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CT scan correlates of gesture recognition

JOSÉ M FERRO, ISABEL P MARTINS, GABRIELA MARIANO, A CASTRO CALDAS

From the Neurobehavioral Unit, Centro de Estudos Egas Moniz, Department of Neurology, Hospital de Santa Maria, Lisboa, Portugal

SUMMARY The ability to recognise gestures was studied in 65 left-hemispheric stroke patients whose lesions were located by CT scan. In the acute stage (first month) frontal lobe and basal ganglia were frequently involved in patients showing inability to recognise gestures. In the later (third to fourth month) and chronic stages (>6 months) parietal lobe involvement was important; lesions causing gesture recognition impairment were larger, had more extensive and frequent parietal involvement and produced less temporal lobe damage than those causing aural comprehension defects. These findings are discussed in the light of recent models of cerebral localisation of complex functions.

Several studies have demonstrated that some aphasics do not recognise gestures.[1–5] This disturbance correlates with comprehension impairment,[1,2,4] alexia[3,4] and constructional apraxia.[4] Gesture recognition impairment in aphasics has been attributed to asymbolia,[1] to an impairment of "semantic understanding",[2] to damage to a "symbolic unit" that relates gestures to the corresponding objects[4] following "plausability" rules,[5] or due to loss of visuokinesthetic motor engrams of gestures.[4]

None of these previous studies addressed the question of the anatomical localisation of the lesions that cause impairment of gesture identification. In a previous work, we found that this disturbance of gesture recognition was more common and severe in global, transcortical and Wernicke's aphasics, independent of the severity of auditory comprehension impairment.[4] This could reflect the major role of posterior left hemispheric areas for gesture identification. The strong correlation found with constructional apraxia could indicate a more extensive involvement of the parietal lobe in aphasic patients displaying defective gesture recognition. These suggestions were investigated in the present study.

Method

The study was of 65 adult right-handed patients, 41 males and 24 females (mean age 56·4 ± 11·6 years) who had suffered a single ischaemic cerebrovascular accident of the left hemisphere. Patients with intracerebral haematomas, cerebral infarct associated with subarachnoid haemorrhage and those in whom CT scan showed multiple or bilateral lesions, or diffuse white matter hypodensity suggestive of arteriosclerotic subcortical encephalopathy[6] were not included in this series. Thirty-two patients were tested during the 1st month after stroke (acute patients), 37 during the 3rd–4th months after stroke (recent patients) and 44 in the six or more months after stroke (chronic patients). Thirty-three patients were observed in more than one period (acute, recent and chronic stages, 18 patients; acute and recent, eight patients; recent and chronic, six patients; acute and chronic, one patient).

All patients had a comprehensive neuropsychological evaluation including testing for aphasia, alexia, agraphia, apraxia and constructional ability. Diagnosis of aphasia was carried out by means of a standardised battery.[4,7] Two tests of auditory comprehension deserve a brief description as their scores were compared with Gesture Recognition Test score. On the test of object identification by name, patients were requested to point to objects (two sets of eight) named by the examiner, while on the test of oral understanding they had to follow four one-step and four two-steps oral commands. Each correct response is scored one point in both tests. Normal controls made no errors on these tasks. Classification by aphasia types was based on taxonomic criteria according to the scores obtained in the tests of speech fluency, naming, understanding of oral commands and repetition of words.[4]

The ability to recognise gestures was assessed by means of the Gesture Recognition Test (GRT).[4] This is a multiple-choice test that requires the patient to point to 12 objects or drawings corresponding to gestures played by the examiner. This test is divided in two parts, preceded by two training items. The training items (spoon, comb) are placed in front of the patient. The examiner pantomimes

Address for reprint requests: José M Ferro MD, Neurobehavioral Unit, Centro de Estudos Egas Moniz, Hospital de Santa Maria, 1600 Lisboa, Portugal.

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their use and instructs the patient (verbally and gestually) to point to the corresponding object. This sequence is repeated twice if necessary. If the patient tries to reproduce the pantomime he is requested to make a pointing response. In part I, six objects are placed in front of the patient and six pantomimes are successively played (glass/drinking, cookie/eating, cigarette/smoking, ball-pen/writing, scissors/cutting, watch/looking at the time). In part II, six items (military salute, cross-sign, playing guitar, typewriting, phoning and blowing) and six different multiple choice cards, each composed of six drawings are presented. Each correct answer was scored one point. An imitation of the gesture was not considered a correct response. A score of 11, that is reached by 93·7% of the controls was used as cut-off score between normal and defective performances.[4] Moderate impairment was defined as a score ranging from 10 to 9, and below 9 points the performance was considered severely impaired, since 100% of the controls exceed this score.

