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Abstract—*Background:* Left caloric vestibular stimulation (CVS) transiently reduces impairments of right-brain-damaged patients with left unilateral neglect, including left hemianesthesia, contralateral to the side of the lesion (contralesional). Conversely, no effect on right contralesional hemianesthesia in left-brain-damaged patients is seen with right CVS. This discrepancy is unexplained. *Methods:* The authors explored the effect of CVS on right- and left-brain-damaged patients with hemianesthesia. One left-brain-damaged patient had an fMRI study during tactile stimulation before and after left CVS. The same fMRI touch study, without CVS, was performed in neurologically unimpaired subjects. *Results:* A transient remission of right hemianesthesia associated with left brain damage was observed, provided that cold CVS was administered to the left ear. In the left-brain-damaged patient studied with fMRI, left CVS modulated the neural response to right hand tactile stimuli of a portion of the secondary somatosensory area (SII) of the right hemisphere. In neurologically unimpaired subjects, fMRI scans showed that the same part of area SII in the right hemisphere was activated by ipsilateral right-sided touches and to a larger extent than area SII in the left hemisphere by left-sided touches. *Conclusions:* Left caloric vestibular stimulation is effective on both left and right hemianesthesia because it modulates the hemisphere that has a more complete representation of, or is capable to attend to, the whole somatosensory surface of the body. These results suggest a hardwired hemispheric asymmetry in hand representation, starting from a somatotopically organized brain region such as area SII.

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In right-brain-damaged patients, the irrigation of the left ear canal with cold water (left caloric vestibular stimulation [CVS]) temporarily ameliorates left hemianesthesia[1] and many manifestations of the syndrome of left spatial neglect.[2-5] On the other hand, the mirror-reversed experimental setting has been unsuccessful so far: Right cold CVS does not remit right hemianesthesia.[1] This hemispheric difference suggests that left hemianesthesia in right-brain-damaged patients is a manifestation of inattention for the left side of space.[1,6] Amelioration of left hemianesthesia after left CVS may be due to a reorientation of attention toward the left side of space or to the reorganization of disrupted spatial representations of it.[1,6] This conclusion is also reinforced by the observation, in a few left-brain-damaged patients with right unilateral spatial neglect, that right hemianesthesia remits after right cold CVS. However, these infrequent patients are likely to have a noncanonical hemispheric lateralization of spatial functions.[1]

There is functional imaging evidence in a right-brain-damaged patient suggesting possible neural mechanisms of the transient recovery from left hemianesthesia after left cold CVS even after damage to somatosensory cortex.[7] In that patient, with a lesion including a large portion of the somatosensory cortex, the neural correlates of the temporary recovery of left hemianesthesia after CVS was the activation of a set of regions in the right hemisphere (insula, right putamen, inferior frontal gyrus in the premotor cortex).[7] Thus, even though the brain lesion was in the right hemisphere, the results suggested that the modulation of somatosensory perception induced by the CVS was mediated by a right hemispheric neural network, putatively involved in somatosensory processing and awareness. Consistent with this interpretation, it is worth noting that in neurologically unimpaired subjects, left cold CVS brings about a bilateral cortical activation (precentral gyrus, premotor cortex, temporoparietal junction), which is more extensive in the contralateral hemisphere (insula, somatosensory cortex SII).[8,9]

In the current study, we explored the hemispheric contribution in the processing and perceptual awareness of somatosensory inputs, through left and right cold CVS in left-brain-damaged patients with right hemianesthesia. To better qualify this neurofunc-

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Table 1 *Demographic and clinical characteristics of left- and right-brain-damaged patients*

