CHAPTER ONE

WAS THAT ME?
THE RIGHT HEMISPHERE AND THE SENSE OF AGENCY:
EMBODIMENT OF OTHERS AND MISATTRIBUTION OF
SELF-GENERATED ACTION

CATHERINE PRESTON AND ROGER NEWPORT
SCHOOL OF PSYCHOLOGY, UNIVERSITY OF NOTTINGHAM (UK)

Introduction

There is a great similarity between the actions of other people and the actions of ourselves, not only in terms of the movements characteristics, but also in the way that these movements are processed in the brain. It is the similarity between our own and others’ actions that allows us to interpret the actions, intentions and desires of other people. Before we can begin to do this, however, we must be aware whether the source of a perceived action is someone else or ourselves. The ability to correctly identify our own actions from those produced by other people (agency attribution) is a fundamental component of human social interaction and self-awareness and while ambiguity of this experience is rare, the process can become compromised following brain injury if mechanisms associated with self-awareness and agency attribution become disrupted. This chapter will examine the evidence for right hemisphere dominance in the attribution of agency and will present experimental data from a patient with cortical disconnection between his right and left hemispheres, plus an experiment in which disruption of the right parietal region with transcranial magnetic stimulation (TMS) leads neurologically intact participants to misattribute self-generated actions and the experiences of patient MP who feels a sense of agency over the movements of others following a right hemisphere stroke.

1.1 Agency attribution and the right hemisphere

Functional imaging has implicated brain regions in the right hemisphere for the successful discrimination between actions made by the *self* and those made by *other* people. For example, Farrer et al. (2003) tested a sample of neurologically intact participants making joystick movements in a scanner while measuring brain activity using positron emission tomography (PET). Participants were given visual feedback of their movements by the presentation of a virtual hand and joystick on a mirror positioned in front of their moving hand. In some trials the visual feedback deviated from their actual movements by 25, or 50 degrees (defined as *other*) while the rest were left unperturbed (defined as *self*). The results of this experiment revealed an increase of blood flow to the right inferior parietal lobe (rILP) when participants made *other* judgments and an increase in blood flow to the insula (primarily right hemisphere) when participants made *self* judgments. The PET study was a follow-up to a previous functional magnetic resonance imaging (fMRI) study that also highlighted the involvement of the anterior insula and rILP for self- vs. other-generated movements respectively (Farrer and Frith 2002).

Further evidence for right hemisphere dominance in the sense of agency comes from neuropsychology. Symptoms following right hemisphere damage frequently involve disorders of self-awareness. Such disorders include: hemi-spatial neglect, a disorder in which a patient is unaware of the side of space contralateral to the lesion, often including their own body parts (Valler and Persn 1986, Driver and Mattingley 1998); anosognosia, in which the patient is unaware of their contralesional disabilities (for
example their hemiplegia) (Paysant et al. 2004, Jehkonen et al. 2006); asomatognosia, in which patients can deny ownership of their own limb (Pia, Neppi-Modona et al. 2004); and some cases of alien limb syndrome (Groom et al. 1999) in which patients report the absence of volitional control over the affected limb - often referring to it in the third person.

A frequently used method to investigate the hemispheric specialization of function is to observe patients who have a damaged or absent corpus callosum. The corpus callosum is responsible for the transfer of cortical information between the cerebral hemispheres. Cases in which this is disconnected (by surgery) or absent (by agenesis) allow stimuli to be presented both visually and (to large extent) proprioceptively to an individual hemisphere (Gazzaniga 2000). Each hemisphere processes information from the contralateral visual field, so when an acallosal individual fixates on a central point a stimulus presented to the left of centre is processed by the right hemisphere and a stimulus to the right of centre by the left hemisphere. The processing of information from the muscles in the limbs is less lateralised as there are a small number of ipsilateral as well as contralateral connections from the proximal muscles. We tested patient TCA, a 65 year old male born without a corpus collosom, who only became aware of his callosal agenesis at the age of 61 years following routine scans to investigate persistent chronic headaches. Prior to this point TCA had no inclination of his condition, with no obvious symptoms and an IQ score well within the normal range (117). This study tested TCA’s performance on a self/other judgment task when visual information was exposed to either his right or his left hemisphere. A cursor rotated perturbation paradigm was used similar to that used by Farrer et al. (2003).

