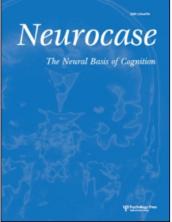
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V. Moro <sup>a</sup>; S. Pernigo <sup>b</sup>; C. Urgesi <sup>c</sup>; P. Zapparoli <sup>d</sup>; S. M. Aglioti <sup>e</sup> <sup>a</sup> Dipartimento di Psicologia ed Antropologia Culturale, University of Verona, Verona, Italy <sup>b</sup> Dipartimento di Scienze Neurologiche e della Visione, Sezione Fisiologia Umana, University of Verona, Verona, Italy <sup>c</sup> Dipartimento di Filosofia, University of Udine and IRCCS 'E. Medea', San Vito al Tagliamento (PN), Italy <sup>d</sup> Servizio di Medicina Fisica e Riabilitazione, Ospedale S.Cuore Don Calabria, Negrar (VR), Italy <sup>e</sup> Dipartimento di Psicologia, University 'La Sapienza' and IRCCS Fondazione S. Lucia, Roma, Italy

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# Finger recognition and gesture imitation in Gerstmann's syndrome

V. Moro,<sup>1</sup> S. Pernigo,<sup>2</sup> C. Urgesi,<sup>3</sup> P. Zapparoli,<sup>4</sup> and S. M. Aglioti<sup>5</sup>

<sup>1</sup>Dipartimento di Psicologia ed Antropologia Culturale, University of Verona, Verona, Italy <sup>2</sup>Dipartimento di Scienze Neurologiche e della Visione, Sezione Fisiologia Umana, University of Verona, Verona, Italy

<sup>3</sup>Dipartimento di Filosofia, University of Udine and IRCCS 'E. Medea', San Vito al Tagliamento (PN), Italy

<sup>4</sup>Servizio di Medicina Fisica e Riabilitazione, Ospedale S.Cuore Don Calabria, Negrar (VR), Italy <sup>5</sup>Dipartimento di Psicologia, University 'La Sapienza' and IRCCS Fondazione S. Lucia, Roma, Italy

We report the association between finger agnosia and gesture imitation deficits in a right-handed, righthemisphere damaged patient with Gerstmann's syndrome (GS), a neuropsychological syndrome characterized by finger and toe agnosia, left-right disorientation and dyscalculia. No language deficits were found. The patient showed a gestural imitation deficit that specifically involved finger movements and postures. The association between finger recognition and imitation deficits suggests that both static and dynamic aspects of finger representations are impaired in GS. We suggest that GS is a disorder of body representation that involves hands and fingers, that is, the non-facial body parts most involved in social interactions.

Keywords: Gerstmann's syndrome; Finger agnosia; Body representation.

#### INTRODUCTION

The association of finger agnosia, left-right confusion, agraphia and dyscalculia characterizes Gerstmann's syndrome (GS) (Gerstmann, 1924; Mayer et al., 1999), which typically occurs as a consequence of vascular lesions involving the angular gyrus of the dominant hemisphere. The nature and occurrence of the syndrome have been debated for many years (Mayer et al., 1999) because it often emerges in an incomplete form or in association with other cognitive deficits. Studies report patients with GS not only after lesions involving the angular gyrus, but also after left parietal or thalamic strokes (Casado, Jarrin, Madrid, & Gil-Peralta, 1995), tumors involving the dominant parietal area (Russell, Elliott, Forshaw, Kelly, & Golfinos, 2005), and traumatic brain damage (Mazzoni et al., 1990) in addition to systemic etiologies (Jung et al., 2001) and Alzheimer's disease (Wingard, Barrett, Crucian, Doty, & Heilman, 2007). Moreover, a form of 'developmental Gerstmann's syndrome' has been described in children with learning disabilities (Suresh & Sebastian, 2000). Reports of patients with GS following damage to the right hemisphere are rare and generally concern ambidextrous or left-handed subjects (Dozono, Hachisuka, Ohnishi, & Ogata, 1997;

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Address correspondence to V. Moro, Dipartimento di Psicologia e Antropologia Culturale, University of Verona, Lungadige Porta Vittoria 17, 37129 Verona, Italy. (E-mail: valentina.moro@univr.it).

