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The virtual bodily self: Mentalisation of the body as revealed in anosognosia for hemiplegia

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ABSTRACT

Despite the coherence and seeming directness of our bodily experience, our perception of the world, including that of our own body, may constitute an inference based on ambiguous sensory data and prior expectations. In this article, I apply a 'psychologised' version of the recently proposed free energy framework to the understanding of certain disorders of neurological unawareness in order to examine how inferential processes may determine our body perception. I specifically consider three facets of body perception in such disorders: namely, the 'external body' as inferred on the basis of exteroceptive signals and related predictions; the 'internal body' as inferred on the basis of proprioceptive and interoceptive signals and related predictions; and lastly the 'impersonalised body' as inferred on the basis of signals from social and third-person perspectives on the body and related predictions. Several conclusions will be drawn from these considerations: (a) there is a deep interdependency of prior beliefs and sensory data; as the brain uses sensory data to update its virtual model of the world, lack or imprecision of sensory prediction errors may lead to aberrant inferences influenced disproportionally by outdated, premorbid predictions; (b) interoception and interoceptive salience have a unique role in our inferences about body awareness and (c) social, 'objectified' prior beliefs about the body may have a silent but potent role in our bodily self-awareness. Finally, the article emphasizes that our learned, virtual model of the body is depended on the nature and thus integrity of the very body that allowed the model to be formed in the first place.

1. Introduction: the 'here and now' as inference

Remembering the past, and being able to project oneself in the future, allows the mind to escape the psychological 'here and now' of experience. Studies in psychology have long established that we do not only project our current self into the future to build a kind of 'as if', imagined future self but we also reconstruct our past self in our memories (Bartlett, 1932). Despite the incredible storage capacity of human memory, what we remember in the now is not always what took place in the past. Instead, the autobiographical incidents that we experience as veridical, coherent and self-defining are frequently unconscious collages of previous recollective attempts, fragments of experienced events, currents thoughts and long term goals (Conway, 2005). In this sense, we have come to understand our autobiographical self as actively, yet unconsciously inferred on the basis of imperfect memory data and current expectations.

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A similar idea for the nature of our experience of current reality, our embodied perception of the world and ourselves in it, has also being long proposed in psychology (e.g. Gregory, 1966). Despite the coherence and seeming directness of our experience, our perception of the world may constitute an inference based on ambiguous sensory data and prior expectations (von Helmholtz, 1878/1971). However, this idea is less established, perhaps given its counterintuitive nature and complex, philosophical implications. We experience the world via our body and the experience of the latter in the 'here and now' is considered as a fundamental aspect of our self-consciousness; our bodily self is the foundation upon which our 'autobiographical', 'narrative' or 'extended' self is built (Gallagher, 2000). If our bodily self is an inference, then our ability to perceive the world and ourselves veridically is called into challenge (see Clark, 2013 for discussion). Leaving aside the majority of the long and complicated philosophical discussions on the nature of reality and our capacity to perceive it, in this paper I will explore similar ideas from the point of view of a recent, influential theory from computational neuroscience. The theory aims to define the idea of perceptual inference using concepts from theoretical physics and mathematics and also aims to ground the same idea on biology and particularly knowledge about the workings of the brain. In the current paper, I will not address the issues of interest in mathematical ways. Instead, I will use a 'psychologised' version of the free energy framework in order to examine some ideas regarding neurological unawareness and ultimately bodily self-consciousness. Specifically, I will use clinical observations, behavioural and neural data from a specific neurological aberration of self-awareness, namely anosognosia for hemiplegia, to explore the possibility that our bodily self-awareness is normally imperfect, in the sense that it is based on a set of inferences about the hidden causes of sensorimotor signals. I also hope to demonstrate that the study of the pathologically exaggerated ways in which we may infer the experience of our own body, can provide insights into the mechanisms of normal perceptual and active inference, and particularly the predictive and social nature of motor awareness.

2. The free energy framework

The starting point of the 'free energy framework' (Friston, 2005) is that humans are biological, self-organising agents that need to occupy a limited repertoire of sensory states for homeostatic reasons (e.g. humans need certain ranges in environmental temperature in order to survive). However, due to the inherent ambiguity and uncertainty of the signals an organism receives from the world, we risk finding ourselves in dangerous states for longer periods than those we could biologically sustain (e.g. in cold climates). We thus need to be able to predict (infer) the causes of our possible sensory states despite the limited or noisy information available to our sensory organs (von Helmholtz, 1878/1971). The framework proposes that our brain engages in a form of probabilistic representation of the causes (e.g. the weather) of our future states (e.g. our temperature) on the basis of noisy sensory data; in other terms, it maintains hypotheses ("generative models") of the hidden causes of sensory input. Furthermore, it uses such input to constantly update its models, so as to reduce its representational errors over time and thus ultimately minimize the risk of surprise (unpredictability, see below for mathematical definition). From a psychological point of view, I will refer to the formation of such models as the 'mentalisation' of sensorimotor signals. Although the term mentalisation is traditionally used in psychology to refer to our cognitive ability to infer the mental states of others and our own, I think the two terms are related (see also Kilner, Friston, & Frith, 2007). In fact, the use of the term 'mentalisation' in this article is intending to ground this traditional concept in its embodied origin.

