The role of reality monitoring in anosognosia for hemiplegia

Paul M. Jenkinson[a,*], Nicola M. J. Edelstyn[b,c], Justine L. Drakeford[d], Christine Roffe[c] and Simon J. Ellis[e]

[a]School of Psychology, University of Hertfordshire, Hatfield, UK
[b]School of Psychology, Keele University, Keele, UK
[c]Research Institute for Life Course Studies, Keele University, Keele, UK
[d]School of Human and Health Sciences, University of Huddersfield, Huddersfield, UK
[e]Department of Neurology, University Hospital of North Staffordshire, Stoke-On-Trent, UK

1. Introduction

Anosognosia for hemiplegia (AHP) refers to a lack of awareness regarding paralysis after stroke. Despite attracting clinical interest for decades, empirical research into AHP has been relatively scarce, and there remains no universally accepted explanation [5]. This is partially due to difficulty characterising the disorder. The term has been applied to both partial and complete lack of awareness, with partial unawareness presenting as a failure to recognise, appreciate the severity, or acknowledge the consequences of paralysis, and more complete cases involving a failure to admit the presence of a paralysis even after its demonstration [8]. The fact that some patients verbally deny their problems, but show behaviours consistent with their paralysis (e.g. executing a bi-manual tasks using a unimanual strategy), while others verbally accept their paralysis but behave in a manner inconsistent with this acceptance (e.g. attempting to walk), suggests that verbal and behavioural awareness are independent [4]. The observation of diverse lesion sites, emotional, perceptual, and cognitive impairments in anosognosia has also resulted in unawareness being considered a multifaceted or multicomponent disorder involving several subtypes [4, 8,10]. As such, different forms of anosognosia may reflect the combination of various deficits, the exact components of which are not currently known [10].

Recent accounts of AHP have employed a model of the motor system, which proposes that awareness involves a comparison of predicted and actual sensory information (Fig. 1). It is suggested that AHP patients fail to register discrepancy between internal sensory predictions and external sensory information [1, 3]. This results in an erroneous feeling of having performed intended movements using the paralysed limb. Recent experiments in AHP support this idea; however, a purely motor account cannot explain several aspects of AHP, such as its delusional character (e.g. resilience to counterargument) and associated affective disturbance [5]. It is likely that other neurocognitive disturbances also contribute to AHP. On the basis of research in other delusional patients [2], and a speculated reality monitoring impairment in an AHP case-report [9], we hypothesised that an inability to discriminate between internally- and externally-generated information (i.e. reality monitor) would contribute to AHP.

We conducted two experiments to examine this proposal [6]. Experiment 1 employed a classic reality monitoring paradigm [7], to test the ability to discriminate between seen (perceived) and imagined drawings of objects in AHP patients ($n = 10$), hemiplegic con-

*Corresponding author: Dr. Paul Jenkinson, School of Psychology, University of Hertfordshire, Hatfield, AL10 9AB, UK. E-mail: dr.paul.jenkinson@gmail.com.
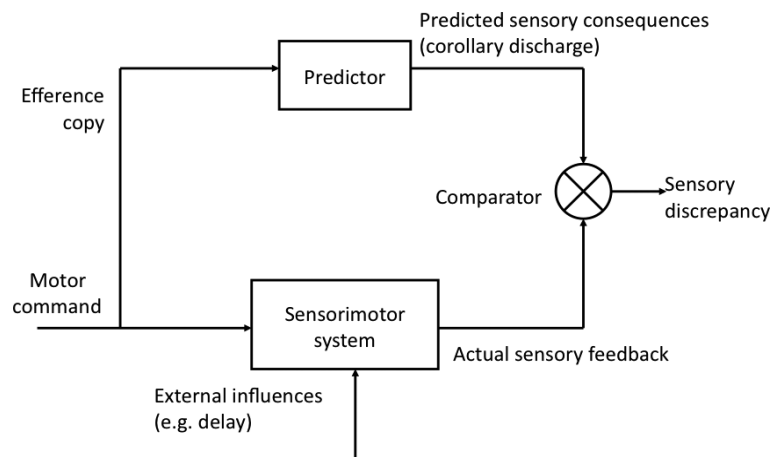
Fig. 1. A simple model of the motor system adapted from Frith et al. [3]. Actual and predicted sensory information are compared to generate a sensory discrepancy which signals movement error.

trol patients without AHP (nonAHP, $n = 7$) and age-matched healthy controls (HC, $n = 20$). During a study phase, subjects were presented with a word (e.g. PEN) followed by either: (i) a picture representing the object (i.e. drawing of a pen), or (ii) an empty circle into which they projected a mental picture of the previous word (i.e. imagine a drawing of a pen). In a test phase, subjects saw previously studied (target) and unstudied (new) words, and had to decide if each word had been studied previously. Following this, words identified as previously studied were assessed in terms of the source of the original image (i.e. "Did you previously *see* or *imagine* a drawing of a [PEN]?"). AHP patients were significantly impaired at this task relative to HCs (omnibus Kruskal-Wallis test $H(2) = 21.23, p < 0.001$) and nonAHP patients (post-hoc Mann-Whitney $U = 6, p = 0.006$), indicating a deficit in discriminating the source of images as real/imagined.

A second experiment explored if this reality monitoring deficit occurs in the motor domain. Adapting the procedure of Experiment 1, AHP ($n = 3$), non-AHP ($n = 6$), and HC ($n = 20$) subjects were presented orally with action phrases (e.g. point to the door), which they either had to execute themselves (perform item), imagine executing (imagine item), or observe the experimenter executing (observe item). The ability to discriminate studied/unstudied phrases, and make source judgements was then assessed using the procedure of Experiment 1 (e.g. "Did you previously [perform/imagine/observe] pointing to the door?"). Results again indicated impaired reality monitoring in patients with AHP compared with HCs ($H(2) = 11.54, p = 0.001$) and nonAHP patients ($U = 1, p = 0.048$). However, the ability to reality monitor movements was also

impaired in nonAHP patients relative to HCs ($U = 24, p = 0.026$). Performance on the task showed a steady decline from HC levels, to mild impairment in nonAHP patients, and greatest impairment in AHP.

Findings of these two experiments suggest a combination of reality monitoring impairments in the pathogenesis of AHP. Experiment 1 showed that the ability to discriminate between real and imagined drawings was impaired in AHP patients only. Experiment 2 showed that the ability to monitor actions is deficient in both AHP and nonAHP patients, but is more impaired in patients with AHP. As such, impaired reality monitoring of movement might be a general consequence of damage to the motor system. From our results it is not possible to identify whether the processes responsible for greater impairment of action reality monitoring in AHP are the same as those underlying the deficit observed in nonAHP patients.

We speculate that this combination of reality monitoring deficits prevents AHP patients from checking the veracity of knowledge about the motor system and their current state generally. This is consistent with the ABC model [10], in which awareness relies on an ability to Check available information, in order to change one's Beliefs, and/or act upon signals of uncertainty arising from subjective Appreciation (experience) of a specific function (e.g. moving). This explanation readily accommodates recent motor explanations of AHP [1,3]: the erroneous belief that one is able to move may arise from a defective appreciation of paralysis, caused by impaired sensory feedback, and/or a failure to register discrepancies between motor intentions and sensory information. This explanation suggests that AHP can result from different deficits which combine to pro-

duce the same clinical endpoint. As such, it is able to account for the multifaceted nature of AHP; however, further research is needed to identify the exact factors which produce AHP.

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