OBSERVATION

Neural Mechanisms of Embodiment

Asomatognosia Due to Premotor Cortex Damage

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Background: Patients with asomatognosia generally describe parts of their body as missing or disappeared from corporeal awareness. This disturbance is generally attributed to damage in the right posterior parietal cortex. However, recent neuroimaging and electrophysiological studies suggest that corporeal awareness and embodiment of body parts are instead linked to the premotor cortex of both hemispheres.

Patient: We describe a patient with asomatognosia of her left arm due to damage in the right premotor and motor cortices. The patient's pathological embodiment for her left arm was associated with mild left somatosensory loss, mild frontal dysfunction, and a behavioral deficit in the mental imagery of human arms.

Conclusion: Asomatognosia may also be associated with damage to the right premotor cortex.

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ASOMATOGNOSIA IS DEFINED as a patient's feeling that parts of his or her body are "missing" or have disappeared from corporeal awareness.[1] Evidence from patients with focal brain damage suggests that asomatognosia is linked to posterior parietal lesions, especially of the right hemisphere, and generally affects the contralesional body.[1-8] Although experimental findings in patients with asomatognosia are rare, these studies showed that asomatognosia may be modified by touching the missing body part or by looking at it, suggesting multisensory mechanisms in awareness and embodiment of body parts.[1,9]

Herein we describe a patient with asomatognosia of her left arm due to 2 small lesions in the right premotor cortex (PMC) and the motor cortex. We discuss asomatognosia with respect to involved brain functions and regions.

REPORT OF A CASE

A 51-year-old, right-handed woman with no neurological or psychiatric antecedents described the following experience with respect to her left arm. Sitting in front of her computer, she unexpectedly felt dizzy and felt that parts of her left arm (**Figure 1** A) had disappeared (Figure 1B). Much to her surprise she could see the table on which she had rested her left arm as if she could see the table *through* the arm, and saw her left arm only above her elbow, with a clear-cut border (Figure 1B). She could not move her left arm or hand. After several minutes, the patient experienced progressive restoration of her left hand and arm starting laterally, then medially (Figure 1C), while leaving 2 holes in the middle of her hand (Figure 1D). Later the 2 holes fused (Figure 1E) until the arm was complete again (Figure 1F), and she was able to move it some minutes later. No other body parts or elements of extrapersonal space were experienced as modified.

The neurological examination showed moderate left-sided hypoesthesia of the arm and lower face (light touch and pinprick). Position sense was normal. There was no left-sided hemianopia or paresis, and muscle tendon reflexes were normal. Finger tapping, index-thumb opposition, diadochokinesis, writing, copying, and drawing were normal. Results of the neuropsychological examination demonstrated a mild executive deficit in the Lurias alternating sequences test and verbal semantic fluency (9.7 words/min; *z* score, −1.50). No deficits were detected in language, calculation, or praxis. Results of the Benton Facial Recognition Test and Benton Judgment of Line Orientation Test, the Culver test,[10] and the Visual Object and Space Perception Battery were normal. There were no signs of visuospatial neglect, unimodal extinction (visual, tactile, or auditory), bimodal extinction (tactile-visual), or allesthesia.[11-13] There was no finger agnosia, astereognosia, agraphesthesia, or topographagnosia and no deficit in right-left discrimination. Findings of computed tomography performed at admission were normal. Magnetic resonance imaging 12 days after lesion onset showed 2 small ischemic lesions in the

