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Peculiarities of insight: Clinical implications of self-representations

> But my eyes and my feelings don't agree, and I must believe my feelings. I know [my left arm and leg] look like mine, but I can feel they are not, and I can't believe my eyes.
>
> - CW Olsen, 1937, as quoted by Feinberg (2001)

Patients who have suffered a stroke may as a result develop hemiplegia. This of course significantly impairs a patient's ability to perform various physical tasks. Yet, some hemiplegics appear to have little or no realization of their paralysis. This curious presentation was first recorded by Joseph Babinski in 1914. He described a woman who, following a stroke, lost function of her left arm. She was otherwise lucid and healthy, but seemed confused when offered treatment, replying, 'I am not paralysed, though,' (Babinski 1914, p 846). When asked to move her arm, the patient would either not respond, or reply, 'here, it's done', without having moved (Babinski 1914, p 846). Babinski coined the term *anosognosia* (meaning 'oblivious to illness') to describe this unawareness. Anosognosia for hemiplegia (AHP) has since been observed in a variety of manifestations. Patients with *asomatognosia*, for example, are unable to recognize the affected limb as their own. Babinski described such a case in which the patient would simply not respond to questions regarding the affected arm, as though the questions were directed at somebody else. This may even extend to a delusional misidentification known as *somatoparaphrenia*, in which patients attribute ownership of the limb to another person (Gerstmann 1942).

What could possibly be happening in the brains of these patients to so displace their self-representations? This ventures into abstract territory, but the translational implications of better understanding self-representations are in fact substantial, and may also extend to several other disorders in which anosognosia manifests, including schizophrenia and Alzheimer's disease. Body ownership as a foundation for a sense of self has become an important focus in cognitive neuroscience in recent years, and is a promising avenue for elucidating how self-representations are formed in the brain. AHP patients in particular offer a uniquely tangible window into the nature of self-awareness.

Several theories of AHP have been proposed over the years since Babinski's original description. The lack of physical and motor self-awareness in AHP has previously been attributed to hemispheric disconnects (Geschwind 1965), unilateral neglect (Vuilleumier 2004), and cognitive deficits in higher-order domains such as memory (Berti *et al.* 1996). While these may contribute to or exacerbate the manifestations of AHP, they can only be fragments of the story, as all of these deficits have shown double dissociations with AHP (Bisiach *et al*. 1986; Starkstein *et al*. 1992; Adair *et al.* 1995; Marcela *et al*. 2004; Berti *et al.* 2005). Using a different approach, Weinstein and Kahn (1955) had suggested that denial of paralysis as seen in AHP is a Freudian defence mechanism against a reality that could induce stress and depression. Asomatognosia, however, can affect individuals with otherwise functional limbs, wherein the requirement of any defence mechanism would be void, and so this also cannot be an exhaustive explanation. Furthermore, from an evolutionary standpoint, denial would be maladaptive when exaggerated to such lengths. Nevertheless, there is some evidence to suggest that this hypothesis may at least be an important starting point. Ramachandran (1996) ingeniously engineered a scenario in which paralysis would be less stressful by injecting the paralysed left arm of an AHP patient with a solution that she was told would induce *temporary* paralysis. The solution was in fact a placebo. She was then asked to move her left arm; she replied that she could not move it, as it was paralysed. The denial had temporarily

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abated in the absence of consequence. When her unaffected *right* arm was later injected with the placebo, her reaction was different. This time, the sensory information was inconsistent with her expectations (she was still able to move her arm), and the denial re-manifested: she brushed off this inconsistency as 'mind over matter'.

However, this 'motivational' evolutionary psychology approach to etiology is somewhat circular and on its own has little translational potential. Ramachandran (1996) therefore exploited the right-hemisphere neuropathology consistently seen in patients with AHP to extend the explanation to hemispheric specialization. He suggested that brain regions in the left hemisphere may be responsible for effecting defence mechanisms for minor, local discrepancies between expectations and reality, to maintain stability of behaviour and avoid aberrant assignment of salience, as seen in schizophrenia. If so, it is further possible that the 'devil's advocate' mechanism that constantly questions the status quo and *overrides* denial when it ceases to be adaptive may be seated in the right fronto-parietal regions. This includes the right sensory and motor cortices, and is the most common site of lesions in AHP. He tested this in another cleverly designed experiment, wherein he induced the illusion of momentary paralysis using a Nielsen box in the *functional* limbs of patients with AHP, by asking them to move the arm, but visually conveying by means of mirrors a stationary arm. In unaffected participants, this elicits a 'jolt' of shock or surprise. AHP patients, however, began to confabulate in this instance as well, making excuses for the lack of movement of their unparalysed arms. This suggests the deficit in anosognosia must lie in the central processing of movement (Ramachandran 1995).