Returning to the biological level, the free energy framework is biologically constrained by the so-called 'predictive coding' models of perception, stemming primarily from biological and behavioural studies in visual perception, with supporting evidence generated in various modalities (e.g. Henson & Gagnepain, 2010; McNally, Johansen, & Blair, 2011). These suggest that a constant filtering of sensations by top-down predictions and a parallel updating of the latter based on prediction errors (signals representing the mismatch between predictions and sensations), with the ultimate goal of minimizing prediction errors, is an imperfect but highly efficient means of perceiving sensations (Rao & Ballard, 1999). Our brain is assumed to achieve the minimisation of prediction errors by recurrent message passing among hierarchical level of cortical systems, so that various neural subsystems at different hierarchical levels minimize uncertainty about incoming information by generating a prediction (or a prior belief, see below) and responding to errors (mismatches) in the accuracy of the prediction, or prediction errors. Such prediction errors are passed forward to drive the units in the level above that encode conditional expectations which optimize top-down predictions to explain away (reduce, inhibit) prediction error in the level below until conditional expectations are optimized. Such message passing is considered neurobiologically plausible on the basis of functional asymmetries in cortical hierarchies; prediction errors are thought to be conveyed via feedforward connections from lower to higher levels in order to optimize representations in the latter. Predictions from higher-levels are transferred via feedback connections that have both driving and modulatory characteristics and can suppress prediction errors in lower levels. This hierarchy is thus reciprocal but asymmetric and models the nonlinear generation of sensory input (Adams, Shipp, & Friston, 2013).

Based on such hierarchical, perceptual schemes, the free energy principle, rests upon the idea that the brain as a whole works as an Helmholtzian inference machine that is trying to optimize its own model of the world by actively predicting the causes of its sensory inputs (Friston, 2005). Moreover, this inferential process is mathematically understood in Bayesian terms (Bayes' theorem describes an optimal procedure for updating the probabilities assigned to a hypothesis in the light of new evidence), in the sense that it relies on a combination of prior beliefs (probability distributions over some unknown cause excluding any sensory data) and new sensory data to update prior beliefs and generate posterior beliefs (probability distributions over some unknown cause after data have been received). Furthermore, in the free energy principle this hierarchical minimization of prediction errors is understood as a minimization of free-energy on the basis of the formal defini-

tion of the latter; a quantity from informational theory that bounds (is greater than) the evidence for a model of data (Hinton & von Camp, 1993). In this case the data is sensory and free energy bounds the negative log-evidence (surprise) inherent in sensory data, under a model of how the data were caused (See Friston, 2010 for the mathematical details). Given some mathematical assumptions, free energy can be thought of as the amount of prediction error in any given level of the system. Minimizing free energy then corresponds to explaining away prediction errors following the principles of Bayes (Friston, 2010).

However, representing the world in constructive ways (perceptual inference) cannot take us far in terms of our ultimate goal; surviving in an uncertain world. Psychologically speaking, we may become better in predicting ('mentalising') the changes in the environment that act to produce sensory impressions on us, but we cannot on this basis change the sensations themselves and hence ultimately their surprise. A highly innovative conceptual move in the free energy principle framework allows us to understand how we do just that. By acting upon the world we can change its states and therefore 're-sample' the world to ensure we satisfy our predictions about the sensory input we expect to receive. By selectively sampling the sensory inputs that we expect we add accuracy to our predictions about sensory states. Thus, action has an intimate relationship with perception, both being governed by the same master principle, namely reduction of free energy; action can reduce free energy by changing sensory input, while perception reduces free-energy by changing predictions.

In sum, the framework is consistent with theories of embodied cognition and enactive perception (see Clark, 2013 for discussion) that stress the role of embodiment in shaping cognition and propose a close link between action and perception. The framework further makes strong claims about cognition consisting of predictions (or priors) that do not represent the world and our bodily state directly. Instead, in order to evade the inherent surprise of the world, our cognition serves a constantly-updated, Bayes-optimal, iterative, self-fulfilling prophecy. via a cascade of multilevel processing across the neurocognitive hierarchy, we progressively minimise our own representational errors in perception and maximize the posterior probability of generating the observed sensory states in action. In doing so, we generate a kind of 'virtual version' of the sources of our bodily signals. The state of the body and its world is, in this sense, never directly available to perception and always inferred.

At this point however, an important clarification needs to be made. The framework does not imply that the mind is (dualistically) divorced from its environment, including its body. The generative models in question are not viewed as mere functions somehow 'housed' in the brain, and 'informed' about the body by sensory states. Instead, the 'mentalisation' of the body implies physical, changes in the structure and function of the body itself, from the periphery to the brain. Indeed, the framework suggests that the structure and physiology of the brain itself are shaped by sensory states in as much as they shape them (in both ontogenetic and phylogenetic development) (e.g. see Adams et al., 2013). In other terms, the inferential, predictive models of possible causes of sensory input are understood as embodied (e.g. reflecting changes in synaptic connectivity) at different levels of the neurobiological hierarchy. It follows that in its totality, the self-organised, agentive virtual model in question is not merely 'corrected' by our embodiment (i.e. affected by sensory prediction errors), but rather it is our embodiment. In this sense, perception of the body and the world is both truly indirect (virtual, predictive) in the 'here and now' and exact in the long-term: it can ultimately only represent itself.

3. Anosognosia for hemiplegia

Anosognosia for hemiplegia (AHP) is defined as the apparent unawareness of one's paralysis (Babinski, 1914), which occurs typically following stroke-induced right perisylvian lesions, and less often following left perisylvian lesions (Cocchini, Beschin, Cameron, Fotopoulou, & Della Sala, 2009). This prototypical, neurological disorder of body unawareness affects our awareness of action; a composite notion that includes at least two facets, the subjective feeling of moving in the here-and-now, and more general beliefs or judgments about one's motor abilities, such as being able to perform certain bilateral actions (see also below for the related distinction of on-line versus off-line awareness, as well as the distinction between illusory versus delusional awareness). In a subset of patients with concomitant body delusions (somatoparaphrenias, Gerstmann, 1942), the right-hemisphere damage can also affects the sense of body ownership (the subjective feeling that our body is separate from the world and other bodies). Such patients may reject the ownership of one's limb (asomatognosia), misattribute it to others, or vice versa (somatoparaphrenia proper), claim they have three or more limbs (supernumerary limbs), or treat the limb as though it was a separate person (personification; Critchley, 1955).