Patient no.	Sex	Age, y	Lesion side	Education, y	Aphasia	Motor deficit	Somatosensory deficit	Visual half-field deficit	Anosognosia	Personal neglect	Extrapersonal neglect
L1	M	57	L	18	+	0/3	3/3	0/3	0/3	0/3	−
L2	M	63	L	5	+	3/3	3/3	3/3	0/3	0/3	−
L3	F	77	L	5	+	3/3	3/3	0/3	0/3	0/3	−
L4	F	63	L	5	+	2/3	3/3	1/3	0/3	0/3	−
L5	F	58	L	10	+	3/3	3/3	0/3	0/3	0/3	−
L6	F	36	L	13	+	3/3	3/3	0/3	0/3	0/3	−
R1	M	39	R	6	−	3/3	3/3	1/3	0/3	0/3	+
R2	F	73	R	10	−	3/3	3/3	1/3	0/3	0/3	+
R3	F	82	R	5	−	2/3	3/3	3/3	3/3	0/3	+
R4	F	82	R	9	−	3/3	3/3	0/3	3/3	0/3	+
R5	F	75	R	8	−	3/3	3/3	3/3	3/3	3/3	+
R6	F	61	R	5	−	3/3	3/3	2/3	1/3	1/3	+

(+/−) = presence/absence of deficit; 0 = no deficit; 3 = maximum deficit.

tional specialization, the study included also right-brain-damaged patients with left hemianesthesia, and lesions were mapped to control for any bias due to differential involvements of somatosensory brain regions. Finally, fMRI scans were also used to clarify the issues raised by the behavioral experiments.

Methods. *Subjects.* Six left-brain-damaged patients and six right-brain-damaged patients with contralesional somatosensory deficits (hemianesthesia) were examined behaviorally. One patient of the six left-brain-damaged patients (Patient L6) and eight neurologically unimpaired control subjects (mean age 30 years, range 25 to 35 years) underwent fMRI scans (see below). All patients were right handed and had no family history of left handedness. All patients had had an ischemic or hemorrhagic stroke. No patient had history or evidence of previous cerebrovascular disease, dementia, or psychiatric disorders. All patients had a CT scan to map the distribution of the brain lesions. Patients' characteristics are summarized in table 1.

Neurologic and neuropsychological assessment. Motor, somatosensory, and visual field deficits (confrontation technique) were assessed by a standard procedure.[1] Unilateral spatial neglect was assessed using letter and line target cancellation tasks.[10,11] A defective performance in at least one of the two tasks was considered evidence of unilateral spatial neglect. Anosognosia for the upper limb motor disorders was rated by a 4-point scale,[12] yielding scores ranging from 0 (no deficit) to 3 (maximum deficit).

One left-brain-damaged patient (Patient L6) was also submitted to fMRI during tactile stimuli prior to and following left CVS. L6 was a 36-year-old woman, who had experienced an embolic stroke in the left frontotemporoparietal cortices 10 years before this study. In particular, the damage involved left somatosensory area SII, a vast part of the left postcentral gyrus, the posterior limb of the internal capsule, and a large portion of the thalamus with a complete destruction of the ventroposterior somatosensory nuclei. At the time of testing, the patient was still right hemiplegic and densely hemianesthesic and did not show visual field deficits, in agreement with a distinct sparing of the lateral geniculate nucleus, the optic radiations, and the primary visual cortex. A moderate residual aphasic disorder was evident, characterized by a nonfluent speech with a relatively preserved comprehension.

CVS. CVS was performed by pouring 20 mL of iced water for 1 minute in the external ear canal. Left-brain-damaged patients received both left and right stimulations (in different occasions, with a time interval of at least 24 hours). In right-brain-damaged patients, only the left external ear was irrigated for ethical reasons, as right cold CVS may induce a worsening of neglect-related symptoms in these patients.[3] CVS produced a brisk nystagmus in all patients, with the slow phase toward the side ipsilateral to the irrigation.

Somatosensory assessment. Tactile perception was assessed before CVS (baseline), immediately after, and 30 minutes after CVS. The baseline and post-CVS blocks included 40 stimuli: 15 brief touches of the examiner's index fingertip to the dorsal surface of the patient's right hand, 15 touches to the dorsal surface of the left hand, and 10 "catch" trials, where no stimulus was given. Stimuli and catch trials were delivered in a random fixed order. Throughout the testing session, patients were blindfolded. Immediately before each touch and catch trial stimulus, the examiner gave a verbal warning ("Now"). Patients had received instructions to report verbally (i.e., "yes") when they perceived a touch to either hand.

