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Touch to See: Neuropsychological Evidence of a Sensory Mirror System for Touch

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The observation of touch can be grounded in the activation of brain areas underpinning direct tactile experience, namely the somatosensory cortices. What is the behavioral impact of such a mirror sensory activity on visual perception? To address this issue, we investigated the causal interplay between observed and felt touch in right brain–damaged patients, as a function of their underlying damaged visual and/or tactile modalities. Patients and healthy controls underwent a detection task, comprising visual stimuli depicting touches or without a tactile component. Touch and No-touch stimuli were presented in egocentric or allocentric perspectives. Seeing touches, regardless of the viewing perspective, differently affects visual perception depending on which sensory modality is damaged: In patients with a selective visual deficit, but without any tactile defect, the sight of touch improves the visual impairment; this effect is associated with a lesion to the supramarginal gyrus. In patients with a tactile deficit, but intact visual perception, the sight of touch disrupts visual processing, inducing a visual extinction-like phenomenon. This disruptive effect is associated with the damage of the postcentral gyrus. Hence, a damage to the somatosensory system can lead to a dysfunctional visual processing, and an intact somatosensory processing can aid visual perception.

Keywords: crossmodal, mirror neuron system, somatosensory cortex, touch observation, visual processing

Introduction

The human brain is able to implicitly transform the sight of touch into an inner representation of touch (Gallese 2003; Keysers et al. 2010). This function might be supported by a visuo-tactile mirror system dealing with both our personal experience of touch and the experience of touch observed in other individuals. Animal and human studies have provided extensive evidence for the existence of neural circuits in the sensorimotor system that are involved in the processing of self as well as others' body-related experiences (Keysers et al. 2010). Empirical evidence from functional imaging studies in humans (Keysers et al. 2004; Blakemore et al. 2005; Schaefer et al. 2005; Ebisch et al. 2008) has revealed the existence of a visuo-tactile mirror system, which matches observed touch with felt touch. Even more intriguing is the report that the mere observation of touch automatically induces the activation of the neural circuitry that is normally involved in our experience of touch, such as the primary (SI) and secondary (SII) somatosensory cortices (Keysers et al. 2004; Blakemore et al. 2005; Bolognini et al. 2011). This somatosensory activation during the sight of touch is reminiscent of the mirror premotor activation following action observation (Rizzolatti and Craighero 2004), although it involves brain regions responsible for somatosensory processing rather than action programming (Keysers et al. 2010).

On a broader behavioral level, this system may be involved in anticipating the effects of the tactile stimulation on our body in a feed-forward fashion as well as in understanding the effects of tactile stimulation experienced by other individuals, as a part of the broader neural circuitry for embodied simulation (Gallese 2005; Grafton 2009).

So far, the behavioral impact of such a mirror somatosensory activity on visual perception remains to be determined. This intriguing question is motivated by findings from both stroke patients and healthy subjects showing that the mere sight of tactile events can be sufficient to evoke somatic sensations in the observer (Halligan et al. 1996; Blakemore et al. 2005). Such somatosensory experiences might be accomplished by the activation of somatosensory areas through a mirror simulation mechanism.

If the vision of a tactile event might automatically be associated with the activation of the cortical network normally involved in our own experience of being touched (e.g., Blakemore et al. 2005; Keysers et al. 2010), there should be some difference in the processing of visual stimuli with or without a tactile component; this difference, in turn, could be linked to the ability to process tactile stimuli following stroke. We therefore expected that, if the tactile system is undamaged, the sight of touch may activate a spared somatosensory representation. In the case of a visual impairment, this additional recruitment of the tactile modality would improve the perception of visual stimuli with a tactile content as compared with visual stimuli without a tactile content. Conversely, a damage to the tactile modality might selectively affect the processing of visual stimuli depicting touch either by disrupting the mirror simulation of the observed touch or by activating an impaired somatosensory representation of the observed touch. In both cases, no advantage would be expected for visual stimuli depicting a touch as compared with those without touch. Finally, when both the visual and tactile modalities are damaged, no crossmodal modulation would be expected in sensory processing by adding a somatic dimension to the visual experience; therefore, any difference between touching and no-touching stimuli should emerge.

To address this issue, the effect of touch observation on visual perception was studied in right brain–damaged patients with visual and/or tactile deficits, namely: patients with visual extinction, which consists in the failure to detect a visual stimulus presented in the contralesional hemifield when a competing stimulus is simultaneously presented in the ipsilesional hemifield (Brozzoli et al. 2006); patients with tactile deficits on the contralesional hand, that is, tactile extinction or hypoesthesia; patients with both contralesional tactile and visual deficits; and patients without sensory (tactile

or visual) deficit. A group of healthy participants was also tested.

Participants performed a visual detection task during which they had to detect the movement of index fingers that could touch the thumb of the same hand (Touch condition) or not (No-touch condition). Egocentric and allocentric viewing perspectives were used in order to determine whether the visual processing of body contact may differ according to the viewing perspective. Since viewing one's own body being touched has an impact on somatosensory processing (Taylor-Clarke et al. 2002), the self-attribution of being touched in the observer (i.e., egocentric view) might affect visual processing to a greater extent as compared with the observation of a touch not attributable to one's own body (i.e., allocentric view).

Materials and Methods

Participants

Patients were recruited from the inpatient population of a Neurological Rehabilitation Unit (S.C. Riabilitazione Specialistica Neuromotoria, Azienda Ospedaliera Carlo Poma, Bozzolo, Italy). Fourteen brain-damaged patients (all right-handed, 8 males; mean age = 68, standard deviation [SD] = 12; mean educational level = 9, SD = 3) participated in the study. The inclusion criterion was the presence of a right hemisphere lesion. All patients had no history of previous neurological disease or psychiatric disorders. Patients did not show any sign of cognitive decline (Folstein et al. 1975).

Twelve neurologically unimpaired subjects (all right-handed, 6 males, mean age = 62, SD = 9; mean educational level = 7, SD = 3) were also tested, and they served as controls.

All participants gave their informed consent to participate in the study. The protocol was carried out in accordance with the ethical standards of the Declaration of Helsinki (British Medical Journal, 1991, Vol. 302, p. 1194), and it was approved by the ethical committee of the University of Milano-Bicocca.