The typical duration of AHP ranges from days to weeks (Vocat, Staub, Stroppini, & Vuilleumier, 2010), but in about one third of patients the symptoms may last beyond the acute stage of illness and even years (see Pia, Neppi-Modona, Ricci, & Berti, 2004). AHP can be highly specific in that some patients deny their plegia, while being simultaneously aware of other neurological, or neuropsychological disturbances (Bisiach, Vallar, Perani, Papagno, & Berti, 1986; Berti, Làdavas, & Della Corte, 1996; Marcel, Tegner, & Nimmo-Smith, 2004). In terms of AHP's 'extension' (what kinds, or objects of awareness can be compromised, Marcel et al., 2004), some patients acknowledge their motor deficits but fail to adjust to their functional consequences, while others show the opposite pattern (Marcel et al., 2004; Moro, Pernigo, Zapparoli, Cordioli, & Aglioti, 2011). Furthermore, some patients claim their limbs have moved even upon demonstration of the opposite (illusory movements, Feinberg, Roane, & Ali, 2000; Fotopoulou, Tsakiris, Haggard, Rudd, & Kopelman, 2008), while others admit their on-line failure, but fail to update their long-term or, 'off-line' body awareness (Carruthers, 2008; Marcel et al., 2004; Moro et al., 2011; Tsakiris & Fotopoulou, 2008). A related, and at times hard to separate, characteristic of AHP is its 'partiality' (whether unawareness of one's deficit is less than total, Marcel et al., 2004). This property is noted in studies that demonstrate implicit awareness of deficits despite explicit unawareness in verbal (Fotopoulou, Pernigo, Maeda, Rudd, & Kopelman,

2010), or behavioural tasks (Cocchini, Beschin, Fotopoulou, & Della Sala, 2010; Moro et al., 2011; Nardone, Ward, Fotopoulou, & Turnbull, 2007), as well as in studies that show higher awareness of plegia in third-person versus first person tasks. For example, patients have been observed to deny their deficits in direct view but admit them in a video replay (Besharati, Kopelman, Avesani, Moro, & Fotopoulou, 2014; Fotopoulou, Rudd, Holmes, & Kopelman, 2009) or in 3rd-person questions (Fotopoulou et al., 2011; Marcel et al., 2004). Similarly, patients who deny the ownership of their arms (see above) in direct view have been shown to admit them in front of a mirror (Fotopoulou et al., 2011; Jenkinson, Haggard, Ferreira, & Fotopoulou, 2013), and even show improved somatosensation when tested from a 3rd-person perspective (Bottini, Bisiach, Sterzi, & Vallar, 2002), or when they use their ipsilesional hand to actively touch their affected, contralesional arm (Van Stralen, van Zandvoort, & Dijkerman, 2011). More recently, we also showed that anosognosia can be momentarily reduced following affective, social feedback (Besharati et al., 2014).

Despite recent rapid progress in the assessment and understanding of AHP (see Fotopoulou, 2014; Jenkinson, Preston, & Ellis, 2011; Orfei, Caltagirone, & Spalletta, 2009 for reviews), little consensus exists regarding its functional and neuroanatomical explanation. Older theories emphasise deficits in afferent (feedback) and bottom-up signals, while more recent hypotheses focus on modular abnormalities in predictive (feedforward) signals and their role in motor awareness (Berti et al., 2005; Frith, Blakemore, & Wolpert, 2000; Heilman, Barret, & Adair, 1998; see also Jenkinson & Fotopoulou, 2010 for review). For example, on the basis of a computational model of motor control (Wolpert, 1997), Frith et al. (2000) have proposed that although patients with AHP are able to predict the expected sensory consequences of intended movements, they fail to register the discrepancy between predicted and actual sensory feedback because of visuospatial neglect or other sensory deficits. Berti and colleagues (see Berti et al., 2007 for review) suggested that this failure may instead relate directly to damage to the lateral premotor cortex (Berti et al., 2005). This group as well as other groups have further produced physiological (Berti et al., 2007; Hildebrandt & Zieger, 1995; but see Gold, Adair, Jacobs, & Heilman, 1994) and behavioural (Garbarini et al., 2012; Jenkinson, Edelstyn, & Ellis, 2009) evidence showing that there are intact motor intentions in AHP. A further study showed for the first time the direct relation between motor intention and awareness (Fotopoulou et al., 2008). The authors were able to show that patients' illusory awareness of movement reflected an abnormal, selective dominance of motor intentions over visual feedback about the actual effects of movement (elicited by a realistic rubber-hand patients assumed was their own), and this effect could not be explained by neglect. Lastly, while as mentioned above, taking a third-person perspective on the self, verbally (e.g. Marcel et al., 2004) or visually by video-replays (Besharati, Kopelman, et al., 2014; Fotopoulou et al., 2009) may improve awareness, there may also be an alternative, motor explanation of the video-replay results. During video viewing, patients receive visual feedback of their paralysis at a time when they are not intending to move and hence forward signals are rendered irrelevant to motor awareness. Patients' awareness during video-replay needs to rely exclusively on the visual or auditory feedback they receive via the video clip.

Despite the clear value of the 'feedforward' hypotheses, it has become apparent to many authors that a strictly modular, motor explanation is not sufficient to account for all the manifestations of AHP. For example, such theories cannot explain why mood induction can temporarily improve AHP (Besharati, Forkel, et al., 2014), nor account for the extension and partiality of AHP (see above). Indeed, recent experimental studies have shown that awareness dissociations between and within patients are linked with different lesion patterns, including limbic areas non-associated with motor functions (Fotopoulou et al., 2010; Moro et al., 2011). Similarly, in a voxel-based lesion-symptom mapping study, Vocat et al. (2010) demonstrated that the neuropsychological and neural profile of AHP patients' changes in time, and different lesion patterns are associated with AHP at different time points. These studies point to a multi-component disorder occurring due to lesions affecting a distributed set of brain regions, including the insula, premotor and parietal regions but also subcortical areas such as the thalamus, basal ganglia and limbic structures.

