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Reality monitoring in anosognosia for hemiplegia

Paul M. Jenkinson and Nicola M. J. Edelstyn

School of Psychology & Research Institute for Life Course Studies, University of Keele, UK.

Justine L. Drakeford

School of Human and Health Sciences, University of Huddersfield, UK.

Simon J. Ellis

Department of Neurology, University Hospital of North Staffordshire, UK.

Correspondence concerning this article should be addressed to: P. M. Jenkinson, School of Psychology, Keele University, Staffordshire, ST5 5BG. Tel: 01782 733317. Fax: 01782 583387. Email: p.m.jenkinson@psy.keele.ac.uk.

Keywords: anosognosia for hemiplegia, reality monitoring, forward model, source memory.

Abstract

Anosognosia for hemiplegia (AHP) is a lack of awareness about paralysis following stroke. Recent explanations use a 'forward model' of movement to suggest that AHP patients fail to register discrepancies between internally- and externally-generated sensory information. We predicted that this failure would impair the ability to recall from memory whether information is internally- or externally-generated (i.e., reality monitor). Two experiments examined this prediction. Experiment 1 demonstrated that AHP patients exhibit a reality monitoring deficit for non-motor information (i.e., perceived vs. imagined drawings), while hemiplegic controls without anosognosia (nonAHP) perform like age-matched healthy volunteers (HVs). Experiment 2 explored if this deficit occurs when AHP patients discriminate performed, imagined, or observed movement. Results showed impaired reality monitoring for movements in AHP and nonAHP patients relative to HVs. Findings suggest that reality monitoring processes not directly related to movement, together with a failure to reality monitor movements, contribute to the pathogenesis of AHP.

Reality monitoring in anosognosia for hemiplegia

The seemingly effortless way that awareness of moving (or not moving) normally occurs might understandably lead to the conclusion that our self-awareness of action is a simple, unambiguous process. However, patients with anosognosia for hemiplegia (AHP) challenge this conclusion, because AHP patients are not aware of being unable to move. More precisely, AHP usually refers to a lack of awareness regarding motor impairment in patients suffering a right hemisphere stroke (Ellis & Small, 1993; 1997). In practice, a variety of clinical presentations are considered characteristic of AHP. Some AHP patients fail to recognise, appreciate the severity, or acknowledge the consequences of paralysis (Orfei et al., 2007). Other patients deny outright any motor impairment, while some acknowledge the presence of a motor deficit, but explain it away (Bisiach & Geminiani, 1991). In some cases AHP co-occurs with unilateral neglect (i.e., a failure to respond to stimuli on the contralesional side), while in others neglect is absent (Berti et al., 2005; Jehkonen, Laihosalo, & Kettunen, 2006). Furthermore, AHP can occur independently at verbal and non-verbal (i.e., behavioural) levels (Jehkonen et al., 2006). That is, some AHP patients refuse to acknowledge their paralysis, but are usually content to remain in bed, whereas other AHP patients may verbally acknowledge their paralysis, but attempt to get out of bed, stand, walk or perform other physical tasks that are clearly impossible (Bisiach & Geminiani, 1991). When asked to make self-evaluations, these patients are often unaware of their inability to execute bilateral tasks requiring use of the hemiplegic limb(s) (e.g., clap hands) (Berti, Làdavas, & Della Corte, 1996; Berti, Làdavas, Stracciari, Giannarelli, & Ossola, 1998; Marcel, Tegnér, & Nimmo-Smith, 2004; Nimmo-Smith, Marcel, & Tegnér, 2005).

Despite several decades of AHP research, we are still without an adequate explanation, capable of accounting for the diverse clinical, emotional, cognitive and neuroanatomical presentation of AHP (see Adair et al., 1997; Berti et al., 1996; Gold, Adair, Jacobs, & Heilman, 1994; Hildebrandt & Zieger, 1995; Small & Ellis, 1996). The

heterogeneous presentation of AHP is one factor that has impeded understanding of the disorder. A lack of consensus on how best to characterise and assess AHP means it is difficult to identify patterns in results across studies. Given the dissociation between verbal and behavioural awareness, AHP should be considered present if a patient displays either type of unawareness (Marcel et al., 2004; Nimmo-Smith et al., 2005). Berti et al. (1996) have developed a rigorous instrument for assessing AHP, which encompasses measures of both verbal awareness and awareness of the behavioural consequences of illness. Employing this type of robust diagnostic method allows more consistent and thorough characterisation of AHP, which can facilitate comparisons across studies and the development of a better understanding of the disorder.

Another major weakness of most AHP studies is a failure to frame their explanations within a robust theoretical model, instead using the disorder itself as a starting point for investigation and constructing an explanation. In contrast, a recent cognitive neuropsychological account of AHP provides a theory-driven, experimentally testable explanation of the disorder (Berti & Pia, 2006). Berti and Pia utilise a 'forward' model (Fig. 1) of normal motor control and awareness (Wolpert, 1997; Wolpert, Ghahramani, & Jordan, 1995), the utility of which has been established by numerous studies in normal, healthy individuals (Blakemore, 2003; Blakemore, Frith, & Wolpert, 1999; Blakemore, Frith, & Wolpert, 2001; Blakemore, Goodbody, & Wolpert, 1998; Blakemore, Rees, & Frith, 1998a; Blakemore, Wolpert, & Frith, 1998b) patients with schizophrenia (Blakemore, Smith, Steel, Johnstone, & Frith, 2000; Frith, 2005; Frith, Blakemore, & Wolpert, 2000) and recent studies of AHP (Fotopoulou et al., in press; Jenkinson, Edelstyn, & Ellis, in press), and anosognosia for dyskinesias (i.e., involuntary movements) in Parkinson's disease (Jenkinson, Edelstyn, Stephens, & Ellis, submitted).