Within 1 month of testing (1st evaluation) all patients had a CT scan. Although in a few cases other types of CT scanners were used, the majority of CT scans were performed with a Siemens Siretom 2000 (matrix 256 × 256) and a GE scanner using a 576 × 576 matrix. In general, CT slices were inclined at 15° to the canthomeatal line. Each slice was 10 mm thick. Using available anatomical and CT atlases,[8-12] the following structures were identified in each scan in order to check their involvement in the ischaemic lesion: prefrontal lobe, Broca's area, white matter deep to Broca's area, internal capsule, caudate and lenticular nucleus, external capsule and claustrum, corona radiata, pre- and post-central gyri, temporal pole, hippocampus, Heschl's gyrus, Wernicke's area, white matter deep to Wernicke's area, posterior and inferior temporal lobe (area 37), angular gyrus, white matter deep to angular gyrus, supramarginal gyrus, white matter deep to supramarginal gyrus, superior parietal lobe (area 7), primary visual area and associative visual areas.

1 The matrix of the CT scan lesion sites provided from the inspection of all individual scans was used to determine the areas that were more often involved in patients with gesture recognition impairment. Inferencial parametric statistic, utilising between-group *t* tests was used to identify loci related to the Gesture Recognition Test (GRT) (method of multiple dissociation for one variable).[13] A series of one-tailed *t* tests were performed for each of the identified anatomical structures, comparing GRT mean scores of subjects having a lesion in each particular area with subjects having the same area spared. It is then possible to obtain a profile of the "critical" lesion sites most likely related to the deficit at each stage of illness. As the population in each period was not the same, comparison of the incidence of impairment at each site, at the different stages of illness, was performed using the Chi-square and Fischer exact tests. This procedure confirms if different critical sites are responsible for gesture recognition impairment at different stages of illness.

2 The outline of the lesions was mapped on a standard lateral diagram of the brain using the method of Mazzocchi and Vignolo[14] and on cross-sectional templates in the so-called B, B + W, W, SM, SM + 1, SM + 2 and SM + 3 CT slices.[15] By overlapping individual outlines, countour composites and areas of greatest overlap were obtained.

3 Lesion size was measured by using a digitiser program to outline the tracings in each cut and adding the values to obtain a volumetric estimate of size.[16] Lesion size was expressed as a percentage of left hemispheric volume (ventricles excluded) obtained by the same procedure.

4 Three measures of diffuse cerebral atrophy were also obtained (measures were taken from the non-involved hemisphere).[17,18]

(1) Frontal/brain (F/B)-ratio of distance between lateralmost margins of lateral ventricular frontal horns and cortical margin-midline distance at the same level.
(2) Caudate/brain (C/B)-ratio of distance between lateral margins of frontal horns at head of caudate nucleus and cortical margin-midline distance at the same level.
(3) Subjective rating of cerebral atrophy using a four-category scale: normal, mild, moderate and severe.

Results

I GESTURE RECOGNITION, LESION LOCALISATION AND SIZE

Table 1 shows patients' diagnosis in the acute, recent and chronic stage and their performance on the Gesture Recognition Test. Gesture recognition disturbance was much more common in the acute (53%) and recent (35%) stages than in the chronic one (18%). Of the 17 acutely impaired patients, six out of 17 retested in the recent period were still disturbed on the Gesture Recognition Test. Ten recent-stage impaired patients were re-evaluated in the chronic stage. Only five remained impaired. Global, transcortical and Wernicke's aphasics were more often impaired than other patients. These results replicate our previous study.[4]

Acute patients

Table 2 indicates the more frequent sites of brain lesions associated with gesture recognition impairment and sites whose lesions produced significantly lower scores in the Gesture Recognition Test ($p < 0·05$). Subcortical fronto-central, basal ganglia and subcortical parieto-temporal areas appeared to be of particular importance in this period. This was confirmed by the lateral (fig 1) and cross-sectional (fig 2) composites of lesions of severe impaired patients. In the lateral composite the area of maximum overlap was in the frontal lobe (areas 44, 45, 46). Two other areas of overlap were located in the parietal lobe (areas 39, 40) and in the temporal lobe (areas 21, 22, 37).

Impaired and non-impaired patients were of similar age (58·1 ± 12·6 vs 53·3 ± 10·4 years, $t = 1·17$, ns), and their lesions did not differ in measures of cerebral atrophy, either in the subjective rating ($\chi^2 = 2·13$, ns), F/B ($t = 0·55$, ns) or C/B ($t = 0·09$, ns) ratios. Lesion size showed moderate negative corre-

Table 1 *Patients' diagnosis and GRT performance*

Diagnosis	*Acute period*	*GRT impaired*	*Recent period*	*GRT impaired*	*Chronic period*	*GRT impaired*
Global aphasia	11	10	7	4	8	2
Wernicke's aphasia	10	4	4	3	4	2
Transcortical aphasia						
Motor			5	2	3	
Mixed	2	2	1		1	1
Sensory	3	1	2	1	3	1
Broca's aphasia	1		4	1	3	1
Conduction aphasia	3				4	
Anomic aphasia			7	2	7	
Word deafness	1		1		1	
Dysarthria	1		1		1	
Alexia with agraphia					1	
Pure alexia			1		2	
Agrafia					1	
No verbal disturbance			3		4	1
Visual agnosia			1		1	
Total	32	17 (53%)	37	13 (35%)	44	8 (18%)