TCA was required to fixate a central point whilst making unilateral reaching movements towards a target bar that disappeared upon trial onset. The two key conditions were RHRP, in which TCA reached with his right hand to a target presented in his right hemifield (all left hemisphere) and LHLP, reaching with his left hand in his left hemifield (all right hemisphere). Visual feedback about the movement of his index finger was given in the form of a cursor presented using a projector and mirror set-up so as to appear in the same spatial location as his actual finger (vision of his real limb was occluded). On half the trials the cursor movements did not deviate from the actual trajectory of his index finger (zero degree perturbation, or self). In the remaining trials a perturbation was applied to the cursor trajectory in the form of a four or sixteen degree cursor rotation (other). At the end of each movement TCA gave a verbal judgment of whether the seen movement was that of self or other. For the duration of the experiment TCA’s eye movements were monitored by a video camera to ensure that fixation was maintained. The results of the experiment are given in Figure 1.1.

![Figure 1.1](image)
Was that me?

Control participants seemed to have difficulty with this task only when the perturbation was small, with their performance dropping close to chance regardless of the side of presentation for 4 degree perturbations. TCA’s performance in the LHLP condition (visual information presented to the right hemisphere) was very similar to that of control participants for all perturbations (in fact his performance was slightly better than controls for the zero degree perturbation). In the RHRP condition, however, when visual information was presented to the left hemisphere, TCA’s performed significantly worse than controls for both the zero and sixteen degree perturbations (his percent correct responses fell outside and below the controls’ 95% confidence intervals) while his responses for the 4 degree perturbations were, like controls, close to chance. So, while performing similarly to controls with his right hemisphere, TCA performed very poorly when forced to rely on his left hemisphere. These data show a clear dissociation between the performance of the two hemispheres when presented with identical visual information and supports the body of evidence implicating a right hemispheric dominance for agency attribution.

1.2 Embodiment of others following right hemisphere damage

Patient MP is a 50 year old male who suffered a right hemisphere stroke complicated by an undetected abscess which blocked blood reperfusion and prohibited recovery. He was left with a large right frontotemporal parietal ring enhancing mass lesion with surrounding oedema and sulcal effacement throughout the frontal and parietal lobes. As a result of this brain damage MP experiences unusual symptoms in which he sometimes feels a sense of agency over movements produced by other people, that is: embodiment of another agent’s limb. MP’s left arm is hemiplegic with a severe loss of sensation as demonstrated by an inability to score on tests of proprioception. The only mild sensation he does have is from the proximal muscles of the limb resulting in a fear of becoming unbalanced when the entire limb is raised in the absence of vision. Interestingly MP also reports that he is unable to imagine his left hand moving and also that he no longer dreams.

The false agency experienced by patient MP is quite striking and unusual as he experiences a feeling of agency over movements that he never makes (and is unable to make). While other examples of disturbed cognition, in terms of embodiment, have been reported, these tend to be an absence of awareness or ownership of a body part rather than feeling embodiment or agency over another person’s limb (e.g. Arzy et al. 2006). In 1999 Sirigu et al. observed patients with left parietal damage who, under experimental conditions, were more likely to accept the spatially coincident movements of the experimenter as their own when they shared the same goal. In these cases the experimenter’s movements were presented in the same location as the patients’ own limbs using mirrors and cameras. In contrast, with MP, his experiences of agency over another’s movements occur spontaneously outside laboratory conditions and occur in the absence of any movement by MP himself. Some phantom limb patients claim to be able to make their phantoms move (thought to be a phenomenon arising from a sense of limb position based on the predicted consequences of a planned, but not executed, movement), but while such phantom limb movements are also without actual self-movement, these are again different to MP as they involve agency over an internally-generated self-movement rather than agency over another person’s limb.

MP reports this transferal of agency or embodiment in situations in which he would normally use his left arm, often in bimanual tasks in which an external agent is assisting him by performing the task that his left hand or arm would normally do. For example, when making a cake, he would stir the mixture with his right hand while another person held the bowl (acting as his left hand); in such situations MP reports a sense of embodiment over the other person’s hand - feeling as if it were his own, despite large morphological differences (including size, sex and colour). This experience seems to be apparent only when the actions of MP and those of the other person share a common goal. When the external agent’s limb makes an unexpected movement this can result in the brief but disturbing experience of his hand moving without his volition. In other situations the experience of embodiment is produced by the action itself. For instance, if, while looking through a newspaper, someone else turns the page for him (acting as his left hand) he often has the experience that it was his own left hand that turned the page. Crucially, this only seems to happen when MP himself is thinking about the need to turn the page.