The forward model is so called because it proposes that the causal (or forward) relationship between intended/planned actions and their sensory consequences are predicted

using an efference copy of motor commands. These predicted actions are believed to form the basis of motor awareness (Blakemore & Frith, 2003). Supporting evidence comes from studies showing remarkably limited knowledge of actual sensory feedback in healthy individuals (Fourneret & Jeannerod, 1998; Slachevsky et al., 2003), and a key role of sensory predictions in several aspects of normal motor awareness, such as correctly attributing actions to the self (i.e., internally produced) or an external source (Blakemore et al., 1999; Blakemore et al., 1998a; Blakemore et al., 1998b). A comparator monitors the congruence between these sensory predictions and the actual consequences of the movement arising from sensory feedback. When predicted and actual movement match no discrepancy signal is generated by the comparator; however, a failure to execute movement as intended results in the comparator signalling a mismatch, which comes into consciousness and produces subjective awareness of an error in performing the action. This is especially noticeable in situations where there is a mismatch between motor intentions, proprioception and/or visual feedback (Fink et al., 1999).

Berti and Pia (2006) propose that pathological awareness in AHP might occur if the ability to predict the expected sensory consequences of movement were preserved, but patients fail to detect when these predictions are not congruent with actual sensory feedback. Under these circumstances motor awareness in AHP becomes based entirely on sensory predictions, which erroneously indicate successful execution of the intended movement. This explanation predicts that patients with AHP retain the ability to form predictions about the consequences of movements, while the ability to monitor discrepancies is defective. In contrast, hemiplegic patients without anosognosia (i.e., nonAHP) possess preserved awareness of their motor impairment, because they are able to detect when the predicted and actual sensory consequences of their movement do not match.

The proposed failure to distinguish between internally-predicted and externally-perceived movement in AHP suggests that patients may have a deficit in *reality testing* ongoing experience (a.k.a. *reality discrimination*, i.e., the process by which information of an

internal versus external origin is discriminated, Johnson, 1991). A failure to monitor ongoing experience might also impair the ability to later recall the origin of information from memory, or *reality monitor* (i.e., the ability to distinguish between *memories* of an internal versus external origin, Johnson, Hashtroudi, & Lindsay, 1993). This link between reality testing and reality monitoring has been demonstrated in healthy volunteers (Rankin & O'Carroll, 1995). Reality monitoring operates on the basis of qualitative characteristics of information (e.g., the amount of perceptual detail supporting memories) in combination with judgement processes that use this information as evidence as to whether something was internally-generated or externally-perceived (Dalla Barba, Nedjam, & Dubois, 1999). AHP patients might adopt an abnormally lax criteria for the evaluation of mental experiences as internally- versus externally-generated, but to our knowledge, no study to date has directly examined reality monitoring in patients with AHP.

Venneri and Shanks (2004) speculated that reality monitoring impairments might be responsible for AHP in their patient EN: an 85-years-old woman who developed AHP following a right hemisphere stroke involving the parietotemporal cortex and frontal regions. This speculation draws on existing evidence indicating that frontal impairments are accompanied by reality monitoring failures, which facilitate the development of abnormal beliefs/delusions (Johnson et al., 1993; Shanks & Venneri, 2002; Venneri, Shanks, Staff, & Della Sala, 2000). Venneri and Shanks base their explanation on this existing evidence, together with findings of structural damage to the frontal lobe in EN, and neuropsychological impairments indicative of frontal lobe pathology appearing in the context of an otherwise unremarkable neuropsychological profile (i.e., EN exhibited poor verbal fluency, and behavioural changes associated with frontal lobe dysfunction, e.g., discussing sexual matters with her son in an inappropriate and disinhibited way). The authors proposed that impaired reality monitoring might form a barrier to natural awareness of hemiplegia, because of a breakdown in the ability to check what is ‘real’ and assess the veracity of mental contents

(Venneri & Shanks, 2004). Unfortunately, the authors did not assess EN's ability to reality monitor, in order to provide direct support for this proposal. Furthermore, although Venneri and Shanks suggest that AHP stems from a reality monitoring deficit, they do not specify that this impairment relates specifically to monitoring movements, as implied by Berti and Pia (2006).

The exact nature of any reality monitoring impairment in AHP is further complicated by experiments in patients with other conditions characterised by abnormal motor awareness (e.g., schizophrenia). An association between delusional beliefs about movement (i.e., delusions of control), and confusion between perceived and imagined images (i.e., reality monitoring), has been repeatedly observed (Anselmetti et al., 2007). Some of the most convincing evidence for this is provided by Brébion and colleagues (Brébion et al., 2000; Brébion, Gorman, Amador, Malaspina, & Sharif, 2002; Brébion, Smith, Gorman, & Amador, 1997), who consistently report a failure to reality monitor in patients with delusions of control in schizophrenia. For example, delusional patients perform worse than healthy controls on a task that requires discrimination of items that are either externally-generated (presented verbally or as a picture by the experimenter) or self-generated (Brébion et al., 2000). This suggests that reality monitoring failures not directly related to movement monitoring might also contribute to abnormal motor awareness.

The cognitive mechanisms leading to impaired reality monitoring have been suggested by Johnson and colleagues. According to Johnson (Johnson, 1997; Johnson et al., 1993) internally- and externally-generated memories tend to have different phenomenological attributes (e.g., internally generated memories contain more information about cognitive operations, whereas externally generated memories contain more perceptual, contextual and semantic information), and most reality monitoring judgements are made by a heuristic process that compares these attributes. Accordingly, reality monitoring errors are likely to occur when the phenomenological characteristics of memories from an internal and external

origin overlap. Several factors have been proposed as leading to such an overlap in patients with schizophrenia: attentional deficits may cause reduced perceptual richness of sensory information, or social isolation can lead patients to engage in frequent imagination, which consequently gains prominence over real-world events (Keefe, 1998). Similar mechanisms might lead to reality monitoring deficits in AHP. For example, patients with AHP frequently exhibit problems with orientation, attention and general arousal/alertness (Pedersen, Jørgensen, Nakayama, Raaschou, & Olsen, 1996; Starkstein, Fedoroff, Price, Leiguarda, & Robinson, 1992), and may experience depression and isolation as a result of their problems (Robinson, 2006). These deficits may reduce the perceptual richness of sensory experience or create an imbalance in the frequency of perceived and imagined events, thereby increasing the likelihood of misattributing internal and external memories about movements.