More broadly, a number of authors have noted that AHP sometimes has delusional features that cannot be explained solely on the basis of sensorimotor deficits (for discussion, see Fotopoulou, 2010; Frith et al., 2000; Ramachandran, 1995; Turnbull & Solms, 2007; Vuilleumier, 2004; Turnbull, Fotopoulou, & Solms, 2014). Feedforward theories are valuable in explaining the *illusion* of moving (Fotopoulou et al., 2008), but AHP patients do not simply claim that they have the phenomenal experience of moving. In fact, typically patients with AHP do not spontaneously complain of any related, subjectively perceived symptom, whether negative (e.g. I am not moving) or positive (I have the impression that I am moving). On the contrary, AHP is diagnosed on the basis of questioning during which patients are typically asked to report on their current experiences (confrontation questions) and infer their more general motor abilities (see also Marcel et al., 2004). Even patients who report illusory experiences of movement during confrontation and hence presumably base their inferences on such impressions (Fotopoulou et al., 2008), they nevertheless simultaneously ignore the wealth of contrary evidence and medical signs indicating that they are paralysed (e.g. their medical results, disabilities, occasional accidents and others' feedback). This perceptual 'selectivity' is not the same as the one observed in other symptoms such as hemispatial or personal neglect, in the sense that patients with neglect can become aware of the fact that they have neglect after their errors are demonstrated to them. They then continue to do such errors but they are not surprised or in denial when these errors are pointed out to them again. In fact, the subset of patients who cannot become aware of their neglect would be diagnosed as anosognosic for these deficits. Moreover, as aforementioned, there is now also experimental evidence that patients with AHP maintain their denial even after they themselves had admitted their paralysis momentarily (e.g. Besharati, Forkel, et al., 2014). It can thus be said that they adhere to the *delusional* belief that they have functional limbs.

If one accepts that anosognosia has delusional features then theoretical loans from the literature on delusions can be allowed (see Fletcher & Fotopoulou, 2014; Fotopoulou, 2010 for discussions). Of particular interest here is the ongoing

debate between one and two-factor theories. According to the former, rational reasoning on the basis of anomalous or unusual experience should be sufficient to ultimately lead to refractory, delusion beliefs (e.g. Maher, 1992). By contrast, two-factor theories claim that delusional beliefs cannot be explained without the role of additional, cognitive dysfunctions such as reasoning biases, or monitoring deficits that are necessary for the generation and maintenance of the false beliefs (e.g. Davies, Coltheart, Langdon, & Breen, 2001; Garety & Freeman, 1999).

Indeed, also in the literature on anosognosia, a third set of recent theories emphasise that the explanation of anosognosic beliefs and attitudes requires the postulation of additional dysfunctions that prevents sensorimotor and other failures to be re-represented at a higher level of cognitive and emotional self-representation, beyond the sensorimotor domain. These accounts stress the necessary combination of bottom-up and top-down deficits and corresponding lesioned brain regions (Davies, Davies, & Coltheart 2005; Levine, 1990; Levine, Calvanio, & Rinn, 1991; Vuilleumier, 2004). For example, considering anosognosia in the more general context of delusional beliefs, Davies et al. (2005) proposed that anosognosic beliefs maybe explained by a two-factor account used to explain other delusions; abnormal beliefs arise due to a first impairment in perception that prompts the abnormal belief and a second impairment that interferes with higher-order, monitoring processes thus allowing the abnormal perceptions to become abnormal beliefs.

These accounts have undoubtedly being useful in emphasizing the multifaceted nature of AHP, and for attempting to link the understanding of anosognosic phenomena with insights about the cognitive processes that may underlie normal and pathological belief formation (see also Fotopoulou, 2010, 2012). However, these accounts have been criticized for not being falsifiable (Vallar & Ronchi, 2006). Moreover, reflecting the modular epistemology of cognitive neuropsychology (Fotopoulou, 2014 for a critical review), these models treat the described deficits as simply 'additive' and as potentially caused by simultaneous damage to functionally independent lesion sites. For example, Vocat et al. (2010) suggested that a combination of lesions to two or more brain areas within the insular, premotor, parietal and temporal cortex, or the white matter connections that link one or more of these areas with subcortical regions, may lead to different combinations of deficits in functions such as proprioception, spatial neglect, and error monitoring, which in turn lead to anosognosia in different patients. While such 'combinations' of lesion sites and deficits are consistent with the multifaceted nature of AHP, what these accounts lack is a more precise neurobiological and neuropsychological description of the dynamic and hierarchical relation between the affected areas and their integrated functional role in body awareness.

At this point, we turn to the aforementioned free energy framework in order to propose an alternative model of AHP that aims to address precisely this limitation, as well as to describe the unique, virtual nature of perception and the social nature of the bodily self. Finally, this model effectively unifies previous one- and two-factor models of anosognosia, as it does not allow for a distinction between perception (experience) and cognition (inference) at any level. Instead, as explained above, all perception (including all conscious experiences) is always an inference. Thus, according to the model, the difference between the various manifestations and possible subtypes of anosognosia cannot be captured on the basis of this distinction between perception and cognition. Instead, one explanatory factor is indeed sufficient to explain all manifestations of anosognosia, but this factor is not anomalous experience. Rather it is the aforementioned, always embodied and always cognitive form of inference that may become aberrant in anosognosia, as in other delusions (Corlett, Taylor, Wang, Fletcher, & Krystal, 2009). We consider the particular kind of aberration that may lead to AHP in the following section.

4. Abnormal inferences about the body

On the basis of the free-energy principle, this paper puts forward the idea that AHP can be best explained as aberrant perceptual inference at various levels of the neurocognitive hierarchy. It is specifically proposed that the observed lesions result in weak, absent or unreliable prediction errors about sensorimotor states of the affected body parts, which ultimately lead patients to base their inference on premorbid, non-updated predictions about their motor abilities and their agency in the world. This faulty relationship between premorbid, habitual predictions and imperfect processing of current prediction errors is thought to take place at different levels of the neurocognitive hierarchy, consistently with the varied phenomenology and critical lesion sites of patients with AHP (e.g. Fotopoulou et al., 2010; Vocat et al., 2010). We consider some of the critical types of such Bayes-optimal, yet aberrant, inference in further detail below.