Clinical Assessment

Contralesional motor, somatosensory, and visual field deficits, including extinction to tactile and visual stimuli, were assessed by a standard neurological examination (Bisiach et al. 1983; Haerer 1992; Anderson et al. 2005). The motor examination of the upper and lower limbs included the clinical assessment of asymmetric strength deficits. The patient was asked, with eyes closed, to hold his/her arms out horizontally (palms up) for 30 s, then observing any sign of weakness (arm drift or pronation: score: 0–3, 0 = no deficit and 3 = maximum deficit). For the lower extremity, the supine patient was asked to flex the thigh on the pelvis 90° and extend the leg on the thigh 90°, maintaining the position for 30 s (score: 0–3) (Bisiach et al. 1983).

Furthermore, none of the patients showed clinical deficit neither of motor coordination nor of tendon reflexes.

Visual field (upper and lower quadrants) was tested by confrontation. The patient was instructed to fixate the nose of the examiner, who performed a conventional manual confrontation test by "finger wiggling," keeping the hands at about 20° of visual angle to the right and left visual fields. The patient had to report the presence and location (left/right/bilateral) of any perceived movement.

The presence of tactile deficits was assessed with manual stimulation through brief and light pressure stimuli delivered with the fingertip on the back of the patient's hand. The patient had to report which hand was touched (left, right, or both).

In both the tactile and visual tests, the patients were warned that the stimulation could be unilateral or bilateral and stimuli were administered in 2 consecutive series: First, only 10 unilateral left and 10 unilateral right stimuli (score = 0–10 omissions for each side) were presented in a random fixed order. In the second sequence, which aimed at looking for the presence of extinction, 5 unilateral left, 5 unilateral right, and 10 bilateral simultaneous stimuli were delivered (score = 0–10 omissions in bilateral trials). The presence of left extinction is indexed by a difference between unilateral left and bilateral stimuli $\geq 20\%$ (Bisiach et al. 1983). For both the visual and tactile tests, whenever the patient correctly reported the occurrence of the stimulus, he/she was always able to correctly localize it.

Furthermore, we evaluated the presence of unilateral spatial neglect (USN), a spatial deficit consisting in the lack of awareness for visual events located in the space contralateral to the damaged hemisphere (typically the left hemispace following a right lesion; Bisiach and Vallar 2000) by using the following tests: 1) Cancellation tasks, that is, Line cancellation, score = 0–40 (Albert 1973); Letter cancellation, score = 0–104 (Diller and Weinberg 1977); and Bell cancellation, score = 0–35 (Gauthier et al. 1989); 2) Line Bisection, score = mean percentage of rightward bias (Wilson et al. 1987); 3) Drawing, that is, Clock Drawing Test, score = 0–12 (Wilson et al. 1987) and 5-element complex drawing, score = 0–10 (Gainotti et al. 1972); in both tests, performance was evaluated considering the completeness and symmetrical arrangement of drawing; and 4) Sentence reading test, score = 0–6 (Pizzamiglio et al. 1992). Personal USN was assessed by asking the patient to touch the contralesional hand with the ipsilesional hand, score = 0–3 (0 = unimpaired performance; 3 = maximum deficit) (Bisiach et al. 1986). A more detailed description of these tests and their scoring is described elsewhere (e.g., Sposito et al. 2010).

The clinical details of each patient are summarized in Table 1.

Based on the neuropsychological evaluation, patients were divided into 4 groups, with respect to their damaged sensory processing: 1) 4 patients with contralesional tactile and visual field deficits at the neurological examination (i.e., visual plus tactile deficit [VTD] group); these patients also exhibited USN; 2) 3 patients with visual extinction but without tactile deficits or USN (i.e., VD group); 3) 3 patients with a tactile deficit on the contralesional hand and no signs of visual deficits or USN (i.e., TD group); for one of these patients, the deficit was present only following bilateral stimulation (i.e., tactile extinction), while for the others, it also followed single contralesional stimulation (i.e., hypoesthesia); and 4) 4 patients without any sensory (tactile or visual) disorder (i.e., D– group) (Note that P4 showed only a mild rightward bias at the line bisection test, at the reading task she made just one error, and at the Bell cancellation task the errors were not spatial specific for the contralesional hemispace [omissions on both the left and right sides]. More important, with respect to the aim of the present study, she did not exhibit any visual or tactile deficits at the neurological examination. Hence, P4 was included in the D– group).

Lesion Data

Figure 1 shows the mapping of the brain lesions for 11 of 14 patients, for whom the original brain scan (in all cases, this was a CT scan) was available. Lesions were mapped using the software MRIcro (Rorden and Brett 2000). We reconstructed the region of interest (ROI) to define the location and the size of the lesion for each patient by using a Template Technique, that is, by manually drawing the lesion on the standard template from the Montreal Neurological Institute. ROIs were created by mapping the regions on each and every 2D slice of a 3D volume. By overlapping the lesions in each group of patients, we could calculate the voxels in which lesions overlapped, as reported in Table 2.

For the 3 patients for whom the original scan was not available, the radiological report indicated the following lesion site: P1 = basal ganglia; P2 = middle cerebral artery stroke, with no sign of focal lesions; and P10 = parietal and temporal areas.

Experimental Task

The visual detection task comprised 3 experimental main factors (see Fig. 2*a*). The factor "touch," referring to the kind of stimulus delivered, comprised the Touch condition, namely the observation of a video clip depicting the index finger touching the thumb of the same hand at the end of the movement, and the No-touch condition, namely the observation of a video clip depicting the index finger making the same movement as in the Touch condition but without touching the thumb. In Touch and No-touch conditions, the amount of the movement of the index finger was matched. The factor "viewing perspective," referring to the configuration of the visual stimuli, comprised the allocentric perspective (in which the depicted hands mimicked the posture of a putative examiner placed in front of the subject) and the egocentric