Moore et al., 1991). Neuropsychological research indicates that different kinds of information contribute to body representations. Indeed, semantic, somatosensory, visuo-spatial and motor knowledge interact in the construction of body representation (Sirigu, Grafman, Bressler, & Sunderland, 1991). Three distinct types of body-related constructs have been postulated (Schwoebel & Coslett, 2005). The first, known as body image, refers to the semantic representation of the body, such as knowledge of the names of body parts, their functions, and their relationships with objects. The second, called body schema, refers to the dynamic representation of the relative position of body parts, which depends on multiple sensory and motor inputs and their interaction with the planning and execution of actions. The third, called body structural description, is a topological map of body locations, which depends primarily on visual inputs, that defines body part boundaries and proximity relationships. While body image and body structural representations seem to rely on the left hemisphere, specifically the temporal lobe (Buxbaum & Coslett, 2001; Schwoebel & Coslett, 2005), body schema seems to be linked to the right hemisphere, in particular the dorso-lateral frontal cortex and the parietal areas (Schwoebel & Coslett, 2005). GS, a condition in which deficits in the semantic representation of the body are typically linked to left hemisphere damage, is considered a disorder of body image (Coslett, Saffran, & Schwoebel, 2002). This is in keeping with a study in which tasks tapping body image and body schema representations were performed by subjects who observed themselves in normal or inverted mirrors. The study demonstrated that in these specific situations right brain-damaged patients exhibit symptoms of body schema deficits and disorders of visuo-spatial body structural representation components rather than body image disorders (Beis, Paysant, Bret, Le Chapelain, & André, 2007).

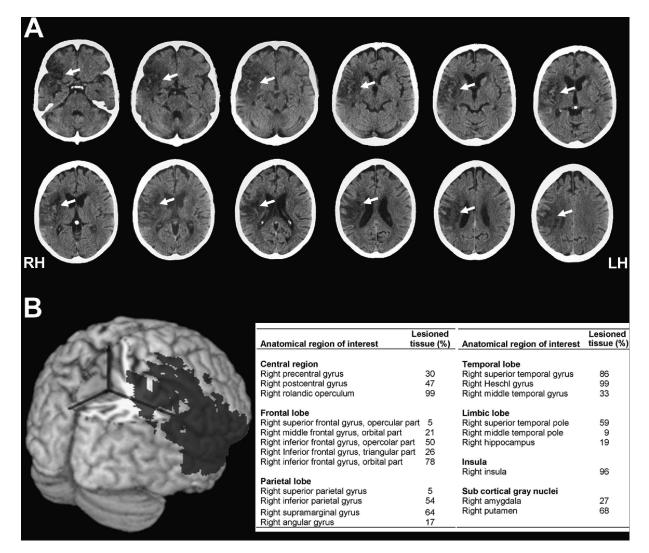
Here we describe a right-handed patient (A.N.) in whom GS is associated with right brain damage. We sought to determine the difference in the influences of semantic as opposed to visuo-spatial representations on the Gerstmann symptoms tetrad by testing distinct components of body representation. Moreover, we adopted an influential model of complex movement production and imitation, namely, the two-route model (Rothi, Ochipa, & Heilman, 1991), to explore the relationship between GS and gestural imitation abilities. This model postulates that the verbal request to perform an action activates the *semantic route*, which is also used for imitation of meaningful gestures. By contrast, the non-semantic (direct) route is the only possible substrate for the imitation of meaningless gestures. Therefore, in the absence of specific semantic representations, the gesture is produced by means of a direct translation of visual inputs into motor outputs. Moreover, two specific predictions have been posited. The first is that body schema disorders are associated with deficits in imitating meaningless gestures and the second that body image disorders are associated with deficits in imitating meaningful gestures (Schwoebel, Buxbaum, & Coslett, 2004). In fact, while the imitation of meaningless gestures is likely related only to dynamic components of body representation, the imitation of meaningful gestures can also be implemented by using semantic body knowledge. We extensively tested patient A.N. in tasks of gesture execution on verbal command and by imitation. This allowed us to detect any dissociations between meaningful and meaningless gesture execution and between the execution of meaningful gestures on verbal command vs. imitation. Moreover, as deficits in meaningless gesture execution are likely associated with body schema deficits (Schowebel, 2004), we compared the performance of patient A.N., who mainly exhibited body image disorders, with that of patient F.C., who presented with anosognosia for hemiplegia, a specific disorder of body schema. Finally, we sought to determine the possible relationship between body representation disorders and specific finger recognition deficits.

