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Anosognosia for hemianaesthesia: A voxel-based lesion-symptom mapping study



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ABSTRACT

Brain-damaged patients affected by hemianaesthesia (i.e., the loss of tactile sensibility on the contralesional side of the body) may deny their deficits (i.e., anosognosia for tactile deficits) even reporting tactile experience when stimuli are delivered on the impaired side. So far, descriptive analysis on small samples of patients reported that the insular cortex, the internal/external capsule, the basal ganglia and the periventricular white matter would subservise anosognosia for hemianaesthesia. Here, we aimed at examining in depth the anatomo-functional nature of anosognosia for hemianaesthesia by means of a voxelwise statistical analysis. We compared two groups of left hemiplegic patients due to right brain damages differing only for the presence/absence of anosognosia for left hemianaesthesia. Our findings showed a lesional cluster confined mainly to the anterior part of the putamen. According to the current anatomical evidence on the neural basis of sensory expectancies, we suggested that anosognosia for hemianaesthesia might be explained as a failure to detect the mismatch between expected and actual tactile stimulation.

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1. Introduction

Anosognosia (from the Greek *nosos* disease and *gnosis* knowledge; an-/a- is a negative prefix) is the lack of awareness for neurological/neuropsychological deficits following focal brain lesions. Such a denial behavior has been reported selectively for motor (e.g., hemiplegia), sensory (cortical blindness, hemianopia, hemianaesthesia), and cognitive deficits (see

Prigatano, 2010 for a review), and it has been taken as evidence of modality-specific disorders of consciousness. Indeed, when different symptoms are simultaneously present due to a brain damage, patients may be unaware of one of them but aware of another revealing that the monitoring of different aspects of behavior is underpinned by discrete brain mechanisms (see Berti, Làdavas, & Della Corte, 1996 for details on this point).

Within the sensory domain, anosognosia for hemianaesthesia (hereinafter AHA) is diagnosed when patients are

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persuaded that they are still able to perceive contralesional tactile stimuli despite the fact that, during the standard neurological examination with eye closed they never report of being touched on the affected side (Bottini et al., 2009; Marcel, 2004; Marcel, Tegnèr, & Nimmo-Smith, 2004; Vallar, Bottini, & Paulesu, 2003; Vallar, Bottini, & Sterzi, 2003). AHA patients may also report an actual tactile sensation when they see a stimulus delivered to their anesthetic body parts (Pia, Garbarini, Fossataro, Fornia, & Berti, 2013; Romano, Gandola, Bottini, & Maravita, 2014). Such a subjective report seems to reflect a real subjective experience of touch rather than a mere verbal confabulation and/or a bias to simply report what is seen, because AHA patients may show normal physiological reactions (i.e., skin conductance response to incoming stimuli delivered to their anesthetic body part; Romano et al., 2014). Interestingly, tactile sensations arise when the physical counterpart is absent (Pia, Garbarini, et al., 2013; Romano et al., 2014).

To the best of our knowledge, only two studies have directly examined AHA (Marcel et al., 2004; Spinazzola, Pia, Folegatti, Marchetti, & Berti, 2008). Both of them demonstrated that when hemianaesthesia (hereinafter HA) co-occur with hemiplegia (i.e., the complete paralysis of the contralesional side of the body; hereinafter HP), AHA can be dissociated from unawareness of HP (hereinafter AHP). In other words, patients can deny their contralesional somatosensory deficits but not their contralesional motor deficits and vice versa. Additionally, Spinazzola and coworkers (Spinazzola et al., 2008) analyzed the individual lesional pattern of four patients affected by AHA reporting that lesions to the insular cortex and to the basal ganglia were crucially associated to AHA (see also (Romano et al., 2014) for similar findings). It was suggested (Spinazzola et al., 2008) that brain damage would impair the ability to distinguish between an internal representation of the sensation and the actual perception of the physical stimulus. The false belief of being still able to perceive tactile stimuli would arise from the intact brain activity within spared areas of the somatosensory system.

In the present paper, we aimed at obtaining a clearer anatomical picture of AHA in order to better understand the nature of the unawareness behavior. As first, we compared the lesional patterns of groups of right brain damaged patients differing only for the presence/absence of AHA. Secondly, on the bases of the anatomical pattern we draw inferences about the functional meaning of the damaged areas.