Table 1
Summary of the clinical data

Patients		Age/gender	Etiology/stroke duration (months)	Neurological examination			Neuropsychological assessment							
				V	SS	M	Line bisection	Drawing test	Sentence reading	Personal neglect	Cancellation			Clock drawing
											Line	Bell	Letter	
D−	P1	90, M	I, 1	−	−	+	−	−	−	−	−	−	−	−
	P2	85, F	I, 1	−	−	+	−	−	−	−	−	−	−	−
	P3	57, F	H, 1	−	−	±	−	−	−	−	−	−	−	−
	P4	72, F	I, 1.5	−	−	−	+	−	±	−	−	−	±	−
VTD	P5	79, M	H, 2	+	+	±	+	+	−	−	+	+	±	+
	P6	52, M	H, 20	+	+	+	+	+	−	−	+	+	+	+
	P7	77, M	I, 1	+	+	+	+	+	−	−	+	+	+	+
	P8	70, M	I, 2	+	+	+	−	+	+	−	−	+	+	+
VD	P9	68, F	I, 17.5	e	−	+	−	−	−	−	−	−	−	−
	P10	62, M	I, 59	e	−	−	−	−	−	−	−	−	−	−
	P11	63, M	I, 15	e	−	+	−	−	−	−	−	−	−	−
TD	P12	45, F	I, 7	−	+	+	−	−	−	−	−	−	−	−
	P13	76, F	I, 1.5	−	e	+	-	−	−	−	−	−	−	−
	P14	65, M	I, 1	−	+	+	−	−	−	−	−	−	−	−

Note: Groups: D−, patients without sensory (tactile or visual) disorders; VTD, patients with tactile and visual deficit; VD, patients with visual extinction; TD, patients with tactile extinction or hypoesthesia. Lesion Aetiology: I/H, ischemic/haemorrhagic. Neurological examination: Motor/Somatosensory/Visual deficit contralateral to the damaged hemisphere, respectively M/SS/V: + = maximum deficit, − = no deficit, ± = minor deficit, and e = extinction to double stimulation. Neuropsychological assessment: + = sign of NSU, ± = minor deficit, and − = no deficit.

perspective (in which the depicted hands assumed a posture compatible to that of subject's own hands). Finally, the factor "side," referring to the location of the moving stimuli, comprised: unilateral stimuli (i.e., movement of either the left or the right index finger), bilateral stimuli (simultaneous movement of both fingers), and catch trials (with just the 2 hands being displayed without any movement), the latter included in order to control for false-positive responses. Overall, the experimental design was a 2 (Touch/No-touch) by 2 (egocentric/allocentric) by 3 (unilateral/bilateral/catch) factorial design.

Each trial started with a picture displayed on a gray background, depicting a left hand and a right hand at 18° of eccentricity from a central fixation point (black cross). The size of the stimuli was 9° × 6°. After checking the subject' fixation, the examiner started stimulus presentation by pressing the space bar on the computer keyboard. Then, after a random interval between 1800 and 2000 ms, subjects were shown a video clip of 100 ms (made up by 4 frames, 25 ms each), depicting the moving fingers. Subjects were required to verbally report how many fingers moved and the side of the movement (left, right, and bilateral); there was no time limit for responding. After a response was given, the experimenter rechecked subject's fixation before delivering the next trial. Subject's verbal responses were recorded for off-line analysis (Fig. 2*b*).

There were 4 experimental blocks delivered in a counterbalanced order across participants, each one featuring one touch condition (Touch/No-touch) and one viewing perspective (egocentric/allocentric). In each block, 24 trials were delivered for each stimulus side (24 right, 24 left, 24 bilateral, and 24 catch trials) for a total of 96 stimuli per block. The total duration of the experiment was about 40 min. The sequence and timing of the stimuli were under computer control (E-prime software, www.psychtoolbox.org).

Results

For the analysis, subjects' responses were categorized as correct visual detections (scored 1), which consisted of both the correct detection of the number of the moving visual stimuli as well as their correct localization; errors (scored 0) consisted in stimulus' omissions (i.e., omission of the isolated visual stimulus in unilateral trials or the contralateral stimulus in bilateral trials), while no mislocalization errors were committed. On catch trials, controls made no false alarms, while only 3 patients committed a total of about 6% false alarms; therefore, false alarms were not further analyzed.

For patients, the mean percentages of correct visual detections were analyzed by means of a repeated measures analysis of variance (ANOVA) with Group (D–, VTD, VD, and TD) as a between-subjects factor, Touch (Touch vs. No-touch), Viewing Perspective (allocentric vs. egocentric), and Side (bilateral, unilateral left, and unilateral right) as within-subjects factors.

A similar ANOVA model was used to analyze the mean percentages of correct visual detections in healthy controls by using a 2 (Touch) × 2 (Viewing Perspective) × 3 (Side) ANOVA.

The partial Eta squared ($p\eta^2$), which measures the proportion of the total variance that is attributable to a main factor or to an interaction (Cohen 1973), was also calculated. Outcomes of the ANOVAs are reported in Table 3.

Post hoc analyses were performed by using the Newman–Keuls test.

The analysis of the patients' performance showed a main effect of Group: VTD (34% correct, $P < 0.01$) and VD patients (67%, $P < 0.01$) were both less accurate than D– (95%) and both less accurate than TD patients (91%). VTD and VD patients also differed from each other ($P < 0.01$), while D– and TD patients did not differ from each other ($P = 0.5$).

The effect of Side was also significant since all groups of patients showed an overall lower accuracy in the detection of left stimuli, that is, on the side contralateral to the damaged hemisphere, in both bilateral (54%, $P < 0.01$) and unilateral left (66%, $P < 0.01$) presentations as compared with unilateral right stimuli (92%). The accuracy on bilateral and unilateral left trials was also different (54% vs. 66%, $P < 0.01$).