#### METHODS AND RESULTS

#### Case report

A.N. is a 78-year-old, right-handed woman who, despite her age, was still working in her ice cream shop when, in January 2006, she had a hemorrhagic stroke in the territory of the right middle cerebral artery and suffered from motor and sensory deficits on the left side of her body. Three months later, when she came under our observation, she was alert and oriented. A CT scan showed a wide frontotemporo-parietal lesion in the right hemisphere that spared the left hemisphere (Figure 1).

Neuropsychological screening established that the patient was not left-handed or ambidextrous (Briggs & Nebes, 1975) and excluded the presence of mental deterioration (Folstein, Folstein, & McHugh, 1975)



**Figure 1.** A.N.'s CT scan performed 30 days after stroke. A) Select transverse cuts showing the lesion (indicated by white arrows). Right hemisphere is on the left side. Note the large cortical and subcortical lesion involving frontal, temporal and parietal structures. Infarction territory was limited to the right hemisphere; the left hemisphere was completely spared. B) Three-dimensional reconstruction of A.N.'s lesion superimposed on the T1-weighted template MRI scan from the Montreal Neurological Institute (right hemisphere is on the right side). Lesion mapping was performed using MRIcro software (Rorden & Brett, 2000) by an examiner who ignored any clinical feature of the patient (Rorden & Brett, 2000). We superimposed the lesion on the Automatic Anatomical Label template provided with MRIcro and calculated the number of lesioned voxels in each anatomical region of interest. The table inserted in the lower right corner of the figure shows the percentage of lesioned tissue for each area affected by the lesion.

(Table 1). Verbal memory was preserved (Spinnler & Tognoni, 1987). Moreover, A.N. had no difficulty performing the Ideational (De Renzi & Lucchelli, 1988), Ideomotor and Oral Apraxia tests (Spinnler & Tognoni 1987). Her language abilities appeared to be preserved, as tested by the Aachener Aphasia Test (Luzzatti, Willmes, & De Bleser, 1991). Nevertheless, a few tasks from the Aachener Test (Token test, Written Comprehension) could not be administered because of the patient's spatial disorders.

Symptoms of left extra-personal neglect were recorded in Line Crossing, Figure and Shape Copying and Representational Drawing (Wilson, Cockburn, & Halligan, 1987). Furthermore, the patient presented with constructional apraxia (Spinnler & Tognoni, 1987), spatial agraphia and alexia. She also showed left visual and tactile extinction of a contralesional stimulus delivered simultaneously with an ipsilesional stimulus. No clear signs of personal neglect were found in the Comb and Razor

 TABLE 1

 Neuropsychological screening

	Patients			
Neuropsychological Screening	<i>A.N.</i>	<i>F.C.</i>		
General Cognitive State				
M.M.S.E. (<21*)	25	21.9		
Verbal Span (<2.75*)	4	4		
Prose Memory (≤11.75*)	13.4	10		
Apraxia (Number of trials)				
Apraxia, ideational task (14)	13	14		
Apraxia, ideomotor task (20)	20	20		
Apraxia, oral task (20)	20	20		
Constructional Apraxia (14)	6	5		
Language (AAT) (Number of trials)				
Oral comprehension (60)	54	Np		
Denomination (120)	116	Np		
Reading (30)	25	Np		
Writing (30)	27	Np		
Repetition (150)	149	Np		
Extrapersonal Neglect				
Line crossing (<34*)	33	6		
Figure and shape copying (<3*)	1	2		
Representational drawing (<2*)	1	0		
Extinction (Number of trials)				
Visual Extinction (10)	0	0		
Tactile Extinction (10)	0	0		
Personal Neglect				
Fluff Test (≤13*)	14	Np		
Comb & Razor (≤–0.11*)	-0.19	-0.53		
Awareness				
Anosognosia Interview	23/23	4/23		

Results on the different tests of patients A.N. and F.C. are reported in the second and third column, respectively. Pathological scores, i.e., below the 5th percentile of normal distribution, are indicated in bold; Np, not performed test; \*indicates the cut-off values.

test (McIntosh, Brodie, Beschin, & Robertson, 2000) or in the Fluff Test (Cocchini, Beschin, & Jehkonen, 2001). To sum up, the patient presented with signs of left extra-personal neglect, a condition typically associated with right hemisphere lesions (Bartolomeo, Thiebaut De Schotten, & Doricchi, 2007). By contrast, cognitive abilities typically lateralized in the left hemisphere (language, praxis, semantic memory) appeared to have been spared, in keeping with a pattern of typical hemispheric lateralization (Table 1).

