

Anosognosia and the Two-factor Theory of Delusions

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Abstract: Anosognosia (denial of impairment), and especially anosognosia for hemiplegia, seems to involve a belief that counts as a delusion by the usual definitions. Existing theories of anosognosia for hemiplegia appeal to impaired feedback from the paralysed side of the body and to cognitive impairments. We show how cases of anosognosia for hemiplegia can be brought within the scope of a generic two-factor theory about the aetiology of monothematic delusions of neuropsychological origin.

Anosognosia is literally ‘unawareness of or failure to acknowledge one’s hemiplegia or other disability’ (*OED*). Etymology would suggest the meaning ‘lack of knowledge of disease’ so that anosognosia would include any denial of impairment, such as denial of blindness (Anton’s syndrome). But Babinski, who introduced the term in 1914, applied it only to patients with hemiplegia who fail to acknowledge their paralysis. Most commonly, this is failure to acknowledge paralysis of the left side of the body following damage to the right hemisphere of the brain. In this paper, we shall mainly be concerned with anosognosia for hemiplegia. But we shall also use the term ‘anosognosia’ in an inclusive way to encompass lack of knowledge or acknowledgement of any impairment. Indeed, in the construction ‘anosognosia for X’, X might even be anosognosia for some Y.

In the literature on anosognosia, rather little has been made of the fact that a patient’s belief that he or she is able to move a limb that is, in reality, paralysed seems to count as a delusion by the usual definitions. It is a false belief that is ‘firmly sustained despite...incontrovertible and obvious proof or evidence to the contrary’ (*DSM-IV*, 1994, p. 765). Given this fact, it is natural to ask whether existing theories of delusions can be applied to cases of anosognosia. Our aim in this paper is to show how at least some cases of anosognosia for hemiplegia fall within the scope of a generic two-factor theory of delusions.

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1. Assessment of Anosognosia and Some Examples

Anosognosia is usually assessed by means of a structured interview, beginning with general questions about the patient's health and why the patient is in hospital and moving on to more specific questions about the impairment in question, such as paralysis of the left arm and leg (Anderson and Tranel, 1989). These more specific questions might begin with 'How is your left arm? Can you move it?'. If the patient verbally denies the paralysis then the examiner may ask the patient to raise the left arm or to reach with the left hand to touch the examiner's hand. When the patient fails to perform the movement, the examiner may ask for some explanation as to why the arm has not moved (Berti, Làdavas and Della Corte, 1996).

On the basis of an assessment interview, patients are classified as having no anosognosia, or mild, moderate or severe anosognosia, in accordance with a scheme such as the following (Bisiach and Geminiani, 1991; Bisiach, Vallar, Perani, Papagno and Berti, 1986):

- 0 = The disorder is spontaneously reported or mentioned by the patient in reply to a general question about his complaints (no anosognosia);
- 1 = The disorder is reported only following a specific question about the affected function (mild anosognosia);
- 2 = The disorder is acknowledged only after its demonstration through routine techniques of neurological examination (moderate anosognosia);
- 3 = No acknowledgement of the disorder can be obtained (severe anosognosia).

1.1. An Example: Patient CC

Berti and colleagues describe the case of an 80-year-old woman, CC, who suffered left-side paralysis following a right-hemisphere stroke. The initial interview with the patient began as follows (Berti, Làdavas, Stracciari, Giannarelli and Ossola, 1998, pp. 28–9):

Examiner: Where are we?

Patient: In the hospital.

E: Which hospital?

P: Santa Orsola.

E: Why are you in the hospital?

P: I fell down and bumped my right leg.

E: What about your left arm and leg? Are they all right?

P: Neither well nor bad.

E: In which sense?

P: They are aching a bit.

E: Can you move your left arm?

P: Yes, I can.

E: [The examiner puts her right index finger in CC's right visual field] Can you touch my finger with your left hand? [CC does not move]

E: What happens?

P: It happens that I am very good.

E: Have you touched my finger?

P: Yes.

E: Can you touch your left hand with your right hand?

P: [Doing it without hesitation] Yes!

Later in the interview, there was the following exchange (1998, pp. 29–30):

E: Could you clap your hands?

P: I am not at the theatre.