1.3 Misattribution of self-generated action in neurologically intact controls
Was that me?

While attribution errors in brain damaged individuals have been studied extensively, reports of misattribution in healthy people are rare. The study described below utilised a relatively new neuroscience technique known as transcranial magnetic stimulation, or TMS, in order to induce agency attribution errors in neurologically intact participants. With TMS, a figure of 8 ‘wand’ is held against the skull over the brain region of interest in order to deliver stimulation. Passing a brief high-current electrical pulse through coils of wire within the wand produces (through electromagnetic induction) harmless stimulation within the brain. The effect of this stimulation is to temporarily and randomly disrupt the neurons directly beneath the wand, inducing a transient virtual ‘lesion’ in the brain. The purpose of this is to disrupt a specific brain region or function while the participant completes a relevant task: a change in performance indicates involvement of the stimulated brain region in performing that task. Because the TMS pulse is noisy and causes a contraction at the focal point on the scalp, performance is often compared against trials in which TMS is applied to a control site not hypothesised to be involved in the task, as well as against trials in which no TMS pulse is delivered. The control site chosen for this experiment was the vertex, which is a frequently used control site to test for non-specific effects of TMS (e.g. Nyffeler et al. 2006, Muggleton et al. 2006, Bestman et al. 2002). Localisation of the experimental site, the right inferior parietal lobe (rIPL), was based on skull co-ordinates from a previous experiment by Nager et al. (2004). We investigated the effect of TMS over the rIPL on self/other judgments in an agency task using a cursor rotation paradigm similar to that used by Farrer et al. (2003). Based on the aforementioned imaging data (Farrer et al. 2003, Farrer and Frith 2002) that revealed an increase in rIPL activity when completing a similar task it was predicted that TMS stimulation would have an effect on percent correct responses when applied to the rIPL and not when applied to the vertex. In order to directly test this, planned comparisons were conducted comparing percent correct responses with and without TMS over the rIPL and the vertex for each size of perturbation.

Each participant (8 female 2 male, right handed mean age 22 years) took part in both rIPL and VERTEX conditions, the order of which was counterbalanced between participants. The experimental set up was similar to that for the callosal agenesis study described above except that this time the movements were represented by a life-sized image of the experimenter’s (female) hand positioned in a pointing posture (extended index finger). A Magstim Rapid TMS machine (the Magstim Company LTD) with double 70mm coil was used to deliver the magnetic pulse to the appropriate areas on the scalp which were marked out using disposable surgical caps. The vertex was found at the intersection between the nasion-inion line and the line between the pre-auricular points. The coil was placed tangential to the skull and was set to stimulate at 110% of motor threshold (defined as the minimal TMS intensity required to cause an involuntary twitch in the contralateral hand in at least 5 out of 10 trials when stimulating the ipsilateral cortical hand area). Participants were required to hold their hand in the same posture as the hand image that represented their movements (pointing posture).

The trajectory and speed of the virtual hand was identical to the actual movement (delay <10ms) on every trial for the first 100mm of the movement before becoming occluded by a virtual black bar (440x130mm). On half the trials, during occlusion, the virtual hand continued to accurately represent the actual reach trajectory (‘self’). On the other half of the trials, while occluded, the image undertook an unseen lateral shift equivalent to a cursor rotation of four degrees (‘other’). Thus, when the hand reappeared on the other side of the occluding bar, its spatial position was either coincident with the actual reach (‘self’) or perturbed by 4 degrees to one side of the actual hand position (‘other’) (remember that the real hand could not be seen, only the representation of the hand). In half of the ‘other’ trials this shift was to the left and in the remaining trials it was to the right. The TMS was applied as a single pulse delivered just before the virtual hand reappeared from occlusion.

The results were analysed by a 2x2x2 repeated measures ANOVA with three factors: STIMULATION (TMS vs. NO-TMS), BRAIN AREA (rPL vs. VERTEX) and PERTURBATION (SELF vs. OTHER). There was no significant main effect of STIMULATION F(1,9)=0.031, p=NS, nor BRAIN AREA F(1,9)=9.09, p=NS. There was a significant main effect of PERTURBATION F(1,9)=17.183, p<.01 with mean percent correct responses being greater for SELF (mean=76.3, SD=16.24) than OTHER (mean=60.4% SD=12.62). A significant two-way interaction between STIMULATION * PERTURBATION F(1,9)=12.27, p<.01. was not very informative as it was collapsed across the brain areas and can be completely explained by the results of the planned comparisons. All other interactions were non significant (min. F(1,9)=2.794, p=NS). Weighted means planned comparisons were conducted to test the prediction that TMS over rIPL would affect self/other judgments compared to no TMS or TMS over the vertex. These revealed significant
Was that me?

differences in the percentage of correct responses made between TMS and NO-TMS trials for both the SELF and OTHER conditions at the rIPL but not at the VERTEX (see Figure 1.2 for details).