Surprisingly, patient A.N. also presented with signs of GS, which is typically associated with left hemisphere lesions. In fact, the occurrence of GS following right hemisphere damage has so far only been reported in patients with reversed cerebral dominance (Moore et al., 1991) or mixed hand preference (Dozono et al., 1997).

#### Gerstmann's syndrome assessment

During the different testing sessions, patient A.N. was seated at a table on which her hands were laid. We ascertained the presence of the tetrad of symptoms by using the tests described below.

#### Finger agnosia

In accordance with previous studies (Mayer et al., 1999), we explored A.N.'s capacity to recognize individual fingers using three types of instructions (verbal, visual, tactile) and three modes of response (verbal, pointing to one's own hand, pointing to specific fingers on a diagram showing the outline of a hand) (Table 2). Finger recognition upon verbal instruction was assessed by asking the patient to denominate the finger used in a specific situation (e.g., the hitchhiking sign used to indicate the direction; see T2.1, Table 2). Moreover, she was requested to indicate the finger named by the examiner on her hand or on the diagram (T2.2–T2.3). In the finger recognition test (with visual instruction) the examiner pointed to a specific finger on the patient's hand and she had to denominate the finger (T2.4) or point to the corresponding finger on the diagram (T2.5).

In tasks 2.6 and 2.7, the examiner either pointed to his own hand or to the diagram and A.N. had to indicate the corresponding finger on her hand. Both hands were tested in all tasks. In the tactile instruction task, the fingers of A.N.'s right hand were touched. The patient was requested to denominate (T2.8) or to indicate the corresponding finger on the diagram (T2.9) or on her contralateral hand (T2.10). Only the right hand was stimulated to avoid the possible effect of left hand somatosensory deficits. To exclude the presence of minor tactile deficits, the patient was also asked to localize the stimulus, to detect touch from a sliding stimulus and to discriminate between single or double stimulation. Although her right-hand performance was perfect in the first two tasks, the patient reported only one touch during double tactile stimulation because of tactile extinction. In all these situations, each finger was stimulated five times in a pseudo-random order. Toe agnosia was investigated by asking A.N. to denominate the toe indicated by the examiner on her foot (T2.11) or to point to it on a diagram (T2.12). We did not use verbal instructions for this task because only the hallux has a specific name in Italian. The other four toes are denominated following a progressive order (second, third, fourth, fifth). Using verbal

Т	Finger agnosia	Left	Right
	Verbal instruction (number of trials)		
2.1	Verbal response (10)		(5)*
2.2	Pointing to one's own hand (25)	16	13
2.3	Pointing on a diagram (25)	17 <b>33/50</b>	16 <b>29/50</b>
	Visual instruction		
2.4	Verbal response (25)	15	13
2.5	The examiner points to specific patient's finger. The patient indicates the correspondent body part on a diagram (25)	11	17
2.6	The examiner points to one of his fingers. The patient points to the correspondent finger on her own hand (25)	19	19
2.7	The examiner points to a specific finger on a diagram. The patient points to the correspondent part on her own hand (25)	22	25
		67/100	74/100
	Tactile instruction		
2.8	Verbal response (25)	Np	13
2.9	The examiner touch to one of her fingers. The patient indicates the correspondent body part on a diagram (25)	Np	18
2.10	The examiner touch to one of her fingers. The patient points to contralateral hand (25)	Np	24
			55/75
	Toe agnosia		
	Visual instruction		
2.11	Verbal response (25)	19	20
2.12	The examiner points to a specific patient's toe. The patient indicates the correspondent toe on a diagram (25)	16	14

 TABLE 2

 A.N.'s performance in finger and toe agnosia tasks

Left, responses referring to left hand; Right, responses referring to right hand; values in parentheses indicate the total number of trials in each test; \*indicates responses not referring to the left or right hand; Np, not performed tasks.

tasks does not therefore prove that verbal instructions tap semantic rather than spatial knowledge. Moreover, tactile localization of the toes was not required because this is very difficult for the elderly. A non-brain-damaged, age-, sex- and education-matched subject served as a control in all tasks. Her performance was without errors.