E: I know. But we just want to see whether you are able to clap your hands.

P: [CC lifts her right arm and puts it in the position for clapping, perfectly aligned with the trunk midline, moving it as if it was clapping against the left hand! She seems perfectly satisfied with the performance]

E: Are you sure that you are clapping your hands? We did not hear any sound.

P: I never make noise.

Thus patient CC denied her left-side paralysis even when she was presented with obvious evidence of it.

Because patient CC did not acknowledge her paralysis, even after it had been demonstrated to her (by her failing to reach out and touch the examiner's right index finger and her failing to clap audibly), she was classified as having severe anosognosia for her hemiplegia.¹

1.2. Awareness, Acknowledgement, and Appreciation of Consequences

Berti and colleagues (1998) report that CC not only claimed that she *could* move her arm and that she *had* moved her arm but also claimed that she *was moving* her arm when questioned *during* the period when the requested action should have taken place (1998, p. 29):

E: So please try. Are you doing it?

P: Yes.

E: Have you already touched my finger?

P: *Not yet . . . NOW!*

¹ Berti and colleagues actually used a three-point scale with severe anosognosia defined specifically in terms of the patient's claim to have reached out and touched the examiner's hand, despite not doing so (1998, p. 28). Within two months of her cerebrovascular accident (CVA), CC's anosognosia had resolved. She acknowledged that she could not move her left arm and that she had not been able to do so for 'three months'. But, she did not acknowledge her earlier denial of her impairment.

They add that, when CC claimed that she was lifting her left arm, their impression was 'that she was really trying to do so and was truly convinced of her action' (p. 38). If this impression is correct then CC not only denied her paralysis in response to interview questions but also was concurrently unaware of her failure to move.

However, although concurrent unawareness of an impairment might be a factor leading to sincere denial of the impairment in an interview, the two are conceptually dissociable. It is possible that a patient might be concurrently *unaware* of her failure to move and yet might *acknowledge* the impairment in an interview after it had been demonstrated to her. It is also possible that a patient might be proprioceptively *aware* of her failure to move her arm and yet might *fail to acknowledge* her paralysis in an interview (perhaps as a result of forgetting the earlier proprioceptive experience). Indeed, Marcel, Tegnér and Nimmo-Smith (2004) report a double dissociation between concurrent unawareness of impairment (as assessed by self-evaluation of performance immediately after being asked to raise the arm or leg with vision precluded) and denial of impairment in an interview.

Unawareness and denial occurred together in patient CC. She was concurrently unaware of her paralysis *and* she failed to acknowledge it in interview. But in some way—'implicitly'—she *appreciated the consequences* of her impairment. For example, when CC was asked what score (out of ten) she would give to her own performance if she had to perform a unimanual action with her right hand or with her left hand (e.g. lift a glass with her right hand; lift a glass with her left hand) she gave high scores (8 or 9) for the right-handed actions and low scores (2 or 3) for the left-handed actions.

Other patients demonstrate the reverse dissociation. They explicitly acknowledge their impairment yet do not appreciate its consequences. Marcel and colleagues (2004) report several patients who, in response to interview questions, acknowledged that their left arm was paralysed yet who overestimated their ability to carry out bi-manual tasks such as tying a knot, clapping hands, or shuffling cards.

House and Hodges (1988) describe the case of an 89-year-old woman who suffered left-side paralysis following a right-hemisphere stroke. When examined six months after the stroke, she could not explain why she was in a nursing home and said that she could not walk because she had hurt her *right* foot. She acknowledged that her left arm was weak, rating the strength of her left hand and wrist as only two out of ten. But she still claimed that 'she would be able to walk, feed and dress herself unaided, and even drive a car although "the left side might be a bit awkward"' (1988, p. 114). When the examiner demonstrated to this patient that her arm was completely paralysed she gave even lower ratings for the strength of her left shoulder, elbow, wrist and hand. Indeed, her rating for the strength of her left hand and wrist was reduced to zero. But she still maintained that she could look after herself, insisting, for example, that she could walk upstairs unaided.