Lesion-mapping analysis. Lesions detected on the best CT scan available for each patient were remapped onto a standard stereotactic space using an MRI template (voxel size $1 \times 1 \times 1$ mm) and MRIcro software (www.mricro.com).[13] The lesion mapping was performed by two independent and experienced operators, and the mean map was used for further analysis. The whole volume of the lesion and its regional distribution were assessed with MRIcro using the Automatic Anatomic Labeling template distributed with the software. Comparison of the distribution of the lesions between right- and left-brain-damaged patients was done using χ^2 and Mann–Whitney statistics.

fMRI scanning and data analysis. fMRI scans were performed on a 1.5 T Philips Infinion scanner (for Patient L6; Best, the Netherlands) and a General Electric Sigma Horizon System (Milwaukee, WI) scanner for eight neurologically unimpaired control subjects. Both scanners were equipped with echo planar imaging (EPI) hardware. Before fMRI scans, scout spin echo sagittal scans were acquired to visualize the anterior and posterior commissures on a midline sagittal section and to facilitate data acquisition, roughly along the bicommissural plane. The volume selected for fMRI scanning was from -16 mm below the bicommissural plane to +80 mm above it. Before fMRI data acquisition, field homogeneity was adjusted by means of "global shimming." Activation images were acquired using an EPI gradient echo sequence (flip angle 90°, echo time [TE] = 60 milliseconds, repetition time [TR] = 3,000 milliseconds, field of view [FOV] = 280×210 mm, matrix = 96×64). The selected volume consisted of 24 contiguous transverse images (4 mm), and these were acquired every 3 seconds. The original sampling volume matrix was resampled to a $64 \times 64 \times 24$ matrix, resulting in a final voxel size of $4.36 \times 4.36 \times 4$ mm. At the end of fMRI data collection, a structural spin echo data set matched to the fMRI images (flip angle 90°, TE = 20 milliseconds, TR = 500 milliseconds, FOV = 280×210 mm, matrix = 256×256) was acquired.

After image reconstruction, raw data visualization and prepro-

cessing were performed with MRIcro. All subsequent data analyses were performed in MATLAB 6.5 (MathWorks, Natick, MA) using Statistical Parametric Mapping software (SPM99, Wellcome Department of Imaging Neuroscience, London, UK). fMRI scans were first realigned to account for any movement during the experiment and were then stereotactically normalized into the standard MRI space using the EPI template provided by SPM99.[14] Areas of signal loss, or corresponding to the lesion in Patient L6, were masked, that is, excluded from the calculations, at the stage of normalization to avoid distortions in the normalization process. At this stage, the data matrix was interpolated to produce voxels of dimensions $2 \times 2 \times 4$ mm. The stereotactically normalized scans were smoothed through a Gaussian filter of $10 \times 10 \times 10$ mm to improve signal-to-noise ratio. Global differences in fMRI signal were compensated for using proportional scaling for all voxels. High-pass filtering was used to remove artifactual contribution to fMRI signal such as, for example, physiologic noise from cardiac and respiratory cycles. Comparisons of blood oxygenation level–dependent (BOLD) signal across tasks were made for all voxels by using the t statistic, thus generating statistical parametric maps of the t values (SPM maps).[15,16]

Stimuli during fMRI. Patient L6. Light touches were delivered alternately to the right or to the left hand in successive 30-second epochs (ten scans per epoch). Within each epoch, the touch was delivered on the same side. The touch rate was 1/s, for a total of 30 touches. Resting state epochs were interleaved with the touch epochs. This basic experiment was performed while the subject was hemianesthesic (first 120 scans), immediately after left CVS (further 120 scans), and 30 minutes after left CVS (final 120 scans). No response to touches was required to the patient during the fMRI scans. However, it is important to note that touch perception was flawless in both hands immediately after CVS as well as at the end of the second fMRI measure (about 10 minutes after the CVS procedure). To assess how the vestibular signals modulate the response of the cerebral cortex when the right hand of the patient was touched, a task by vestibular stimulation interaction effect was computed, according to a factorial design. To control for generic time-dependent effects, the interaction effect "vestibular stimulation by touch" for the right hand was compared with the same effect and time course for the left hand.