Table 2

More frequent sites of lesion (%) in GRT impaired patients		*Sites with lower GRT scores multiple dissociation method*		
		Acute patients		
			(*n*)	(*a*/*b*)
in >50% of impaired patients	Subc. Broca's area	Caudate N.	6/7	3·6/8·9
	Pre. postcentral gyr	Ant. limb. int. cap.	7/11	
	Corona radiata	Prefrontal lobe	6/9	4·6/8·7
	Lenticular N.	Broca's area	6/10	5·3/8·6
		Sub. Broca's area	8/13	5·4/9·1
>40%	Ant. limb. int. cap.	Corona radiata	9/15	5·5/9·2
	Ext. cap.	Sup. parietal lobe	4/4	4/8·2
	Insula			
	Subc. Wernicke's area			
	Subc. supramarg gyr			
		Recent patients		
			(*n*)	(*a*/*b*)
in >50% of impaired patients	Subc. angular gyr.	Sup. parietal lobe	5/5	6·4/10·6
	Corona radiata	Post. inf. temporal lobe	5/7	8·1/10·4
	Subc. Wernicke's area			
>40%	Angular gyr.			
	Supramarg gyr.			
		Chronic patients		
in >50% of impaired patients	Angular gyr.	No significant difference in any area		
	Subc. Wernicke's area			
	Lenticular N.			
>40%	Corona radiata			
	Subc. angular gyr.			
	Supramarg. gyr.			
	Subc. supramarg. gyr.			

(a) mean score of patients whose lesion involved the specified area.
(b) mean score of patients whose lesion did not involve the specified area.
(n) number of impaired patients/total number of patients whose lesion involved the specified area.

lation with Gesture Recognition Test scores (Pearson's $r = 48$, $p < 0{\cdot}01$). Impaired patient had larger lesions ($0{\cdot}108 \pm 0{\cdot}09$) than non-impaired ($0{\cdot}04 \pm 0{\cdot}03$) ($t = 2{\cdot}37$, $p < 0{\cdot}025$).

Recent patients

In recent patients the more frequent sites of lesion were mostly in the parietal lobe and sites with lower Gesture Recognition Test scores were parietal (area 7) and temporal (area 37) (table 2). In the lateral composite, all patients overlapped in the angular gyrus (fig 1). In cross-sectional composites, one of the areas of maximum overlap was also in the parietal lobe (fig 3). Comparison of the incidence of impairment at each anatomical site showed no significant difference between recent and acute patients. The degree of cerebral atrophy had no influence on Gesture Recognition Test performance (F/B, $t = 0{\cdot}03$, ns; C/B, $t = 0{\cdot}01$ ns; subjective rating $\chi^2 = 2{\cdot}20$, ns). Correlation between lesion size and Gesture Recognition Test scores was weak and non-significant ($r = -0{\cdot}26$). However, impaired patients

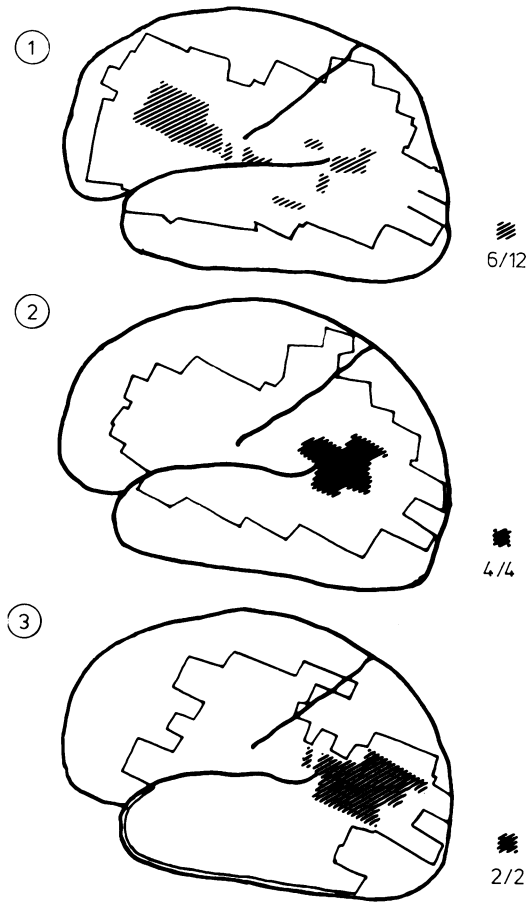

Fig 1 *Lateral composite contour of lesions and areas of maximum overlap in acute (1), recent (2) and chronic patients (3). Number of overlapping cases is indicated on the right of the diagrams.*

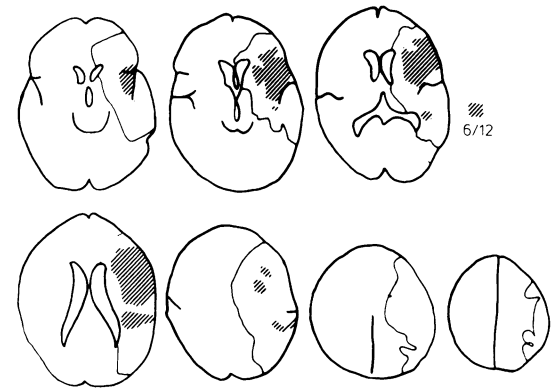

Fig 2 *Acute patients. Cross-sectional composite contour of lesions and areas of maximum overlap. Number of overlapping cases is indicated on the right of the diagram.*