Crucially, the Group by Side interaction showed that VD patients were impaired in detecting bilateral stimuli (32%) as compared with both unilateral left stimuli (73%, $P < 0.01$) and unilateral right stimuli (95%, $P < 0.01$); this is typically observed in visual extinction (Brozzoli et al. 2006). Instead, VTD patients were impaired in detecting both bilateral (7%, $P < 0.01$) and unilateral left (11%, $P < 0.01$) stimuli as compared with unilateral right stimuli (83%), without any difference between bilateral and left stimuli ($P = 0.5$); this pattern of response is likely to be ascribed to the additional presence of USN. Finally, in D– and TD patients, there was no difference in visual detections with respect to the side of the stimuli (D–: bilateral = 94%, unilateral left = 96%, and unilateral right = 96%, $P = 0.9$ for all comparisons; TD: bilateral = 87%, unilateral left = 92%, and

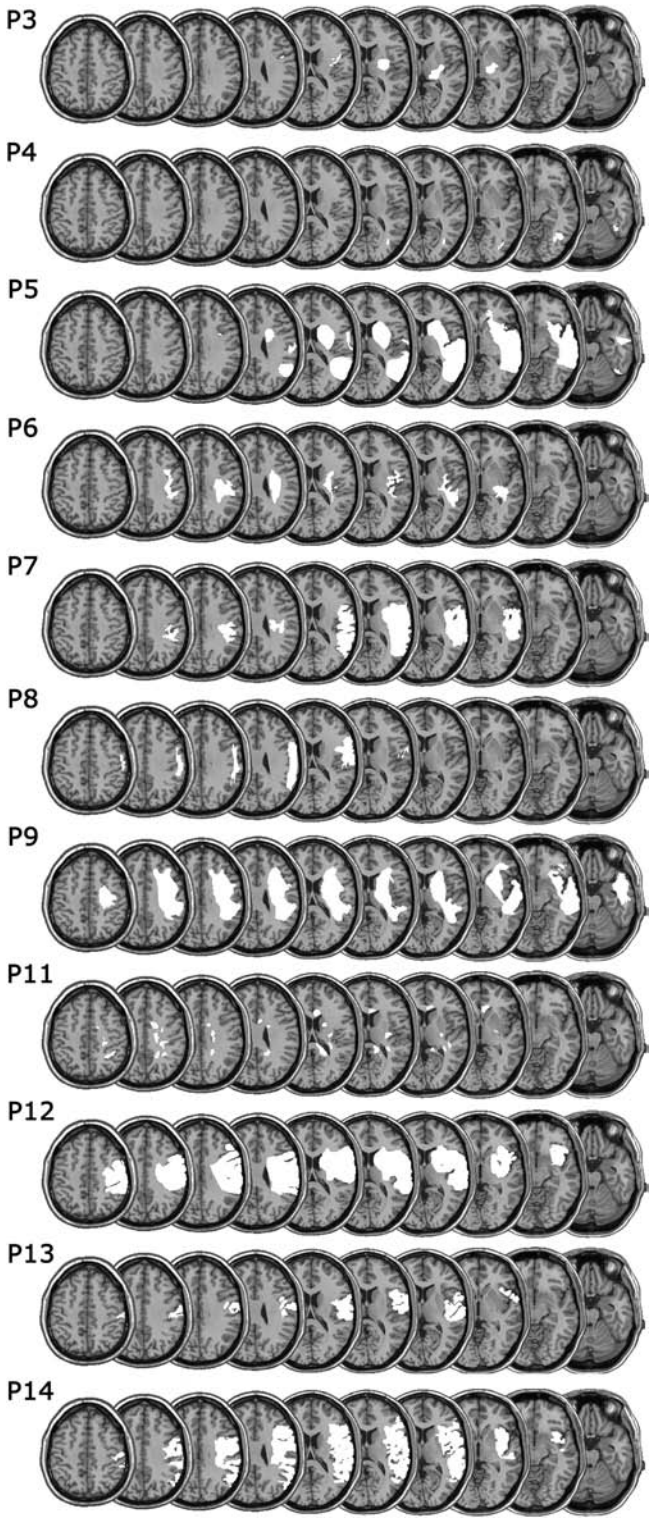

Figure 1. Lesion mapping for each single patient. White areas represent the extension of the lesion of each single patient.

unilateral right = 95%, P = 0.6 for all comparisons). These findings replicate those obtained at the neurological examination, confirming the appropriateness of our visual task for assessing unilateral and bilateral visual deficits and supporting the validity of our allocation of the patients in the different experimental groups.

Table 2
Number of voxels in each ROI are given for each group of patient

	D–	VTD	TD	VD
Precentral gyrus	104	3190	17 330	5003
Frontal sup	—	—	2964	602
Frontal mid	—	366	1383	2954
Frontal inf	76	3574	18 455	5868
Rolandic oper	196	8942	10 622	2643
Insula	400	9036	12 749	4511
Cingulum	—	19	27	747
Hippocampus	—	358	—	143
Fusiform	690	1922	—	—
Occipital mid	—	775	204	102
Occipital inf	779	—	—	—
Postcentral gyrus	—	6079	18 562	3845
Parietal sup	—	—	80	123
Parietal inf	—	59	2315	1266
Supramarginal gyrus	—	8883	10 665	8704
Angular gyrus	—	2580	4228	1270
Caudate	—	—	3501	3432
Putamen	1139	7507	7088	7739
Pallidum	261	117	1578	1658
Thalamus	1519	163	73	1986
Heschl	—	1740	1657	825
Temporal sup	—	25 838	19 275	17 568
Temporal mid	—	29 455	8400	16 367
Temporal infer	725	7974	—	3028

The significant main effect of the Viewing Perspective showed a better performance for the allocentric (73%) as compared to the egocentric perspective (69%, $P < 0.01$).

The key finding with respect to our experimental hypothesis was the significant Group by Touch by Side interaction, which highlighted how the sight of touch can modulate visual perception in a fashion that is specific to the patients' sensory deficit, as shown in Figure 3*a*. In VD patients, seeing visual stimuli depicting a touch improved visual extinction on bilateral trials (39%, $P < 0.01$) as compared with bilateral visual stimuli without the tactile component (24%). For unilateral left-sided and right-sided stimuli, the difference between Touch and No-touch conditions was not significant (unilateral left: Touch = 76% vs. No-touch = 70%, $P = 0.07$; unilateral right: Touch = 94% vs. No-touch = 97%, $P = 0.9$). In striking contrast, in TD patients, the sight of touch impaired the performance on bilateral trials in an extinction-like fashion; in other words, when the tactile modality is affected, seeing touches reduced the accuracy on bilateral trials as compared with bilateral No-touch trials (78% vs. 93%, $P < 0.01$). This effect was specific for the bilateral stimuli, being absent for unilateral left stimuli (Touch = 91% vs. No-touch = 93%, $P = 0.9$) and for unilateral right stimuli (Touch = 96% vs. No-touch = 95%, $P = 0.8$). Instead, in both VTD and D– patients, there was no difference between Touch and No-touch conditions for all stimulus locations (i.e., bilateral, unilateral left, and unilateral right; $P > 0.2$, for all comparisons).