Normal control subjects. Neurologically unimpaired control subjects received the same light touches to the right or to the left hand in alternate blocks (overall 120 scans per subjects; 60 resting scans, 30 scans for right and 30 for left hand touches). No vestibular stimulation was performed in the normal control subjects, as this touch experiment was aimed to determine the receptive field characteristics of the somatosensory regions activated by touch.

In neurologically unimpaired subjects, we computed the simple main effects associated with right or left hand activation and the existence of hemisphere by side of the stimulation interaction effects. The hemispheric differences for stimulation of the hand ipsilateral to a given hemisphere (e.g., right hemispheric activations for right hand touches larger than left hemispheric activations for left hand touches; or left hemispheric activations for left hand touches larger than right hemispheric activations for right hand touches) were calculated following the procedures described below.

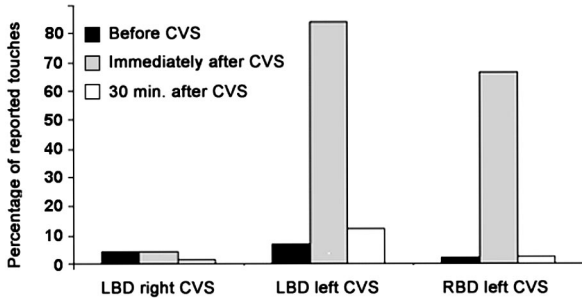

Figure 1. Percentage of touches delivered to the contralesional hand (left hand in right-brain-damaged patients, right hand in left-brain-damaged patients) reported before, after caloric vestibular stimulation, and at a 30-minute delay. LBD = left-brain-damaged patients; RBD = right-brain-damaged patients.

We first performed a voxel-wise analysis of the simple main effects of right hand and left hand touch in each individual according to a fixed-effect analysis. These analyses generated for each subject contrast images containing the statistical information about the fMRI signal change observed for a given statistical comparison (e.g., right touches compared with rest). Further, individual neurologically oriented (i.e., images where the anatomic right is displayed on the right side of the image) contrast images for right hand stimulation were compared with radiologically oriented (i.e., images with the anatomic right on the left side of the image) contrast images for left hand stimulation. Using two opposite pairwise t-test contrasts ($1\ -1$ or $-1\ 1$), we therefore assessed the existence of any greater activation in the right or in the left hemisphere for ipsilateral stimuli. This analysis conforms to a random effect analysis valid for population inference.[17] Whereas all other analyses in the patient and controls involved the whole brain, this lattermost analysis was constrained to the SII region, operationally defined as the voxels in the vicinity of the parietal operculum that were activated by either left or right hand stimulation.

Results. The behavioral experiments replicate the observation that left CVS induces a transient remission of left hemianesthesia in right-brain-damaged patients, whereas right CVS has no such effect on right hemianesthesia in

Table 2 *Percentage of touches, delivered to the hand contralateral to the side of the lesion, reported before, immediately after CVS, and at a 30-minute delay in left- and right-brain-damaged patients*

	Left CVS			Right CVS				Left CVS		
Left-brain-damaged patients	Before CVS	CVS	30 min after CVS	Before CVS	CVS	30 min after CVS	Right-brain-damaged patients	Before CVS	CVS	30 min after CVS
L1	7	93	13	20	20	0	R1	14	91	0
L2	20	87	7	7	0	7	R2	0	67	0
L3	0	87	0	0	0	0	R3	0	15	0
L4	0	67	0	0	0	0	R4	0	65	0
L5	7	93	53	0	0	0	R5	0	100	0
L6	7	80	0	0	7	0	R6	0	63	15
Mean	6.8	84.5	12.2	4.5	4.5	1.1	Mean	2.3	66.8	2.5
SD	7.3	9.8	20.7	8.1	8.1	2.9	SD	5.7	29.6	6.1

CVS = caloric vestibular stimulation.