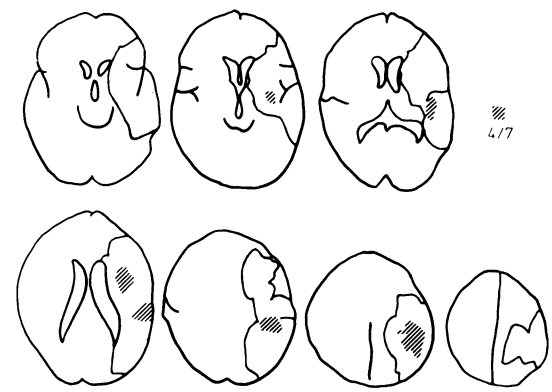

Fig 3 *Recent patients. Cross-sectional composite contour of lesions and areas of maximum overlap. Number of overlapping cases is indicated on the right of the diagram.*

had larger lesions (0·117 ± 0·08 vs 0·069 ± 0·07, t = 1·85, $p < 0·05$) and were older (impaired 62·3 ± 9·2 vs 50·8 ± 10·6 years, t = 2·53, $p < 0·01$) than non-impaired patients.

Chronic patients

There were few patients impaired on the Gesture Recognition Test in this stage. This may contribute to the failure of finding significantly lower Gesture Recognition Test scores with lesions of any specific area. However, results in this group were rather comparable with those of the recent group. More frequent sites of lesion (table 2) and areas of maximum overlap (fig 1) were located in the parietal lobe (areas 39, 40). In cross-sectional composites a subcortical frontal area of overlap was also found (fig 4). Comparison of the incidence of impairment at each anatomical site showed no significant differences between chronic and recent patients. Chronic impaired patients had less frequent caudate, ($p = 0·05$) insular ($\chi^2 = 7·77$, $p < 0·01$), corona radiata ($\chi^2 = 5·38$ $p < 0·025$) and subcortical supramarginal gyrus ($\chi^2 = 5·72$, $p < 0·025$) involvement than acute impaired patients. There was no influence of the degree of cerebral atrophy, but impaired patients were older than non-impaired (t = 3·01, $p < 0·001$). Differences in lesion size were nonsignificant (impaired patients = 0·132 ± 0·09, non impaired = 0·090 ± 0·09, t = 1·32, ns). Correlation between Gesture Recognition Test scores and lesion size was weak ($r = -0·30$), but significant ($p < 0·05$).

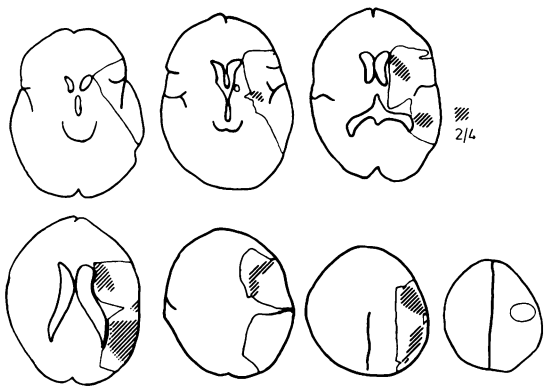

Fig 4 *Chronic patients. Cross-sectional composite contour of lesions and areas of maximum overlap. Number of overlapping cases is indicated on the right of the diagram.*

II GESTURE RECOGNITION AND LANGUAGE COMPREHENSION

Some of the areas whose lesions were associated with impairment on gesture recognition are also known to be important in the understanding of oral language. In order to separate the crucial areas for the recognition of gestures, from those crucial to language comprehension, we compared the lesion sites of patients having severe impaired Gesture Recognition Test scores and normal scores on the test of object identification by name, with those having normal Gesture Recognition Test scores and impaired performance on both the object identification by name and the oral understanding tests (table 3). As fluent aphasics with impaired comprehension appear to display a bimodal curve on Gesture Recognition Test performance (Wernicke's: 9 impaired/9 non-impaired; Transcortical sensory: 3 impaired/5 non-impaired), we also compared the lesion sites of Wernicke-transcortical sensory aphasics with gesture recognition disturbance with those having normal Gesture Recognition Test scores.

Only four patients presented severely impaired gesture recognition despite good oral comprehension at the word level (fig 5). This small number of patients does not allow generalisation concerning their lesion sites. The frequent involvement of the centro-parietal white matter (corona radiata) should, however, be noted. Thirteen patients with normal gesture recognition displayed a severe aural comprehension defect. Although in four cases the angular gyrus was involved, the bulk of the lesions was centered on Wernicke's area (12 out of 13 cases), Heschl's gyrus (7/13), insula (7/13), and supramarginal gyrus (6/13), confirming the importance of these areas for language comprehension.

