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The affective modulation of motor awareness in anosognosia for hemiplegia: Behavioural and lesion evidence



Sahba Besharati ^{a,b,c}, Stephanie J. Forkel ^{c,d}, Michael Kopelman ^a,
Mark Solms ^b, Paul M. Jenkinson ^e and Aikaterini Fotopoulou ^{c,*}

^a King's College London, Institute of Psychiatry, UK^b Department of Psychology, University of Cape Town, South Africa^c Clinical, Educational & Health Psychology, Division of Psychology & Language Sciences, University College London, UK^d King's College London, Department of Neuroimaging, Natbrainlab, Institute of Psychiatry, UK^e Department of Psychology, School of Life and Medical Sciences, University of Hertfordshire, UK

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ABSTRACT

The possible role of emotion in anosognosia for hemiplegia (i.e., denial of motor deficits contralateral to a brain lesion), has long been debated between psychodynamic and neurocognitive theories. However, there are only a handful of case studies focussing on this topic, and the precise role of emotion in anosognosia for hemiplegia requires empirical investigation. In the present study, we aimed to investigate how negative and positive emotions influence motor awareness in anosognosia. Positive and negative emotions were induced under carefully-controlled experimental conditions in right-hemisphere stroke patients with anosognosia for hemiplegia ($n = 11$) and controls with clinically normal awareness ($n = 10$). Only the negative, emotion induction condition resulted in a significant improvement of motor awareness in anosognosic patients compared to controls; the positive emotion induction did not. Using lesion overlay and voxel-based lesion-symptom mapping approaches, we also investigated the brain lesions associated with the diagnosis of anosognosia, as well as with performance on the experimental task. Anatomical areas that are commonly damaged in AHP included the right-hemisphere motor and sensory cortices, the inferior frontal cortex, and the insula. Additionally, the insula, putamen and anterior periventricular white matter were associated with less awareness change following the negative emotion induction. This study suggests that motor unawareness and the observed lack of negative emotions about one's disabilities cannot be adequately explained by either purely motivational or neurocognitive accounts. Instead, we propose an integrative account in which insular and striatal lesions result in weak interoceptive and motivational signals. These deficits lead to faulty inferences about the self, involving a

* Corresponding author. CEHP Research Department, University College London, 1-19 Torrington Place, London WC1E 7HJ, UK.

E-mail addresses: sahba@besharati.com (S. Besharati), a.fotopoulou@ucl.ac.uk (A. Fotopoulou).

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difficulty to personalise new sensorimotor information, and an abnormal adherence to premorbid beliefs about the body.

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1. Introduction

Neurological disturbances of body awareness provide a useful way of investigating the bodily self; a fundamental facet of self-consciousness (Gallagher, 2000). Anosognosia for hemiplegia (AHP; i.e., the denial of motor deficits contralateral to a brain lesion) is a prototypical example of a disturbance in body awareness. AHP occurs more frequently following right perisylvian lesions, and less often following left-hemisphere perisylvian lesions (Cocchini, Beschini, Cameron, Fotopoulou, & Della Sala, 2009). AHP can take various clinical forms, ranging from blatant denial of limb paralysis and associated delusional beliefs to milder forms of motor unawareness (see Fotopoulou, 2014; Jenkinson, Preston & Ellis, 2011; Marcel, Tegner & Nimmo-Smith, 2004). Although the exact aetiology of AHP remains debated, the clinical variability of AHP suggests that it is a multifaceted and heterogeneous phenomenon (Fotopoulou, 2014; Marcel, Tegner, & Nimmo-Smith, 2004; Orfei et al., 2007; Vocat, Staub, Stroppini, & Vuilleumier, 2010). Accordingly, explanations have varied from selective deficits in motor planning, to multi-factorial accounts involving both basic sensorimotor and higher-order cognitive deficits (see Fotopoulou, 2014; Jenkinson & Fotopoulou, 2010 for reviews). These cognitive deficits have been associated with either particular lesion sites such as the premotor cortex (Berti et al., 2005) and the insula (Karnath, Baier & Nagele, 2005), or involvement of a more varied pattern of cortical and subcortical regions and their connections (Fotopoulou, Pernigo, Maeda, Rudd, & Kopelman, 2010; Moro, Pernigo, Zapparoli, Cordioli & Aglioti, 2011; Vocat et al., 2010).