In summary, despite several previous attempts to explain AHP, an adequate explanation of the disorder has failed to emerge. A recent cognitive neuropsychological account of AHP (Berti & Pia, 2006) offers an explanation that is theory-driven and empirically testable. This account suggests that AHP stems from a failure to discriminate between internally-predicted and actual movement. A recent AHP case report (Venneri & Shanks, 2004), and reality monitoring experiments in patients with schizophrenia (Anselmetti et al., 2007; Brébion et al., 2000; Brébion et al., 2002; Brébion et al., 1997) also suggest that impaired reality monitoring may contribute to the psychopathology of AHP. However, it is uncertain whether this impairment involves reality monitoring processes not directly related to movement (as implied by Venneri & Shanks, 2004; Brébion et al., 2000; Brébion et al., 2002; Brébion et al., 1997; Anselmetti et al., 2007), or is purely motor (as implied by Berti & Pia, 2006). The aim of this study was to provide the first direct examination of reality monitoring in patients with AHP. We conducted two experiments to explore the contribution of reality monitoring impairments to AHP. Experiment 1 was designed to investigate whether AHP patients have a reality monitoring deficit for non-motor information, and the purpose of

Experiment 2 was to specifically test whether AHP patients have a deficit in reality monitoring movements.

EXPERIMENT 1: NON-MOTOR REALITY MONITORING

Experiment 1 tested the hypothesis that, like abnormal motor awareness in schizophrenia, AHP might involve a reality monitoring failure for information not directly related to movement. The ability to discriminate between memories of imagined and perceived images was tested in hemiplegic stroke patients with AHP, hemiplegic control patients without AHP (nonAHP), and age-matched healthy volunteers (HVs). It was predicted that the AHP group would be defective at reality monitoring relative to the nonAHP and HV groups. In contrast, both control groups should perform at comparable levels.

Method

Participants

Seventeen patients with a dense left hemiplegia (8 male, 9 female, mean age = 68.41, S.D. = 10.13) participated in the study. Patients were recruited from consecutive admissions to acute stroke wards at the University Hospital of North Staffordshire, and were selected on the basis of routine clinical and brain imaging (i.e., CT or MRI) evidence of a right hemisphere stroke. Lesions were typical of those reported in the literature (see Pia, Neppi-Modona, Ricci, & Berti, 2004), comprising various combinations of damage to the right frontal, parietal, temporal and occipital lobe, frontal deep white matter, parietal deep white matter, basal ganglia, thalamus, and minor ischeamic changes in the left hemisphere. Clinical scans were not of sufficient detail or control to warrant volumetric analysis or lesion mapping. Muscle power in the left upper and lower limbs was measured using the Medical Research Council (MRC) Scale (Guarantors of Brain, 1986), which grades power on an ordinal scale from 0 (*no contraction*) to 5 (*normal power*). The extent of motor impairment varied from complete flaccidity (MRC score 0) to slight movements (MRC score 1-2); however, none of the participants were able to execute controlled movements with the affected limbs. Patient

performance was compared with 20 age-matched HVs (9 male, 11 female; mean age = 68.20, S.D. = 5.44). Exclusion criteria comprised existing neurological or psychiatric illness (except stroke in the patient groups), concurrent left hemisphere damage other than minor ischaemic changes (patients only), learning disability, or history of drug or alcohol dependency. Participation was dependent on compliance with the testing schedule, so those patients with severe cognitive impairment (i.e., score of <18 on the Mini Mental State Examination, MMSE; Folstein, Folstein, & McHugh, 1975) or a reduced consciousness level were also excluded. Patients were screened for unilateral visuospatial neglect using the star cancellation task (Halligan, Marshall, & Wade, 1989), and personal neglect using the 'comb/razor test' (McIntosh, Brodie, Beschin, & Robertson, 2000). An estimate of pre-morbid intelligence was also obtained (National Adult Reading Test, NART; Nelson & Willison, 1991). All participants were right hand dominant, native English speakers, with normal or corrected-to-normal vision. Local NHS research ethics committee approval was granted for the study and all participants gave fully informed, written consent.

Assessment of Anosognosia for Hemiplegia

Assessment of AHP followed the method of Berti et al., (1996), which includes a structured interview to measure verbal awareness, and self-evaluations of the potential capacity to perform actions. Patients were classified as anosognosic if they demonstrated unawareness of their motor impairment on either measure.

Verbal awareness of upper limb motor impairment.

Patients were first asked to answer a few preliminary questions (Berti et al., 1996, p. 429), about their present condition: "Where are we? Why are you in the hospital/current location? How is your left arm? Can you move it?" If the patient answered "no" to the last question then he/she was asked "Why can you not move your left arm?" A second set of questions was asked if the patient verbally denied left upper limb motor impairment: "Please touch my hand with your left hand" (the experimenter put his hand in the patient's right visual

field). The patient was then asked “Have you done it?” If the patient answered “no”, then he/she was asked “Why have you not done it?” If the patient answered “yes”, then he/she was asked “Are you sure? It is very strange because I have not seen your hand touch my hand.”

Responses were documented verbatim and later scored for anosognosic content by PMJ and SJE independently, according to the following criteria: 0 = the patient answered correctly to the first group of questions (normal), 1 = the patient acknowledged being in the hospital and/or being affected by a stroke, but denied his or her upper limb impairment; however, the patient acknowledged that the left arm did not reach the examiner’s hand (mild anosognosia), 2 = the patient claimed that he/she had reached the examiner’s hand (severe anosognosia).