Figure 6 compares lesion overlaps of Wernicke-transcortical sensory aphasics with normal and with poor Gesture Recognition Test scores. After obtaining the cross-sectional CT tracing overlaps corresponding to 70% of each Wernicke-transcortical sensory group (with and without gesture recognition impairment), we superimposed these two areas so that an area common to both groups and areas specific to each group were outlined. This method of subtracting overlaps (modified from Blunk *et al*[19]) enables the isolation of areas specifically associated

Table 3 *Lesion sites of patients showing dissociation between gestural and language comprehension*

GRT	*severe impaired <9*			*normal 11–12*		
Object identification by name	*normal 16*			*severe impaired ≤14*		
Stage	*Acute*	*Recent*	*Chronic*	*Acute*	*Recent*	*Chronic*
Number of patients	*2*	*2*	—	*9*	*3*	*1*
Sites of lesion (retrorolandic and subcortical)						
external and extreme capsules	1	1		2	2	
internal capsule				1	1	
lenticular n.		1		1		
insula	1			4	2	1
pre and post rolandic g.				1	1	1
corona radiata	1	2		1	1	1
temporal pole					1	
post-inf. temporal L.	1			2	1	1
Heschl's gyrus				4	2	1
cortical and subc. Wernicke's area	1			8	3	1
cortical and subc. supramarginal g.				4	2	
cortical and subcortical angular gyrus	1					
sup. parietal lobe	1				1	
assoc. visual areas	1			3		1

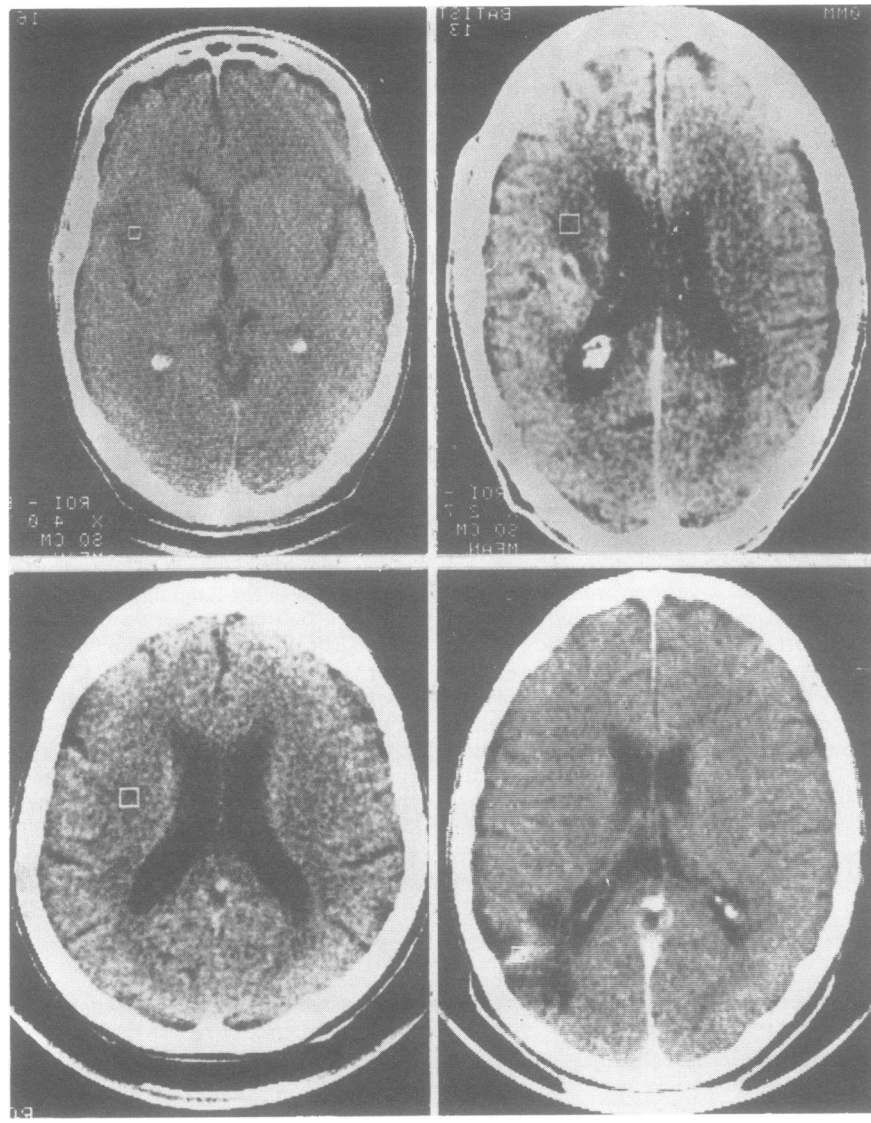

Fig 5 *CT scans of 4 patients who were severely impaired on Gesture Recognition Test, but had good aural comprehension.*

