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Selective imitation impairments differentially interact with language processing

Paola Mengotti,[1,*] Corrado Corradi-Dell'Acqua,[1,2,*] Gioia A. L. Negri,[1,3] Maja Ukmar,[4] Valentina Pesavento[5] and Raffaella I. Rumiati[1]

1 Neuroscience Area, SISSA, Trieste, Italy
2 Swiss Centre for Affective Sciences, University of Genève, Genève, Switzerland
3 IRCCS E. Medea, Polo Friuli-Venezia Giulia, Italy
4 U.C.O. di Radiologia, Azienda Ospedaliero-Universitaria 'Ospedali Riuniti', Trieste, Italy
5 S.C. Medicina Riabilitativa, Azienda Ospedaliero-Universitaria 'Ospedali Riuniti', Trieste, Italy

*These authors contributed equally to this work.

Correspondence to: Raffaella Rumiati,
Neuroscience Area, SISSA, Via Bonomea 265,
34136 Trieste, Italy
E-mail: rumiati@sissa.it

Whether motor and linguistic representations of actions share common neural structures has recently been the focus of an animated debate in cognitive neuroscience. Group studies with brain-damaged patients reported association patterns of praxic and linguistic deficits whereas single case studies documented double dissociations between the correct execution of gestures and their comprehension in verbal contexts. When the relationship between language and imitation was investigated, each ability was analysed as a unique process without distinguishing between possible subprocesses. However, recent cognitive models can be successfully used to account for these inconsistencies in the extant literature. In the present study, in 57 patients with left brain damage, we tested whether a deficit at imitating either meaningful or meaningless gestures differentially impinges on three distinct linguistic abilities (comprehension, naming and repetition). Based on the dual-pathway models, we predicted that praxic and linguistic performance would be associated when meaningful gestures are processed, and would dissociate for meaningless gestures. We used partial correlations to assess the association between patients' scores while accounting for potential confounding effects of aspecific factors such age, education and lesion size. We found that imitation of meaningful gestures significantly correlated with patients' performance on naming and repetition (but not on comprehension). This was not the case for the imitation of meaningless gestures. Moreover, voxel-based lesion-symptom mapping analysis revealed that damage to the angular gyrus specifically affected imitation of meaningless gestures, independent of patients' performance on linguistic tests. Instead, damage to the supramarginal gyrus affected not only imitation of meaningful gestures, but also patients' performance on naming and repetition. Our findings clarify the apparent conflict between associations and dissociations patterns previously observed in neuropsychological studies, and suggest that motor experience and language can interact when the two domains conceptually overlap.

Keywords: apraxia; aphasia; motor system; grounded cognition

Abbreviations: AAT = Aachener Aphasie Test

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Introduction

Traditionally limb apraxia is defined as a deficit in producing voluntary movements in absence of elementary sensorimotor or coordination deficits, language comprehension deficit or severe mental deterioration. The most popular classification of limb apraxia closely derives from the two-step model of action control proposed by Liepmann (1920). According to his conceptualization, a failure to generate the mental image of the intended gesture gives rise to ideational apraxia, which is better captured by asking patients to use objects (Steinthal, 1871; Morlaas, 1928; Poeck, 1982; De Renzi and Lucchelli, 1988; Goldenberg and Hagmann, 1998; Rumiati *et al.*, 2001). In contrast, a faulty ability to implement this image into the appropriate motor output corresponds to ideomotor apraxia and it is observed, according to Liepmann's view later endorsed by De Renzi (1990), when patients are not only asked to execute a gesture demonstrated by the examiner (i.e. visuo-imitative apraxia), but also when asked to pantomime them on verbal command [see Goldenberg, (2009) for detailed discussions on the model]. As to the brain correlates of these two main manifestations of apraxia, although ideational apraxia has been primarily associated with damage to the parietal lobe (Rumiati *et al.*, 2001; Goldenberg and Spatt, 2009), ideomotor apraxia has been observed after damage predominantly to the left parietal and premotor cortices (De Renzi *et al.*, 1980, 1983; Goldenberg *et al.*, 1996; Tessari *et al.*, 2007; see Goldenberg, 2009; Rumiati *et al.*, 2010, for recent reviews). This two-step distinction of limb apraxia has been replaced by other accounts as it does not fully capture all the observed dissociations and it does not adequately explain the types of errors apraxic patients make (Poizner *et al.*, 1995).

As in right-handed individuals, left-brain damage frequently (although not necessarily) impairs both linguistic and action abilities, it has been suggested that apraxia and aphasia might be caused by the disruption of a single underlying mechanism that, over the years, has been differentially referred to as 'asymbolia' (Finkelnburg, 1870), deficit of 'abstraction' (Goldstein, 1948), of 'conceptualization' (Bay, 1962) or of 'use of symbols for communication' (Duffy *et al.*, 1975). For instance, de Ajuriaguerra *et al.* (1960) tested 415 patients and found that ~90% of patients suffering from apraxia (42/47 with ideomotor apraxia and 10/11 with ideational apraxia) were also aphasic. Similarly, De Renzi *et al.* (1968) reported a strong correlation between a deficit in language comprehension and ideational apraxia. These associations between apraxic and aphasic symptoms in neuropsychological patients converge with the results from neuroimaging studies on healthy volunteers in which activations in the fronto-parietal motor system were observed when they processed action-related words and sentences (Hauk *et al.*, 2004; Tettamanti *et al.*, 2005; Ruschemeyer *et al.*, 2007; Postle *et al.*, 2008; Péran *et al.*, 2010; Willems *et al.*, 2010). These neuropsychological and neuroimaging results have, in recent years, been interpreted as evidence for grounded (or embodied) theories of cognition (Pulvermüller, 2005; Gallese and Lakoff, 2005; Barsalou, 2008). Accordingly, access to the meaning of a verb or sentence denoting a given action activates brain regions that are involved in the execution of the same action. On this perspective, understanding the meaning of verbs such as 'grasping' or 'throwing' implies the re-enactment of the action representations that enable actual grasping and throwing to occur, and that are thought to be damaged in apraxic patients.

In contrast with the associations of deficits in large groups of patients (and by neuroimaging data on healthy individuals), reports of isolated patients exhibiting aphasia in the absence of apraxia and *vice versa* suggest how linguistic and motor abilities can functionally dissociate. For instance, Liepmann (quoted by de Ajuriaguerra *et al.*, 1960), described seven non-aphasic patients with apraxia, six of whom had right-sided hemiplegia. Subsequently, Kertesz *et al.* (1984) studied 177 cerebrovascular patients and found that six of them with severe aphasia did not show any apraxic deficit. Critically, Papagno *et al.* (1993) tested 699 patients with a shortened version of a comprehension test (Token test, De Renzi and Faglioni, 1978) to assess language abilities and an imitation test (De Renzi *et al.*, 1980), and found that 149 were aphasic but not apraxic, and 10 apraxic but not aphasic. The double dissociations between apraxic and aphasic deficits reported in the above studies suggest that the linguistic ability and the praxic ability are functionally separable and that they may each rely on a different neural network.

The striking divergence between association and dissociation patterns was confirmed by two recent studies in which 37 (Negri *et al.*, 2007) and 12 (Papeo *et al.*, 2010) unilaterally brain-damaged patients were required to perform motor and verbal tasks on the same set of stimuli. Although at the group-level reliable correlations between patients' performance in motor and linguistic tasks were documented, when the performance of individual patients was considered, double dissociations between object use and linguistic processing of the corresponding nouns were observed (Negri *et al.*, 2007; Papeo *et al.*, 2010). There are several possible explanations as to why conflicting results are found in the literature: (i) the tests used to assess language and imitation, which tend to vary considerably across studies, typically tap either language or imitation as if they each were a single process; (ii) patients' averaged performance does not show the single cases who may dissociate (i.e. the artifact of the mean, Shallice, 1988); and (iii) the brain regions sustaining language and imitation are contiguous and therefore a vascular lesion may affect both abilities.

As far as imitation is concerned, only a handful of neuropsychological reports analysed at single-case level patients' dissociating imitative performance on meaningful and meaningless gestures (Goldenberg and Hagmann, 1997; Peigneux *et al.*, 2000; Bartolo *et al.*, 2001; Tessari *et al.*, 2007). This evidence suggests that imitation does not rely on a single but on multiple mechanisms (see also Tessari and Rumiati, 2004, for evidence from healthy individuals). Overall, three patients had more difficulty imitating meaningful gestures than meaningless gestures, whereas 11 showed the opposite pattern. The lesions of 7 of 11 patients with a specific deficit at imitating meaningless gestures seem to affect the left angular gyrus (Patients LK and EN in Goldenberg and Hagmann 1997; one patient described in Peigneux *et al.*, 2004; Cases 12, 13, 19 and 23 in Tessari *et al.*, 2007), whereas two of three patients with a specific deficit in imitating meaningful

gestures had lesions also involving the middle and superior temporal gyri as well as the hippocampus.