One facet of AHP that has received less empirical attention, despite a long history of clinical observations and theoretical debates (Bisiach & Geminiani, 1991; Weinstein & Kahn, 1955), is the role of emotional factors. On clinical examination, patients typically manifest some degree of blunted affect or ‘indifference’ for their paralysis and its consequences. This indifference (anosodiaphoria, Babinski, 1914) can exist with or without concomitant explicit denial of deficits. On the contrary, depressive symptoms and ‘catastrophic reactions’ (sudden influx of strong, negative feelings and related behaviours; Goldstein, 1939) are encountered rarely. Moreover, there are some clinical indications that as unawareness decreases over time, depressive symptoms begin to emerge in patients who were previously emotionally unresponsive towards their paralysis (Besharati, Kopelman, Avesani, Moro, & Fotopoulou, 2014; Fotopoulou, Rudd, Holmes & Kopelman, 2009; Kaplan-Solms & Solms, 2000). Exceptionally, some patients with or without explicit denial of deficits have been noted to show a strong hatred towards their paralysed limbs (misoplegia; Critchley, 1974), or a disproportionate exasperation with irrelevant, minor disappointments, despite their

apparent indifference for their paralysis (Fotopoulou & Conway, 2004; Kaplan-Solms & Solms, 2000; Weinstein & Kahn, 1950).

Some authors have argued that this lack of affect, or misattribution of negative emotions, is caused by purely psychogenic ‘defence’ mechanisms. According to the now classic theory of Weinstein and colleagues (e.g. Weinstein, 1991; Weinstein & Kahn, 1955), denial and related premorbid coping mechanisms prevent patients from explicitly acknowledging their paralysis, and self-attributing the associated negative emotions. Alternatively, this lack of emotional reactivity has been considered to be the direct consequence of damage to the right (frontal) hemisphere, regarded by some authors as specialised for the processing of negative, withdrawal-related emotions (Davidson, 2001; see Gainotti, 2012 for review). However, neither of these two approaches has been fully supported by empirical evidence. Specifically, the psychodynamic account of AHP fails to explain the relative neuroanatomical and behavioural specificity of anosognosic behaviours (Bisiach & Geminiani, 1991; Heilman & Harciarek, 2010). The ‘valence’ hypothesis has similarly not been supported in the literature; although patients with AHP do typically score lower than control patients in self-report measures of depression and anxiety (e.g., Fotopoulou et al., 2010), more sensitive investigations have shown that they do not differ from controls groups in their ability to experience such emotions (Turnbull, Evans, & Owen, 2005; Vocat et al., 2010). They also show appropriate, negative emotional reactions to their deficits when the latter are evoked implicitly (Fotopoulou et al., 2010; Nadrone, Ward, Fotopoulou, & Turnbull, 2007). Thus, it appears that the relation between AHP and emotion is more complex than suggested by either the psychodynamic or the valence hypothesis.

More generally, such rigid distinctions between purely psychodynamic and neurocognitive explanations have been challenged recently (Fotopoulou, 2012) and integrative accounts of AHP have been put forward (Fotopoulou, 2010; Turnbull et al., 2005; Turnbull & Solms, 2007; Vuilleumier, 2004; see also Turnbull, Fotopoulou & Solms, 2014). According to such theories, complex imbalances between cognition and motivation may be caused directly by damage to insular, striatal, or limbic regions that have recently been found to be selectively associated with AHP (Fotopoulou et al., 2010; Moro, Pernigo, Zapparoli, Cordioli, & Aglioti, 2011; Vocat et al., 2010). For example, Vuilleumier and colleagues have suggested that damage to the basal ganglia may obstruct the “discovery” of deficits, as patients have reduced affective drive to respond to errors and revise beliefs based on new perceptual evidence (Vocat, Saj, & Vuilleumier, 2013; Vuilleumier, 2000, 2004). Similarly, within a computational framework, Fotopoulou and colleagues have suggested that insular and basal ganglia

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