Table 3 *Distribution of brain lesions in left- and right-brain-damaged patients*

Brain region	Left-brain-damaged patients		Right-brain-damaged patients	
	No. of patients with damage	Lesion volume (mm^3)	No. of patients with damage	Lesion volume, ($mm^{3)}$
Global lesion volume	6/6	82,258 (113,820)	6/6	126,316 (120,042)
Inferior frontal gyrus (operculum)	3/6	1,568 (3,234)	3/6	3,434 (3,954)
Precentral gyrus	4/6	4,697 (7,582)	3/6	1,052 (1,461)
Postcentral (area SI)	4/6	4,246 (6,020)	3/6	3,837 (5,041)
Rolandic operculum (area SII)	5/6	2,551 (2,900)	5/6	5,930 (4,707)
Supramarginal gyrus	4/6	3,069 (3,080)	4/6	4,885 (5,641)
Insula	4/6	3,780 (6,084)	5/6	7,289 (4,998)
Internal capsule (posterior limb)	3/6	Nm	6/6	Nm
Corona radiata	5/6	Nm	6/6	Nm
Thalamus (whole volume)	2/6	422 (742)	5/6	1,878 (2,559)
Thalamus (VPL)	2/6	Nm	5/6	Nm

Values in parentheses are SD.

VPL = ventroposterolateral nucleus; Nm = not measured.

left-brain-damaged patients (figure 1; table 2). The absence of an effect of right CVS on the right hemianesthesia of left-brain-damaged patients was not explained by the left-sided lesions being larger or more complete in terms of the involvement of somatosensory areas such as SI, SII, the insula, the ventral premotor and frontal opercular cortices, the supramarginal gyrus, the ventroposterolateral thalamus, the internal capsule, and the corona radiata (table 3). The absence of behavioral effects on right hemianesthesia by right CVS in left-brain-damaged patients was associated neither with overall larger left-sided lesions nor with a more systematic or significantly larger involvement of somatosensory cortical areas or of the somatosensory thalamus (χ^2 tests and Mann–Whitney tests, all not significant; see table 3).

However, figure 1 also illustrates a novel behavioral finding: Right hemianesthesia in left-brain-damaged patients fully recovered for about 30 minutes following left CVS, much as did left hemianesthesia in right-brain-damaged patients. This observation rules out the possibility that the behavioral effect is due to the lateralization of the some spatial cues: The side of the effective stimulation (left) was opposite to the side of the body (right) in which the behavioral phenomena of recovery of hemianesthesia were observed.

FMRI scans in left-brain-damaged Patient L6 provide evidence concerning the brain areas modulated by the vestibular signals. Patient L6, in the behavioral CVS study performed before the fMRI experiment, showed a dramatic, although transient, improvement of left hemianesthesia.

Figure 2 illustrates the areas where an increased activation ($p < 0.05$ corrected for multiple comparison) was observed for touches delivered to the right hand after left CVS as opposed to touches delivered to the right hand before left CVS. The statistical effect was assessed as a task by vestibular stimulation interaction effect, the fMRI signal being also compared with the same time course for the left hand. The modulatory effect of vestibular stimulation involved the right temporoparietal junction, including area SII and the supramarginal gyrus, which were significantly activated after left CVS and touches to the right hand, with a significant touch-by-CVS-by-hand interaction effect (stereotactic coordinates and Z scores of the interaction effect: $x = 54$; $y = -32$, $z = 24$, Z score = 5.8; $x = 5$ 2; $y = -40$; $z = 26$, Z score = 5.4).

The right hemispheric effect in Patient L6 suggests a special competence of that hemisphere in touch detection. The fMRI data in the normal control subjects support this conclusion. Comparison of touch stimuli vs resting state caused activations ($p < 0.001$ or more) of the contralateral ventroposterior lateral nucleus of the thalamus, of somato-

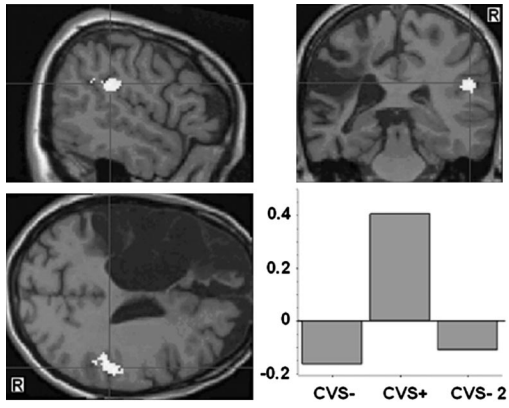

Figure 2. Physiologic effect of left caloric vestibular stimulation (CVS) on touch in Patient L6. Area of significant interaction of left CVS with right-sided touches in Patient L6 (left) displayed on her MRI scan. The bar plot shows the average fMRI blood oxygenation level–dependent signal change (compared with rest in that brain region) before CVS (−), after CVS (+), and at a 30-minute delay (CVS-2). The values of signal change are mean centered. Images are displayed with the anatomic right on the right side. R = anatomic right.