A.N.'s results indicate severe impairment in finger identification, denomination and recognition. This deficit does not seem to depend on instruction modalities in that A.N.'s performance was comparable on verbal, visual, and tactile instruction tasks ( $\chi^2 = 4.1, p = ns$ ). By contrast, the type of response influenced A.N.'s performance. In fact, she was more accurate when asked to point to fingers on her own body (78.9%) than to indicate fingers on a diagram (63.2%;  $\chi^2 = 8.93, p = .002$ ) or to denominate the finger (54.1%;  $\chi^2 = 16.93, p = < .001$ ). Her performance was comparable when she pointed to a diagram and in finger denomination conditions ( $\chi^2 = 0.188, p = ns$ ). The possible detrimental effect of left sided sensorimotor deficits in a left finger identification impairment was

tested by comparing left and right hand performance. No difference in response accuracy was found for the left (66.7%) or the right hand (70.2%;  $\chi^2 = 0.53$ , p = ns). Interestingly, the agnosic deficit was not confined solely to fingers but also involved toes (see Table 2).

#### Left-right discrimination

To test impairment in left–right discrimination tasks, A.N. was verbally asked to point, with her eyes open, to left or right parts of the body (not fingers or toes) on her own body, on a model facing away from the patient, on the examiner's body, or on a diagram facing the patient. Note that performing the latter two tasks implies moving from an egocentric to an allocentric observation perspective. For each task, 12 trials were administered on each side of the body. A.N. failed to point to the correct side when she had to point to her own body parts, those of the examiner, or to the model's body parts (Table 3). As no significant difference was observed between pointing to left

TABLE 3           Left–right discrimination tasks					
Right-Left Discrimination	Left (12)	Central (10)	Right (12)		
Patient's own body	5	9	5		
Examiner body	7	10	5		
Model facing the patient	6	10	2		
Model facing away from the patient	4	9	4		

As shown in the central column, A.N. was able to indicate specific body parts in all conditions but failed to discriminate lateralized body segments on both the right and the left side.

and right body parts ( $\chi^2 = 0.24$ , p = ns; left: 22/48, right: 16/48), the possible influence of spatial attentional disorders can be ruled out. Identification of centrally positioned body parts (e.g., nose, chin) was almost perfect (38/40). Furthermore, changing the observation perspective did not affect performance ( $\chi^2 = 0.68$ , p = ns).

#### Dyscalculia and dysgraphia

The patient performed without errors in the Number Repetition and Number Writing (after dictation) sub-tests of Miceli and Capasso's (1991) battery. By contrast, Number Reading and Quantity Comparison were impaired mainly because of deficits in the detection of targets in the left hemispace. However, the signs of dyscalculia observed in the Mental and Written Calculation tasks were not simply due to concurrent spatial problems. Indeed, the patient was unable to perform additions and subtractions because she had a conspicuous impairment in using the rules of calculation. For example, she started additions by calculating from the left-hand digit, did not follow the sequence of the digits, and forgot the amount to be carried. The minor signs of dysgraphia shown in the Writing after Dictation task (omission of double consonants or of other letters, stress-omissions and unfinished letters) likely depend on spatial neglect.

#### Assessment of body representations

Based on the evidence that GS is rare after right hemisphere lesions, we tested separately the three components of body representation, that is, body image, body schema and body structural description (Coslett et al., 2002), capitalizing on experimental tasks used in relevant previous studies (Semenza & Goodglass, 1985; Schwoebel et al., 2004). A non-brain damaged woman (BC), matched to A.N. for age and education, served as control in these tasks (Table 4).

Body image was assessed by asking subjects to choose the body part denominated by an examiner from four drawings (T4.1) and to denominate individual body parts (T4.2). Subjects were also asked to name the body part used for specific functions (e.g., 'What is the body part used for stepping on a cigarette?' - T4.3; or 'Where do you wear gloves?' -T4.4). The subjects were also asked to describe verbally the spatial position of individual body parts (e.g., 'Where is the elbow?' - T4.5). Table 4 shows that A.N. failed in two of these tasks: matching body parts and functions and describing the spatial position of body parts. To rule out the possibility that the body processing impairment observed was due to non-specific spatial deficits, we asked the subjects to perform a bicycle part description task (e.g., 'Where is the mudguard?' - T4.6) and to denominate specific parts on a picture of a bicycle (T4.7). A.N.'s performance in these tasks was almost errorless.