![Figure 1.2: The effects of TMS over rIPL and the vertex on correct self vs. other responses for perturbed and unperturbed trials. Planned comparisons revealed a significant difference between TMS and no-TMS trials over rIPL for both unperturbed (F[1] = 11.96, p < 0.01) and perturbed trials (F[1] = 6.43, p < 0.05), but not over the vertex for either unperturbed (F[1] = 0.33, NS) or perturbed trials (F[1] = 4.31, NS).](image)

The results of this experiment show that when TMS is applied over the rIPL, participants are more likely to give a judgment of other for both present and absent perturbations (compared to when no TMS was applied); percent correct responses were reduced for the zero-degree perturbation, but increased for the four-degree perturbation). This TMS effect was not observed when stimulation was over the vertex. At first glance these results may seem counter-intuitive given the results of previous imaging experiments using similar paradigms. For example, Farrer and colleagues (Farrer et al. 2003 and Farrer and Frith 2002) reported increased activation in the rIPL when participants made other judgments compared to self judgments in tasks involving perturbed vs real feedback of cursor or joystick movements. In accordance with these findings one might expect that disrupting the area thought to be responsible for other attribution (rIPL) would lead to a disruption of the ability to make other judgments - and hence an increase in self judgments. However, the results are compatible with forward model accounts of the processes underlying rIPL activity. Frith et al. (2000) formulated a comparator model of agency attribution which attempted to explain the delusions of control symptoms often observed in schizophrenic patients. Such patients often attribute self-generated actions to another agent, a comparable experience to that induced in this experiment by rIPL TMS.

1.4 A forward model explanation of agency attribution

Delusions of control, otherwise known as passivity, are a common symptom of schizophrenia. Such patients can accurately carry out movements as intended, but feel as if their movements are under the control of another (external) agent. A forward model account of abnormalities in the awareness of action, proposed by Frith et al. (2000), helps us to understand the processes that might underlie this experience (see Figure 1.3). Furthermore, this model can be used to account for the TMS and patient data contained within this chapter. Whenever the CNS (central nervous system) plans a movement, a copy of the motor command is generated (called an efference copy) and this can be used by the CNS to predict the consequences of that movement. Such a prediction mechanism (forward model) can be used in many ways, but importantly it allows the CNS to anticipate and correct movement errors, filter expected sensory input and help maintain the estimate of the current state of the motor system. An accurate representation of one’s own current limb
position depends on accurate sensory feedback as well as accurate current state predictions using forward modelling. Lesions to such a system can produce a variety of abnormalities of limb awareness and, as a consequence, abnormalities of agency attribution (Frith et al. 2000).

![Diagram](https://example.com/diagram.png)

Figure 1.3: A forward model account of agency attribution based on Frith et al. 2000. See text for details.

Starting from the top left of the diagram (Figure 1.3) and working down the left hand side: the intended goal of an action is necessary to specify the desired (next) state of the limb and also the movement required to achieve that state. At this stage a motor command is generated to execute the necessary movements and a parallel efference copy of that command is sent to the predictor in order to calculate the consequences of that particular motor command. In addition to this, and following on from the motor command, there is the (estimated) actual state of the motor system based on the outgoing motor commands and sensory feedback. This is an iterative loop and is constantly active both before and throughout the movement and as the movement unfolds there are a number of comparisons that can be made in order to monitor and update it. Comparisons (represented by crossed circles in Figure 1.3) can be made between the desired state and the predicted state, between the desired state and the actual state and between the actual state and the predicted state. Any discrepancies can be used to modify and correct the motor command on-line during the movement and so make it as accurate as possible. Thus, feelings of agency are apparent when the comparisons match, but if the discrepancies between any of the comparisons become too large then the CNS may treat any observed or internally monitored self movement as belonging to, or being under the influence of, an external agent. When the system dysfunctions, therefore, perhaps through brain abnormality or lesion, misperceptions of agency can occur.