Other effects did not reach significance (Table 3).

Noteworthy, the size effects in the ANOVA showed that the significant effects were quite consistent, notwithstanding the comparatively small number of patients (Table 3).

Finally, the analysis of healthy control subjects did not showed any significant effect (Table 3).

Comparison between Patients and Controls

To assess for the presence of any significant dissociation in patients' performance, we further compared the detection accuracy on bilateral trials in Touch and No-touch conditions

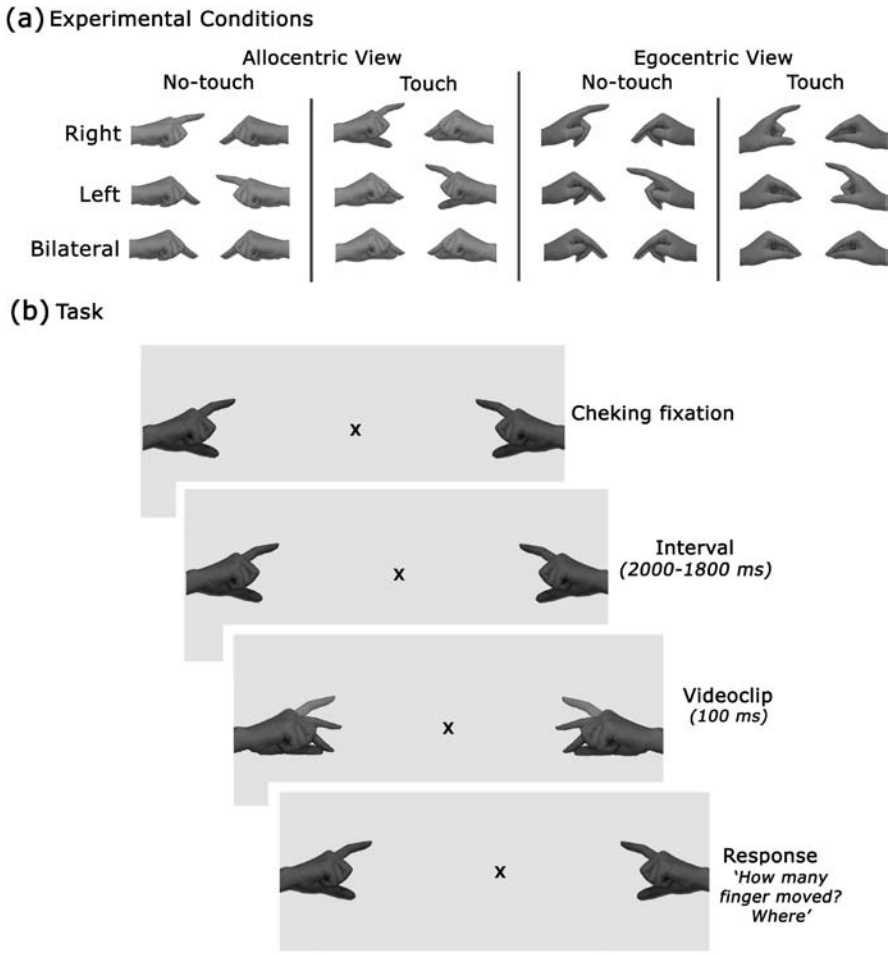

Figure 2. Experimental procedure. (*a*) Experimental conditions of the visual detection task: No-touch and Touch stimuli, in egocentric or allocentric perspectives, presented unilaterally (i.e., movement of the left or the right index finger) or bilaterally (simultaneous movement of both fingers). In catch trials, 2 hands were displayed without any movement (not shown in the figure). (*b*) Sequence of events: for example, the bilateral stimulus of the Touch/Allocentric condition. With their eyes at fixation, patients had to verbally report the movement of the fingers and the side of the movement.

Table 3
F, *P* level, and $p\eta^2$ values from the ANOVAs

	Factors (df)	*F*	*P* level	$p\eta^2$
Patients	Group (3,10)	47.03	0.0001	0.9
	Viewing Perspective (1,10)	7.02	0.02	0.4
	Touch (1,10)	0.026	0.8	0.01
	Side (2,20)	82.53	0.0001	0.9
	Viewing Perspective × Touch (1,10)	0.17	0.7	0.02
	Viewing Perspective × Side (2,20)	0.47	0.6	0.04
	Touch × Side (2,20)	1.16	0.3	0.1
	Viewing Perspective × Touch × Side (2,20)	1.13	0.3	0.1
	Group × Viewing Perspective (3,10)	2.15	0.16	0.3
	Group × Touch (3,10)	2.31	0.14	0.4
	Group × Side (6,20)	28.1	0.0001	0.9
	Group × Viewing Perspective × Touch (3,10)	0.49	0.7	0.1
	Group × Viewing Perspective × Side (6,20)	0.36	0.9	0.1
	Group × Touch × Side (6,20)	4.95	0.01	0.6
	Group × Viewing Perspective × Touch × Side (6,20)	1.07	0.4	0.2
Controls	Touch (1,11)	0.49	0.5	0.01
	Side (2,22)	1.85	0.2	0.04
	Viewing Perspective × Touch (1,11)	1.90	0.2	0.1
	Viewing Perspective × Side (2,22)	0.37	0.7	0.03
	Touch × Side (2,22)	0.09	0.9	0.01
	Viewing Perspective × Touch × Side (2,20)	1.07	0.4	0.01

Note: df = degrees of freedom.

with that of the healthy controls. The comparison was performed by *t*-tests performed following the procedure by Crawford and Garthwaite (2005). This procedure allows to estimate the abnormality of the patient's score at each test (i.e., the estimate of the percentage of the control population that would obtain a lower score). Then, using the same statistical methodology, we compared the difference between Touch and No-touch conditions in each patient with that of the control sample (Crawford and Garthwaite 2005).

Every VD patient was significantly impaired in detecting bilateral stimuli in both Touch and No-touch conditions ($P < 0.05$ for all comparisons), but they also showed a significant difference between these conditions consisting in an advantage for the touching stimuli (difference between the Touch and No-touch conditions: P9 = 19%, $t_{11} = 1.7$, $P < 0.05$; P10 = 13%, $t_{11} = 4.7$, $P < 0.01$; and P11 = 13%, $t_{11} = 5.8$, $P < 0.01$) (see Fig. 4).