These neuropsychological findings can be interpreted within the dual-pathway model of gesture imitation by Rumiati and colleagues (Tessari and Rumiati, 2004; Tessari *et al*., 2007), based on the elaboration of the seminal model originally put forward by Rothi *et al.* (1991); (see also Cubelli *et al*. 2000, for a similar model). According to this view, gesture imitation is subserved by both a 'direct' pathway, that allows the slavish reproduction of the seen gestures, irrespective of their content, and a 'semantic' pathway, through which gestures are produced by access to their meaning in the semantic memory. Whereas the semantic pathway is exclusively dedicated to meaningful, familiar gestures, the direct pathway prevalently processes meaningless gestures, but it could theoretically be used with meaningful gestures too as, for instance, when meaningful and meaningless gestures are presented intermingled within a block (Tessari and Rumiati, 2004).

Models such as the one just described (Tessari and Rumiati, 2004; Tessari *et al*., 2007; but see also Rothi *et al*., 1991; Cubelli *et al*., 2000) offer a possible account of the apparent inconsistency in the extant literature between patients' linguistic performance and praxic performance. When patients attempted to imitate meaningful gestures by relying on a damaged semantic pathway, potentially subserving also some aspects of linguistic tasks (Fig. 1), correlations/associations of aphasia and imitation apraxia for meaningful gestures were observed (even though, in principle, patients could have selected the direct pathway to imitate meaningful gestures; Bartolo *et al.*, 2001, see also Tessari *et al*., 2007, for an explanation of why patients with a faulty semantic pathway do not choose the direct pathway to imitate meaningful gestures). On the other hand, dissociations between language and motor action performance might reflect patients' attempts to engage the same direct pathway used prevalently, but not exclusively, for imitating meaningless gestures. Thus correlations/associations of linguistic and praxic deficits should be expected only when patients imitate meaningful gestures (but not meaningless) gestures. Unfortunately, given that in the extant literature the number of single-cases with a selective deficit in imitation of meaningful (three patients, one of which with a documented aphasia) or meaningless gestures (11 patients, three of which with a documented aphasia) is very small, such prediction remains untested.

In the present study we tested 57 patients with damage to the left hemisphere for their ability to process language [Aachener Aphasie Test (AAT), Luzzatti *et al*., 1996], to use objects (test from De Renzi *et al*., 1968) and to imitate meaningful and meaningless gestures. Two different tests were used for assessing patients' imitation performance, one developed by De Renzi *et al.* (1980), administered to 25 patients, and one developed by Tessari *et al.* (2011), administered to the remaining 32. We used partial correlation analysis to assess possible relations between linguistic and imitation performance (while controlling for potential effects of aspecific variables, such as age, education, lesion size, etc.). Moreover, to identify the brain regions specifically associated with patients' behavioural scores in all the tests used, we then implemented the voxel-based lesion-symptom mapping analysis (Bates *et al*., 2003). In particular, we predicted that only imitation of meaningful gestures would correlate with patients' linguistic deficit; if this prediction turned out to be correct, it would suggest that language and action are likely to interact in the conceptual system. In contrast, we predicted that imitation of meaningless gestures should dissociate from patients' linguistic abilities, revealing regions specifically recruited for action reproduction, independently of the language network.

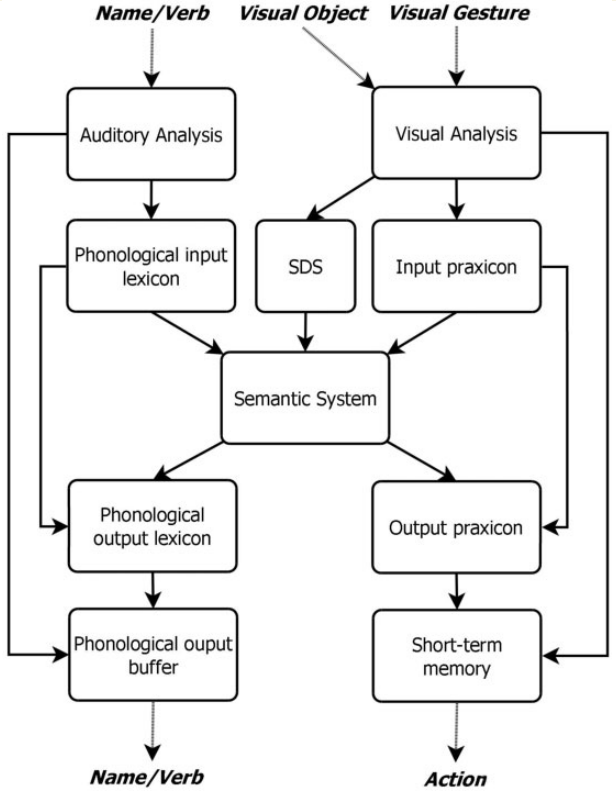

Figure 1 A modified version of the model of praxis originally proposed by Rothi *et al*. (1991). According to this model, imitation of familiar gestures relies on the semantic, indirect route that encompasses the input praxicon, the semantic system, the output praxicon, and the short term memory; imitation of new gestures relies on the non-semantic, direct route that, from visual analysis, leads directly to the short-term memory. Originally published in Rumiati *et al.* (2010). SDS = structural description system.

Materials and methods

Participants

Fifty-seven patients (24 females, mean age = 64.05 ± 9.83 years; mean education = 10.54 ± 4.09 years; mean time post stroke = 5.83 months, range: 15 days to 77 months) who had suffered a single left-hemisphere cerebrovascular accident entered the study. Patients had no previous neurological history, were <85 years of age, and had at least 5 years of education. Patients' handedness was assessed using the Edinburgh Handedness Inventory (Oldfield, 1971) and their scores are shown in Table 1. They were tested at the Rehabilitation Unit of

Table 1 Patients' scores on neuropsychological tests

Patient initials	Oldfield	Raven's CPM	VOSP screen	VOSP o.d.	Span fwd	Span bwd	Corsi	P&P pictures	TMT-A	TMT-B	TMT B-A	WEIGL	WCST n.cat.	WCST pers	REY imm	REY def	REY rec	WARR. faces
A.N.	100	32.8	18	19	6	6	4.5	50	26	46	20	n.a.	5	6	35	6	26/32	n.a.
B.V.	83	21	20	12	3.25	3	4	48	203	p.u.	p.u.	6.75	n.a.	n.a.	18	2	38/46	n.a.
B.R.	100	36.8	20	16	n.a.	n.a.	4.5	48	37	p.u.	p.u.	p.u.	p.u.	p.u.	n.a.	n.a.	n.a.	n.a.
B.U.	100	21.2	14	16	p.u.	n.a.	2.25	46	p.u.	p.u.	p.u.	7.25	n.a.	n.a.	n.a.	n.a.	n.a.	16
B.Z.	92	32.6	20	16	n.a.	n.a.	4	49	22	275	253	10	n.a.	n.a.	n.a.	n.a.	n.a.	24
C.A.	n.a.	34.9	20	17	5.5	4	5.25	n.a.	41	116	76	4.75	n.a.	n.a.	13.7	1.1	23/32	22
C.N.	-83	26.6	20	13	4.25	3	3	44	67	p.u.	p.u.	6	p.u.	p.u.	17	2	26/32	n.a.
C.C.	100	20.8	19	12	p.u.	n.a.	n.a.	47	194	p.u.	p.u.	1.5	n.a.	n.a.	n.a.	n.a.	n.a.	p.u.
C.G.	100	19.8	19	17	3.75	2	4.25	n.a.	47	276	229	4.5	n.a.	n.a.	31	7	44/46	n.a.
C.O.	n.a.	34.3	20	18	n.a.	n.a.	3.5	n.a.	59	179	120	5	n.a.	n.a.	n.a.	n.a.	n.a.	25
C.R.	n.a.	7.2	19	12	n.a.	n.a.	3.75	n.a.	94	p.u.	p.u.	0	p.u.	p.u.	n.a.	n.a.	n.a.	23
C.S.	83	36	20	17	n.a.	n.a.	4.75	50	10	92	82	8.5	n.a.	n.a.	n.a.	n.a.	n.a.	25
C.U.	100	30.8	20	19	6.75	5	4.75	51	63	164	101	n.a.	6	0	37	9	32/32	n.a.
C.Z.	n.a.	14.4	18	15	4.75	2	3.25	n.a.	325	p.u.	p.u.	2.25	n.a.	n.a.	n.a.	n.a.	n.a.	23
D.C.R.	n.a.	30.1	19	18	6.5	6	4	52	21	177	175	n.a.	6	0	33.3	6.4	41/46	n.a.
D.C.C.	n.a.	n.a.	20	16	n.a.	n.a.	4	n.a.	n.a.	n.a.	n.a.	6	n.a.	n.a.	15	1	n.a.	n.a.
D.B.	100	31	20	16	n.a.	n.a.	4	51	48	76	28	10.75	n.a.	n.a.	n.a.	n.a.	n.a.	25
D.P.	100	24.5	20	15	5.5	3	4.25	n.a.	24	263	239	11	n.a.	n.a.	22	4	45/46	24
D.O.	100	32.3	20	20	7	4	3.5	52	20	64	44	n.a.	6	0	24	4	40/46	n.a.
D.R.	100	32.3	20	19	3.75	n.a.	4.75	51	n.a.	n.a.	n.a.	n.a.	6	1	p.u.	p.u.	p.u.	19
F.R.	100	21.8	20	15	2.75	n.a.	4	47	133	303	170	3	n.a.	n.a.	n.a.	n.a.	n.a.	20
F.N.	100	26.8	20	19	4.5	4	4.75	52	122	p.u.	p.u.	n.a.	2	3	25	1	40/46	n.a.
F.U.	67	21.2	20	15	5.5	4	3.75	50	43	128	85	9	n.a.	n.a.	45	10	31/32	n.a.
F.L.	n.a.	33.8	20	17	4.75	5	6.25	n.a.	36	104	68	7.5	n.a.	n.a.	45	9.2	n.a.	n.a.
F.S.	100	29.8	19	16	5	3	4	51	65	276	211	n.a.	4	15	15	0	25/32	n.a.
G.C.	n.a.	33.4	19	19	4	3	4.75	52	44	340	296	12.5	n.a.	n.a.	n.a.	n.a.	n.a.	24
G.V.	n.a.	33.5	19	18	p.u.	p.u.	4.5	51	21	155	106	n.a.	6	1	n.a.	n.a.	n.a.	25
G.O.	n.a.	19.5	19	n.a.	3	n.a.	p.u.	n.a.	303	p.u.	p.u.	2	n.a.	n.a.	p.u.	p.u.	p.u.	13
G.U.	100	32	20	14	4.25	n.a.	4.25	32	238	p.u.	p.u.	3.75	n.a.	n.a.	n.a.	n.a.	n.a.	23
H.B.	n.a.	17	n.a.	n.a.	6.25	2	3	n.a.	n.a.	n.a.	n.a.	3.75	n.a.	n.a.	n.a.	n.a.	n.a.	18
J.N.	83	29.8	20	17	2.75	n.a.	4	52	39	134	95	11.75	n.a.	n.a.	n.a.	n.a.	n.a.	16
L.E.	100	27.6	20	19	2.5	2	3.75	48	35	339	304	n.a.	5	7	5	2	32/46	n.a.
L.N.	100	29.8	20	19	5	4	4.75	51	70	149	79	n.a.	3	5	20	3	41/46	n.a.
L.I.	n.a.	29.8	20	17	n.a.	n.a.	4.5	n.a.	36	189	153	9.75	23	2	n.a.	n.a.	n.a.	n.a.
L.C.	n.a.	23.3	20	16	4.75	4	5.75	n.a.	103	243	140	6.75	n.a.	n.a.	23	3.5	n.a.	20