In summary, a patient is usually classified as anosognosic on the basis of denial of an impairment in an interview. In the case of anosognosia for hemiplegia, this explicit denial in the context of an interview may or may not co-occur with unawareness of failure to move the paralysed limbs at the time when the failure

occurs. And the explicit denial of paralysis may or may not be accompanied by lack of appreciation of the consequences of the impairment for the activities of daily life or for the likelihood of the patient's returning to work.

2. Theories of Anosognosia: Cognitive Impairments

Studies of large groups of patients provide information about the incidence of anosognosia and about correlations between anosognosia and other impairments. Smaller group studies and single-case studies allow more detailed investigation of associations and dissociations and provide evidence for or against claims that such-and-such an impairment is either a necessary or a sufficient condition for anosognosia. Further evidence about factors that may figure in the aetiology of anosognosia for hemiplegia is provided by studies of patients with epilepsy who undergo the Wada test in preparation for surgery to remove part of the temporal lobe of the brain. The Wada test involves injection of a barbiturate into one or the other carotid artery with the result that one hemisphere of the brain is selectively anaesthetised. During this procedure, subjects suffer weakness of the side of the body opposite to the injection.

2.1. Disorientation and Confusion

In an early study of 100 patients with hemiplegia (95 following a cerebrovascular accident—CVA), Nathanson and colleagues (Nathanson, Bergman and Gordon, 1952) found twenty-eight patients with anosognosia for their paralysis. All twenty-eight showed some degree of disorientation, particularly in time and place. Cutting (1978) observes that in three out of four studies conducted during the 1950s and 1960s (including the study by Nathanson and colleagues) every patient with anosognosia for hemiplegia showed disorientation, while in the remaining study the patients with anosognosia who were correctly oriented in time and place still showed a milder degree of confusion. In Cutting's own study of 100 patients with hemiplegia, examined within a few days of onset of their paralysis, nineteen out of twenty-eight patients with anosognosia for their left-side paralysis and all three patients with anosognosia for their right-side paralysis showed disorientation.

These findings and others (Anderson and Tranel, 1989; Wagner and Cushman, 1994) are consistent with the suggestion that disorientation or confusion may be a factor in some cases of anosognosia (Johnson, 2001). But they also show that anosognosia for hemiplegia can occur without disorientation, at least in patients with right-hemisphere damage. For, in Cutting's (1978) study, nine patients with anosognosia for their left-side paralysis were correctly oriented in time and place.

The possibility of anosognosia for hemiplegia without disorientation, confusion, or general intellectual impairment is confirmed by many subsequent studies. For example, Jehkonen and colleagues (Jehkonen, Ahonen, Dastidar, Laippala and Vilkki, 2000) found that anosognosia for hemiplegia could occur in patients who

scored in the normal range on the Orientation subtest of the Wechsler Memory Scale (WMS; Wechsler, 1945) and that, considered as a group, patients with anosognosia for their hemiplegia did not differ significantly on the Orientation subtest from patients who acknowledged their paralysis. In a study of thirty-four right-CVA patients, Berti, Làdavos and Della Corte (1996) assessed general intellectual capacity using a version of Raven's progressive matrices and found no difference on this measure between patients with and without anosognosia for their left-side motor impairments. Several studies have used the Mini-Mental State Examination (MMSE; Folstein, Folstein and McHugh, 1975) to assess cognitive function. The MMSE is a brief standardised assessment of orientation, attention, immediate and short-term recall, and language and it provides a screen for dementia. Small and Ellis (1996) found that only nine out of twenty patients with anosognosia for their hemiplegia scored below cut-off on the MMSE. And Marcel and colleagues (2004) found that patients with anosognosia for their motor impairments were no more likely to score below cut-off on the MMSE than those who acknowledged their impairments.