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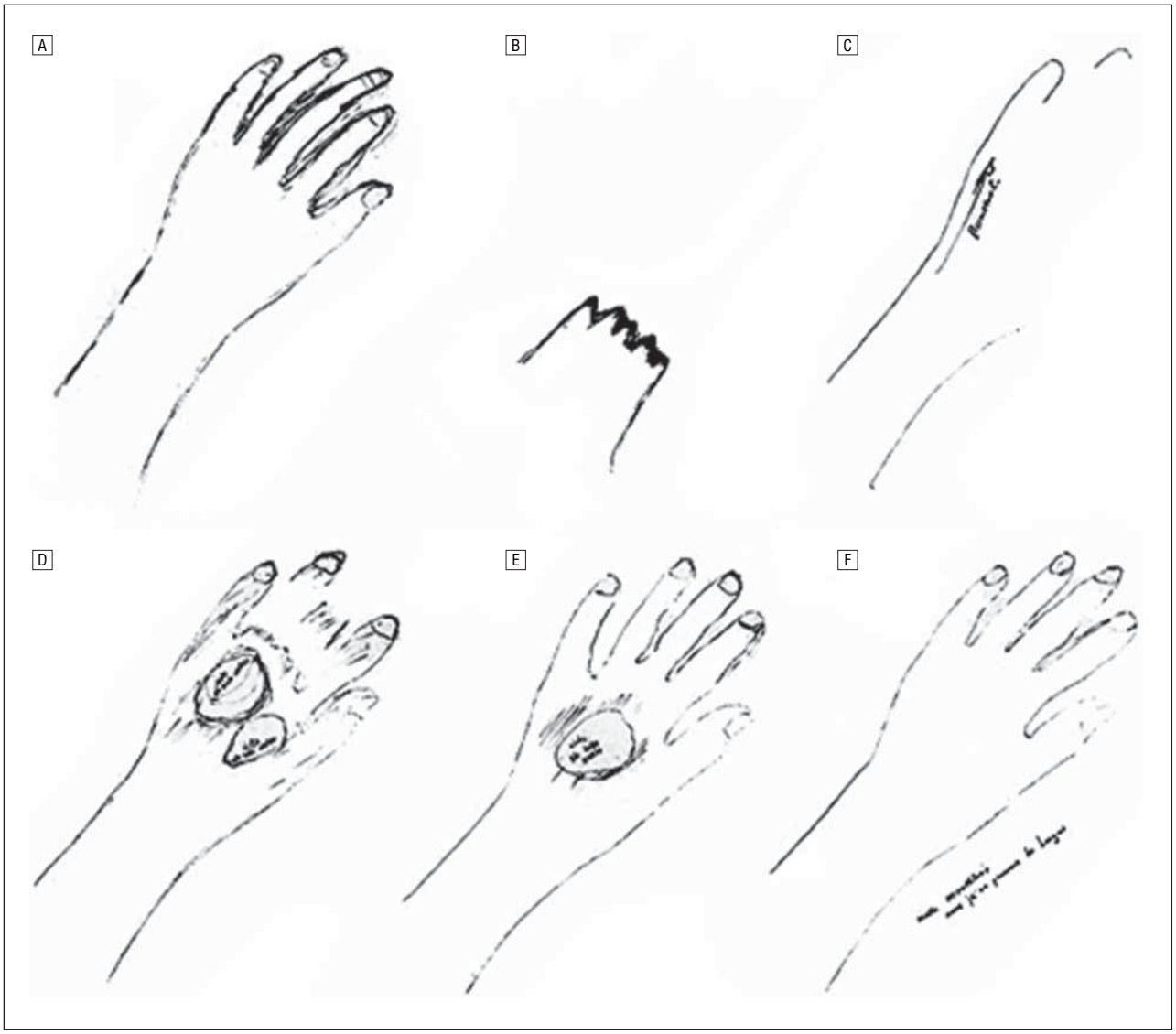

Figure 1. Illustrations drawn by the patient describing asomatognosia. A, Normal left arm; B, "disappeared" left arm; C, "restored" left arm beginning laterally; D, "holes" in the left hand; E, fusion of 2 holes; and F, full restoration.

PMC and the primary motor cortex (**Figure 2**) of cardiac embolic origin.