(continued)

Table 1 Continued

Patient initials	Oldfield	Raven's CPM	VOSP screen	VOSP o.d.	Span fwd	Span bwd	Corsi	P&P pictures	TMT-A	TMT-B	TMT B-A	WEIGL	WCST n.cat.	WCST pers	REY imm	REY def	REY rec	WARR. faces
M.N.	100	32.8	20	15	4.75	4	4.75	52	23	116	93	8	n.a.	n.a.	38	8	29/32	n.a.
M.R.	n.a.	32	20	16	5.25	n.a.	5,5	n.a.	58	129	71	11.5	n.a.	n.a.	n.a.	n.a.	n.a.	24
M.E.	100	26.3	18	18	3.75	3	4.75	51	29	201	172	1.75	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
M.L.	n.a.	32.8	20	19	6.75	5	4.5	n.a.	23	69	46	7.5	n.a.	n.a.	48.7	14.3	n.a.	n.a.
N.V.	n.a.	16.2	18	13	5.5	n.a.	4.25	46	112	n.a.	n.a.	4.25	n.a.	n.a.	n.a.	n.a.	n.a.	21
O.B.	n.a.	31.4	20	18	4.5	3	3.25	n.a.	p.u.	p.u.	p.u.	5	n.a.	n.a.	15	1	n.a.	n.a.
P.A.	n.a.	31.4	20	19	6.5	5	4	n.a.	37	138	101	15	n.a.	n.a.	39.3	7.2	29/32	n.a.
P.T.	100	23	20	18	5.25	2	3.25	50	194	p.u.	p.u.	5.75	n.a.	n.a.	21	1	28/32	n.a.
P.R.	100	25.3	18	16	4.25	2	4.25	n.a.	36	173	137	8.25	n.a.	n.a.	30	5	25/32	n.a.
P.I.	100	28	18	16	2.25	3	4.25	48	n.a.	n.a.	n.a.	8.75	5	2	n.a.	n.a.	n.a.	n.a.
R.A.	100	18.6	19	11	6.25	n.a.	3.25	46	n.a.	n.a.	n.a.	5	n.a.	n.a.	n.a.	n.a.	n.a.	19
S.L.	n.a.	32.8	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	8	n.a.	n.a.	n.a.	n.a.	n.a.	12
S.R.	100	26	20	13	4.25	2	3.25	46	n.a.	n.a.	n.a.	2.75	3	9	n.a.	n.a.	n.a.	23
S.A.	100	28	20	13	p.u.	n.a.	6.25	39	94	p.u.	p.u.	9.75	n.a.	n.a.	n.a.	n.a.	n.a.	21
S.O.	n.a.	30.6	20	20	4.25	4	5.25	n.a.	66	128	62	13	n.a.	n.a.	n.a.	n.a.	n.a.	24
S.N.	100	34.6	20	18	5.25	4	5	52	19	137	118	8	2	5	24	1	18/32	n.a.
S.V.	100	17.3	19	17	3	n.a.	3.75	47	n.a.	n.a.	n.a.	n.a.	6	3	n.a.	n.a.	n.a.	24
S.S.	100	28.9	20	16	p.u.	n.a.	1	47	63	p.u.	p.u.	4.75	n.a.	n.a.	n.a.	n.a.	n.a.	22
S.T.	100	32.3	19	16	n.a.	n.a.	4.75	49	n.a.	n.a.	n.a.	10.5	2	5	n.a.	n.a.	n.a.	n.a.
T.R.	n.a.	28.3	20	19	4.25	3	4	n.a.	32	181	149	10.25	n.a.	n.a.	18.6	5.3	29/32	n.a.
U.L.	83	23.5	20	17	4.5	n.a.	3	47	p.u.	p.u.	p.u.	5	n.a.	n.a.	n.a.	n.a.	n.a.	3
V.I.	n.a.	22	20	17	p.u.	n.a.	3.5	52	79	p.u.	p.u.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	25

Scores are corrected according to tests' norms. Corsi = Corsi test, spatial short-term memory (Spinnler and Tognoni, 1987); n.a. = not administered; Oldfield = handedness (Oldfield, 1971); P&P pictures = Pyramids and Palm Trees Test (Howard and Patterson, 1992); p.u. = patient unable to complete the task.; Raven's CPM = Raven Coloured Progressive Matrices (Carlesimo *et al.*, 1996); REY = 15 words memory test (Rey, 1964); REY def = deferred recollection; REY imm = immediate recollection; REY rec = recognition; Span bwd = digit span backward; Span fwd = digit span forward (Orsini *et al.*, 1987); TMT-A,-B,B-A = Trail making test (Giovagnoli *et al.*, 1996); VOSP = Visual Object and Space Perception battery (Warrington and James, 1991). VOSP screen = screening task; VOSP o.d. = object decision task; WARR faces = Warrington for faces test (Warrington, 1996); WCST = Wisconsin Card Sorting Test (Caffarra *et al.*, 2004); WCST n.cat. = number of categories; WCST pers = number of perseverations; WEIGL = Weigl's sorting test (Spinnler and Tognoni, 1987).

the Ospedali Riuniti in Trieste and at the Azienda Ospedaliera-Universitaria 'Santa Maria della Misericordia' in Udine. All patients (or a relative in the case of aphasia) read and signed a written informed consent. The study was approved by the SISSA Ethics Committee and conducted in accordance with the Declaration of Helsinki.

Neuropsychological assessment

All 57 patients were administered an extensive neuropsychological assessment evaluating language, praxis, visuo-spatial abilities, memory and executive functions. Scores are reported in Table 1.

The AAT (Luzzatti *et al.*, 1996) was used to evaluate patients' linguistic abilities. The AAT provides an output in terms of presence or absence of aphasia together with a definition of the specific type of aphasic syndrome and related probabilities, based on the results on different linguistic tasks (Token test, Naming, Comprehension, Repetition and Writing). The cut-offs were determined based on the performance of a group of 88 healthy participants (mean age: 52 years; age range: 20–85 years). Scores on these tasks are reported in Table 2.

All patients were assessed for their ability to use objects by means of a test developed by De Renzi *et al*. (1968), in which patients are asked to use seven common objects. Maximum score on this test is 14, which also represents the cut-off. The cut-off was determined based on the performance of a group of 40 control participants without brain damage. Pathological performance on this task has been interpreted as a sign of ideational apraxia (e.g. De Renzi and Lucchelli 1988; Rumiati *et al.*, 2001).