Instead, TD patients showed a selective impairment only in the Touch condition ($P < 0.05$) with the performance in No-touch condition being within the normal range. Crucially, every TD patient showed a significant difference between the 2 conditions consisting in a disadvantage for the Touch condition at variance with the VD group (difference between the 2 conditions: P12 = –27%, $t_{11} = 8.6$, $P < 0.01$; P13 = –9%, $t_{11} = 4.3$, $P < 0.01$; and P14 = –10%, $t_{11} = 2.5$, $P < 0.01$) (Fig. 4).

Finally, VTD patients were impaired in both the Touch and No-touch conditions ($P < 0.01$), and they did not showed any

difference between these 2 conditions ($P = 0.2$); D– patients scored normal on both conditions ($P = 0.5$), without difference among them ($P = 0.7$).

Association between the Crossmodal Effect of the Sight of Touch and the Anatomoclinical Data

Multiple correlations (Pearson correlation) were performed to examine the relation between the effect induced by the sight of touch on bilateral trials (facilitation or impairment of visual detections, i.e., Touch minus No-touch, with positive values indicating that the sight of touch improves visual detections and negative values indicating that the sight of touch impairs visual detections) and the following variables: age; time from stroke (in months); visual, tactile, and motor deficit as assessed at the neurological examination; and performance at the neuropsychological assessment. All the 14 patients were considered in these analyses. Only a significant correlation was found: The difference between Touch and No-touch on bilateral trials was positively correlated with the difference between visual and tactile extinction (tactile detections on bilateral trials minus visual detections on bilateral trials) ($r = 0.7$, $P < 0.01$). This suggests that the presence of a tactile impairment, without visual extinction, was associated with a visual impairment in the Touch condition (i.e., the greater the tactile deficit, the greater the disruption of visual perception in the Touch condition). By contrast, the presence of visual extinction, without tactile extinction, was associated with a benefit in the Touch condition (i.e., the greater the extinction of visual rather than tactile stimuli, the greater the benefit in the Touch condition).

With respect to the lesion profile, correlation analyses were performed between the change in visual detections between Touch and No-touch stimuli on bilateral trials and the lesion size (number of voxels) in each ROI. We considered all the 11 patients for which the lesion reconstruction was made. Only 2 significant effects were found. A negative correlation was found between the difference between Touch and No-touch stimuli and the size of the lesion in the postcentral cortex ($r = -0.65$, $P < 0.04$). This means that the bigger the damage to the postcentral gyrus, the greater the impairment induced by the sight of touch; conversely, the greater the tactile-induced benefit, the smaller the damage to the postcentral gyrus.

Additionally, there was a positive correlation between the difference between Touch and No-touch stimuli and the size of the lesion in the supramarginal gyrus ($r = 0.71$, $P < 0.02$), suggesting that the larger the damage to this area, the greater the benefit induced by seeing touches. The results from the correlation analysis are reported in the Supplementary Tables S1 and S2.

To further explore the relation between the lesion site and the dissociation in performance between VD and TD patients, we looked for those regions that were more damaged (in percentage) in TD than in VD patients by using a subtraction method (Rorden and Brett 2000). Figure 3*b* depicts the resulting subtraction plot, showing the hotspots indicating regions that were more damaged in the TD group than in the VD group. The more damaged areas in TD patients were the postcentral gyrus, the inferior frontal gyrus, and the precentral gyrus; instead, in VD patients, the inferior and middle temporal cortices and the supramarginal gyrus were more largely involved (see also Table 2).

Discussion

Our findings showed that viewing a touch can differentially modulate visual perception, depending on the underlying damaged sensory modality: If the visual modality is affected (VD patients), the sight of touch can improve the visual disorder, whereas in case of a damage confined to the tactile modality (TD patients), the sight of touch impairs visual perception. These visuo-tactile effects were present only on bilateral trials, which is the most attentional demanding condition. This supports the view that the somatosensory mirror system might produce a behaviorally relevant cross-modal modulation of visual perception only within a certain perceptual threshold (Blakemore et al. 2005).

Conversely, patients with both visual and tactile disorders or patients without visual and tactile disorders did not show any visual effect induced by the observation of touch. Hence, the crossmodal modulation of visual perception by touch does not occur when sensory awareness is deeply impaired across vision and touch (VTD, who also exhibited USN) or, else, completely undamaged (D–, as well as in healthy controls or in general for processing ipsilesional stimuli).

Correlation analyses clearly indicate that the deficit of sensory processing predicts the effect induced by the sight of touch: TD is associated to the disruption of visual processing induced by touch observation, while visual extinction is associated to the visual improvement induced by touch observation. Instead, other clinical factors, such as motor- and USN-related disorders, stroke duration, or age, were not associated to the effects induced by the sight of touch (although we must be cautious about absent correlations since the sample size was pretty small).

The effect of seeing touches was unrelated to the attribution of the observed touch to oneself (egocentric view) or to somebody else (allocentric view), in line with previous evidence (Keysers et al. 2004; Schaefer et al. 2005). Rather, we found that all groups of patients were more impaired in detecting visual stimuli seen from an egocentric perspective than from an allocentric perspective. These findings are consistent with the idea of a right hemisphere specialization in the egocentric processing of spatial information (Stein 1989; Iachini et al. 2009). The right hemisphere also plays a special role in the ability to distinguish the self from others (Decety and Chaminade 2003) and the recognition of self body parts (Frassinetti et al. 2008). Hence, a lesion to the right hemisphere may have affected any possible advantage/disadvantage by self-referential processing of the observed hands, regardless of the tactile component.

Overall, the behavioral results provide compelling support that the processing of visual stimuli conveying a tactile content involves different mechanisms as compared with the processing of visual stimuli without a tactile content. Indeed, the former seems to automatically engage an additional somatosensory representation, likely through a mirror simulation process, in order to build a crossmodal representation of the seen touch. In patients with visual deficits, but spared somatosensation, the recruitment of this undamaged representation in the tactile modality favors their defective visual perception. This effect may be viewed as a compensatory crossmodal mechanism for visual processing, activated by the sight of touch. Conversely, in patients with tactile deficits, the sight of touch impinges on visual perception negatively.