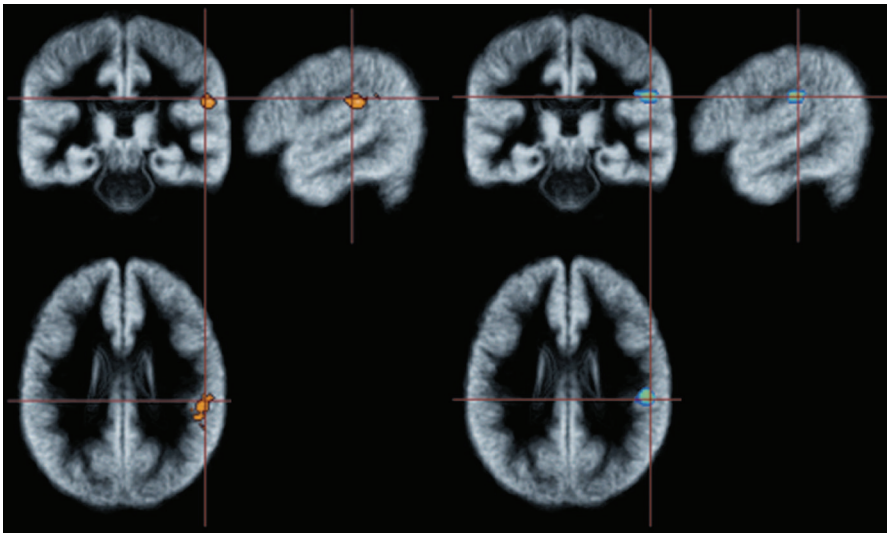

Figure 3. Interaction of left caloric vestibular stimulation with right-sided touches in Patient L6 and brain areas showing larger activations for ipsilateral stimuli in neurologically unimpaired control subjects. fMRI activation data of Patient L6 (on the left, yellow) and of control subjects (on the right, blue) are displayed on an average high-resolution gray matter MRI template. Images are displayed with the anatomic right (R) on the right side.

sensory areas SI and the supplementary motor area, and of area SII bilaterally. We then explored the hypothesis of hemispheric asymmetries in the cortical responses for tactile stimulation, namely, that the right hemisphere may have a mapping of the right, ipsilateral, side of the body, more comprehensive than that of the left hemisphere for the left side of the body. This hypothesis was assessed by a voxel-by-voxel analysis of the parietal operculi containing area SII and the supramarginal gyrus, brain areas known for having abundant neurons with bilateral and ipsilateral somatosensory receptive fields in the monkey.[18] A significantly greater activation for ipsilateral hand stimuli was seen in the right hemisphere compared with the left hemisphere; this was located in the parietal operculum (area SII) in stereotactic locations virtually not distinguishable from those of the interaction effect seen in Patient L6 ($x = 52$; $y = -24$; $z = 28$; Z score = 2.3; $x = 42$; $y = -34$; $z = 16$; Z score = 2.1; figure 3). No reversed effect (left hemispheric larger activation for left hand stimulation) was seen in any location within the left parietal operculum, indicating that the lack of a reversed effect was not simply due to different locations in the two hemispheres of an ipsilateral body representation within area SII.