Verbal awareness of lower limb motor impairment.

Patients were asked the following questions: “How is your left leg? Can you move it? Can you walk without any problem?”, and responses scored according to the following criteria: 0 = the patient either spontaneously reported the motor impairment of the lower limb when first asked about the reasons for his/her being in the hospital (see above) or acknowledged the paralysis when specifically questioned about the left leg (normal), 1 = the patient answered “well/fine” to the first question, but acknowledged the impossibility of walking (mild anosognosia), 2 = the patient claimed that he/she was able to walk (severe anosognosia).

Self-evaluations of potential ability to perform actions.

Patients were asked to rate their potential to perform several actions requiring use of the upper or lower limb. Five monomanual actions involving the left (impaired) or right (intact) upper limb (i.e, drink from a glass, open a door, eat with a fork, lift a small object) and ten bimanual actions (i.e., clap hands, wash hands, wash face, put on gloves, open a jar, open a bottle, deal cards, tie a knot, light a cigarette with a match, put on socks) were rated on a scale from nought (*perform very badly*) to ten (*perform very well*). Five locomotor actions

involving the lower limbs (i.e., walk, jump, climb stairs, drive, ride a bicycle) were also rated on the same scale. Mean scores were calculated for left and right monomanual upper limb actions, bimanual upper limb actions, and lower limb actions separately. Patients' ratings for monomanual actions involving the right (intact) upper limb were used to check compliance and understanding of the scaling system, since all such tasks were performable and should result in a high mean score. For the remaining three measures, a mean score of between nought and five was considered normal (i.e., not anosognosic), and a score of six or more was considered evidence of anosognosia (Berti et al., 1996).

Reality monitoring task

Stimuli and apparatus.

One hundred and twenty black and white line drawings of common objects (i.e., concrete nouns) were selected from the standardised illustrations produced by Snodgrass and Vanderwart (1980). Each drawing had a corresponding written concrete noun. For each participant, 40 of the written nouns were randomly chosen from the pool as presentation stimuli and 20 nouns were used as new items during the test phase. Stimuli were presented on a laptop computer with a 14 in. display.

Procedure.

The task was organised into two phases: acquisition and test (Fig. 2), both of which were completed in a single session. Participants sat in a comfortable chair approximately 50cm from the computer. During the acquisition phase, participants were instructed to read aloud a written noun (e.g., pen) that appeared in the centre of the screen for 3 s. Following this, a circle (11cm in diameter) appeared for 3 s, which either contained a corresponding drawing (i.e., perceived items, n=20), or was blank (i.e., imagined items, n=20). For imagined items, participants were instructed to visualise the blank circle containing a line drawing corresponding to the presented written noun. Participants were instructed to rate the quality of each image (perceived or imagined) as *good*, *fair* or *poor*, in order to facilitate engagement

and concentration. Items were presented in random order. Participants were not instructed to memorise the word items, or warned that they would be required to recall these at a later time. A practice, comprising three items, was completed by all participants prior to the actual task. During this practice the laptop display was repositioned (where necessary) to ensure that stimuli were clearly visible to the participant, so that performance would not be influenced by the presence of unilateral neglect. Visibility of the stimuli was established by participants correctly reading aloud the practice words.

The test phase immediately followed acquisition. Participants were presented with previously studied (termed *target*) and unstudied (termed *new*) items in random order, and asked to identity whether or not they had been presented with the item during acquisition. If the participant thought the item was a target, they were required to recall its source: i.e., whether a drawing of the item had been displayed (*perceived*) or they had mentally generated a drawing of the item (*imagined)*. A *familiar* response was also allowed in instances when the item was identified as a target, but participants were unable to make a source attribution. Participants responded orally and their choices entered into the computer by the experimenter. No time restriction was imposed, so items remained on the screen until a response was entered. Participants completed a practice, comprising three items, prior to the actual test phase.

Data analysis.

Two types of performance data were considered: firstly, a studied-unstudied judgement which provided a measure of recognition memory *discrimination accuracy* (i.e., the ability to discriminate between target and new items, irrespective of source); and secondly, reality monitoring (i.e., identification of internally generated [perceived] and externally generated [imagined] items). Studied-unstudied recognition memory judgements were assessed using the Two-High-Threshold Theory (2HTT, Corwin, 1994). Correct identification of a target was defined as a *hit*, whilst false recognition of a new item was

termed a *false alarm*. The measure of discrimination accuracy (denoted by the index Pr) was calculated accordingly: [number of hits + 0.5 / number of targets + 1] – [number of false alarms + 0.5 / number of new items + 1]. The 2HTT also provides a measure of *response bias* (denoted by Br), which reflects the probability of a participant saying 'yes' to an item when in an uncertain state, and is calculated accordingly: [number of false alarms + 0.5 / number of new items + 1] / [1 – Pr]. A value of 0.5 indicates a neutral response bias (i.e., equal number of 'yes' and 'no' responses when uncertain), values greater than 0.5 are indicative of a liberal bias, whereas values less than 0.5 reflect a conservative bias.

The reality monitoring data are reported as a *source proportion* (i.e., the number of items assigned to a correct source divided by the total number of hits). This calculation takes into account the fact that the maximum possible number of items attributed to the correct source depends on the absolute number of hits. Source misattributions (i.e., attributing items to the wrong source) were also tallied and used to create the following two error indices: (i) internal misattribution, i.e., the tendency to misattribute events (perceived plus new items) to an internal (imagined) source; and (ii) external misattribution, i.e., the tendency to misattribute events (imagined plus new items) to an external (perceived) source.