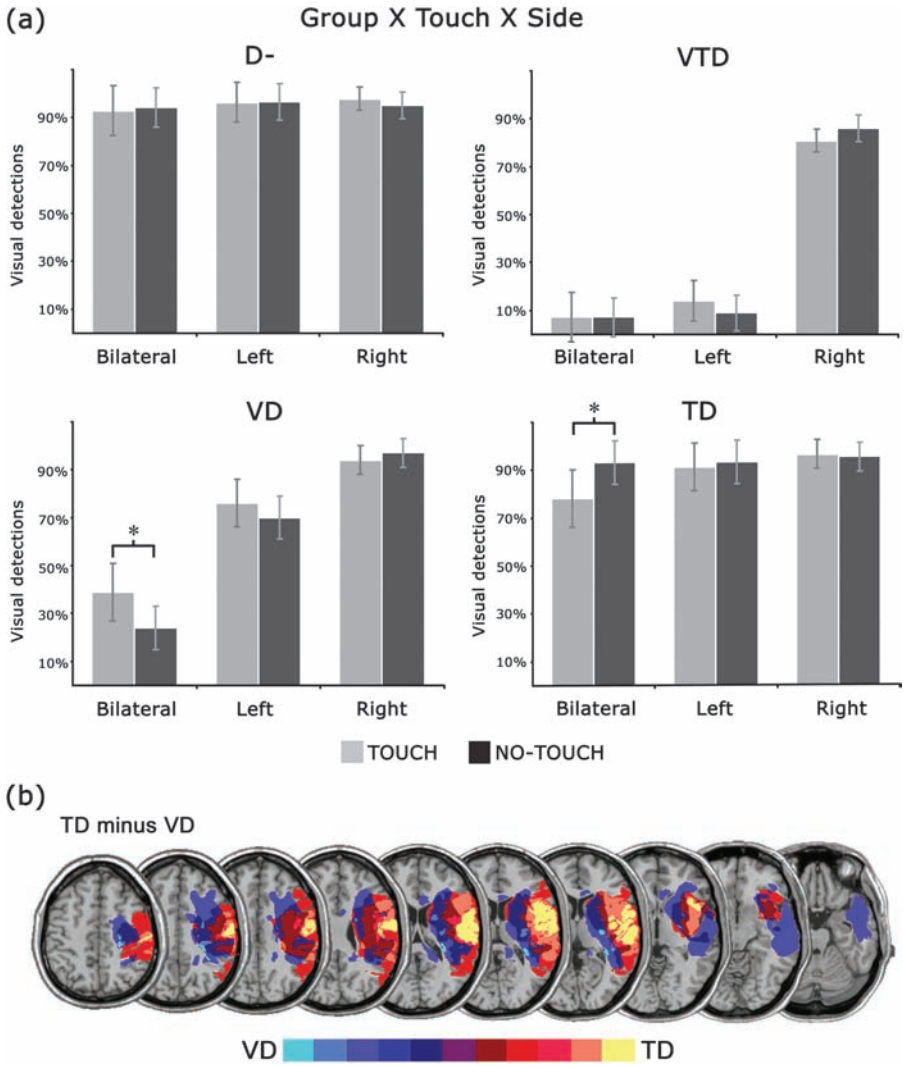

Figure 3. (*a*) Modulation of visual perception by the sight of touch in right brain–damaged patients: D− = no sensory or spatial disorders, VTD= visual plus tactile deficit, VD= visual deficit, TD= tactile deficit. Light gray bars = accuracy in Touch trials; dark gray bars = accuracy in No-touch trials. Asterisks indicate the significant difference in bilateral trials for Touch and No-touch stimuli in VD and TD patients. Error Bars = standard error of the mean. (*b*) Overlapping of VD and TD lesions. Each bar represents 20% increments: lightest blue areas = the maximum percentage of specificity of the VD group for that region; lightest yellow areas = maximum percentage of specificity of the TD group for that region; and purple = regions where there is an identical percent of VD and TD.

The striking finding is that the damage to the tactile modality actually reverses the crossmodal effect of the sight of touch, although does not abolish it, suggesting that mirror system for touch is spared and still functioning. In fact, the impaired tactile processing appears now associated to the activation of a "disrupted" somatosensory representation of the observed touch, which selectively hampers visual perception of touch, even if visual processing is undamaged per se.

Worth mentioning, in our design, the blocked nature of Touch and No-touch trials (which likely provided clues as to whether a Touch or No-touch trial was about to occur) could have induced patients to rely more on the visual or tactile representation to detect the visual stimulus, depending on the block of trials, hence fostering the difference between Touch and No-touch conditions. However, this is still perfectly compatible with the role of a putative imitative mechanism for touch in biasing the participant's performance.

With respect to the anatomoclinical correlation of the present visuo-tactile effects, the lesions of VD and TD patients differed mainly with respect to the involvement of regions in the postcentral gyrus, suggesting a likely role of cortical areas underpinning tactile sensation in matching observed and felt touch. In fact, the size of the lesion in the postcentral gyrus was associated with the effect induced by the sight of touch. A larger damage to the postcentral gyrus corresponded to a greater visual impairment induced by the tactile component of the observed visual event; conversely, the sparing of this area was positively associated with the benefit induced by the sight of touch. This finding is in line with functional neuroimaging evidence showing the activation of somatosensory areas by the sight of touch (Keysers et al. 2004; Blakemore et al. 2005; Schaefer et al. 2005; Ebisch et al. 2008). Noteworthy, mirror-touch synesthesia, that is, people reporting on their own skin the feeling of a touch observed on other person, is associated with an unusual hyperactivation of somatosensory cortices during touch observation (Blakemore et al. 2005). Conversely, complete haptic deafferentation impairs the ability to infer