The evidence emerging now supports one particular model of how movement is centrally processed. 'Bayesian inference' or 'predictive coding' is a concept derived from computer science and has become central to many cognitive theories of information processing in recent years. This theory suggests that the brain interacts with its environment by constructing probabilistic internal models based on accumulated experience, which allow it to make predictions about the outcomes of actions and situations (Helmholtz 1896; Friston 2012). These models are constantly re-evaluated as sensory input is received in order to improve their accuracy. In their famous paper, "Why can't you tickle yourself?", Blakemore, Wolpert and Frith (2000) outlined a 'forward model' of motor awareness centered in the cerebellum, in which the current state of the motor system is used to predict the next state. These predictions are then confirmed by their outcomes in a feedback loop. A prediction error (a discrepancy between the predicted outcome and the actual outcome) generally elicits surprise or confusion. For people with intact motor predictions, a self-generated command will have few discrepancies, and therefore cannot elicit surprise (hence we cannot tickle ourselves). When the command is externally generated (by another person, for example), there is a greater likelihood of prediction errors.

Earlier theories proposed that AHP is a result of absent motor predictions, such that the subsequent lack of movement does not constitute a discrepancy, so AHP patients do not realize that they have not moved (Heilman *et al.* 1998). More recent studies have found strong evidence to the contrary. Frith *et al.* (2000) proposed that AHP patients in fact have intact motor predictions (e.g. the position the limb should move to), but the neural system monitoring inconsistencies of the actual outcomes (e.g. where the limb actually ended up – in this case, it does not move) against those predictions must be damaged. Reminiscent of Ramachandran's right fronto-parietal 'devil's advocate', they conclude that the brain region that evaluates discrepancies between predictions and outcomes (or, in this case, internal 'feelings' and visual feedback, as described by the patient CW Olsen, above) is likely to be in the right perisylvian region that is usually affected in AHP patients. This was congruous with Fink *et al.*'s (1999) findings in healthy subjects, who were PET-scanned during predictive incongruity tasks and exhibited significant activation in right fronto-parietal regions. Berti *et al.* (2005) expanded on this by studying the anatomical distribution of lesions associated with denial in AHP patients, finding associations with motor programming areas (Brodmann's premotor areas 6 and 44, motor area 4 and the somatosensory cortex). Later studies of patients with AHP have supported the notion of dominance of motor intentions over visual feedback (Fotopoulou *et al.* 2008). Most recently, in an elegant study by Garbarini *et al.* (2012), AHP patients and unaffected controls were asked to perform a simple task: drawing circles with the

left hand and straight lines with the right. Due to bimanual interference, the normal subjects were unable to perfectly execute the task – the straight lines drawn with the right hand looked more circular. Even though their left arms were stationary, the AHP patients showed the *same* interference and circular pattern drawn by their right hands. This beautifully illustrates the intact motor predictions of patients with AHP.

All of these findings have contributed to an abstract model of motor unawareness in AHP as a product of deficits in brain areas monitoring discrepancies between predictions and outcomes, a faculty inevitably critical to insight. More importantly, however, this model lays the foundation for questions regarding treatment of AHP patients. If indeed motor predictions have dominance over visual feedback when responding to discrepant information, it would logically follow that allowing patients to see themselves from an 'offline' third person perspective, without the intention to move, should facilitate their awareness of visual information, and therefore alter their self-representations. Fotopoulou *et al.* (2008) found that this was indeed the case in a patient with severe AHP who underwent a radical and immediate recovery of awareness when shown a video replay of herself being asked to move her arm. Besharati *et al.* (2015) replicated these results with a further two AHP patients, coupling the video-replays with social support to ease the stress of accepting the paralysis. It would seem that although right hemisphere damage may affect physical self-representations, third-person self-representations in AHP are still intact, and could provide an avenue to restoring first-person awareness. Fotopoulou *et al*. (2011) went on to test whether this would also apply to the sense of body ownership by studying two somatoparaphrenic patients who consistently attributed ownership of their own arms to their husband and daughter, respectively. When shown a mirror, the patients' delusions were momentarily reversed: they were able to recognize the arm in question as their own. When the mirror was removed, however, the misidentification re-manifested, and the patients once again attributed ownership of their arms to their husband/daughter. Perhaps most interestingly, the patients exhibited no surprise at these moment-to-moment changes in representation. This does not quite constitute treatment for somatoparaphrenia, but is certainly very promising, particularly considering that previous attempts at treatment such as vestibular stimulation, transcutaneous nerve stimulation, and prism adaptation showed only temporary effects (Bisiach and Geminiani 1991; Beschin *et al*. 2012), and did not directly target the cause of the problem.

It would therefore seem that the most promising route to treatment lies in the intrinsic link between first-person and third-person perspectives, and restoring this connection. In order to do so, we would need to better understand how the two are related. The co-development of 'self' awareness and 'other' awareness in human children by means of second-person ('you and me') interactions (Fuchs 2013) may be the key to defining this relationship. Pertinently to the predictive coding theory, interactions with a primary caregiver allow an infant to construct an 'internal working model' of 'self' and 'other' and the systems of attachment between them (Mikulincer 1995). The *bodily* self in particular is a concept largely learnt in childhood for most individuals, and is constantly updated by interactions with other people (Cowie *et al.* 2013). In his 1948 lecture, 'Man seen from outside', the French philosopher Merleau-Ponty expresses this other-dependent development of self-representations aptly:

> We do not start out in life immersed in our own self-consciousness (or even in that of things) but rather from the experience of other people… An infant of a few months is already very good at differentiating between [emotional expressions] in others at a stage when he could not have learned the physical signs of these emotions by examining his own body… [As an adult] the contact I make with myselfis always mediated by a culture, or at least by a language that we have received from without and which guides us in our self-knowledge' (Merleau-Ponty 2004, p 86).