To further explore the functional mechanisms of asomatognosia and body-part processing, we tested the patient's capacity to mentally imagine human body parts. For this, we compared the patient's performance in a mental rotation task involving body parts (arms) with performance using noncorporeal external objects.[12] Both types of stimuli were presented in 7 different angles (0°-180°) and were in a normal view or an inverse view (for body parts, the contralateral hand was attached to the ipsilateral arm; alphanumeric characters were presented in a mirror-reversed view) (**Figure 3**A-D). The patient and 7 age-matched healthy control subjects had to determine as quickly as possible whether the stimulus was the correct one or the inverse one.[12] The patient had significantly longer reaction times for the arms (mean ± SD, 1795 ± 121 milliseconds) than control subjects (mean ± SD, 907 ± 70 milliseconds; $t_7 = 6.3$; $P < .001$) (Figure 3E), but she had similar reaction times for external objects (mean ± SD, 505 ± 80 milliseconds; controls, 698 ± 93 milliseconds; $t_7 = -1.6$; $P = .14$) (Figure 3F). The same difference was found in the error rates. The patient made significantly more errors for arms (mean ± SD, 16.5% ± 3.0%) than did control subjects (mean ± SD, 6.3% ± 1.1%; $t_7 = 3.2$; $P < .01$), but she had the same number of errors for external objects (mean ± SD, 5.3% ± 2.6%; controls, 8.0% ± 3.9%; $t_7 = -0.58$; $P = .57$). In addition, the patient showed a global mental rotation function for the external objects (reaction times increasing linearly with the angle of rotation) but not for the arms (Figure 3E-F).

COMMENT

Patients with asomatognosia may describe that "the left arm and leg seem to be 'missing'" or that the affected body part seems "to disappear, or to fall out of corporeal awareness."[1(p237-238)] Asomatognosia is generally attributed to posterior parietal lesions, especially of the right hemisphere[1,7,8] Our patient's symptoms, affected body side, and hemisphere lesions are concordant with previous cases