4.1. Anosognosia for hemiplegia and the 'External Body'

AHP typically occurs in the context of a number of concomitant sensory impairments, including primary exteroceptive (signals relating to the state of world and the external surface of the body) deficits as well as related, higher order impairments such as visuospatial or, personal neglect (see Vallar & Ronchi, 2006; Small & Ellis, 1996 for reviews). During the 1980s and 1990s, studies under the epistemological remit of cognitive neuropsychology, attempted to establish whether any of these deficits or any given combination of deficits could explain the occurrence of one or more of the above anosognosic phenomena (see Fotopoulou, 2014 for review). These studies revealed double dissociations between AHP and such impairments, suggesting that none was necessary for AHP to occur (e.g. Bisiach et al., 1986; Marcel et al., 2004).

Nevertheless, under the remit of the free energy principle, these dissociations do not exclude the possibility that some of these deficits may act as predisposing, or contributing factors. Exteroceptive signals about the left side of the body, as represented in the connections of right hemisphere subcortical areas (e.g. the thalamus), or re-represented and organised in cor-

tical functional networks of the right-hemisphere (Berti et al., 2005; Fotopoulou et al., 2010; Moro et al., 2011; Vocat et al., 2010) may be weak, or even absent due to damage to one or more of these areas. Such damage may therefore allow predictive signals at these levels to continue to operate 'unchecked' by appropriate prediction errors about the current, exteroceptive state of the body. In other terms, there will not be sufficient or sufficiently precise (see also below) signals to update one's normally, predictive motor awareness. It is further expected that the more and the greater the deficits in one or more of these domains, the greater the likelihood of faulty (anosognosic) inferences. Indeed, a recent paper revealed a positive correlation between the degree of AHP and the combined quantity of such deficits (Vocat et al., 2010).

However, as aforementioned, it is unlikely that these deficits are sufficient to explain the richness of anosognosic phenomena and are best equipped to explain the illusion of moving, rather than the more general delusional beliefs and attitudes s that patients with AHP show in several cognitive and emotional domains. If such deficits were the sole cause of AHP, it would be unclear why other, unaffected bottom-up information about the body (e.g. internal, homeostatic signals, see below) could not provide the necessary prediction errors to update one's beliefs about the current state of the body. It would also be unclear why other top-down predictions about the body (e.g. the prediction that one will not fall after moving one's left leg) were not used to update beliefs about the body.

4.2. Anosognosia for hemiplegia and the 'Internal Body'

A further set of important signals about the body arises from within the body's skin boundary. These include proprioceptive and more generally kinesthetic sensations about the position and dynamic properties of the body in space arising from the vestibular system and from muscles and tendons, as well as interoceptive sensations about the physiological conditions of all internal organs (Craig, 2009).

Deficits in prioprioception are not sufficient to cause AHP but they have been shown to be among the most common deficits in AHP patients (Vallar & Ronchi, 2006; Vocat et al., 2010). The vestibular system is also thought to be affected in AHP, given the fact that vestibular stimulation has been shown to temporarily reinstate awareness (Cappa, Sterzi, Vallar, & Bisiach, 1987; Ramachandran, 1995; Ronchi et al., 2013). Such deficits are potentially important when trying to understand motor awareness, as according to the free energy framework, predictive signals during action do not constitute forward signals on the basis of efference copies of motor commands but rather sensorimotor predictions (e.g. proprioceptive predictions) about the effects of movement (for detailed discussions, see Adams et al., 2013; Friston, 2010). This perspective thus implies that some patients may have reduced ability to generate novel predictions about their bodily and spatial effects of their potential left-sided movements (see also Heilman et al., 1998 for a similar proposal based on a previous computational model of motor control and awareness). Although this deficit may contribute to unawareness in some patients, previous studies have shown that at least some patients with AHP have intact ability to generate sensorimotor predictions (see above). Moreover, if patients with AHP are unable to generate such predictions, the fact that some patients insist that they have moved as desired, i.e. fulfilled kinesthetic predictions, is not easy to explain.

Importantly, even in patients whose proprioceptive and vestibular systems are intact, there is a degree of paresis due to damage to the motor system. In fact prototypical cases of motor unawareness are considered the ones who show complete paralysis of their left limbs. Thus, an important source of disruption may be the mere fact that patient *can no longer fulfill their proprioceptive and other related priors by active sampling of prediction errors* (i.e. transmitting such descending somatomotor predictions to the peripheral motor system and moving their affected limbs so as to generate reafference). AHP patients should be able to generate such somatomotor predictions in spared premotor and parietal cortex areas (Berti et al., 2005; Karnath, Baier, & Nägele, 2005). However, damage further down the hierarchy would mean that such predictions are not fulfilled by the motor system. Nevertheless, unlike the aforementioned effects of missing or weak prediction errors that are passed up the cortical hierarchy in order to modify perceptual inference, such disruptions in active inference at the spinal cord or subcortical level should not have an effect of motor awareness. The normal role of such somatomotor (mainly proprioceptive) reafference seems to be to modify descending predictions at spinothalamic and spino-cerebellothalamic circuits, thus allowing, fast, 'automatic' correction and control of movement. Indeed, this lack of active inference does not seem sufficient to cause AHP as the syndrome occurs in a minority of patients with stroke-induced hemiplegia.