There are also reasons to question whether general cognitive impairments, even if they were present, could by themselves provide a complete explanation of anosognosia. It is not easy to explain, in terms of general cognitive impairments alone, why a patient would be anosognosic for one impairment but not for another. Yet such dissociations have been found. For example, Nathanson and colleagues (1952) note that patients with anosognosia for their hemiplegia generally did not deny other illnesses and that several patients acknowledged that they had heart disease while denying their paralysis. Bisiach and colleagues (1986) report patients with severe anosognosia for their visual field defects but no anosognosia or only mild anosognosia for their hemiplegia. Jehkonen and colleagues (2000) report a double dissociation between anosognosia for hemiplegia and anosognosia for unilateral neglect and Berti, Làdavos and Della Corte (1996) report a double dissociation between anosognosia for hemiplegia and anosognosia for unilateral neglect manifested in drawings (drawing neglect). There are even reports of patients with anosognosia for their paralysis of the left arm but not for their paralysis of the left leg or *vice versa* (Bisiach *et al.*, 1986; Berti, Làdavos and Della Corte, 1996).²

2.2. Intracarotid Barbiturate Injections

The results of studies of patients undergoing the Wada procedure provide an additional reason to doubt that a complete explanation of anosognosia for hemiplegia can be provided by appeal to disorientation, confusion, or general

² Bisiach and Geminiani (1991, p. 24) mention a second problem for accounts of anosognosia that appeal to general confusion or intellectual impairment; namely, the fact that anosognosia for left-side paralysis sometimes disappears after irrigation of the left ear with iced water or the right ear with warm water (Cappa, Sterzi, Vallar and Bisiach, 1987).

intellectual impairment. Because left-side injections produce not only right-side weakness but also language impairments, it is not possible to assess the results of both left- and right-side injections by asking patients questions at the time of their weakness. For this reason, many studies proceed by asking the patients questions after the effects of the barbiturate have resolved, so that neither weakness nor language impairment is present at the time of the enquiry. A typical finding from such studies is that, in response to these questions, many patients fail to acknowledge their earlier weakness. But this denial of weakness cannot be explained in terms of disorientation, confusion, or general intellectual impairment since the effects of the injection have resolved before questioning begins (Heilman, Barrett and Adair, 1998).

For example, in a study of thirty-one epilepsy patients by Carpenter and colleagues (Carpenter, Berti, Oxbury, Molyneux, Bisiach and Oxbury, 1995), twenty-seven patients failed to recall having had left-arm weakness when questioned after a right-side barbiturate injection and twelve patients denied having had right-arm weakness when questioned after a left-side injection. The interview with one patient, thirteen minutes after a right-side injection, was as follows (1995, p. 248):

Examiner: Did you ever have any weakness of the arms or legs?

Patient: No.

E: Were you able to keep the arms up in the air all the while?

P: Yes.

E: Do you think there was any difference between what happened to the right arm and what happened to the left arm?

P: I don't think so.

E: No? If I suggested to you that you might have been weak in one arm would you agree with me or disagree?

P: Disagree.

E: Disagree. Right. So I cannot persuade you that at any time there was a weakness of the left arm, for instance?

P: No.

This patient clearly denied her earlier left-arm weakness. But Carpenter and colleagues state explicitly that the mean IQ of the thirty-one patients was in the normal range and that none was confused at the time of questioning about their left-arm weakness (1995, p. 250).

2.3. Memory

Nathanson and colleagues (1952) did not find clear evidence of memory problems in patients with anosognosia for hemiplegia and Cutting (1978) found that only four of the twenty-eight patients with anosognosia for their left-side paralysis had memory impairments. But, despite these unpromising findings, it is sometimes

suggested that memory disorders play a role in the aetiology of anosognosia (Cocchini, Beschin and Della Sala, 2002; see below, section 7.2.). Indeed, it is intuitively very plausible that failure to remember relevant evidence would impact negatively on a patient's ability to make an accurate assessment of the nature and extent of his or her motor impairments or other illness.

However, recent studies support the findings of Nathanson and colleagues (1952) and Cutting (1978) that memory disorders—particularly disorders of short-term verbal memory as assessed, for example, by digit span or long-term verbal memory as assessed by recall of a short story—do not play an essential role in the aetiology of anosognosia (Berti, Làdavas and Della Corte, 1996).

Studies in which patients are asked questions after the effects of a barbiturate injection have resolved also raise issues about the role of memory impairments in denial of paralysis. For these studies leave open the possibility that a patient's denial may be the result of amnesia induced by the barbiturate rather than a manifestation of lack of knowledge of the weakness at the time when it occurred. These issues have been investigated further by asking patients questions about their weakness both during and after the Wada procedure.