They were also assessed for their ability to imitate actions using two different tests, the first in which meaningful and meaningless gestures are presented in separate blocks, the second in which the two action types are presented intermingled. The first test, developed in our lab (Tessari *et al.*, 2011), requires patients to imitate, one after the other, 18 meaningful intransitive gestures and 18 meaningless gestures derived from the meaningful gestures by modifying the spatial relationship between the effector and the main body axis. If a correct response was not produced on the first trial, a second trial was allowed. For each gesture a score of 0, 1, 2 is given according to the performance (0 = no imitation, 1 = correct imitation in the second trial, 2 = correct imitation in the first trial), with the cut-off varying according to age and years of education (age between 30 and 50: cut-off meaningful $\leqslant 32$, cut-off meaningless $\leqslant 31$, cut-off total $\leqslant 63$; age between 51 and 70: cut-off meaningful $\leqslant 31$, cut-off meaningless $\leqslant 28$, cut-off total $\leqslant 59$; age of 71 and above: cut-off meaningful $\leqslant 25$ if education <6 years and cut-off meaningful $\leqslant 30$ if education $\geqslant 7$ years, cut-off meaningless $\leqslant 24$ if education <6 years and cut-off meaningless $\leqslant 24$ if education $\geqslant 7$ years, cut-off total $\leqslant 50$ if education <6 years and cut-off total $\leqslant 58$ if education $\geqslant 7$ years) for a total score of 72 maximum. The cut-offs were determined based on the performance of a group of 111 healthy participants (age range: 30–90 years). The second test, developed by De Renzi *et al*. (1980), requires patients to imitate 24 gestures of which 12 are meaningful (half distal and half proximal) and 12 are meaningless (half distal and half proximal). Each gesture was presented up to three times and a score from 0 to 3 was assigned depending on patients' performance (0 = no imitation, 1 = correct imitation in the third trial, 2 = correct imitation in the second trial, 3 = correct imitation in the first trial), for a maximum total score of 72. A score <53 was considered as pathological. The cut-offs were determined based on the performance of a group of 100 control participants without brain damage (mean age: 52.6 years). Pathological performance on these tasks has been interpreted as a sign of ideomotor apraxia (De Renzi *et al*., 1980; Tessari *et al*., 2011).

Gestures were considered as incorrect if one of the following errors was present: (i) spatial error: the gesture is recognizable but the arm or the hand movement followed the wrong direction or axis; (ii) visuo-semantic error: the performed gesture is visually similar and semantically related to the proper gesture; (iii) visual error: the performed gesture is visually similar to the proper one, or it combines two different gestures already presented, or it is visually similar to a gesture that was already presented in the list; (iv) omission: the gesture is not reproduced; and (v) unrecognizable: the performed gesture is unrecognizable. Patients imitated all the gestures with the ipsilesional left hand. Scores on the praxic tests are reported in Table 3.

Behavioural data analysis

To ascertain the relation between praxic and linguistic performances, we carried out partial correlation analyses aimed at assessing the amount of shared variability between patients' performance in two tests while controlling for potential effects of aspecific variables, such as age, gender, education, lesion size and illness length. Please note that all linear effects of the confounding variables were simultaneously removed from each of the two conditions of interest. As a measure of linguistic proficiency, we took patients' performance in the AAT. Thus, for each AAT subtest, we took patients' score as a percentage of the maximum possible score. Likewise, as a measure of ideational apraxia, we took participants' score on the test from De Renzi *et al.* (1968) as a percentage of the maximum possible score. Finally, for ideomotor apraxia, we considered separately, items in which patients imitated a meaningful and a meaningless gesture, thus for each kind of gesture we took patients' score as a percentage of the maximum possible score. Furthermore, as ideomotor apraxia was investigated in 24 patients, with the test from De Renzi *et al.* (1980) and, in the remaining 33 patients, with the test from Tessari *et al.* (2011), we subtracted from the percentage score of each patient the average score from the test used. Statistical analysis was carried out with R.11.1 (http://cran.r-project.org//) open source software.

Lesion analysis

Computed tomography or MRI scans were available for each patient. An experienced neuroradiologist (M.U.), blind to the aims of the study and to the behavioural deficits of the patients, mapped the lesioned areas of each patient onto the normalized MNI template (http://www.bic.mni.mcgill.ca/cgi/icbm_view), with a voxel size of $1 \times 1 \times 1\,mm^3$, using MRIcro software (http://www.mricro.com; Rorden and Brett, 2000).

Neural correlates of language and praxis measures were further investigated using voxel-based lesion-symptom mapping (Bates *et al*., 2003). This technique allows analysing the relationship between lesion data (described, for each voxel, as a variable reflecting the putative presence of a lesion) and continuous behavioural measures. To this purpose, lesion masks were converted into ANALYZE format volumes, in which 0 corresponded to undamaged tissue and 1 to the lesioned part of the brain. The images were subsequently smoothed with an 8-mm full-width at half-maximum kernel, thus allowing non-integer values around the lesion borders. Each behavioural measure (percentage scores of meaningless, meaningful, ideational apraxia, AAT, etc.) was fed into a general linear model in which it was related to the lesion intensity. The analysis was restricted to those voxels for which there were at least five patients with and five patients without a lesion (i.e. those voxels in which the sum of the smoothed lesion

Table 2 Summary of all patients' performance on linguistic tests

Patient	AAT					
	Output	Token	Repetition	Writing	Naming	Comprehension
A.N.	No aphasia	0	148	90	120	120
B.V.	Not classified	15	134	78	109	99
B.R.	Global aphasia	48	39	12	0	41
B.U.	Global aphasia	36	44	9	15	40
B.Z.	Wernicke's aphasia	50	9	22	15	91
C.A.	No aphasia	4	146	89	112	112
C.N.	No aphasia	4	140	85	108	105
C.C.	Wernicke's aphasia	50	0	0	0	22
C.G.	No aphasia	10	136	84	112	113
C.O.	Global aphasia	36	39	30	17	83
C.R.	Wernicke's aphasia	50	14	0	27	49
C.S.	Wernicke's aphasia	25	102	47	61	92
C.U.	Broca's aphasia	18	142	68	91	98
C.Z.	No aphasia	14	139	68	102	88
D.C.R.	No aphasia	0	105	80	115	112
D.C.C.	Wernicke's aphasia	11	88	56	63	95
D.B.	Wernicke's aphasia	39	71	44	83	99
D.P.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
D.O.	No aphasia	n.a.	146	n.a.	n.a.	n.a.
D.R.	Broca's aphasia	1	94	15	69	95
F.R.	Transcort. aphasia	41	95	58	34	62
F.N.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
F.U.	No aphasia	3	142	88	120	117
F.L.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
F.S.	Not classified	13	133	82	109	117
G.C.	Not classified	5	141	86	102	113
G.V.	Not classified	20	90	41	69	112
G.O.	Wernicke's aphasia	26	110	41	62	92
G.U.	Wernicke's aphasia	29	134	71	29	55
H.B.	No aphasia	16	145	68	108	87
J.N.	Conduction aphasia	6	134	74	109	118
L.E.	Wernicke's aphasia	47	35	58	81	79
L.N.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
L.I.	Broca's aphasia	18	9	12	0	101
L.C.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
M.N.	No aphasia	4	131	84	119	118
M.R.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
M.E.	Broca's aphasia	15	123	75	110	102
M.L.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
N.V.	Amnestic aphasia	20	139	37	93	90
O.B.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
P.A.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
P.T.	Amnestic aphasia	20	141	57	102	89
P.R.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
P.I.	Not classified	31	96	55	29	87
R.A.	Transcort. aphasia	40	139	22	59	44
S.L.	Global aphasia	p.u.	p.u.	p.u.	p.u.	38
S.R.	Transcort. aphasia	39	147	29	67	87
S.A.	Broca's aphasia	36	101	19	29	46
S.O.	No aphasia	1	140	82	118	119
S.N.	No aphasia	2	149	89	114	118
S.V.	Broca's aphasia	37	97	18	10	72
S.S.	Broca's aphasia	40	94	31	43	53

(continued)

Table 2 Continued

Patient	AAT					
	Output	Token	Repetition	Writing	Naming	Comprehension
S.T.	Broca's aphasia	21	73	25	0	64
T.R.	No aphasia	n.a.	n.a.	n.a.	n.a.	n.a.
U.L.	Broca's aphasia	36	122	15	88	50
V.I.	Broca's aphasia	7	100	47	98	108

Italian norms (Luzzatti *et al*., 1996); Token = token subtest; Transcort. = transcortical; n.a. = not administered.