It thus seems that while exteroceptive, proprioceptive and motor deficits may be important contributors to the symptomatology of some patients with AHP, they are unlikely to be its primary or central causes. By contrast, another facet of the internal body may have a more central role in AHP. Recent lesion mapping studies have highlighted that areas such as the insula, limbic structures and subcortical white matter connections may be selectively associated with AHP (Fotopoulou et al., 2010; Karnath et al., 2005; Moro et al., 2011; Vocat et al., 2010). Such areas and their connections are linked with interoception and motivation and are specifically implicated in bodily salience and interoceptive awareness (Craig, 2009; Critchley, Wiens, Rotshtein, Öhman, & Dolan, 2004). Thus, we propose that weak or imprecise (see also below) interoceptive and emotional signals about the current (physiological) state of the body, may lead to difficulties in affectively *personalising* new sensorimotor information about the affected body parts. This would be coupled with a persistent, necessary adherence to past expectations of how the affected body parts should *feel,* ultimately leading to the aberrant beliefs about any available contrary information about the body. In support of this hypothesis, a recent study (Romano, Gandola, Bottini, & Maravita, 2014) shown that right hemisphere patients who show somatoparaphrenic beliefs about their affected body parts, also show reduced physiological reactions to the threat of the same body parts, as measured by skin conductance responses.

Moreover, given the higher position of such priors in the neurocognitive hierarchy (see Friston, 2013), such faulty inference may also 'explain away' contrary exteroceptive signals during instances of multisensory integration. To use the words of one anosognosic patient who also denied the ownership of his paralysed limbs "But my eyes and my feelings don't agree, and I must believe my feelings. I know they [left arm and leg] look like mine, but I can feel they are not, and I can't believe my eyes." (C.W. Olsen, 1937, cited in Feinberg, 1997). The degree to which such interoceptive deficits are linked to delusions of ownership more frequently than delusions of motor awareness remains to be specified in future studies.

Furthermore, a related deficit in the processing of salience from the affected body parts needs to be emphasized. As aforementioned, activity in areas such as the insular and the limbic cortex is not only linked with interoception and emotion but more generally with interoceptive salience and motivation. In the free energy framework, these notions are linked to the concepts of 'precision' (mathematically inverse dispersion or variance, and hence the inverse of uncertainty) and its neurochemical equivalent, neuromodulation (Friston et al., 2012). Specifically, precision is linked mainly with the neuromodulation of synaptic gain that encodes the uncertainty of random fluctuations about predicted states. It follows that neuromodulators of synaptic gain (such as dopamine and acetylcholine), do not signal (reward or pleasure) prediction errors about sensory data but the context in which such data were encountered. In other words, such neuromodulators report the salience of sensorimotor representations encoded by the activity of the synapses they modulate. This is important, especially in hierarchical schemes, where precision controls the relative influence of bottom-up prediction errors and top-down predictions. In psychological terms, the processing of salience expectancy allows the organism to control the significance it attributes to the sensory data it uses to update its predictions or to explain away prediction errors.

The above considerations have added potency in AHP given the above critical lesion cites, as well the recently identified lesions in fronto-striatal circuits (Fotopoulou et al., 2010; Moro et al., 2011; Venneri & Shanks, 2004; Vocat et al., 2010). Such lesions may lead to a more general difficulty in optimizing the precision of prediction errors (Friston et al., 2012), affecting their salience and ultimately both short- and long-term learning (suboptimal synaptic gain and plasticity, Friston, 2010). Indeed, the functional role of the basal ganglia and particularly the striatum has been linked with prediction error-driven learning (O'Doherty et al., 2003) as well as the aberrant salience theories of psychosis (Gray, Feldon, Rawlins, Hemsley, & Smith, 1991; Kapur, 2003). In AHP such deficits can be linked with both specific instances of aberrant motor monitoring in functionally specialised systems (Berti et al., 2005), or more generally in global error monitoring (Davies et al., 2005; Venneri & Shanks, 2004; Vocat, Saj, & Vuilleumier, 2012), mental flexibility (Levine et al., 1991) and 'surprise detection' (Ramachandran, 1995) deficits.

Indeed, a recent study showed that AHP patients had the tendency to 'jump to conclusions' on the basis of limited and rather vague information and then to subsequently get stuck to their former "false" beliefs instead of modifying them based on novel, arguably more salient information (Vocat et al., 2012). Another study (Besharati, Forkel, et al., 2014) further found that anosognosia can be temporarily reduced by the induction of negative mood, presumably because negative emotions prime the organism for defensive action and increase the salience of sensorimotor signals (see Pereira et al., 2010; Gentsch, & Synofzik, 2014). These 'neuromodulatory' deficits may explain some of the delusional features of AHP that are harder to explain on the basis of deficits in sensorimotor signals per se, be those exteroceptive or interoceptive. For example, they provide some insight into how patients can remain in denial of their paralysis and/or apathetic towards the normally alarming sight of a paralysed left arm and its related consequences.

More generally, this explanation of anosognosia as an inability to update body awareness in a way that takes into account and 'personalises' new motor (agentive), interoceptive (emotional) or, just salient information about the affected body parts has the advantage of unifying the hypotheses put forward previously by modular (e.g. Berti et al., 2005; Karnath et al., 2005) and multi-factorial theories (e.g. Davies et al., 2005; Vuilleumier, 2004) on a single, unified and neurobiologically-plausible formulation. Moreover, this formulation integrates both bottom-up and top-down mechanisms of bodily perception, action and belief formation (see also Fotopoulou, 2012; Fotopoulou, 2014). Furthermore taken together, the above considerations on AHP in the light of the free energy framework highlight, not only how our awareness of our own body is based on habitual predictions of its state in the world, but also how this learned, virtual model of the body is depended on the integrity of the very body that allowed the model to be formed in the first place (see introduction). I now turn to a final aspect of body awareness formation that seems less intuitive than the rest and can hopefully be made more evident via the study of AHP.