 TABLE 4

 Assessment of body representation

Т		AN	BC
	Body image		
4.1	Sorting among 4 body parts (18)	16	18
4.2	Denomination of single body parts	14	14
4.3	Matching of body parts and functions (12)	6	12
4.4	Where do you wear $\dots$ ? (13)	13	13
4.5	Where is? (14) Bicycle	6	14
4.6	Where is? (14)	13	14
4.7	Indication of parts (14)	14	14
	Body structural description		
	Indication of body parts		
4.8	One's own body with closed eyes (18)	16	18
4.9	One's own body with opened eyes (18)	18	18
4.10	Examiner's body (18)	18	18
4.11	On a manikin (18)	18	18
4.12	On a drawing (18)	18	18
4.13	Body parts localization (18) Denomination of body parts	16	18
4.14	One's own body	18	18
4.15	Examiner's body Body schema	18	18
4.16	Mental rotation of hand (48)	32	47
4.17	Mental rotation of object (48)	31	48

Scores of A.N. and B.C. (non-brain damaged age and education matched woman) in tasks assessing body representation. Presence of deficit is indicated in bold.

*Body structural descriptions* were explored by using body part indication and denomination tasks (Table 4). In the former, the examiner named a specific body part (other than fingers) and the subjects had to point to it on their own body (T4.8–T4.9), on the examiner's body (4.10), on a manikin (T4.11), and on a drawing of a human body (T4.12). In addition, we used a body part localization task (T4.13) in which the examiner touched a specific part of the subjects' body while they kept their eyes closed. The task was to open their eyes and point to the corresponding body part on the examiner's body (Semenza & Goodglass, 1985).

In the body part denomination tasks, the subjects were requested to name the body parts indicated by the examiner on their own body (T4.14) or on the examiner's body (T4.15). It is relevant that in the body structural description tasks, A.N.'s performance was not different from that of the non-brain damaged control subject.

To investigate *body schema*, we used a hand laterality task (Schwoebel et al., 2004). This was modified so as to minimize the influence of the left-right discrimination impairment. On a computer screen we presented two digitized color pictures, each representing a hand. One of the hands was always in a vertical position, whereas the other could be vertical or rotated 60, 120, 180, 240, or 300 degrees. The stimuli were presented at the subjects' midline with the palm facing toward the front or the back. In each of 48 trials, the subjects had to decide whether the two images referred to the same hand (e.g., left-left) or to opposite hands (e.g., leftright). To verify the presence of mental rotation disorders non-specific for the body, the same task was proposed with inanimate stimuli. We presented a picture of two cars, each with a black headlight, either on the right or on the left. Subjects were asked to judge whether the two cars were the same or different. As A.N. failed on both tasks, we cannot exclude the possibility of a general mental rotation disorder.

In any case, she did not show any signs of anosognosia for hemiplegia, disownership of contralesional hand, somatoparaphrenia or supernumerary phantom limb perception, all of which are typical of body schema disorders.

#### Finger agnosia and gesture execution

We tested the relationship between body representation and gesture execution by assessing whether A.N., who showed body image deficits,

was impaired in action production. We used tasks developed by Buxbaum, Giovannetti, and Libon (2000) in which the spatial components of actions are very well controlled. As we were interested in whether gesture imitation was related to specific deficits in body representations in relation to the dual-route model (Rothi et al., 2001), the action tasks were also carried out with F.C., a 58-year-old right-handed man suffering from signs of body schema disorders following a hemorrhagic stroke involving the right basal ganglia. This patient also showed a lesion on the left temporal lobe, which, however, as inferred from its appearance on the scan, was likely due to a previous asymptomatic stroke. F.C. was selected as a control subject because, like A.N., he presented with sensorimotor, controlesional deficits as well as clear signs of left visuo-spatial neglect and personal neglect (Table 1). However, unlike A.N., patient F.C. showed clear signs of body schema disorders. Indeed, he denied his paralysis and stated he was able to move his left limbs normally, thus showing anosognosia for hemiplegia at a specific interview (modified from Marcel, Tegnér, & Nimmo-Smith, 2004).