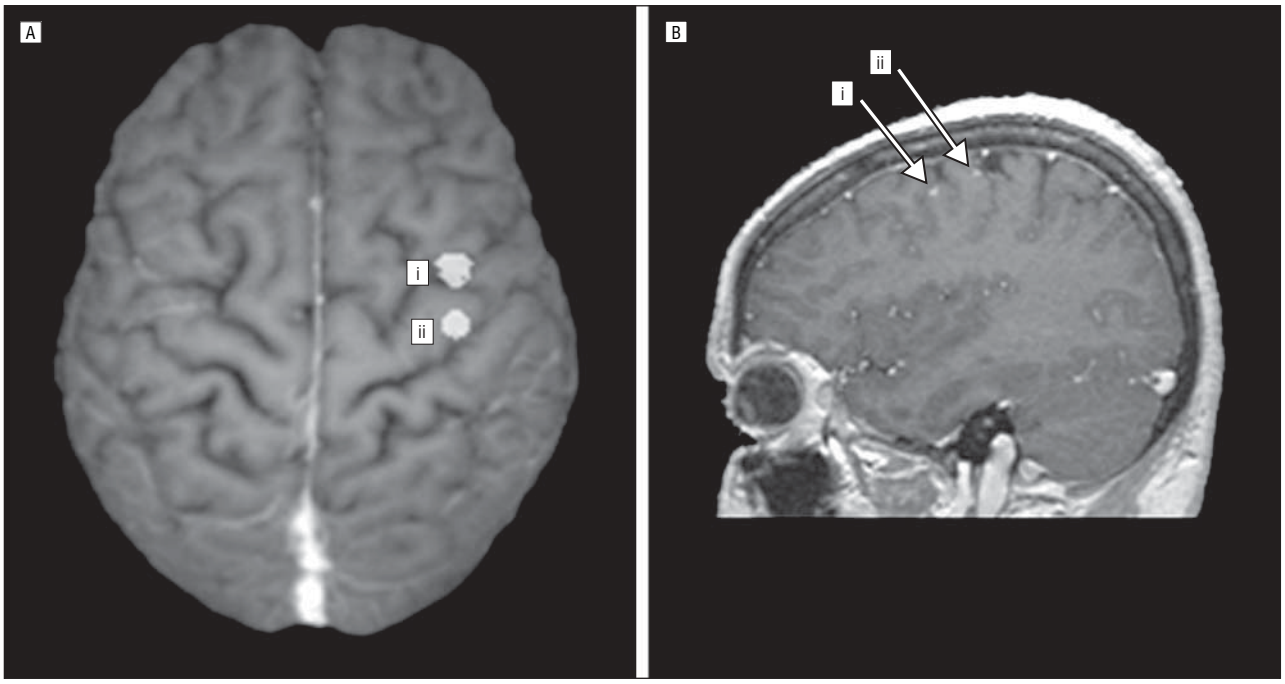

Figure 2. Lesion location. A, Three-dimensional reconstruction of a T1-weighted magnetic resonance image. The 2 small lesions confined to the right premotor cortex (i) and motor cortex (ii) are projected on the cortical surface. B, Magnetic resonance image (T1-weighted, with gadolinium enhancement; sagittal section) showing the 2 hypointense small ischemic lesions.