Table 3 Summary of all patients' performance on praxic tests

Patients	Ideomotor apraxia						Ideational apraxia De Renzi *et al*. (1968)
	De Renzi *et al*. (1980)			Tessari *et al*. (2011)			
	Meaningful	Meaningless	Total	Meaningful	Meaningless	Total	
A.N.	36	34	70				14
B.V.				28	31	59	12
B.R.	27	26	53				14
B.U.				17	19	36	9
B.Z.				19	25	44	14
C.A.				35	32	67	14
C.N.	35	28	63				14
C.C.				12	19	31	7
C.G.	32	31	63				14
C.O.				20	29	49	14
C.R.				25	18	43	11
C.S.				34	31	65	13
C.U.	30	31	61				14
C.Z.				20	24	44	11
D.C.R.	33	28	61				14
D.C.C.				18	30	48	14
D.B.				36	36	72	14
D.P.	33	36	69				14
D.O.	36	36	72				14
D.R.	25	29	54				8
F.R.				28	28	56	14
F.N.	33	27	60				14
F.U.	30	30	60				14
F.L.				32	32	64	14
F.S.	26	26	52				14
G.C.				25	28	53	14
G.V.	26	30	56				12
G.O.				24	28	52	5
G.U.				35	34	69	14
H.B.				23	17	40	12
J.N.				15	20	35	14
L.E.	33	35	68				14
L.N.	32	31	63				14
L.I.				25	33	58	14
L.C.	32	30	62				14
M.N.	36	31	67				14
M.R.				35	36	71	11
M.E.	35	31	66				14
M.L.				33	34	67	14
N.V.				26	26	52	12
O.B.				17	25	42	14
P.A.				36	36	72	14

(continued)

Table 3 Continued

Patients	Ideomotor apraxia						Ideational apraxia De Renzi et al. (1968)
	De Renzi et al. (1980)			Tessari et al. (2011)			
	Meaningful	Meaningless	Total	Meaningful	Meaningless	Total	
P.T.	27	24	51				12
P.R.	31	31	62				14
P.I.	33	25	58				14
R.A.				21	26	47	5
S.L.				5	27	32	3
S.R.	30	24	54				11
S.A.				26	29	55	14
S.O.				33	32	65	14
S.N.	35	34	69				14
S.V.	28	22	50				11
S.S.				33	32	65	14
S.T.	27	26	53				14
T.R.				36	32	68	14
U.L.				29	28	57	14
V.I.				18	16	34	10

masks ranged between 5 and 52, corresponding to a search area of 177 521 mm^3). We used permutation techniques (Kimberg *et al.*, 2007; Baldo *et al.*, 2010) to apply to our data a family-wise correction for multiple comparisons at the voxel level. Specifically, we randomly reassigned patients' behavioural scores 5000 times and, for each permutated data set, we refit the general linear model and recorded the largest *t*-value across all voxels. *T*-values in the original unpermuted data set were considered as significant only if they exceeded the 95th percentile of the largest-*t* distribution collected in the permuted data sets. Such analysis ensures that, if the null hypothesis is true (and, therefore, if no real relation between lesion and behaviour exists), the probability of a *t*-value to be larger than the cut-off across all voxels would be <5%. The analysis was carried out using the SnPM package (http://go.warwick.ac.uk/tenichols/snpm) implemented in SPM8 (http://www.fil.ion.ucl.ac.uk/spm/) freeware software.

As behavioural measures on different tests might correlate, their statistical maps are likely to describe overlapping neural structures. We therefore tested also the neural correlates using residual scores from which linear effects of the potentially confounding measures were removed. For instance, to isolate the neural structures that were involved in the imitation of meaningful gestures, accounting for potential confounds of patients' repetition performance, we first carried out a linear regression analysis with meaningful imitation as dependent variable and repetition as independent variable. The residual meaningful scores were then calculated and fed into the same voxel-based lesion-symptom mapping procedure described above. This approach ensures that the results of the mapping procedure cannot be considered confounded by repetition.

Results

Behavioural analysis

We found a significant correlation between patients' imitation of meaningful gestures and their performance on Naming (Meaningful–Naming: $r = 0.32$, $P < 0.02$). No relation was found between Naming performance and either the imitation of meaningless gestures (Meaningless–Naming: $r = 0.23$, not significant) or ideational apraxia (Ideational apraxia–Naming: $r = 0.10$, not significant). Similar results were found when taking into account patients' Repetition performance, which correlated with imitation of meaningful gestures (Meaningful–Repetition: $r = 0.37$, $P < 0.005$) but not with other praxic measures (Meaningless–Repetition: $r = 0.18$, not significant; Ideational apraxia–Repetition: $r = 0.08$, not significant). Instead, patients' Comprehension correlated with the imitation of meaningless gestures (Meaningless–Comprehension: $r = 0.30$, $P < 0.05$), but not with meaningful gestures (Meaningful-Comprehension: $r = 0.22$, not significant), or with ideational apraxia (Ideational apraxia–Comprehension: $r = 0.24$, not significant). Overall, we found that patients' linguistic performance and imitation performance correlated depending on the subtest taken into account. In particular, whereas patients' ability to imitate meaningful gestures only correlated with their Naming and Repetition performance, the imitation of meaningless gestures was correlated only with patients' Comprehension. Note that these were partial correlations in which the potential confounding effects of age, gender, education, lesion size and illness length were simultaneously controlled. Thus, positive correlations cannot be considered confounded by patients' aspecific cognitive impairment.

In the single case analysis, for each of the three tests considered (AAT, ideomotor apraxia, ideational apraxia) we found patients who showed a selective impairment in one of the three tests, with normal performance on the other two. The cut-offs considered for this analysis were derived from the tests, with no further correction (De Renzi *et al.*, 1968, 1980; Luzzatti *et al.*, 1996; Tessari *et al.*, 2011). Whereas six patients showed a selective aphasic impairment (Patients D.B., G.U., L.E., M.E., S.S. and U.L.), five patients showed ideomotor apraxia (Patients C.G., D.C.R., F.U., F.N. and O.B.) and only one patient (Patient M.R.)

showed ideational apraxia. The individual scores of the patients with selective impairments are reported in Table 4.

Lesion analysis

Unless stated otherwise, we report results that survived a height threshold corresponding to $P < 0.05$ family-wise corrected for multiple comparisons for the whole search area under rigorous non-parametrical permutation test (see 'Materials and methods' section). All suprathreshold effects are listed in Tables 5 and 6.

We focused on testing the neural correlates of patients' performance in each AAT subtest of interest (i.e. Repetition, Naming and Comprehension). The analysis of the lesions associated with impairments on all three subtests indicated the involvement of the superior temporal gyrus and the insular cortex (Fig. 2). Furthermore, whereas Naming and Repetition also implicated more dorsal and posterior regions, extending to the supramarginal gyrus, Comprehension was associated also with damage around the putamen.

We then assessed the neural structures associated with patients' praxic abilities (Fig. 3). Ideational apraxia performance was associated with damage to a restricted portion of the supramarginal gyrus and the superior temporal gyrus. A drop in imitation performance of meaningful gestures was associated with damage to an extended region, which included the supramarginal and the superior temporal gyri, the insula and the inferior frontal gyrus. Please note that the structures associated with meaningful imitation (and to a lesser extent with ideational apraxia) were also implicated when testing patients' linguistic skills (especially in Naming and Repetition subtests). This was not the case for imitation of meaningless gestures that, at least under rigorous correction for multiple comparisons for the whole search area [critical threshold: $t(55) > 4.05$], led to no suprathreshold regions. However, under a less conservative threshold [$t(55) > 3.25$, corresponding to $P < 0.001$ uncorrected], a drop in performance when patients imitated meaningless gestures was associated with lesions involving a portion of the angular gyrus [$x = -55$, $y = -58$, $z = 37$; $t(55) = 3.92$, cluster size 772 mm^3], located posteriorly and dorsally to the cluster in the supramarginal gyrus associated with the imitation of meaningful gestures (Fig. 3).

Given that patients' linguistic and praxic abilities, to a certain extent, correlated (see 'Behavioural analysis' section), we tested which brain regions were associated with patients' praxic proficiency when potential linguistic confounds were accounted for. This was achieved by feeding to the voxel-based lesion-symptom mapping procedure unconfounded values, which were obtained by removing from the original percentage scores in praxic tests putative linear effects of all three AAT subtests simultaneously (Repetition, Naming and Comprehension). Results are listed in Table 6. Following this approach no significant effects were found either for ideational apraxia [local maxima $t(55) = 4.19$; critical threshold $t(55) > 4.67$] or for the imitation of meaningful gestures [local maxima $t(55) = 3.42$; critical threshold $t(55) > 3.82$]. Instead the analysis of imitation of meaningless gestures led to significant effects in the angular gyrus (Fig. 4), over the same cluster previously isolated when testing meaningless original percentage scores (Fig. 3). Interestingly, this latter effect survived rigorous correction for multiple comparisons, although the previous angular cluster was identified under a more liberal threshold. Thus, controlling for potential confounds of linguistic abilities had beneficial effects for the analysis of the neural correlates of meaningless imitation. Figure 4 displays the average performance in meaningless imitation and in linguistic tests in patients with and without damage to the cluster's local maxima, and shows how lesions to the angular gyrus lead to a drop in performance exclusively in the imitation of meaningless gestures.

We further explored the role played by language in imitation of meaningful gestures and in tool use, not by considering Repetition, Naming and Comprehension together, but by controlling for one AAT subtest at a time. No suprathreshold effects were found for ideational apraxia, when controlling for Repetition, Naming and Comprehension alone. Likewise the analysis of performance on meaningful gestures failed to lead to significant effects when controlling for Naming or Repetition. Instead, small portions of the supramarginal gyrus, extending to the border of the superior temporal cortex, were found for imitation of meaningful gestures when controlling for Comprehension effects. Figure 4 displays the average performance on imitation of meaningful gestures and on linguistic tests in patients with and without damage to the supramarginal's local maxima. Our results clearly show how damage to the supramarginal gyrus is associated with a drop not only in imitation of meaningful gestures, but also in performing the AAT subtests. Interestingly, Comprehension performance seems the least impaired by the lesion. This might explain why supramarginal effects for imitation of meaningful gestures could still be found only when controlling for this AAT subtest.