In the first task, A.N. and F.C. were verbally requested to perform 10 transitive gestures, imagining holding and using specific implements (i.e., "Show me how to use a "hammer", or "a comb" ...), and five intransitive gestures (i.e., 'Show me how to signal "stop" or "come here" . . .). (Verbal command condition). In the second task, the two patients were asked to imitate the aforementioned 15 gestures (meaningful imitation condition) and 15 meaningless gestures performed by the examiner (meaningless imitation condition). Each meaningless movement maintained the characteristics of the meaningful gesture from which it derived with respect to: plane of movement (vertical/horizontal), joint moved (shoulder/elbow/wrist/fingers), type of grip (hand open/clenched/partially open) and oscillations (present/absent). For example, if the meaningful gesture is 'to saw', the meaningless analogue is 'fingers fanned, arm move forward/ back', which corresponds to the 'to saw' gesture for the plane of action (radial), the joints activated (shoulder and elbow) and the oscillation (of the arm) (Buxbaum et al., 2000). In the third task, the two patients were asked to perform 10 transitive gestures by actually holding and using a given implement (use condition). Following specific criteria (Buxbaum et al., 2000), two independent experimenters rated the four components of the

Gesture components	Grasp		Trajectory		Amplitude		Timing		Score	
	<i>A</i> . <i>N</i> .	<i>F.C.</i>	<i>A</i> . <i>N</i> .	<i>F.C.</i>	<i>A</i> . <i>N</i> .	<i>F.C.</i>	A.N.	<i>F.C.</i>	<i>A</i> . <i>N</i> .	<i>F.C.</i>
Meaningful (command) (60)	14	14	13	12	14	13	13	14	55	53
Meaningful (imitation) (60)	10	14	14	13	15	11	15	14	54	52
Meaningless (imitation) (60)	9	8	14	7	15	11	15	13	53	39
Use (40)	9	9	10	9	9	9	10	9	38	36
Score	42	45	51	41	53	44	53	50		

 TABLE 5

 Performance of patients A.N. and F.C. in the gesture imitation tes

Scores in bold indicate presence of deficit.

gesture production performance, namely, grasp, trajectory, amplitude and timing. Scores ranged from 0 (non-recognizable gesture) to 4 (correct execution). In the case of discordant ratings, subjects were asked to perform the action again.

The performance of the two patients in the gesture execution tasks is reported in Table 5. Both patients exhibited impaired performances which, however, differed on a qualitative level. F.C. showed a pattern of behavior reminiscent of the patient reported by Schwoebel et al. (2004). Indeed, he performed significantly worse in the imitation of meaningless actions (65% of hits) than in the other conditions (imitation of meaningful actions = 86.7% of hits; verbal command condition = 88.3%of hits) ( $\chi^2 = 12.71$ , p = .002). By contrast, no significant difference was observed between the four action components ( $\chi^2 = 5.13$ , p = ns; grasp = 81.8%, trajectory = 74.5\%, amplitude = 80%, timing = 90.9\%).

Interestingly, A.N. showed a specific deficit in finger orienting and positioning during the imitation of both meaningful and meaningless actions. During imitation of combing, for example, she did not close her fingers to grasp the comb, but put her hand in a tight claw position. Similarly, she kept her fist clenched during imitation of the greeting gesture. Her performance was significantly more impaired for the grasp than for the other components, which were not different from one another ( $\chi^2 = 17.43$ , p = .001; grasp = 76.4%, trajectory = 92.7%, amplitude = 96.4%, timing = 96.4%). Note that the errors in the grasp component regarded only finger positions and never the wrist or the spatial orienting of the hand. By contrast, no differences between the four conditions (imitation of meaningful and meaningless actions, verbal command condition and use of objects) was found ( $\chi^2 = 0.37, p = ns$ ).

#### DISCUSSION

GS typically occurs following a lesion to the left, language-dominant hemisphere. Although direct cortical stimulation of the right angular gyrus produces a clear interference in calculation, finger recognition, and writing tasks in a patient who has undergone intraoperative surgical mapping of cortical functions (Roux, Boetto, Sacko, Chollet, & Trémoulet, 2003), reports of GS following right hemisphere lesions are rare (Mayer et al., 1999) and mainly associated with evidence of reversed functional cerebral dominance and reversed anatomic cerebral asymmetries. Moore et al. (1991) reported a well documented case of a woman with GS following a right hemispheric lesion. However, the patient was left-handed and her reversed cerebral dominance was confirmed by the presence of posterior aphasia and alexia. Moreover, a CT scan indicated that the patient had a larger planum temporale on the right than on the left hemisphere, which is typical of non-standard patterns of cerebral asymmetry. Another patient with right parieto-occipital hemorrhage was described by Dozono et al. (1997). This patient was ambidextrous and also showed clear signs of ideational, constructional and dressing apraxia. Moreover, the patient had suffered a previous lesion that affected his left parietal lobe and he obtained a pathological score on the MMSE (15/24).