4.3. Anosognosia for hemiplegia and the 'Impersonalised Body'

As explained above, the proposal regarding the inability to shape unconscious inferences about the body by prediction errors signaling information about the affected body parts as 'personal' and relevant to the self can explain a lot of anosognosic features and beliefs. However, a fundamental question remains. Patients seem unable to use other higher-order knowledge, including social feedback, to update their beliefs. This failure is not easy to explain on the basis of any sensorimotor deficits. Of course, their feelings that the paralysed body parts are of limited self-relevance partly explains why they would disregard social feedback for some time. As the aforementioned patient claimed, one has learned to trust one's feelings about the body over and above other sources of information. This is also consistent with the assumed hierarchy of the proposed model; brain areas assumed to subserve interoception and emotion are thought of as higher in the neurocognitive hierarchy than areas subserving exteroception (Friston, 2013). However, why are aberrant interoceptive inferences not updated by priors at even higher levels? For example, the model includes the possibility of other, already formed generative models about the self with predictions represented at higher levels (e.g. 'my family and doctors would not lie to me about serious health

issues') that could presumably be used to send predictions down the neurocognitive hierarchy and eventually influence the inferences based on faulty interoception and salience. Given that patients' beliefs about their motor abilities seem uninfluenced by such knowledge we infer that these processes are not taking place. This assumption is consistent with the aforementioned clinical observation that patients with AHP frequently refer to other people's opinions on their bodily state, without altering their self-awareness. For example, they say phrases like 'I know the doctors think I am paralysed, but I do not believe it', 'everyone says I cannot move, but I know I can'. If such third-person knowledge about the self is available why is it not affecting their own opinion about themselves?

The answer could be that this particular aspect of perception-cognition, particularly as applied to the affected body parts, is also damaged by the right hemisphere damage in question. In this section, I will try to describe the nature of this particular impairment as manifested in AHP and the nature of the presumed corresponding function in the undamaged brain. Borrowing insights from Merleau-Ponty (1945/1962) I will call this aspect of body representation, the 'impersonalised' body. Related concepts include the 'habit' body (Merleau-Ponty (1945/1962)), the off-line (vs. on-line) body representation (Carruthers, 2008; see also Tsakiris & Fotopoulou, 2008) and of course the various versions of the old and highly problematic distinction between body schema and body image. The proposed aspect of body representation is not proposed as fitting any of these terms exactly, nor being part of a simple contrast between the personal and the impersonal, or social body. As should be obvious in light of what I have written above, no simple dichotomy of body representations would be sufficient to account for the multiple ways in which the body is represented in the mind and our existence is embodied in brain functioning. However, a proper discussion of all these concepts and terms escape the scope of this article. Instead, I use the term 'impersonalised' body in order to emphasise with a single, heuristic term the 'objectified' aspect of this domain of bodily perception, as well as its personal and interpersonal origins.

Perceiving the world, including my own body, also entails the perception of the body's action possibilities in the same world. Indeed, my perception of the world is rarely confined to the characteristics of the input that reaches my few, sensory organs from my current, unique position and perspective in the world. Instead, my prior experiences of different sensations, possibilities and positions in the world define how I perceive the world in any given time and space. For example, my multisensory expectations and reaching movements adjust to the characteristics and affordances of a glass on a table by the mere sight of it from one particular egocentric perspective and prior to any current movements, or tactile feedback. Such expectations constitute the world as real in relation to my body, even if they remain implicit most of the time. These ideas, present in phenomenology, have been more recently explored in cognitive neuroscience and are accepted by scholars that subscribe to the ideas of 'multisensory integration' and 'enacted perception'. Moreover, such subpersonal processes are also compatible with the reading of AHP in the light of the free energy framework; the perception of the world entails (unconscious) inferences based on the agents prior embodied experiences with the same world. As Merleau-Ponty (1945/1962) first speculated, the affordances and multisensory characteristics of the world continue to appeal to the habitual body of the anosognosic patient, in the same hidden way that they appealed to it prior to the paralysis. Moreover, as I have outlined, the framework explicitly proposes that perception and action as operating in a continuum, being essentially governed by the same operating principles.

There is however an aspect of such perceptual and active inference that seems to have received less attention within the framework. The perception of the world, as a coherent canvas of multiple interrelated sensations, action possibilities and perceptual perspectives, entails the implicit possibility that any given object can simultaneously be perceived by different agents. When I look at the front of a chair, my awareness of the chair as one thing entails my tacit perception of its back. In this sense, I may be aware that the chair can be perceived by different positions, by anyone, or by any-body (Taipale, 2014). In more explicit ways, my perception of the world is developmentally shaped by learning opportunities afforded by solitary prior instances of active and perceptual inference, as well as by the active presence of other agents (see Krahe, Springer, Weinman, & Fotopoulou, 2013 for related ideas in the domain of pain perception). A glass on a table is the object that others and myself can manipulate at any given time, from any of our unique perspectives. This rich experience of others interacting with the same world as ourselves is presumably at the basis of our everyday, intuitive sense of 'veridical' and 'shared' perception. We perceive the world as containing unique, whole-in-themselves objects despite the fact that our actual perception of them from a subjective point of view will always be limited to the constraints of our body in time and space, e.g. the position of our eyes on the head. This human, adult ability to simultaneously perceive the world, from many silent, potential perspectives and the action possibilities they entail, seems to suggest not only that our perception relies on inference but also that such inferences about the world is deeply embedded in the social world. Indeed, other than classic phenomenological views on intersubjectivity (see Gallagher, 2008; Taipale, 2014; Zahavi, 2001 for recent considerations), such ideas have been mostly developed in certain stands of developmental psychology (e.g. Reddy, 2008; Rochat, 2009).

This perspective however entails a kind of tension when applied to the body. Signals in the above mentioned domains of the perceived body, including the exteroceptive and interoceptive domains, are ultimately integrated in egocentric coordinates. The 1st-person perspective (spatial and mental) remains fundamental for the perception of the body as mine, as under my volitional control and as separate from other objects in the world (e.g. Damasio, 1994). In other terms, although the body can be perceived (objectified) as a kind of socially perceived, impersonal, unique object, it is also always the subject of all experiences. As aforementioned, this tension has received many names in the history of mind and brain fields, and this paper cannot even begin to address the complexities behind such questions. However, attempting to understand this tension in the

context of AHP and the free energy framework, as outlined in this article, may generate some insights, particularly as our everyday conscious perception of the body does not include two, separated experiences of the body.