2. Materials and methods

2.1. Participants

We retrospectively selected the participants of our study from a series of stroke patients with right hemispheric lesions (documented by computerized tomography) and no history of substance abuse/previous neurological diseases, admitted to different rehabilitation centers from 2005 to 2013. The prerequisite to be included in the study was the presence of HA. Additionally, in order to focus exclusively on the neural correlates AHA, patients affected by AHP were not included. As a result, twenty-seven patients (ten women) affected by HA

(sixteen with and eleven without AHA), participated in the study approved by the local ethic committee after signing a written informed consent. In order to perform the anatomical comparisons, HA patients were divided into three subgroups: those who suffered from HP (hereinafter HA_HP group; $n = 11$), those who suffered from both HP and AHA (hereinafter AHA_HP group; $n = 11$), and those who suffered from AHA only (AHA group; $n = 5$). The three groups did not differ (Mann Whitney U Test or Student's *t* test) in terms of age (AHA = mean 69, SD = 7.54; AHA_HP = mean 65.72, SD = 7.86; HA_HP = mean 64.09, SD = 13.41. AHA vs AHA_HP, $p = .61$; AHA vs HA_HP, $p = .9$; AHA_HP vs HA_HP, $p = .73$), educational level (AHA = mean 9.8, SD = 4.6; AHA_HP = mean 8.54, SD = 4.69; HA_HP = mean 10.09, SD = 4.1. AHA vs AHA_HP, $p = .46$; AHA vs HA_HP, $p = .9$; AHA_HP vs HA_HP, $p = .42$) and illness onset (AHA = mean 55, SD = 37.1; AHA_HP = mean 46.63, SD = 12.97; HA_HP = mean 60.27, SD = 33.8. AHA vs AHA_HP, $p = .9$; AHA vs HA_HP, $p = .65$; AHA_HP vs HA_HP, $p = .22$).

2.2. Neurological and neuropsychological assessment

Contralesional visual, motor and tactile deficits were assessed according to a standardized protocol (Bisiach, Pattini, Rusconi, Ricci, & Bernardini, 1997; Bisiach, Vallar, Perani, Papagno, & Berti, 1986) in which scores range from 0 (no deficit) to 3 (severe deficit). What follows is part of the routine neurological diagnosis of HA. Patients blindfolded first receive ten single light touch stimuli applied on the dorsal surface of either the hands or the feet (in random order). They have to report the touch by answering “right” or “left”. The score is assigned on the basis of the performance of healthy participants (100% of detections) as follows: score 3 = 3 to 0 stimuli are reported on the contralesional limb; score 2 = 7 to 4 stimuli are reported on the contralesional limb. When patients score 2 (i.e., who reported some contralesional stimuli), they are administered ten double (symmetrical and simultaneous) stimuli (“right”, “left” or “right and left” answers). They receive the score 1 if they report 7 to 0 stimuli on the contralesional limb, and 0 if they report 8 to 10.

After the motor and somatosensory examination, AHA and AHP were evaluated according to a standard protocol (Pia, Garbarini, et al., 2013; Romano et al., 2014; Spinazzola, Bellan, Pia, & Berti, 2014). For the diagnosis of AHA, HA patients were first assessed with four questions related to tactile perception (see Spinazzola et al., 2014 for details), two for the upper limb (How is sensation in your arm?, Are you able to perceive a light touch on your left hand?) and two for the lower limb (How is sensation in your leg?, Are you able to perceive a light touch on your left foot?). For each question, HA patients had to rate their own perceptual abilities by means of a verbal judgment: normal perception, perception with difficulties, no perception. Awareness of the potential ability to feel sensations was scored comparing the examiner's judgment with the patient's self-evaluation, as follows: no AHA (score 0, full accord in all questions), moderate AHA (score 1, disagreement in one or two questions), severe AHA (score 2, disagreement in all questions). AHA was diagnosed with score 1 or 2 (0 was the cut-off score since healthy participants and patients without HA do not show any disagreement; Spinazzola et al., 2014).

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