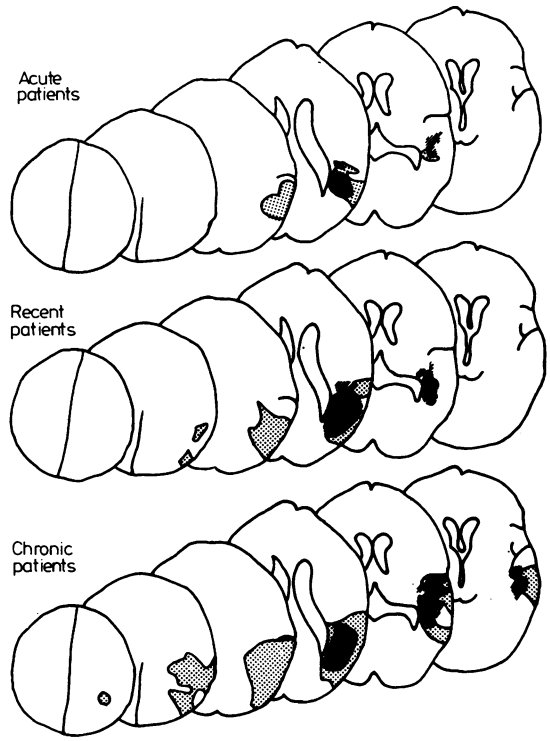

Fig 6 *Areas associated with gesture recognition impairment (dotted) auditory comprehension defects at the word level (striped) or both (solid area) obtained from CT tracing overlaps of Wernicke-transcortical sensory aphasics.*

with either gesture or language comprehension defects.

Table 4 contrasts lesion sites in Wernicke-transcortical sensory aphasics with normal and poor Gesture Recognition Test performance. Patients with Gesture Recognition Test impairment had larger lesions (0·111 ± 0·07 vs 0·047 ± 0·03, $t = 3·01$, $p < 0·005$) and more frequent involvement of angular gyrus (12/13 versus 9/16, $\chi^2 = 4·67$, $p < 0·05$) and superior parietal lobe ($\chi^2 = 3·91$, $p < 0·05$). The area of maximum overlap in Wernicke-transcortical sensory aphasics with normal recognition of gestures included primary auditory areas, Wernicke's area, white matter deep to Wernicke's area, and supramarginal gyrus, while in Wernicke-transcortical sensory aphasics showing impaired

Table 4 *Lesion sites and GRT scores of fluent aphasics with impaired oral comprehension (Wernicke-transcortical sensory)*

	Impaired GRT performance (<11)			*Normal GRT performance (11–12)*		
Stage	*Acute*	*Recent*	*Chronic*	*Acute*	*Recent*	*Chronic*
No. of patients	*5*	*4*	*4*	*9*	*3*	*4*
Lesion sites (post rolandic)						
Insula	1	1	1	3	1	2
Temporal pole	1	1	1			
Post inf temporal L.	2	1	3	3	2	1
Herschl's gyrus	1	1	1	4	1	3
Cortical and subc. Wernicke's area	5	4	3	9	3	4
Supramarginal g	3	4	3	5	1	1
Cortical and subc angular gyrus	5	3	4	6	2	1
Superior parietal L	2	2	3	1	1	1
Assoc visual areas	3	2	2	2	1	1

Gesture Recognition Test scores, it extended to upper cuts (SM, SM + 1 and SM + 2), in the area of the supramarginal and angular gyrus and in the posterior parietal lobe. All the five cases whose lesions were restricted to the temporal lobe had normal Gesture Recognition Test scores.

Discussion

The fact that some areas responsible for a defective Gesture Recognition Test performance are different in the acute and in the chronic stages points to the need for considering the length of disease in the study of anatomical/behavioural correlations. This need has been, with a few exceptions,[16 20] rarely recognised. In the acute stage, basal ganglia and other subcortical sites and the frontal lobe appear very important for Gesture Recognition Test performance. They tend to loose their role in the recent and chronic stage, while cortico-subcortical parietal sites remain crucial for the understanding of gestures.

Frontal and subcortical lesions can disrupt Gesture Recognition Test performance during the acute stage by several mechanisms:

(a) by severing thalamic radiations, namely those travelling through the anterior limb of the internal capsule[21] and lowering cortical arousal. The fact that the "mass effect," that is, a significant influence of lesion size, was more striking acutely, is in accordance with this interpretation.

(b) by impairing active visual scanning of pantomimes or of objects or drawings to be matched with them. Indeed, acute cortical frontal lesions overlapped around the "frontal eye field".

(c) by impairing cognitive operations necessary for performing the Gesture Recognition Test. To match pantomime with the corresponding object is an easy, although somewhat artificial, task. Subjects need to use contextual semantics, that may need the participation of the frontal lobes.

(d) disrupting a subcortical "primitive" system of gesture recognition, whose lesion would have only a transient effect on Gesture Recognition Test performance. The crucial participation of the frontal lobe and basal ganglia in motor programming is well known. Recently several receptive functions have been attributed to the striatum.[22 23] Our results suggest that those anatomical structures which receive efferents from the visual areas and parietal cortex are not only important for the execution of complex motor acts, but also for their recognition. It is possible that spatio-temporal engrams of the gestures[24] that are necessary for its imitation and recognition are stored there, at least those related to common and overlearned gestures.

The importance of the multimodal parietal lobe for gesture recognition is in accordance with the finding of Heilman *et al*[25] of disturbed gesture recognition in apraxics with parietal lobe lesions. This zone may be either the anatomical locus of the proposed "symbolic unit" that relates pantomimes to corresponding objects through plausability rules[5] or the storage area of visuo-kinesthetic motor engrams of gestures.