We mostly conceive of the body we see in the mirror as the one who feels itself to be standing in front of the mirror. It turns out that some patients with AHP do not share this experience. When looking at their paralysed body parts directly, they believe they are able body parts, and if they are somatoparaphrenic, they may believe they belong to someone else. However, when confronted with mirror images or video replays of their own body in the third-person perspective, the same patients describe the reflected body parts as being paralysed and as belonging to themselves, respectively (Fotopoulou et al., 2009, 2011; Jenkinson et al., 2013; Besharati, Kopelman, et al., 2014). These findings confirm the distinction between first and third-person perspectives on body perception and they further highlight the primacy of the first person perspective in conscious perception: it is the (delusional) content of the first person perspective that dominates their conscious awareness when mirrors are not made available to them. We can thus confirm Merleau-Ponty's (1945/1962, p. 82) intuitions:

In the case under consideration, the ambiguity of knowledge amounts to this: our body comprise as it were two layers: that of the habit body and that of the body in this moment. In the first appear manipulatory objects that have disappeared from the other and the problem how I can have the sensation of still possessing a limb I no longer have amounts to finding out how the habitual body can act as guarantee for the body at this moment. How can I perceive objects as manipulatable when I can no longer manipulate them? The manipulatable must have ceased to be what I am now manipulating, and become what one can manipulate; it must have ceased to be a thing manipulatable for me and become a thing manipulate in itself. Correspondingly, my body must be apprehended not only in an experience which is instantaneous, peculiar to itself and complete in itself, but also in some general aspect and in the light of an impersonal being.

These observations also reveal another intriguing aspect of this distinction. Although these patients seem able to perceive their body 'correctly' from a third-person perspective, they do not seem surprised by the difference between the content of the two perceptual instances. Even though they may not have 'seen' their own arm for a month or so, they do not scream, 'oh there it my arm' when we place a mirror in front of them. Nor do they blame us for suddenly giving them paralysis when we take the mirror away. And they are not even surprised by the fact that they themselves have given a different answer about the ownership and agency of the same body part, just seconds ago. It thus seems that apart from their deficits in updating their body representation in the first-person, they have also lost the ability to perceive their body as an object in the world that needs to have a unique, socially-shared existence. The fact that they can recognize third-person, images of the body as theirs does not seem sufficient for the proper 'objectification' of the body (i.e. the perception of the body as a thing in itself). The latter, 'impersonalised' sense of the body therefore seems to involve cognitive and perhaps emotional operations that extend the mere self-recognition in third-person perspectives and rather bizarrely seem to also depend on a kind of grounding in the subjective body, or at least a kind of flexible, abstract perception and integration of 1st and 3rd person perspectives on the body.

The cognitive and emotional integration between first and third person perspectives on the bodily self is thought to take place progressively in development but developmental psychologists, as well as phenomenological and psychoanalytic thinkers, seem to stress different aspects in these processes of self-objectification and awareness. In fact, the majority of studies and theories focus on how we come to understand or, infer other minds via their bodies, or how we come to regulate our own emotions. Far less attention is paid to the mentalisation (see definition above) of one's own body via the influence of other people. While this discussion extends the scope of the current paper, I note certain possibilities as regards AHP here. The loss of the 'impersonalised body' in AHP can be explained in at least two ways: (a) body awareness from a first person perspective, including the processing of both interoceptive and exteroceptive signals is higher in the neurocognitive hierarchy (Friston, 2013), thus prediction errors relating to the 'objectified' body are simply explained away by predictions about the subjectively felt body and its related salience (see also section on the 'Internal Body'); (b) the very faculty that allows individuals to engage in the act of flexible perspective-taking and integrate first and third person perspectives is impaired. The latter interpretation would be consistent with the observed damage in AHP patients in brain areas such as the temporoparietal junctions and the superior temporal sulcus (e.g. Besharati, Forkel, et al., 2014; Fotopoulou et al., 2010; Moro et al., 2011) and we have preliminary data showing that such lesions in patients with AHP are selectively associated with deficits in perspective taking and theory of mind abilities (Besharati et al., in preparation).

5. Conclusion

In this paper, I described the counterintuitive syndrome of anosognosia for hemiplegia, the striking, apparent unawareness of paralysis following right hemisphere stroke. I further put forward a large-scale framework from computational neuroscience, namely the free energy framework in order to account for the clinical variability of AHP and unite previous, seemingly divergent hypotheses about its pathogenesis. The framework proposes a view of human perception that relies on inferring the self and the world in both perception and action on the basis of prior expectations and ambiguous sensory signals. Contrary to intuition, our perception of the world is rarely confined to the characteristics of the input that reaches our sensory organs from our current, unique position and perspective in the world. Instead, our prior experiences of different possibilities and positions in the world define how we perceive the world in any given moment and position in space. In this sense, cognition can be thought of as imperfect, yet highly efficient (in a Bayes-optimal sense) strategy for self-organization in an ambiguous world. Anosognosia for hemiplegia, as a prototypical disorder of body unawareness, represents an exaggeration of such 'imperfect' body awareness system. I hope that the description of this neuropathology in the light of the free

energy framework served to highlight the deep interdependency of prior beliefs and sensory data; as the brain uses sensory data to update its virtual model of the world, lack or imprecision of sensory prediction errors may lead to aberrant inferences influenced disproportionally by outdated, premorbid predictions. Finally, I hope that this consideration of anosognosia stresses that our learned, virtual model of the body is depended on the nature and thus integrity of the very body that allowed the model to be formed in the first instance.

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