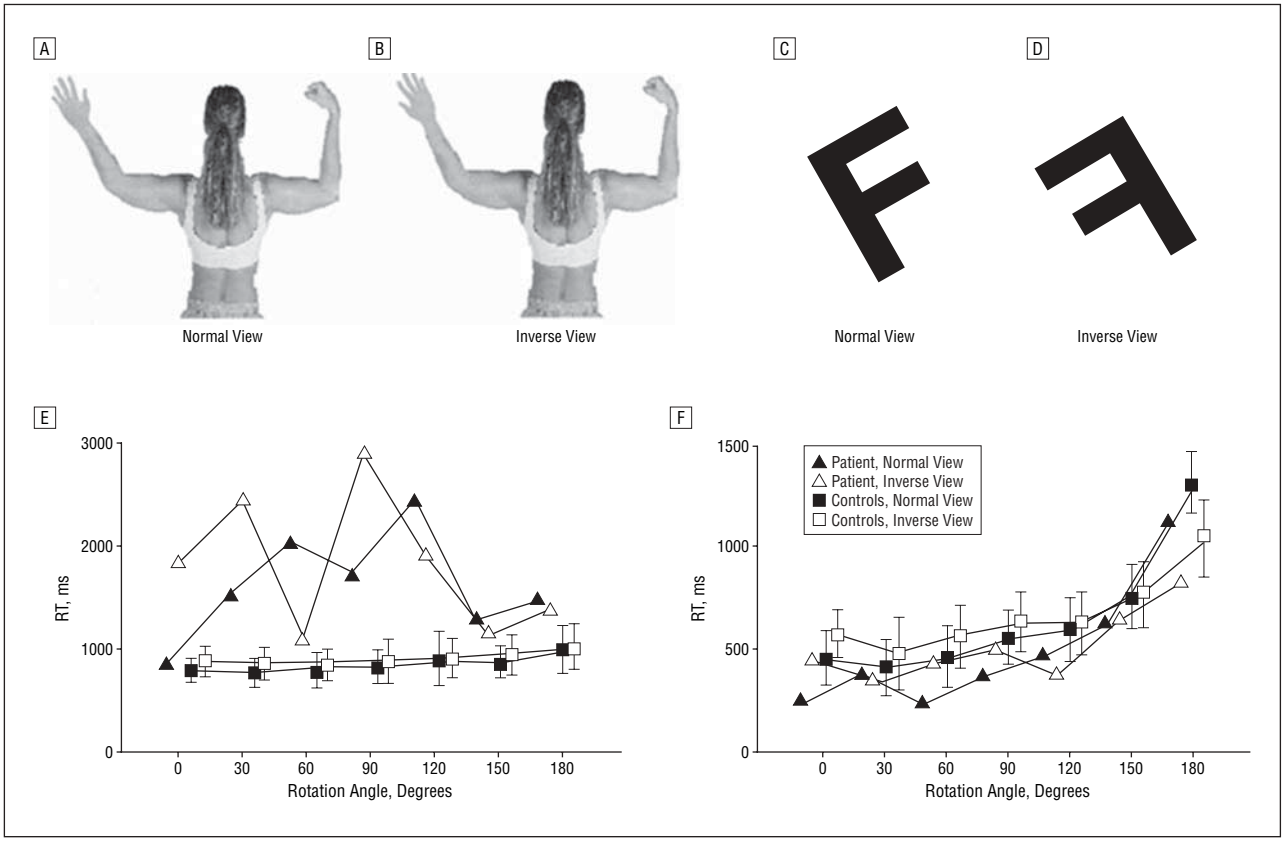

Figure 3. Behavioral findings. A-D, Illustration of the stimuli used including body parts (arms) (A and B) and external objects (letters) (C and D) that were in a normal view (A and C) or in an inverse view (B and D). The stimuli were presented in 7 different angles. Patient and control subjects had to determine as quickly as possible whether the stimulus was the correct one or the mirror-reversed one. E and F, Mean reaction times as a function of orientation are plotted separately for the patient (black) and control subjects (open) in normal and inverse view for body parts (E) and external objects (F). RT indicates reaction time.

of asomatognosia. We report the following novel observations: (1) the predominantly visual character in our patient, (2) the associated behavioral deficit, and (3) the restricted lesions to the right motor cortex and PMC. These observations are discussed with respect to the multisensory coding of body parts and higher-level aspects of one's own body perception and embodiment.

The importance of vision of one's own body for somatosensory perception was investigated by Tipper et al,[14] who showed that visual inspection of a body part, independent of proprioceptive orienting, enhances the detection of somatosensory stimuli. Multimodal visuotactile extinction and processing[15] also illustrate the importance of visual and somatosensory modalities in one's own body perception,[1,11,13] as do reports of patients with asomatognosia whose experiences of missing body parts can be corrected by enhanced input from vision, touch, and passive or active body part movements.[1-3,9] Based on the visual character of asomatognosia in the present patient, we suggest that "disappearance from corporeal awareness"[1] may not only be experienced as a somatosensory loss, but also as a visual loss, probably due to involvement of multisensory mechanisms. This visual loss may lead to the experience of not seeing one's own body parts and seeing other objects at its position in the visual field.[16]

Our patient's disturbed body perception was corroborated by a behavioral deficit in the mental rotation of body parts.[12] Mental imagery per se was not disturbed, as the mental rotation of external objects was comparable to that of healthy subjects. The association of a (negative) visual illusion restricted to the patient's body,[17] loss of embodiment, brachiofacial hypoesthesia, and a selective deficit in the mental rotation of body parts corroborates the importance of multisensory and sensorimotor mechanisms in the perception of body parts and embodiment.[16] Mental rotation of external objects is associated with parietal activation, whereas mental rotation of body parts involves a larger network, including the PMC and motor and parietal cortices.[18]

The links among embodiment, multisensory processing for body parts, and the neural substrates of this embodiment and multisensory processing in the frontoparietal cortex have also been examined in healthy subjects using functional magnetic resonance imaging. Illusory embodiment was induced by investigating the "rubber-hand illusion," in which tactile sensations are referred to an alien limb.[19] Although illusion-related activity was found in the posterior parietal cortex and PMC, only premotor activation correlated with the strength of the illusion. This suggested that embodiment may be correlated with activity in the PMC, a finding that is concordant with the location of brain damage in our patient.[19] Our data add causal evidence to correlative functional magnetic resonance imaging evidence that embodiment of one's upper extremity may also be related to the PMC and motor cortex. However, most clinical evidence speaks against this finding, as most cases of asomatognosia are linked to posterior parietal damage.[1-8] Furthermore, electrophysiological studies in monkeys[20,21] and neuroimaging studies in humans[15,19] showed the involvement of the PMC as well as the posterior parietal cortex in the coding of body parts and embodiment. Therefore, it was proposed that separate visual and somatosensory inputs converge on premotor neurons, as the PMC contains neurons that share visual and tactile receptive fields, or that the PMC receives input from parietal regions integrating visual, tactile, and proprioceptive information.[20,21]

Based on our clinical, neuropsychological, and neuroimaging evidence, we conclude that the posterior parietal cortex and PMC are involved in the coding of embodiment. Given that the underlying neurons share many functional characteristics, it is plausible that interference with either area, but especially in the right hemisphere, may lead to pathological forms of embodiment.

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