Fotopoulou (2012) suggests that the bodily self rests upon on two systems, interoceptive (the 'felt' physiological condition of the body; Craig 2003) and exteroceptive ('observed' information from the surrounding environment). Many irregularities of body ownership, from AHP to anorexia nervosa, are a product of discrepancies between these 'felt' and 'observed' selves (Feinberg 2001; Herbert and Pollatos 2012; Emanuelsen *et al.* 2015) Accessing the interoceptive system by means of interactions with others may therefore be the key to restoring a consistent sense of self. One particular such means that has emerged is 'affective touch': slow, gentle stimulation (experimentally standardized as 3cm/s

stroking movements) of C-Tactile (CT) afferents in hairy skin, which stimulates neural activation in the posterior insula and triggers the release of the 'social bonding' neuropeptide oxytocin, resulting in feelings of pleasantness and attachment (Krahé *et al.* 2015). Affective touch is a significant component of caregiver–child interactions and, as attachment styles are largely developed in the early years of childhood, this oxytocin-based stimulation may be crucial to the socialization of the child in later life (Gromov 2013). To put this in terms of predictive coding theory, Brown and Brüne (2012) posited that interpreting social stimuli (understanding the minds of others and making predictions about their intentions) and behaving appropriately in response is imperative to existing as a social organism. Human social interactions in particular are so exceedingly complex that every social situation gives rise to several levels of uncertainty. Predictive coding, they suggest, is therefore fundamental to navigating these situations. However, the extent of complexity in human interactions is such that, in each instance, there may be conflicting and converging effects from several internal models. It is conceivable from this model that children with inconsistent information from their primary caregivers develop internal working models of social interactions with more conflicting components than children with reliable caregivers. This may as a result make inconsistently parented children more prone to social prediction errors, eliciting maladaptive or insecure attachment behaviours.

Crucianelli *et al.* (2013) tested the applicability of affective touch to enhancement of body ownership using the rubber hand task, in which a rubber hand model is placed next to participants' own hand. Both are stroked synchronously such that subjects often begin to visually re-encode their internal models and feel internally that the rubber hand is their own (Botvinick and Cohen 1998). Most describe this as a bizarre sensation, but the tendency to submit to this illusion varies between individuals. Crucianelli *et al.* (2013) found that participants were indeed more willing to submit to the rubber hand illusion when they were stroked at the optimum velocity for stimulating CT afferents. A further clinical question that emerges from this finding is whether a similar effect may be observed between asomatognosic patients and their limbs. This has not been experimentally studied, but anecdotally seems plausible. For example, Fotopoulou (2014) described a patient who would often be seen stroking her disowned arm. The patient explained,

> I woke up and called this arm 'a beast.' It was not my arm, it was some foreign fellow. But then you touched it and I caress it and I decided to love it again (Fotopoulou 2014).

Perhaps most centrally, these experiments with affective touch have also revealed the key role of oxytocin in the interoceptive system and development of 'self' and 'other' concepts. Tying several of these avenues together, Quattrocki and Friston (2014) proposed a theory of predictive coding with oxytocin as a mediator. Predictive coding would neurologically depend upon both short-term neuroplasticity required to navigate perceptual uncertainty, as well as more enduring synaptic changes that reflect the development of internal working models and ultimately self-representations. They posit that oxytocin may enable this synaptic flexibility, although there is little known as yet about the specific mechanisms by which it would do so. They suggest, however, that the property of oxytocin to induce synchronous neuronal activity may be highly relevant, as the flexible coherence of activity between different neural networks is thought to underlie cognitive flexibility as a whole (Fries 2005). In addition to AHP, there are a great many common disorders in which anosognosia manifests, such as Alzheimer's disease and schizophrenia. There are also a great many common disorders that can be modelled upon the predictive coding framework, including autism. Quattrocki and Friston (2014) propose that the social, sensory and motor impairments in children with autism may result from an abnormal oxytocin system in infancy, which compromises the child's ability to make accurate predictions about the salience of interoceptive signals (for example, those elicited by social cues such as eye contact). Indeed, studies have shown that administration of oxytocin can improve many factors of social interaction, including eye contact, in people with autism (Hollander *et al.* 2007; Auyeung *et al.* 2015). Furthermore, in light of the recent influx of studies into mindfulness and cognitive behavioural therapy expounding the psychological benefits of well-developed insight even in healthy individuals, it is clear that the implications of identifying neurobiological mechanisms underlying self-representations in the brain are vast. There is much

ground to be covered, but several clear trajectories for study are emerging, with predictive coding as a framework and oxytocin as a highly promising starting point.

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