In a study by Adair and colleagues (Adair, Gilmore, Fennell, Gold and Heilman, 1995), patients were questioned while they were affected by a right-side injection of the short-acting barbiturate methohexital and again after the effects had resolved. The results suggest that, at least when this barbiturate is used, post-procedure denial does not depend on memory failure but is a good indicator of lack of knowledge of left-arm weakness at the time when it occurred (see also Heilman, Barrett and Adair, 1998).

From the study by Carpenter and colleagues (1995), using a longer-acting barbiturate, a more complicated picture emerges. Five out of nine patients who were questioned early after right-side injection—while the effects of the barbiturate were still present—denied their left-arm paralysis. The interview with one patient was as follows (1995, p. 247):

Examiner: Can you lift your arms up in the air now?

Patient: [lifts right arm only]

E: Is there anything the matter with your arms?

P: No.

E: Can you lift this one up in the air? [E points to and then touches left arm]

P: [makes no response]

E: Fine. Is the left one all right?

P: Yes.

E: Nothing the matter with the left one?

P: No.

E: Why isn't it up in the air?

P: So sorry.

E: Can you lift it up?

P: Yes [still not moving the left arm, but pushing the right arm further up].

These five patients also denied their weakness when they were questioned about fifteen minutes after the injection, when the effects had resolved.

But also, three of the four patients who acknowledged their left-arm paralysis at the time when it occurred *failed to recall it* when questioned later, even though the barbiturate did not induce a general memory impairment in these patients. The upshot is (p. 249): '[I]n some patients failure to recall left arm weakness can be attributed to unawareness at the time. In others it seems to be due to a specific memory deficit'. Furthermore, Carpenter and colleagues suggest that the relevant memory function may be subserved by structures in the right temporal lobe of the brain.

3. Theories of Anosognosia: Feedback and Feedforward

We have considered a number of problems for theories of anosognosia that appeal to cognitive impairments. Anosognosia can occur without disorientation, confusion, or general intellectual impairment; and general cognitive impairments cannot account for anosognosia for one impairment in the absence of anosognosia for other impairments. The issue that concerns us now is that the patient's concurrent unawareness of failure to move a paralysed limb is plausibly a factor in at least some cases of anosognosia for hemiplegia, yet theories that appeal only to cognitive impairments do nothing to explain this concurrent unawareness. Given this fact, it is natural to postulate a lack of sensory information from, or a failure to direct attention towards, the paralysed side of the body.

3.1. Feedback: Sensory Loss and Unilateral Neglect

Cutting (1978) found that sensory loss was more common amongst patients with anosognosia for their hemiplegia than amongst patients with a normal attitude towards their paralysis. But the fact that some patients with anosognosia had no sensory loss, while some patients without anosognosia had suffered a sensory loss, shows that sensory loss does not provide a full explanation of the occurrence of anosognosia for hemiplegia. Bisiach and colleagues (1986) demonstrated that anosognosia for motor impairments was doubly dissociated from somatosensory impairment, although anosognosia in the absence of sensory loss was relatively rare. Similarly, Berti and colleagues (1996) found that, while sensory loss was common amongst patients with anosognosia for their hemiplegia, two out of nine patients with anosognosia for their hemiplegia were able to detect tactile stimuli applied to their paralysed left hand.

The studies mentioned so far tested touch sensation but not proprioception. But Small and Ellis (1996) tested joint position sense in twenty patients with anosognosia for hemiplegia and found that proprioception was intact in ten patients. And Marcel and colleagues (2004) tested both touch and proprioception

in sixty-four patients with hemiplegia following stroke and found that those with sensory loss were no more likely than those without to be anosognosic for their paralysis.

Anosognosia for hemiplegia often co-occurs with unilateral neglect. Patients with neglect fail to respond to stimuli presented on the side opposite to their lesion—the *contralateral* side. Unilateral neglect is not a unitary condition and one important distinction is between extrapersonal neglect and personal neglect. In *extrapersonal neglect*, patients neglect contralateral stimuli external to their body; for example, neglecting to copy the contralateral side of a picture or neglecting to cross out lines or other targets on the contralateral side of a sheet of paper. In *personal neglect*, patients neglect the contralateral side of their body; for example, combing their hair only on the ipsilateral side (that is, on the same side as the lesion).