One of the major issues on gestural behavioural studies is whether the frequent occurrence of language and gestural impairment, after posterior left hemispheric lesions, is due to the anatomical coincidence of areas serving both functions or due to an impairment of an underlying general communicative or cognitive ability. The association between language and gesture comprehension impairment and the correlation between their severity varies among series [1-5 26 27] reflecting different test procedures, different aphasia severity and different length of disease.

Correlation between gesture and language comprehension varies with type of aphasia, being highly significant in global aphasia but non-significant in Wernicke's.[4] Moreover among aphasics with similar language impairment some are impaired in gesture recognition whether others are not.[4] The possible anatomical explanation for these issues was addressed by comparing (a) lesion sites of patients showing striking dissociation between Gesture Recognition Test and auditory recognition performances and (b) lesion site of Wernicke's and transcortical sensory aphasics with normal and impaired Gesture Recognition Test scores. Involvement of centroparietal white matter can cause gesture comprehension disturbance while sparing auditory comprehension, at least at the word level. Severe comprehension defect without gesture recognition disturbance was associated with temporal lesions and a relative sparing of the parietal lobe. Moreover, lesion sites of Wernicke-transcortical sensory aphasics with and without gesture recognition impairment showed important differences: while the former had larger lesions and more frequent involvement of the parietal lobe (Broadman's areas 39 and 7), the latter had more frequently primary auditory and subcortical temporal lesions. Primary auditory and subcortical temporal lesions severing auditory radiations produce auditory comprehension impairment by disturbing phonemic decodification. They also prevent access of verbal auditory information to the posterior part of Wernicke's area and adjacent parietal areas, which play a major role in semantic comprehension.[28] The loci specifically associated with gesture recognition impairment correspond to parietal supramodal cortical areas that

can integrate visual and somesthetic information and the exploration of extrapersonal space.[29]

Although some brain areas were mainly associated either with gesture or language comprehension defects, in some other loci (for example, part of Wernicke's area and supramarginal gyrus) both disturbances could be found with comparable frequency. This partial overlap of the areas crucial to those two functions explains why some posterior aphasics with comparable language disturbances can show striking discrepancies in gestural behaviour.

Lesions producing auditory comprehension defects, were smaller than those causing gesture recognition impairment suggesting a multicomponent cortical representation of gestures. Gestures can have at least three different cortical counterparts: (1) linguistic referents, generally a verb describing the use of an object (or objects) or its goal. These linguistic referents are part of the semantic fields for objects and can be labelled as their "action fields"[30]; (2) at the perceptual/conceptual-symbolic level some perceptual features of gestures and of their situational context that restrict the object to be matched with them, to those which are symbolically or functionally compatible; (3) finally, the kinesthetic-motor engrams of the gestures, that is, the spatio-temporal sequence of elementary movements that compose them.[24] We propose that the multimodal parietal lobe is involved in representations 2 and 3. When the "symbolic/conceptual" unit is damaged patients are no longer able to match gestures with objects, because they had lost the plausability and functional rules necessary to this kind of matching. However they can imitate gestures. When visuo-kinesthetic motor engrams are lost, patients cannot recognise or imitate gestures, or manipulate objects. When these engrams and the "symbolic unit" are disconnected from the visual areas, subjects are unable to imitate and recognise gestures or to pantomime the use of objects, although they could use them. Patients displaying such dissociations have already been reported.[4 25 31] In some cases large (especially chronic) left hemispheric lesions or dominant parietal lesions were not followed by gesture recognition impairment. This suggests that in some subjects the comprehension of gestures is a bi-hemispheric function. They may have either a bilateral representation of kinesthetic motor engrams, or rely mainly on a perceptual/contextual strategy to identify gestures as opposed to a verbal categorial one. The association between gesture recognition and other left hemispheric functions is largely dependent on the partial anatomical overlap of the areas that constitute their anatomical support. This is in accordance with the network approach to cerebral localisation of complex functions[32] that considers them in terms of several component processes, each of which has a distinct localisation. These sites are interconnected and constitute an integrated network subserving each particular function. Meanwhile, each of these sites had additional functional specialisations that are components of intersecting but distinct networks.

Severe and lasting impairment usually requires involvement of several components of the network, although impairment on gesture recognition may follow a lesion in one of the several areas of the network (basal ganglia, centro-parietal white matter, parietal cortex). Lesions of some of these sites, for example parietal lobe, result in multiple defects, and explain the close association between gesture recognition and reading performances[3 4] and constructional apraxia.[4]

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References

[1] Duffy RJ, Duffy JR, Pearson RL. Pantomime recognition in aphasics. *J Speech Hearing Res* 1975;**18**:115–32.

[2] Gainotti G, Lemmo MA. Comprehension of symbolic gesture in aphasia. *Brain Lang* 1976;**3**:451–60.

[3] Varney NR. Linguistic correlates of pantomime recognition. *J Neurol Neurosurg Psychiatry* 1978;**41**:564–8.

