree than the left (dominant) hemisphere. After a lesion of the left hemisphere or corpus callosum, this subdued right hemisphere praxic capacity could be released from inhibition and allowed fuller expression (Ibid., p. 197). This hypothesis could be reformulated to encompass the functions of the two SMAs. Accordingly, it is conceivable that in normal conditions one of the two SMAs (generally the left, but in our 'crossed' patient the right) is dominant and controls the gestures of both sides. Therefore, a lesion of the dominant SMA would leave the patient's gestures under the sole control of the subordinate SMA, giving rise to asymmetry between the performance of the ipsilateral and contralateral hand (as shown by GP).

In conclusion, we maintain that cases as GP do not falsify the classic view of a strict link between praxic control and hemispheric manual dominance (Liepmann, 1920; Goldstein, 1948; Brown and Wilson, 1973; Geschwind, 1975; Margolin, 1980; Jason, 1983a, 1983b; Castro-Caldas et al., 1987; Alexander et al., 1989). Rather, we maintain that exceptions to the rule, such as GP's apraxia localisation, are due to individual variability (De Renzi, 1989), which can go as far as a complete right-sided dominance. These exceptions are probably more likely to be found after lesions of the mesial frontal cortex, which is known to show less functional asymmetry between the two hemispheres (Kolb and Milner, 1981) and might be more frequent than previously thought.

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