Statistical analyses

The data for each measure did not have a normal distribution; therefore, differences between AHP patients, nonAHP patients and HVs (i.e., MMSE, NART, age, star cancellation, comb/razor test, hits, false alarms, Pr, Br, source proportion and source misattributions) were analysed using the Kruskal-Wallis test, with post hoc analyses using multiple Mann-Whitney *U* tests and applying a Bonferroni correction to obtained p-values[1]. Gender distribution was analysed using Fisher's Exact test. The Mann-Whitney *U* was used to analyse differences

[1] Because an omnibus test comparing three groups indicates whether or nor the *greatest* difference between groups is significant (i.e., group with largest summed rank ≠ group with smallest summed rank), post hoc tests did not repeat this analysis and involved only the two remaining comparisons. This avoided us being too conservative in our statistical (i.e., Bonferroni) corrections, which might have obscured potentially meaningful patterns in the data by making type II errors.

between AHP and nonAHP patients in the number of days since insult, and degree of hemiplegia (i.e., MRC power) . All tests were two-tailed.

Results

The upper part of Table 1 shows the clinical characteristics and neuropsychological profile of each group. Ten patients fulfilled the criteria for AHP and AHP was absent in 7 control/nonAHP patients. The proportion of males and females did not differ across HVs, AHP and nonAHP groups (all *p*-values >.10). The three groups were matched for age (p=.991), and both patient groups were matched in terms of the length of time between stroke and participation in the study (p=.189), degree of hemiplegia (left upper limb, p=.158; left lower limb, p=.263), NART (p>.99), and MMSE *(*p=.281). However, compared with HVs, both AHP and nonAHP patients had significantly lower NART (p<.001 in both groups), and MMSE scores (p<.001 and .004 for AHP and nonAHP patients respectively). Both patient groups showed some degree of visuospatial neglect on the star cancellation task (AHP vs. HV, p<.001; nonAHP vs. HV, p=.034), and neglect was more severe in AHP patients compared with nonAHP patients (p=.038). Scores on the comb/razor test of personal neglect did not differ between groups (p=.286).

Table 1 and 2 about here

Reality Monitoring Task

The lower part of Table 1 summarises the raw and corrected studied-unstudied recognition memory performance measures, and reality monitoring scores for each group. A detailed breakdown of the raw data from the reality monitoring task is presented in Table 2. The columns indicate the source of the items (i.e., perceived or imagined), and the rows indicate the participants' responses.

Hits, False Alarms, Discrimination Accuracy (Pr) and Response Bias (Br).

The raw number of hits was significantly fewer in AHP patients ($H(2)=9.38$, $p=.009$) and nonAHP patients ($U=28.00$, $p=.035$) compared with HVs, but was comparable in the two patient groups ($p>.99$). The number of false alarms produced by the AHP patients was significantly greater than HVs ($H(2)=16.72$, $p<.001$) and marginally more than nonAHP patients ($U=13.00$, $p=.057$). False alarms were comparable in the HV and nonAHP groups ($p=.289$).

The HV group displayed significantly higher discrimination accuracy scores (Pr) compared with both AHP patients ($H(2)=20.64$, $p<.001$), and nonAHP patients ($U=30.00$, $p=.048$). Discrimination accuracy in AHP and nonAHP patients also approached significance ($U=13.00$, $p=.059$), suggesting a tendency for poorer performance in AHP patients.

Br scores did not differ significantly between the three groups ($p=.473$). However, the Br score was higher in the AHP group than in both the nonAHP and HV control groups, indicating a liberal response bias in the AHP patients and a conservative response bias in the control participants.

Reality monitoring.

Source proportion was significantly lower in AHP patients compared with both HVs ($H(2)=21.23$, $p<.001$), and nonAHP patients ($U=6$, $p=.006$), but did not differ significantly between nonAHP patients and HVs ($U=36.50$, $p=.128$). Analysis of internal misattributions showed no difference between groups ($p=.123$). However, external misattributions were significantly higher in AHP patients compared with HVs ($H(2)=8.17$, $p=.026$), indicating a tendency to misattribute items to an external source. No other differences between groups were significant (all p-values $>.10$).

In summary, the results of Experiment 1 revealed that AHP patients exhibit a reality monitoring impairment for information not directly related to movements (i.e., images of objects), while nonAHP patients are able to reality monitoring this type of information at a level comparable to HVs. This finding is consistent with research into delusions of control in

patients with schizophrenia (Brébion et al., 2000; Anselmetti et al., 2007; Brébion et al., 2002; Brébion et al., 1997), who display a reality monitoring impairment in which imagined *images* are misattributed to an external source, despite their delusion focusing on movement. This finding raises the further question of whether the observed reality monitoring impairment can be demonstrated in the domain of movement. No existing study has examined this question experimentally; therefore, a second experiment was conducted to address this issue.

EXPERIMENT 2: ACTION REALITY MONITORING

The aim of Experiment 2 was to expand the findings from Experiment 1, and test whether patients with AHP have a deficit in reality monitoring movements. Using the same method as Experiment 1, we examined the ability of participants to discriminate between movements that were performed, imagined, or observed. Our predictions were the same as for Experiment 1; that the AHP group would be defective at reality monitoring [movements] relative to nonAHP and HV groups, whereas the nonAHP and HV groups would perform at comparable levels.

Method

Participants

Three patients with AHP and 6 nonAHP control patients took part in the experiment. Patients were a subgroup of those who took part in Experiment 1, with the exception of two naïve nonAHP control patients. Patient performance was compared with the 20 age-matched HVs from Experiment 1. Participant demographics and clinical characteristics are summarised in Table 3.

Stimuli and Apparatus

Ninety action phrases were constructed using existing literature on memory for actions (Hornstein & Mulligan, 2005; Mulligan & Hornstein, 2003; Leynes & Bink, 2002). Each was a simple action involving the upper limb(s), and containing a verb and noun. Half of the actions were unimanual (i.e., usually performed using one hand; e.g., press a button), and half

were bimanual (i.e., usually performed using both hands simultaneously; e.g., clap your hands). Action phrases were presented on a laptop computer. For each participant, the computer randomly assigned 20 actions (10 unimanual and 10 bimanual) to each of three item types: perform, imagine, and observe. *Perform* items were enacted by the participant. *Imagine* items were not enacted, but participants were instructed to imagine themselves performing the action. *Observe* items were enacted by the experimenter while the participant watched without moving. The remaining 30 action phrases were used as new items during the test phase.