Finally, using a similar approach we tested which brain regions were associated with patients' linguistic proficiency when potential praxic confounds were accounted for. This was achieved by inserting in the voxel-based lesion-symptom mapping procedure unconfounded values, which were obtained by removing from the original percentage scores on linguistic tests putative linear effects of imitation of meaningful or meaningless gestures and object use (ideational apraxia). Results (listed in Table 6) are strongly similar to those observed when testing AAT original percentage scores (displayed in Fig. 2), with the exception that the regions outlined were confined to the temporal and insular cortex (and, for the case of comprehension, also to the putamen) whereas no effect in the supramarginal gyrus was found.

Discussion

That language normally interacts with motor experience is widely accepted (Mahon and Caramazza, 2008; Pulvermüller and Fadiga, 2010; Bedny and Caramazza, 2011). The main evidence in support of this argument is based on group studies in which correlations and associations of aphasic and apraxic deficits have been documented in brain damaged patients (Saygin *et al*., 2004; Buxbaum *et al*., 2005; Negri *et al*., 2007; Papeo *et al*., 2010), and on imaging studies in which motor regions in the brain were activated when healthy individuals were presented with action-related words (Hauk *et al*., 2004; Tettamanti *et al*., 2005; Ruschemeyer *et al*., 2007; Postle *et al*., 2008; Péran *et al*., 2010; Willems *et al*.,

Table 4 Performance on linguistic and praxic tasks of patients who showed a selective deficit in one of these tasks and areas of the Brodmann areas lesioned in each of patient

	Patient	AAT output	Ideomotor apraxix	Ideational apraxia	Brodmann areas lesioned
Selective impairment on language	D.B.	**Wernicke's aphasia**	72	14	19, 20, 21, 22, 37, 39, 41, 42, 48
	G.U.	**Wernicke's aphasia**	69	14	20, 21, 22, 34, 38, 48
	L.E.	**Wernicke's aphasia**	68	14	19, 20, 21, 22, 37, 39, 40, 41, 42, 48
	M.E.	**Broca's aphasia**	66	14	2, 3, 4, 6, 20, 22, 38, 41, 42, 43, 45, 47, 48
	S.S.	**Broca's aphasia**	65	14	6, 11, 20, 22, 25, 34, 41, 42, 43, 47, 48
	U.L.	**Broca's aphasia**	57	14	11, 20, 25, 34, 47, 48
Selective impairment on gesture imitation	C.G.	No aphasia	**63**	14	7, 19, 39, 40
	D.C.R.	No aphasia	**61**	14	20, 34, 48
	F.U.	No aphasia	**60**	14	10, 11, 32, 45, 46, 47, 48
	F.N.	No aphasia	**60**	14	1, 2, 3, 4, 5, 6, 7, 17, 18, 19, 20, 21, 22, 23, 26, 29, 30, 37, 39, 40, 41, 42, 48
	O.B.	No aphasia	**42**	14	20, 27, 37, 41, 48
Selective impairment on tool use	M.R.	No aphasia	71	**11**	48

Impaired performance is highlighted in bold.

Table 5 Voxel-based lesion symptom mapping: effect of each subtest

	Coordinates			t(55)	Cluster size [mm^3]
	x	y	z		
Comprehension (critical $t = 4.25$)					
Superior temporal gyrus	−51	−13	−2	6.20	13 970
Insular cortex	−39	5	−4	5.65	
Putamen	−25	18	9	5.03	1508
Naming (critical $t = 4.07$)					
Superior temporal gyrus	−53	−9	−3	7.98	45 360
Middle temporal gyrus	−67	−28	2	6.32	
Insular cortex	−43	4	−4	7.15	
Supramarginal gyrus	−60	22	32	4.43	
Repetition (critical $t = 4.41$)					
Superior temporal gyrus	−59	−32	17	8.08	56 585
Middle temporal gyrus	−65	−28	3	7.97	
Supramarginal gyrus	−59	−40	31	6.68	
Insular cortex	−44	−12	8	6.07	
Ideational apraxia (critical $t = 5.11$)					
Supramarginal gyrus	−65	−46	32	5.79	288
Superior temporal gyrus	−42	−24	6	5.47	255
Meaningful gestures (critical $t = 4.19$)					
Supramarginal gyrus	−64	−45	33	5.53	19 733
Temporo-parietal junction	−64	−31	24	5.28	
Insular/opercular cortex	−45	18	−3	4.80	4631
Inferior frontal gyrus	−41	18	19	4.79	

Regions significantly associated with patients' performance in AAT, ideomotor apraxia and ideational apraxia subtests. Coordinates (in standard MNI space) refer to maximally activated foci in *t*-map: *x* = distance (mm) to the right (+) or the left (−) of the midsagittal line; *y* = distance anterior (+) or posterior (−) to the vertical plane through the anterior commissure; *z* = distance above (+) or below (−) the intercommissural (anterior–posterior commissure) line. All listed regions are in the left hemisphere.

Table 6 Voxel-based lesion symptom mapping: dissociated effects

	Coordinates			*t*(55)	Cluster size [mm^3]
	x	*y*	*z*		
Meaningless gestures, controlling for repetition, naming and comprehension (critical $t = 4.05$)					
Angular gyrus	−48	−55	46	4.07	2
Meaningful gestures, controlling for comprehension only (critical $t = 3.92$)					
Supramarginal gyrus	−39	−38	38	4.15	181
Temporo-parietal junction	−65	−31	24	3.98	126
Comprehension, controlling for ideational apraxia, imitation of meaningful and meaningless gestures (critical $t = 4.29$)					
Putamen	−22	19	5	4.76	1483
Insular cortex	−37	5	−6	4.86	267
Superior temporal gyrus	−51	−13	−3	4.52	123
Naming, controlling for ideational apraxia, imitation of meaningful and meaningless gestures (critical $t = 4.12$)					
Superior temporal gyrus	−52	−10	−4	5.68	6708
Middle temporal gyrus	−66	−28	1	4.58	
Insular cortex	−42	5	−5	5.25	
Repetition, controlling for ideational apraxia, imitation of meaningful and meaningless gestures (critical $t = 4.20$)					
Superior temporal gyrus	−58	−32	16	4.61	7123
Middle temporal gyrus	−65	−11	0	5.83	
Insular cortex	−44	−13	1	4.74	675

Regions significantly implicated in (i) patients' praxic abilities when potential linguistic confounds are accounted for; and (ii) patients' linguistic abilities when potential praxic confounds are accounted for.

2010). However, when performance is analysed at the level of individual patients, double dissociations between linguistic ability and praxic ability have repeatedly been observed (de Ajuriaguerra *et al*., 1960; Kertesz *et al*., 1984; Papagno *et al.* 1993; Negri *et al*., 2007; Papeo *et al*., 2010; Papeo and Rumiati, 2013). With the present study we aimed at offering an alternative account that reconciles apparently conflicting associations and dissociations patterns previously reported in the literature.

Fifty-seven left-brain damaged patients performed tests exploring their linguistic and praxic abilities. Presence of aphasia was assessed through three AAT subtests tapping patients' repetition, naming and comprehension wheras ideomotor apraxia was assessed by engaging patients in imitation of meaningful and meaningless actions. In the present study patients were not asked to imitate transitive gestures or to pantomime on verbal command, to avoid the possible interference of aphasia with the apraxia assessment. Moreover, the possible presence of ideational apraxia was tested by asking patients to use real objects. At the group level, we carried out partial correlation analyses that revealed how patients' performance on naming and repetition (but not on comprehension) significantly correlated with imitation of meaningful gestures, while imitation of meaningless gestures mildly correlated with patients' performance on comprehension (but not on naming and repetition).

Moreover, voxel-based lesion-symptom mapping analysis revealed a dissociation between the left angular gyrus, whose damage specifically affected imitation of meaningless gestures (but not linguistic abilities), and the left superior temporal sulcus and the insula, whose damage specifically affected linguistic (but not praxic) proficiency. In contrast, there was no specific region that, when lesioned, led to a deficit in imitating meaningful gestures that was not also causing a linguistic deficit. However, damage to the supramarginal gyrus affected imitation of meaningful gestures (damage to this region was also associated with a drop in patients' naming and repetition), and damage to the superior temporal gyrus and the insular cortex was associated with poor scores on linguistic tests.

With this study we clearly demonstrated that praxic performance and linguistic performance tend to associate when the gesture to be imitated has a meaning for the imitator, and dissociate when the gesture to be imitated carries no meaning.

Towards a reconciliation of embodied and disembodied hypotheses

There are two alternative views competing over the interpretation of why motor and premotor regions are recruited during linguistic tasks. According to the grounded or embodied accounts of language understanding (Gallese and Lakoff, 2005; Pulvermüller, 2005; Barsalou, 2008), motor activations play a particularly critical role in understanding motor-related words. Reading or listening to the verb 'to grasp', for instance, automatically activates, through a motor resonance mechanism, the motor representation that is associated with the word. Within this approach, the word–motor coupling is said to have been established during a life-long Hebbian learning: exposure to the word acts as a probe that re-enacts the associated motor representation.