[4] Ferro JM, Santos ME, Castro-Caldas A, Mariano MG. Gesture recognition in aphasia. *J Clin Neuropsychol* 1980;**2**:277–92.

[5] Seron X, Van der Kaa MA, Remitz A, Van der Linden M. Pantomime interpretation and aphasia. *Neuropsychologia* 1979;**17**:661–8.

[6] Loizou LA, Kendall BE, Marshall J. Subcortical arteriosclerotic encephalopathy: a clinical and radiological investigation. *J Neurol Neurosurg Psychiatry* 1981;**44**:294–304.

[7] Castro-Caldas A. *Diagnóstico e Evolução das Afasias de Causa Vascular.* Lisboa: Faculdade de Medicina de Lisboa, 1979.

[8] Gonzalez CF, Grossman CB, Palacios E. *Computed Brain and Orbital Tomography. Technique and Interpretation.* New York: John Wiley and Sons, 1976.

[9] Shipps FC, Jones JM, D'Agostino A. *Atlas of Brain Anatomy for CT scans.* Springfield: Charles C Thomas, 1977.

[10] Gado M, Hanaway J, Frank R. Functional anatomy of the cerebral cortex by computed tomography. *J Comput Assist Tomogr* 1979;**3**:1–19.

[11] Damásio H, *A computerized tomography guide to the*

identification of cerebral structures. Arch Neurol 1983;**40**:138–42.

[12] Matsui T, Hirano A. *An Atlas of Human Brain for Computerized Tomography.* Tokyo: Igaku-Schoin, 1978.

[13] Gordon WP, Rosencrantz PL. *Statistical methods of analysing CT scan data based upon neuroradiological localization. INS Bull* 1982;18.

[14] Mazzocchi F, Vignolo LA. Computer assisted tomography in neuropsychological research: a simple procedure for lesion mapping. *Cortex* 1978;**14**:136–44.

[15] Hayward RN, Naeser MA, Zatz LM. Cranial computed tomography in aphasia. Correlation of anatomical lesions with functional deficits. *Radiology* 1977;**123**:653–60.

[16] Kertesz A, Harlock W, Coates RK. Computed tomographic localization, lesion size and prognosis of aphasia and non-verbal impairment. *Brain Lang* 1979;**8**:34–50.

[17] Wu S, Schenkenberg T, Wing SD, Osborn AG. Cognitive correlates of diffuse cerebral atrophy determined by computed tomography. *Neurology (Ny)* 1981;**31**:1180–4.

[18] Huckman MS, Fox J, Topel J. The validity of criteria for the evaluation of cerebral atrophy by computed tomography. *Radiology* 1975;**116**:85–92.

[19] Blunk R, De Bleser R, Willmes K, Zeumer H. A refined method to relate morphological and functional aspects of aphasia. *Eur Neurol* 1981;**20**:69–79.

[20] Mazzochi F, Vignolo LA. Localization of lesions in aphasia: clinical-CT scan correlations in stroke patients. *Cortex* 1979;**15**:627–54.

[21] Kienet J, Kuypers HGJM. Organization of thalamocortical connexions to the frontal lobe in the rhesus monkey. *Exp Brain Res* 1977;**29**:299–322.

[22] Purpura DP. *Physiological Organization of the Basal Ganglia.* In: Yahr MD, ed. *The Basal Ganglia.* New York: Raven Press, 1976.

[23] Teuber HL. *Complex functions of basal ganglia.* In: Yahr MD, ed. *The Basal Ganglia.* New York: Raven Press, 1976.

[24] Heilman KM. Apraxia. In: Heilman KM, Valenstein E, eds. *Clinical Neuropsychology.* Oxford: Oxford University Press, 1979;159–185.

[25] Heilman KM, Rothil LJ, Valenstein E. Two forms of ideomotor apraxia. *Neurology (Ny),* 1982;**32**:342–6.

[26] Feyereisen P, Seron X, Macar MD. L'interpretation de differentes categories de gestes chez des sujets aphasiques. *Neuropsychologia* 1981;**19**:515–21.

[27] Duffy RJ, Duffy JR. Three studies of deficits in pantomimic expression and pantomimic recognition in aphasia. *J Speech Hear Res* 1981;**24**:70–84.

[28] Cappa S, Cavallotti G, Vignolo LA. Phonemic and lexical errors in fluent aphasia: correlation with lesion site. *Neuropsychologia* 1981;**19**:171–7.

[29] Mountcastle VB, Lynch JC, Georgopoulus A, Sakata H, Acuna C. Posterior parietal cortex of the monkey: command functions for operations within extrapersonal space. *J Neurophysiol* 1975;**38**:871–908.

[30] Roy EA. *Neuropsychological Perspectives on Apraxia* and *Related Action Disorders. Advances in Psychology,* Amsterdam: North Holland Co (in press).

[31] Assal G, Regli F. Syndrome de disconnexion visuoverbale et visuo-gestuelle. Aphasic optique et apraxie optique. *Rev Neurol (Paris)* 1980;**136**:365–76.

[32] Mesulam MM. A cortical network for directed attention and unilateral neglect. *Ann Neurol* 1981;**10**:309–25.