Procedure

The procedure was adapted from the Experiment 1. The participant and experimenter sat facing each other at a distance of approximately one meter. During the acquisition phase, participants were told they would hear a series of actions, some of which they should *perform* (i.e., perform item), some of which they should *imagine* performing (i.e., imagine item), and some of which they should *observe* the experimenter performing (i.e., observe item). The experimenter read each action aloud from the laptop display, which was not visible to the participant. Each item was preceded by the instruction "perform", "imagine" or "observe", to indicate the item type to the participant. If actions involved an object (e.g., pick up a pen), participants were instructed to pantomime the object. The pace of presentation was controlled by the experimenter, who read action phrases at a pace of approximately one every 5 s. Participants were reminded to pay careful attention to the whole action phrase (i.e., both the action and item type), and told that each action phrase would be *either* perform, imagine, or observe. Three practice items were completed prior to the actual task.

During the test phase participants were orally presented with previously studied (i.e., target) and unstudied (i.e., new) action phrases in random order. Participants were asked to indicate whether each item was previously performed, imagined, observed, or new. A *familiar* response was available when participants identified an item as a target, but were unsure of the original source. All other aspects of the procedure replicated Experiment 1.

Data and Statistical Analysis

Hits, false alarms, Pr, Br, and source proportion were calculated following the procedures described for Experiment 1. Source misattributions were also calculated to provide the following three error indices: (i) performed misattributions, i.e., the tendency to misattribute actions (observe, imagined and new items) as being performed, (ii) imagined misattributions, i.e., the tendency to misattribute actions (performed, observed and new items) as being imagined, and (iii) observed misattributions, i.e., the tendency to misattribute actions (performed, imagined and new items) as being observed. Statistical analyses were performed as described for Experiment 1; however, in light of the low statistical power that is inevitable when dealing with an extremely small sample such as the size of our AHP group, a more liberal strategy of statistical inference was adopted (i.e., reported *p*-values were not Bonferroni corrected). Although this more liberal strategy increases the likelihood of Type I errors (i.e., false positives), it decreases Type II errors (i.e., false negatives) that might obscure potentially meaningful findings.

Results

Table 3 and 4 about here

The upper part of Table 3 shows the clinical characteristics and neuropsychological profile for each group in Experiment 2. Because participants in Experiment 2 were mostly a subset of those from Experiment 1, their characteristics repeated the previous pattern of results. The proportion of males and females did not differ across HVs, AHP and nonAHP groups (all *p*-values>.10). The three groups were matched for age (p=.371), and both patient groups were matched in terms of the length of time between stroke and participation in the study (p=.119), degree of hemiplegia (left upper limb, p>.99; left lower limb, p=.50), NART (p>.99), and MMSE (p=.190). However, compared with HVs, both AHP and nonAHP

patients had significantly lower NART (both $p<.001$), and MMSE scores ($p<.001$ and .009 respectively). Both patient groups again showed some degree of visuospatial neglect on the star cancellation task (AHP vs. HV, $p=.016$; nonAHP vs. HV, $p<.001$); however, AHP patients and nonAHP patients were matched in severity of visuospatial neglect ($p=.595$). Comb/razor test scores did not differ between groups ($p=.628$).

Hits, false alarms, discrimination accuracy (Pr), and response bias (Br)

The lower part of Table 3 summarises the reality monitoring task performance measures, and Table 4 presents a detailed breakdown of the raw data. Like Experiment 1, the number of hits did not differ between groups ($H(2)=2.11$, $p=.366$). False alarms also replicated the pattern found for Experiment 1; that is, AHP patients made significantly more false alarms than HVs ($H(2)=8.74$, $p=.007$), and marginally more than nonAHP patients ($U=2.00$, $p=.095$), but were comparable in HVs and nonAHP patients ($U=45.00$, $p=.318$).

Discrimination accuracy (Pr) and response bias (Br) also followed the same pattern of results as found in Experiment 1. Pr was significantly higher in HVs compared with AHP patients ($H(2)=10.92$, $p=.001$) and nonAHP patients ($U=22.00$, $p=.018$). AHP patients showed a tendency for lower Pr than nonAHP patients ($U=2.00$, $p=.083$). Br did not differ significantly between the three groups ($H(2)=3.26$, $p=.203$), but was higher in AHP patients than nonAHP patients and HVs, indicating a more liberal response bias.

Reality monitoring

Source proportion was significantly lower in AHP patients compared with HVs ($H(2)=11.54$, $p=.001$) and nonAHP patients ($U=1.00$, $p=.048$), again mirroring results of Experiment 1. However, unlike the results of Experiment 1, source proportion was also significantly lower in nonAHP patients compared with HVs ($U=24.00$, $p=.026$).

Analysis of source misattributions revealed no differences between groups in imagine misattributions ($H(2)=1.98$, $p=.362$) or observe misattributions ($H(2)=1.71$, $p=.453$); however, AHP patients made significantly more perform misattributions than HVs ($H(2)=10.79$,

p=.001), as did nonAHP patients (*U*=28.00, *p*=.028). The number of perform misattributions did not differ between AHP and nonAHP patients (*p*=.143).

In summary, the results of Experiment 2 are partially consistent with our prediction, in that AHP patients were impaired at reality monitoring movements relative to the nonAHP and HV control groups. However, contrary to prediction, nonAHP patients were impaired relative to HVs. The former finding is consistent with the account of AHP proposed by Berti and Pia (REF), while the latter finding is not expected according to this account.