Frith and colleagues suggest that schizophrenic patients suffering from passivity have a problem with predicted state representations and that they perceive a false discordance between the predicted and actual states of their movement. As a consequence of this they feel as though an external agent is controlling their actions – even though the intended goal is still achieved (the rest of the system remains intact so the patient can still successfully construct and execute the desired movement and their intended goals match their perceived outcome). This is a similar process to that observed in the aforementioned TMS experiment. The model predicts that disrupting the predicted state representation would lead to an increase in other judgments and this is what happens when rIPL function is disrupted by TMS. This experiment helps place
Was that me?

these processes in the right hemisphere, specifically the right parietal region. Activity observed in this area in imaging studies when making other judgments could represent comparator activity: when the predicted and actual representations match there is little activity compared to when there is a discordance and the greater the discordance the greater the activity (Farrer et al. 2003). An increase in activity has also been observed in schizophrenic patients suffering from passivity compared to both schizophrenics not suffering from passivity and non-schizophrenics (Spence et al. 1997).

Although comparator mechanisms are probably bilateral, the weight of evidence suggests that the use of forward model comparisons for the attribution of action, or the feeling of control over one’s own actions, is firmly rooted in the right hemisphere – specifically the right inferior parietal lobe (Farrer et al. 2004; Decety and Chaminade 2003). Within this framework the results from our colossal agenic patient, TCA, are relatively straight-forward. In the isolated left hemisphere condition (RHRP) visual (and perhaps proprioceptive) information is unavailable to the relevant comparators, making judgments close to chance. In the isolated right hemisphere condition (LHLP) the same information is available to the comparators and therefore TCA’s results were the same as normals. In control participants presenting information only to the left hemisphere does not present a problem as relevant information regarding the position of the target and limb can be transferred effectively to the comparator via the corpus callosum.

With patient MP we propose that he also has a deficit in movement prediction, but instead of forming inaccurate predicted state representations (like passivity schizophrenics and our TMS participants) he is unable to form any such representation at all, accurate or otherwise. Although we have no direct evidence for this, his self-report of being unable to imagine limb movements (even when pressed to do so) strongly suggests that this is the case. Making imaginary movement and making actual movements rely heavily on common neural substrates (see Wilson 2003; Decety and Grezes 2006 for reviews). Indeed, Schwoebel et al. (2002) reported the unusual case of a man with bilateral parietal lesions who unknowingly executed imagined movements. Frith et al. (2000) have suggested that representations of imagined limb movements may correspond to predicted state representations, meaning that an inability to form imaginary movement representations could also mean an inability to form the predicted state representations necessary for successful agency attribution. This idea is supported by data from schizophrenic patients suffering from passivity (proposed by this model to have disrupted predicted state representations) as they have also been found to have a deficit in motor imagery (Maruff et al. 2003). If MP is unable to construct and compare predicted state representations then in the absence of comparisons between his predicted state and his actual or desired state he must rely upon comparisons between his actual state and the representation of his original goal when making judgments about the ownership of movements. The formulation of movement goals, being associated with the left hemisphere (Hamilton et al. 2006), should be spared in MP, which leaves the problem of matching the intention to the actual movement (of which there is none). In some of MP’s experiences, the feeling of agency involves a stationary limb (in the example of the hand that holds the mixing bowl) and in these cases the intended limb position matches the actual limb position, even though it is not MP’s own limb (from which he gets no incongruent proprioceptive or visual feedback if it is out of sight). In cases in which the external agent’s hand is moving, again MP’s overall goal (e.g. turning the page of a newspaper) matches the perceived movement, which, in the absence of motor prediction and proprioceptive feedback, is entirely reliant on visual input. Clearly, in the absence of motor prediction and actual state feedback, minor differences in limb posture and movement kinematics go unnoticed. Quite why MP fails to detect gross differences in colour, sex and age between his own and the external agent’s limb is still a mystery, but may reflect a further global deficit in the representations of self or all body parts.

1.5 Summary

Despite the apparent ease with which humans make accurate judgments about the ownership of their own and other people’s actions, the processes that underlie these decisions are fragile and abnormalities within the system can lead to remarkable and bizarre attribution errors. To date, the bulk of the evidence suggests that the right hemisphere plays a pivotal role in determining whether movements are generated by ourselves or by another person. In particular the right inferior parietal lobe appears to be crucial for detecting the ‘otherness’ of an action. Disconnection, disruption or damage to this area impairs our ability to say for certain whether it was ourselves that were responsible for making perceived movements. Many attribution errors can be explained by a forward model mechanism in the brain, in which errors arise from an impaired ability to compare the predicted state of our own limbs with their current actual state. Because
Was that me?

our predicted state arises directly from our intended control over the movement any discrepancies must be the result of action by an external agent.

1.6 References


Was that me?