Discussion. The current results elucidate the neural processes whereby left cold CVS ameliorates both left[1,19] and right hemianesthesia, pointing to a main role of the right hemisphere and suggesting some possible hard-wired mechanisms. In particular, our results explain why previous attempts to use cold right CVS in right hemianesthesia failed.[1] That study, using the mirror-reversed paradigm of the successful left CVS for left emianesthesia, somewhat assumed a equal role of the right and left hemisphere for lateralized tactile stimuli, if not for attention toward the body. Our behavioral and functional anatomic data strongly suggest that this is not the case and help to decide among competing interpretations on the mechanisms underlying the interaction of vestibular signals with hemianesthesia in left- and in right-brain-damaged patients. The amelioration of right hemianesthesia after left CVS in left-brain-damaged patients, rather than after right CVS, does not support the interpretation that recovery of touch perception might be due to a lateral cue (i.e., the CVS), bringing about an exogenous orienting of attention toward the affected right side,[20] as elicited by a sensory (CVS) stimulation delivered to the same side.[21] It remains in principle possible that the final result of left CVS on right hemianesthesia consists in the recovery of a pathologic attentional bias, namely, a right somatosensory hemi-inattention.[22] Interpretations in terms of the temporary recovery of a disrupted somatosensory representation of the left side of the body are equally possible.[1] In any case, the current findings are consistent with the view that the right hemisphere possesses a neural machinery supporting somatosensory attention or representation for the whole body space.[6,23,24]

On the basis of the current data, an interpretation in terms of exogenous attentional cueing toward the side of the stimulated ear cannot be excluded for right-brain-damaged patients with left hemianesthesia, who receive left CVS. It should be noted, however, that right-brain-damaged patients transiently recover from left spatial neglect[3] and left hemianesthesia[19] also following right warm CVS. To summarize, the complete behavioral pattern of CVS is better accounted for in terms of higher-order effects, attentional or representational in nature,[25] that do not reflect a lower-level lateral cuing toward the side of the stimulated ear. It is the physiologic characteristics of the CVS and its central neural effects that matter, rather than the side to which the CVS is delivered.

The behavioral effects of left CVS, together with the evidence that this stimulation mainly activates the right hemisphere,[8,9] suggest a predominant role of the right hemisphere in tactile conscious perception, independent of the side on which touches are delivered. The current fMRI data support this conclusion, as well as localize a specific region of the right hemisphere with such properties: area SII.

In left-brain-damaged Patient L6, with a complete destruction of somatosensory afferents from the tha-

lamic level and with a vast cortical lesion involving the somatosensory cortices, transient recovery from hemianesthesia was associated with the activity of right hemispheric somatosensory neurons. The same right hemispheric cortical field seen in the patient was activated in a population of normal subjects by ipsilateral stimuli. This suggests that the neurons that generated this BOLD effect response have ipsilateral receptive fields. In principle, ipsilateral receptive fields in somatosensory cortex could be due to uncrossed somatosensory afferents from the spinal cord or to callosal connectivity.[26] In the cat, there are thalamic neurons with ipsilateral receptive fields based on connections with the neospinothalamic tract.[27] The existence of ipsilateral spinothalamic afferents in humans, sufficient to guarantee some crude tactile perception, is supported by a case of a spinal lesion sparing only one lateral white matter spinal column: One month after the lesion, this patient was able to detect touch stimuli on both sides of the body below the lesion.[28] In neurologically unimpaired subjects, magnetoencephalography data also show ipsilateral responses in area 3b with time latencies incompatible with a callosal origin.[29] For Patient L6, with her lesion involving both the somatosensory thalamus and the posterior limb of the internal capsule, the right hemisphere could not rely on somatosensory inputs arriving from the left hemisphere. We may then conclude that right hemispheric neurons in area SII have some somatosensory representation of the right hand through an ipsilateral input, modulated by the vestibular signals. In neurologically unimpaired subjects, a response of right area SII for ipsilateral right-sided stimuli larger than the response of left SII for left-sided stimuli also implies a hard-wired asymmetry in the surface representation of the hand. It remains to be established whether this asymmetry also applies to other segments of the body. Its functional relevance should be considerable, however, given the prominent role of the hand in spatial behavior, such as pointing and reaching. The existence of such a hard-wired asymmetry for tactile perception may contribute to understanding the neural bases of hemispheric specialization that underpins the clear-cut hemispheric difference in the clinical manifestations of complex spatial disorders, such as unilateral spatial neglect, a disorder more frequent after right hemispheric lesions.[6,22,30]

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