In contrast, neuropsychological studies (Negri *et al.* 2007; Papeo *et al.* 2010; Papeo and Rumiati, 2013) showed that the integrity of motor representations is not necessary to understand action words. More recently, transcranial magnetic stimulation and

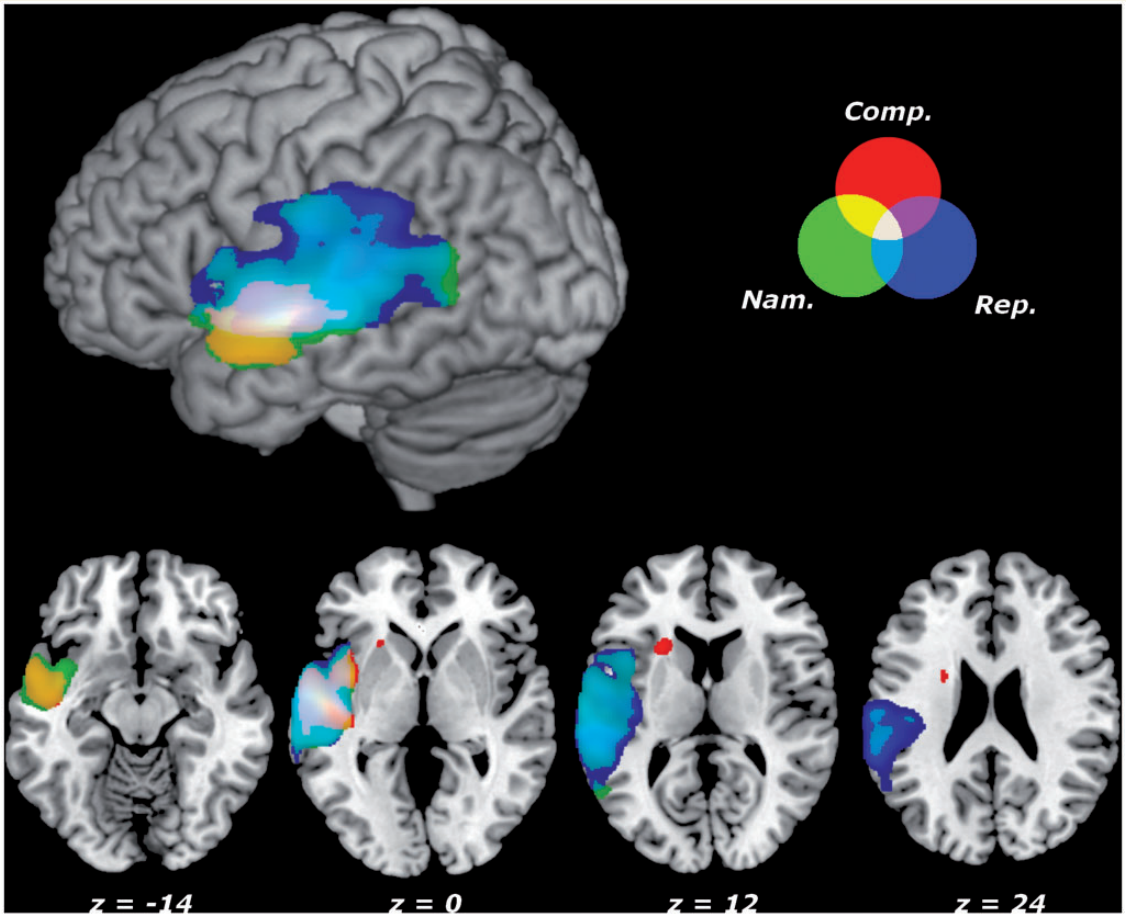

Figure 2 Surface rendering and axial ($z = -12; 0; 12; 24$) sections displaying regions whose damage is associated with a significant drop in performance in AAT subtests. Red, green and blue areas refer to Comprehension (Comp), Naming (Nam) and Repetition (Rep), respectively. Dark yellow describes regions associated with pathological Comprehension and Naming, whereas light blue describes regions associated with pathological Repetition and Naming. Light pink describes regions associated with an impairment in all three subtests.

imaging studies demonstrated that the involvement of the motor system in action word understanding is not fast and it is contingent upon the task or the context in which the word is presented (Ruschemeyer *et al*., 2007; Tomasino *et al*., 2008, 2010; Papeo *et al*., 2009, 2011, 2012; Raposo *et al*., 2009). Using functional MRI, Tomasino *et al*. (2010), for instance, showed that when participants silently read action verbs the activations of motor and premotor cortices were modulated by the context in which those verbs were inserted. More specifically, motor and premotor activations decreased in response to action verbs presented as negative imperatives (e.g. 'Don't write'), in comparison with positive imperatives ('Do write'). In a different functional MRI study (Papeo *et al*., 2012), participants were asked to silently read action and state verbs and, before words were presented, they had to perform a mental rotation task, using either a motor or a non-motor strategy. The type of strategy used induced a particular cognitive context that could be transferred to the immediately subsequent verb reading. Indeed, results showed that reading after the motor strategy was applied led to an increase of the activation in primary motor, premotor and somatosensory cortices compared with reading after the non-motor strategy. The activations of the motor systems were independent of the identity of the verb, being action or state verb, thus suggesting a predominance of the context over the semantics of the stimuli presented. To conclude this section, there is strong evidence now suggesting that motor resonance for linguistic stimuli can be dependent on contextual factors and that the locus of the interaction between language and action is not the system for action production (see also Papeo *et al*., 2010; Papeo and Rumiati, 2013).

With the present study, we offer a novel interpretation of how the action system and language system may interact. Although the differential processing of action versus non-action verbs was analysed in other studies performed by our group (Papeo *et al*., 2009, 2011, 2012), in the present study we focused on how imitation of meaningful and meaningless gestures may be influenced by different linguistic processes. The correlational results suggest that imitation of familiar gestures relies on the language system, most likely because these gestures are linked to the corresponding lexical-semantic representations. Thus, as shown by our data, there is an overlap between brain regions underlying linguistic abilities and the ability to imitate meaningful gestures. In contrast, imitation of unknown gestures cannot rely on lexical-semantic

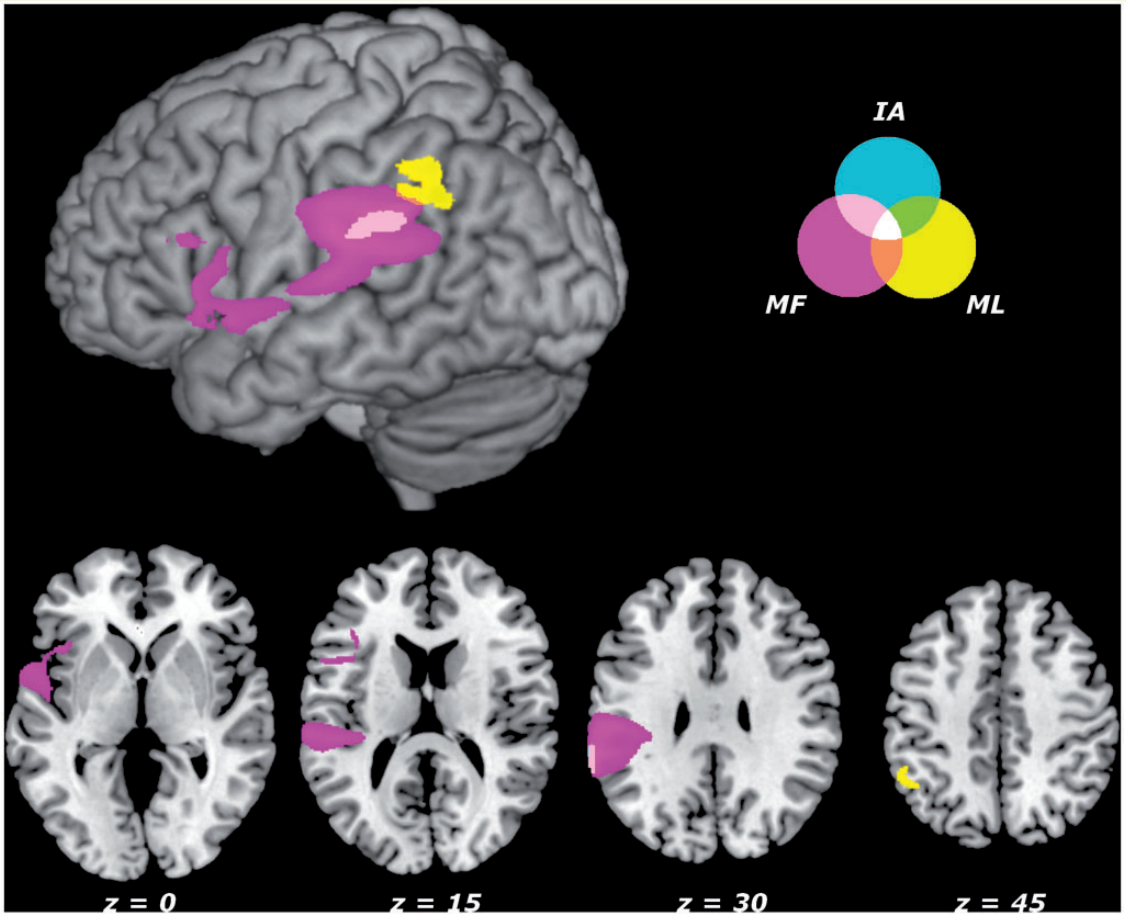

Figure 3 Surface rendering and axial ($z = 0$; 15; 30; 45) section displaying regions whose damage is associated with a significant drop in performance in ideomotor apraxia and ideational apraxia tests. Cyan, magenta and yellow areas refer to ideational apraxia (IA), imitation of meaningful (MF) and meaningless (ML) gestures, respectively. Light pink describes regions associated with an impairment in both ideational apraxia and imitation of meaningful gestures. Areas associated with impairment in imitation of meaningless gestures are displayed at a less stringent threshold, corresponding to $P < 0.001$ uncorrected.

representations, as they are novel to the patients, but on a visuo-motor conversion mechanism whose neural correlate has often been identified with the angular gyrus (Goldenberg and Hagmann 1997; Peigneux *et al*., 2004; Rumiati *et al*., 2005; Tessari *et al*., 2007). This pattern of results can be fully explained within the dual-pathway model of gesture imitation (Tessari and Rumiati, 2004; Tessari *et al*., 2007), that predicts relatively independent neurocognitive mechanisms for familiar and unknown stimuli.