Discussion

This study provides the first direct examination of reality monitoring in AHP. Experiment 1 examined reality monitoring processes not directly related to movement (i.e., for perceived and imagined images of objects) in AHP, thereby mirroring existing studies of reality monitoring in patients with schizophrenia and abnormal motor awareness (i.e., delusions of control). Experiment 2 further examined the effects found in Experiment 1, by exploring the idea that AHP patients suffer impairment to their ability to reality monitoring movements. These experiments are highly pertinent to understanding AHP, as no study has hitherto directly examined reality monitoring impairments in these patients. This is despite the compelling theoretical prediction of such impairments, arising from existing AHP case reports (Venneri & Shanks, 2004), reality monitoring studies in patients with schizophrenia and delusions of control (Brébion et al., 2000; Anselmetti et al., 2007; Brébion et al., 2002; Brébion et al., 1997), and accounts of AHP utilising well-established models of normal motor control and awareness (Berti & Pia, 2006).

The results of Experiment 1 revealed that, compared with nonAHP patients and HVs, patients with AHP exhibited a significant impairment in their ability to correctly identify the source of an image as perceived or imagined. This ability was preserved in nonAHP patients, with performance being comparable to that of HVs. Patients with AHP showed a propensity to report memories as originating from an external (i.e., perceived) source. Interestingly, they

did not make misattributions in the opposite direction (i.e., external/perceived items misattributed as internal/imagined). We also found both patient groups to be impaired in their studied-unstudied recognition memory. Discrimination accuracy (Pr) was significantly lower in AHP and nonAHP patients relative to HVs, and was marginally lower in AHP patients relative to nonAHP patients. Together these results suggest that discrimination accuracy lies on a continuum in HVs, nonAHP patients and AHP patients.

Examination of hits and false alarms indicated that both patient groups were matched in the number of hits they produced, which was significantly fewer than HVs. However, the AHP patients produced more false alarms than nonAHP patients and significantly more than HVs, while the number of false alarms was comparable in nonAHP patients and HVs. The AHP patients also showed evidence of significantly poorer discrimination accuracy compared to the HVs. However, discrimination accuracy scores were not significantly different between the two patient groups which suggests that memory impairment alone cannot explain the AHP patients' elevated number of false alarms. This proposal receives further support from the response bias data, which revealed that the AHP patients showed evidence of a liberal response bias whereas the nonAHP patients (and the HVs) showed a conservative bias.

The low number of hits suggests both patient groups have difficulty encoding distinctive memory representations. Due to this encoding impairment, patients do not have access to high quality, item-specific information which is critical for discriminating between studied and unstudied items. Assuming that item-specific memory was compromised in both AHP and nonAHP patients, as reflected by their significantly lower discrimination accuracy scores relative to the HVs, why the AHP group's score was poorer than the nonAHP patients still needs to be explained. The AHP group showed a liberal response bias whilst the nonAHP group performed at the same level as the HVs in showing a conservative bias. As previously explained, response bias reflects the decision strategy adopted under conditions of uncertainty, when information immediately in memory is insufficient to determine whether an

item was studied or not. An explanation of liberal response bias in AHP therefore indicates abnormal criterion setting and decision processes underlying the production of false alarms beyond that which would be predicted on the basis of random guessing.

In situations where item-specific contextual information is lacking, participants may use idiosyncratic approaches to decision-making. For example, participants may rely on gist memory, which provides an overall impression of general stimulus characteristics, but it is insufficiently detailed to distinguish between items which share a high degree of inter-item similarity. According to fuzzy-trace theory, gist memory is the primary source of false alarms (Brainerd, Reyna, & Kneer, 1995). Thus, the elevated false alarms exhibited by our AHP patients may be underpinned by a reduced access to detailed item-specific memory representations, which results in patients adopting an uncritical/more lenient decision criteria and accepting gist-based feelings of familiarity as diagnostic of the item having been presented during the acquisition phase.

The results of Experiment 2 demonstrated studied-unstudied recognition memory impairments that mirrored exactly the pattern found in Experiment 1, thereby providing additional support for our initial findings regarding recognition memory in AHP. Results of Experiment 2 further showed that AHP patients are impaired at reality monitoring movements relative to HVs and nonAHP patients. This finding is consistent with the idea of a breakdown in the monitoring of internally-generated and externally-perceived movements in AHP (Berti & Pia, 2006). However, an unexpected finding of Experiment 2 was impaired reality monitoring for movements in nonAHP patients, relative to HVs. This differs from the findings of Experiment 1, where nonAHP patients' reality monitoring was at a level comparable to that of HVs. The overall pattern of findings from Experiment 2 indicate that the ability to reality monitor movements is significantly impaired in AHP patients compared with nonAHP patients, which in turn is impaired compared to normal levels. This suggests

that deficits in reality monitoring movements might be a general consequence of damage to the motor system, and lie on a continuum in AHP patients, nonAHP patients and HVs.

Unfortunately, there are no previous experimental investigations of reality monitoring in AHP against which to compare the present findings. Venneri and Shanks (2004) propose that a reality monitoring impairment might be responsible for AHP, while existing research in patients with schizophrenia and delusions of control provides compelling evidence that abnormal motor awareness stems from a deficit in discriminating between self-generated (i.e., imagined) and externally-generated (i.e., perceived) events (Brébion et al., 2000; Brébion et al., 2002; Brébion et al., 1997). Our finding of a non-motor reality monitoring deficit in AHP is consistent with this research. It has been suggested that these impairments in schizophrenia might be a result of diminished perceptual richness of externally-generated memories. As a result, memories of imagined events might appear to have equal contextual support as memories derived from an external source, making confusion between memories of an internal and external origin more likely (Johnson, 1997; Johnson et al., 1993; Keefe, 1998). We speculate that a similar mechanism might operate in AHP. For example, reduced attention, arousal or social interaction might result in imagined events having more prominence in AHP, leading internally predicted movement to be mistaken for externally produced movement.