In the present study, we report a right-handed patient with a lesion involving the right hemisphere who exhibited clear signs of GS. Indeed, all the signs which, according to Schilder (1931), are typically associated with GS (finger agnosia, finger aphasia, visual finger agnosia, constructive finger apraxia, and apraxia for finger choice) were present in our patient who, in keeping with a previous report (Tucha, Steup, Smely, & Lange, 1997), also showed toe agnosia. Interestingly, the neuropsychological assessment excluded the possibility that the patient had reversed hemispheric lateralization, thus making the case even rarer. Moreover, signs of GS were triggered by verbal and non-verbal instructions, thus ruling out the influence of language impairment. A.N. performed better when she referred to her own body as compared to the examiner's or the model's body, thus suggesting, in keeping with Gerstmann's original hypothesis, that representations regarding other people's bodies involve one's own body representation (Gerstmann, 1940).

Patient A.N. was impaired not only in the mental rotation of body parts but also in the mental rotation of non-corporeal objects. Moreover, errors in left-right discrimination did not seem to be related to mental rotation abilities in that the patient's performance did not change with respect to egocentric or allocentric perspectives. Therefore, our study does not allow us to draw any conclusions about the hypothesis that deficits in visuo-spatial mental manipulation may be the link between the various GS symptoms (Ardila, Concha, & Rosselli, 2000; Carota, Di Pietro, Ptak, Poglia, & Schnider, 2004; Mayer et al., 1999). A.N.'s finger recognition impairments hint at a regionally specific deficit that regards both semantic and online representations of fingers. This is confirmed by the results of the gesture imitation tasks in which A.N.'s deficit in orienting and positioning body parts was similar for both meaningful and meaningless gestures but was specific for fingers. Indeed, we observed several instances of correct orienting of arm and wrist with defective orientation of fingers. This result is in keeping with a recent report on body part specific imitation deficits with disturbed imitation of hand postures associated with inferior parietal and posterior cortical areas, and disturbed imitation of finger postures associated with lesions of the inferior frontal gyrus, insula, putamen and caudate nucleus (Goldenberg & Karnath, 2006). It is interesting that all of the above areas were severely damaged in patient A.N. (see Figure 1B). Our data complement and extend the study of Goldenberg and Strauss (2002) in which right brain lesions were shown to impair imitation of meaningless finger as compared to meaningless hand gestures, whereas the opposite was true for left brain lesions. Based on this pattern of hemispheric division of labor, the authors posited that while hand coding is linked to body knowledge, finger coding requires fine-grained discrimination and integration of perceptual cues. Note that Goldenberg and Strauss (2002) used meaningless finger gestures that were entirely novel to the patients. By contrast, we used complex and dynamic finger gestures and found that disorders in finger positioning affected both meaningless and meaningful actions which may or may not be novel. Moreover, we not only examined the final static position of a given posture but also the different components of the action. The results suggest that A.N.'s impairments are linked to her difficulty in representing specific body parts (i.e., the fingers), rather than to a non-specific perceptual deficit. It is also worth noting that the patient's performance in more general tests of imitation was perfect, thus ruling out the possibility of a general disorder in action organization.

Patient F.C. performed within the normal range in the meaningful gestures imitation task. By contrast, he performed deficiently in the meaningless gestures imitation task; his errors concerned all components of action except timing. This result supports the hypothesis of an association between body schema disorders and impairment in imitation of meaningless gestures (Schwoebel et al., 2004). F.C.'s performance is also in keeping with the dualroute model (Rothi et al., 1991). In fact, the model postulates that body schema is relevant for meaningless movements, typically performed via the non-semantic (or direct) route that provides direct translation of visual input into motor output. A.N's performance, however, does not fit completely with the model according to which only gesture execution on verbal command, but not on imitation (where the non-semantic route can be used), should be impaired. Indeed, A.N. exhibited a specific finger imitation deficit. In conclusion, in keeping with Gerstmann's original hypothesis we suggest that A.N. presents a specific disorder in finger recognition that regards both semantic and online sensorimotor representations. In this vein, finger agnosia may represent a localized disorder of both static and dynamic finger cognition that impairs the organization of actions. A strict version of the distinction between body schema and body image, normally connected with left or right hemispheric lesions, seems inadequate to highlight the subtle links between body representation and action.

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