Imitation is not a single process

According to cognitive models of praxis (Rothi *et al*., 1991; Cubelli *et al*., 2000; Buxbaum, 2001; Rumiati *et al*., 2010), different processing stages are required depending on the type of stimulus to be processed and the response to be produced. Thus, the pattern of deficits in reproducing gestures shown by neuropsychological patients can be explained by the disruption of one or more of these processing stages. Our study provides novel evidence for how the neural structures underlying imitation of meaningful and meaningless gestures comprise different subportions of the inferior parietal cortex, with the more dorsal portion of the angular gyrus ($z = 37$, 46) specifically implicated in the imitation of meaningless gestures, and the ventral/anterior portion of the supramarginal gyrus ($z = 33$, 38) associated with meaningful gestures (Figs 3 and 4). Consistently, the large group of patients that took part in our study ($n = 57$) allowed us to better characterize the findings of previous neuropsychological reports, in which damage to the inferior parietal cortex was associated with either type of gesture (Goldenberg and Hagmann, 1997; Tessari *et al*., 2007).

The functional differentiation of the parietal cortex, along the dorsal-to-ventral axis, as dependent on the semantic content of a gesture is consistent with the one provided by previous neuroimaging studies on healthy volunteers (Peigneux *et al*., 2004; Rumiati *et al*., 2005). When testing for meaningful (versus meaningful) gestures (Rumiati *et al*., 2005), or for gestures which were novel (versus familiar) to the performer (Peigneux *et al*., 2004), an increase of neural activity in the intraparietal and superior parietal cortex (average z from all local maximas ~50), together with the

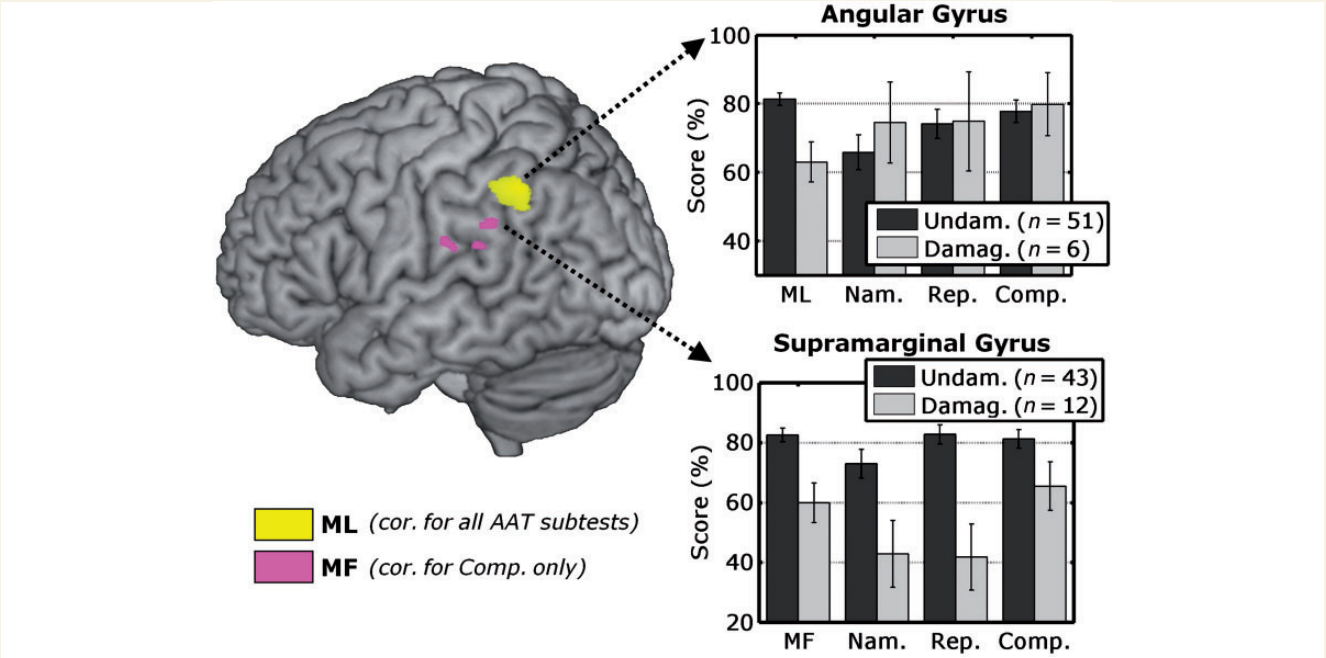

Figure 4 Surface rendering displaying regions associated with praxic effects when potential linguistic confounds are accounted for. Yellow regions refer to effects of imitation of meaningless (ML) gestures while controlling for Repetition (Rep), Naming (Nam) and Comprehension (Comp). Magenta regions refer to effects of imitation of meaningful (MF) gestures while controlling for Comprehension only. Regions are displayed with the same threshold as in Fig. 2 (for magenta areas, $P < 0.05$ corrected; for yellow areas, $P < 0.001$, uncorrected). For each region we also plotted patients' average performance in praxic and linguistic tests against the presence/absence of a lesion in the region's local maxima. Dark grey bars refer to the performance of those patients in which the region is undamaged, whereas light grey bars refer to those patients in which the region is damaged. Graph legends also report how many patients fall in each category. Error bars refer to standard errors of the mean.

precuneus, the inferior temporal gyrus and the parahippocampal gyrus, was observed. However, when testing the imitation of meaningful (versus meaningless), or familiar (versus novel) gestures, the authors reported activations of the inferior parietal cortex (average $z \sim 37$), as well as the middle frontal gyrus, the left superior temporal gyrus, right parieto-occipital and occipito-temporal junctions.

Our results clarify the previous functional findings in two ways. First, lesion data reveal the causal contribution of the different parietal portions towards the individuals' ability to reproduce gestures. Recent studies (Rizzolatti and Matelli, 2003; Binkofski and Buxbaum, 2012; Mahon *et al*., 2013) suggested that the dorsal pathway in the parietal cortex can be divided into two specialized components, the dorso-dorsal stream and the ventro-dorsal stream. The parietal cluster found in the present study is part of the ventro-dorsal stream, thus suggesting a further specialization of this pathway when gesture imitation is considered. Second, our results clearly indicate that only imitation of meaningful gestures and its neural correlates are influenced by the lexical-semantic processes. As we have argued above, these findings are accommodated within the dual-pathway model of gesture imitation (Tessari and Rumiati, 2004; Tessari *et al*., 2007), with the angular gyrus being associated with the direct pathway triggered by any gesture irrespective of its content but prevalently recruited for meaningless gestures, and the supramarginal gyrus being recruited by the semantic pathway, whenever the gesture to be is reproduced by relying on the corresponding lexical-semantic representation.

Dissociating patterns

In general, associations of aphasic and apraxic deficits are often observed in brain-damaged patients. However, evidence that the linguistic abilities and the ability to imitate gestures, as a key test for ideomotor apraxia, can dissociate has also been documented (de Ajuriaguerra *et al*., 1960; Kertesz *et al*., 1984; Papagno *et al*., 1993; Negri *et al*., 2007; Papeo *et al*., 2010; Papeo and Rumiati, 2013), thus suggesting the independence of these two abilities. Likewise dissociations between linguistic abilities and the ability to use objects, as the key test for the presence of ideational apraxia, have also been known since many years (Negri *et al*., 2007; Papeo *et al*., 2010). Moreover, even though ideational apraxia is less common than ideomotor apraxia (defined here as a prevalent deficit in using objects and in gesture imitation, respectively), they have been observed to dissociate in patients (De Renzi *et al*., 1968; De Renzi and Lucchelli, 1988; Rumiati *et al*., 2001; Lunardelli *et al*., 2011) thus ruling out the account that ideational apraxia is simply a more severe form of ideomotor apraxia (Sitting, 1931; Zangwill, 1960).

Our present data confirm that aphasia, ideomotor apraxia and ideational apraxia can be observed in isolation. Dissociations are fundamental in neuropsychological studies, as they provide strong evidence in support of functional independence of the dissociating abilities (Shallice, 1988). However, our findings go beyond the inference that dissociating processes must be associated with discrete neural substrates but they rather suggest that, even when considering independent processes, these brain networks can partially overlap. Thus, functional independence does not exclude the possibility of an interaction between the systems, as showed by the lack of brain correlates exclusively dedicated to the imitation of meaningful gestures (controlling for the influence of language).

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