Berti and Pia (2006) suggest that AHP arises from a failure to register discrepancies between internally- and externally-generated movement. We predicted that this failure would manifest as a failure to reality monitoring movements in AHP. Experiment 2 supported this prediction. The AHP group made more source attribution errors – more often erroneously misattributing memories about movements to a performed source. A possible basis of this impaired reality monitoring in the AHP group may be the previously described *reduction* in item-specific contextual information (Keefe, 1998). If there is a disruption in the coding of these memories initially, the context of the input of the stimulus is likely to be reduced. The

internal comparator proposed by the forward model might fail to detect this reduction in contextual input, making discrimination of source unreliable, since the phenomenological characteristics of internal and external events are less distinctive.

Based on existing theory and the findings of our two experiments, it is possible to speculate, if only tentatively, how a combination of reality monitoring impairments might contribute to the pathogenesis of AHP. First, a deficit in the ability to reality monitor movements following brain injury might impair the ability to discriminate between internally-generated information about intended movements and externally-generated sensory information concerning actual movement (or lack thereof). A tendency to attribute events to an external source means that internally represented movements are misattributed to an external (i.e., performed) origin. Second, a breakdown of reality monitoring processes for information not directly related to movement might preclude indirect verification of whether or not movement has occurred as intended. The outcome of these combined reality monitoring impairments in AHP is knowledge of the motor system based entirely on internal motor information, which erroneously indicate successful execution of intended movement. In contrast, although nonAHP patients also exhibit impaired reality monitoring for movement, this is of a lesser extent than in AHP, such that the discrimination of internal and external movement is generally more accurate. In addition, nonAHP patients are able to use extended reality monitoring processes to check knowledge about their general state of healthy, since they do not suffer a reality monitoring deficit for information other than movements. In combination, these relatively preserved reality monitoring processes allow nonAHP patients to conclude that their ability to move is impaired.

Some possible limitations of the study should be considered. It might be argued that our results were influenced by pre-morbid intelligence (i.e., NART) and current mental state (i.e., MMSE), which were impaired in both patient groups relative to HVs. However, it is unlikely that these deficits account for the differences in studied-unstudied recognition

memory and reality monitoring of our groups, since AHP and nonAHP groups were matched for NART and MMSE scores, but showed differences in recognition memory and reality monitoring. This negates the possibility that differences in the performance of these two groups only reflect the influence of pre-morbid intelligence or current mental state.

The findings also cannot be attributed to the presence of unilateral neglect. Although some patients did exhibit spatial neglect, we used our screening tests to ensure that stimuli were presented in the patients' non-neglected hemispace. Thus, any differences in performance on the reality monitoring task cannot simply be a confounding effect of unilateral neglect.

Another factor that should be taken into consideration when drawing conclusions from our study is the more liberal strategy adopted for statistical inference in Experiment 2. This was done in order to counteract the inherent loss of statistical power that occurs as sample size decreases, but introduces the possibility of increased Type II errors. We believe that our method was appropriate in light of the small AHP group sample size in Experiment 2. Furthermore, the pattern of results was consistent with that found in Experiment 1, and supports our overall interpretation of the findings.

A final consideration regarding our findings is the ability to establish a causal relationship between AHP and reality monitoring impairments. Although the results of the group analyses are largely consistent with our predictions, in order to establish a causal link all AHP patients should show the same reality monitoring deficit. While the majority of AHP patients exhibited impaired reality monitoring on Experiments 1 and 2 relative to nonAHP patients, this relationship was not steadfast. This constraint on our findings does not negate our conclusion that the combination of reality monitoring failures observed might contribute to the pathogenesis of AHP; however, a direct causal relationship cannot be inferred.

A task for future research will be to explore the exact nature of the observed breakdown in discriminating the origins of information. For example, the present study

employed a *reality monitoring* paradigm (i.e., distinguishing between *memories* of an internal versus external source) to examine the ability to discriminate between internally- and externally-generated information in AHP. However, AHP patients exhibit a motor monitoring deficit *during* the execution of movements (i.e., an 'online' monitoring deficit). Johnson (1991), uses the term reality *testing* to refer to the processes that discriminate internally- and externally-generated information during ongoing experience. A link between reality testing deficits and a failure to reality monitor is suggested by existing research in healthy volunteers (Rankin & O'Carroll, 1995). However, it would be interesting to confirm the deficits found in our study using a reality testing task (e.g., by adapting an existing task, in which recurring items must be discriminated from non-recurring items during repeated runs of a recognition memory task; see Schnider & Ptak, 1999).

In conclusion, our study is the first to examine reality monitoring in AHP. Results suggests that AHP may be linked to a combination of reality monitor failures, both for movement and information not directly related to movement. Findings are consistent with existing investigations of reality monitoring in psychiatric patients with abnormal motor awareness (i.e., delusions of control in schizophrenia), and support recent accounts of AHP, which propose that motor awareness depends on comparison of the predicted and actual sensory consequences of movement.

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Figure Captions

Figure 1. A simple forward model of the normal motor system (from Blakemore, Frith & Wolpert, 2001). Predictions of sensory feedback are made by a movement predictor, using an efference copy of the motor command. These predictions are compared to the actual sensory feedback. In hemiplegic patients without anosognosia damage is located in the production of movement; therefore, comparison of actual and predicted sensory feedback produces a discrepancy and normal awareness of the motor deficit. Hemiplegic patients with anosognosia fail to execute this comparison successfully and register a discrepancy. Consequently, knowledge of the motor system is constructed from sensory predictions, resulting in a false belief of being able to move.

Figure 2. Schematic of reality monitoring task. During the acquisition phase participants are presented with a written, concrete noun followed by either a line drawing or an empty circle into which they visualise a line drawing. During the test phase participants are presented with written nouns and make a studied-unstudied recognition memory judgement followed by a reality monitoring judgement.