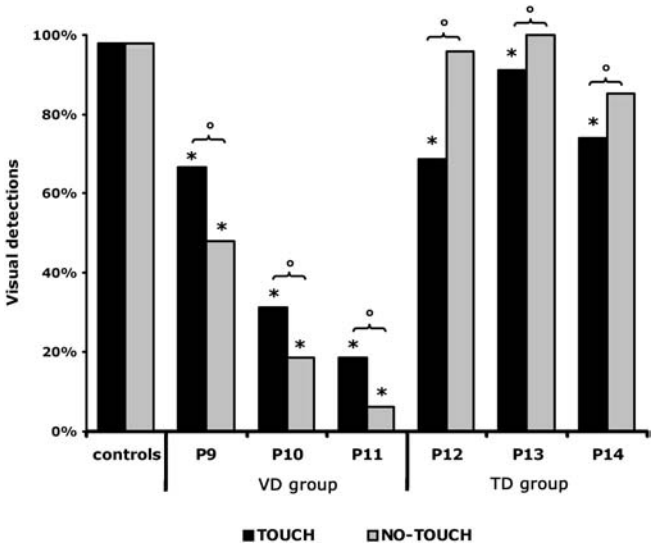

Figure 4. Visual detection accuracy on bilateral trials in healthy controls, VD, and TD patients. Stars: significant difference between each patient's performance and that of the control group for each Touch and No-touch conditions. Circles: significant difference between Touch and No-touch conditions in the individual patient as compared with the same average difference in the control group.

someone else's sensory expectations through the observation of their actions (Bosbach et al. 2005).

On the other hand, the amount of involvement of the supramarginal gryus (SMG) was associated to the visual facilitation induced by the sight of touch. In other words, touch observation seems to provide a facilitatory effect, which is likely mediated by crossmodal somatosensory activation, that can compensate an impaired visual processing due to an SMG damage. The right SMG is important for strategic orienting of spatial attention (Perry and Zeki 2000; Corbetta and Shulman 2002), particularly within the visual modality. The temporary disruption of this area can impair the ability to strategically orient toward visual stimuli but without affecting the shift of attention to somatosensory stimuli in the same location (Chambers et al. 2004). Moreover, the SMG is a crucial site of multisensory convergence, being involved in the integration of visual and tactile information for space and body representation (Macaluso and Maravita 2010). Finally, functional magnetic resonance imaging evidence in brain-damaged patients shows that crossmodal visuo-tactile extinction reflects the interaction of competing information from vision and touch within surviving multisensory parietal areas along the right SMG (Sarri et al. 2006). This last finding is also in accordance with the selectivity of our crossmodal effects for bilateral trials.

Overall, the lesion data suggest that the visuo-tactile effects observed in the present study may involve a local mechanism within somatosensory areas as well as the interaction between somatosensory and posterior parietal areas. First, some SI neurons react to visual stimuli that are behaviorally associated with tactile information (Zhou and Fuster 1997), while SII is involved in visuo-tactile integration of body-related information (Carlsson et al. 2000; Avikainen et al. 2002). Second, both SI and SII have afferent and efferent cortical connections with multisensory regions of the posterior parietal cortex (PPC) (Keysers et al. 2010). PPC contains neurons sensitive to both vision and touch (Andersen 1997; Rizzolatti et al. 1997; Duhamel et al. 1998) and shows strong visuo-tactile interactions for conscious perception, body, and space representation (Halligan et al. 2003; Maravita et al. 2003; Farne et al. 2005; Brozzoli et al. 2006; Bolognini and Maravita 2007; Macaluso and Maravita 2010; Pasalar et al. 2010). Therefore, the crossmodal influences induced by the sight of touch on visual processing may arise as a result of an interaction between the visual input and the tactile input, being the last provided by the somatosensory cortex through a simulation process. These interactions may take place in multisensory convergence zones and/or attentional control structures of the posterior parietal cortex (Macaluso and Driver 2005; Stein and Stanford 2008).

With respect to the supposed involvement of the mirror neuron system in the observed crossmodal effects, seminal studies in monkeys report that the viewing of actions performed by other individuals activates frontal and parietal cortical areas (Rizzolatti and Craighero 2004). However, it has been recently proposed that motor and parietal areas might play a differential role in simulating, respectively, efferent (i.e., motor) and afferent (somatic) components of observed actions. The visual observation of movements eliciting somatic sensations in the onlooker, ranging from pain to the sensation of being touched, activates a large sensorimotor parietal network, including SI (Avenanti et al. 2007). In this regard, our findings suggest that the sensory-parietal, but not the motor-frontal, component of the mirror neuron system is likely to affect the ability of simulating the afferent (somatic) components of observed actions.

In future experiments, it would be interesting to include a control task with nonhuman stimuli depicting touches, in order to assess whether the visuo-tactile mirroring mechanism for touch applies to the sight of "any" touch or whether it is restricted to human contact. There is evidence suggesting that mirror areas are preferentially activated by "social" touch (i.e., the observation of touch to another human, relative to the contact between objects) and, more generally, when the target of the action is biological rather than inanimate (Tai et al. 2004; Blakemore et al. 2005).

In conclusion, the perception of visual events conveying a tactile content seems to involve not only the visual but also the somatosensory modality. This is true when there is a visually based encoding of somatic sensations, such as touch, that is likely to activate an inner mirror-like simulation of the observed tactile sensation (Gallese 2003; Blakemore et al. 2005; Keysers et al. 2010; Bolognini et al. 2011). The pervasiveness of the functional interplay between vision and touch is such that a damage to the somatosensory system can lead to a dysfunctional visual processing, whereas an intact somatosensory mirroring mechanism can even aid an impaired visual perception. On a broader perspective, the activation of a shared circuit for visual and somatosensory processing of tactile events might allow an automatic and implicit coding of the sense of touch through vision, in line with the theories of embodied simulation (Rizzolatti and Craighero 2004; Keysers et al. 2010).

Our findings may also have clinical implications. After ischemic insults, there may be an increase in axonal sprouting, unmasking of existing connections, and other occurrences of neuronal plasticity. These changes are likely to create the opportunity for crossmodal functional rewiring (Ro et al. 2007; Beauchamp and Ro 2008), which may promote plastic visuo-tactile interactions, usually masked under normal conditions. Crossmodal rehabilitation approaches may be useful to prevent

the formation of inappropriate crossmodal deficits (as the visually induced impairment in TD patients) or may subserve crossmodal compensation (as the improvement in visual perception induced by the sight of tactile stimuli in VD patients).

Supplementary Material

Supplementary material can be found at: http://www.cercor.oxfordjournals.org/

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