It is intuitively plausible that, following right-hemisphere damage, unilateral neglect could contribute to concurrent unawareness of left-side paralysis. But the results from several studies suggest that unilateral neglect is neither necessary nor sufficient for anosognosia for hemiplegia. For example, Bisiach and colleagues (1986) demonstrated a double dissociation between anosognosia for motor impairments and extrapersonal neglect. And Small and Ellis (1996) found that two of the twenty patients with anosognosia for their hemiplegia did not have extrapersonal neglect—indeed, these two patients had neither extrapersonal neglect nor proprioceptive loss. So extrapersonal neglect was found not to be necessary for anosognosia for hemiplegia and, since the Small and Ellis study included a control group of patients with hemiplegia and neglect but no anosognosia, neglect was clearly not sufficient for anosognosia either.

In many studies, personal neglect is assessed by a test in which the patient is asked to use the ipsilateral hand to reach across the midline of the body and touch or grasp the contralateral hand (Bisiach *et al.*, 1986). Personal neglect assessed by Bisiach's test is sometimes referred to as *asomatognosia*. Single and double dissociations between anosognosia for hemiplegia and asomatognosia have been demonstrated (Cutting, 1978; Bisiach *et al.*, 1986; Berti, Làdavas and Della Corte, 1996; Marcel *et al.*, 2004). Assessment of personal neglect by tests involving combing the hair, using a razor or powder compact, and putting on spectacles (Zoccolotti and Judica, 1991) is less common in studies of anosognosia for hemiplegia. But in a large study by Appelros and colleagues (Appelros, Karlsson, Seiger and Nydevik, 2002), forty-eight patients showed signs of anosognosia for hemiplegia while only nineteen demonstrated personal neglect on the tests just mentioned and twenty-one on either those tests or Bisiach's test.

In summary, theories of anosognosia that appeal to somatosensory loss or to extrapersonal or personal neglect have in common the aim of explaining denial of hemiplegia in terms of impaired feedback from the paralysed side of the body. But the findings from many studies suggest that anosognosia for hemiplegia cannot be adequately explained in these terms.

3.2. Feedforward: Prediction and Comparators

As an alternative to all feedback theories of anosognosia, Heilman (1991) has proposed a feedforward theory—so-called because it appeals to a malfunction in a comparator system that includes a forward model. The basic ideas about efference copies and forward models are familiar. The subject's representation of an *intended state* leads to the generation of motor instructions and these have two consequences. The motor instructions are sent to the muscles and actual movement results. Feedback from this movement allows the generation of a representation of the *actual state*. But, in addition, predictors use the motor instructions (the efference copy) to generate a representation of the *predicted state*. This is the forward model. Comparisons amongst these three representations are important for motor control and for our awareness of our actions and their consequences.

For example, an attempt to move the left arm would normally result in the forward model representing movement. If the arm were paralysed, so that no actual movement ensued, then there would normally be a mismatch between the predicted state and the actual state, and the subject would become aware of his or her paralysis. Thus (Heilman, Barrett and Adair, 1998, p. 1908): 'weakness is detected when there is a mismatch between the expectancy of movement and the perception of movement'. However, if the systems that generate motor instructions and send copies of these instructions to the predictors were damaged, then it might happen that no movement would even be predicted. In that case, there would be no mismatch between the predicted state and the actual state and so no experience of being paralysed. As Heilman puts it (1991, p. 59): 'if the intentional-preparatory systems are damaged along with the motor-effector system, there is no mismatch and one does not recognize paralysis'.

Some support for Heilman's feedforward hypothesis is provided by an electrophysiological investigation in which subjects were asked to squeeze their left or right hand forcefully while contraction of the pectoral muscles was measured by electrodes placed on the skin (Gold, Adair, Jacobs and Heilman, 1994). In normal subjects, squeezing either hand activated the muscles on both sides. This was also true for patients with hemiplegia but without anosognosia. In contrast, in the case of a patient with anosognosia for his left hemiplegia, squeezing with his right hand increased activation in the muscles on both sides, but when he was asked to squeeze with his left hand there was no increase in muscle activation over the resting condition.

Unfortunately, studies of anosognosia often do not include tests that would directly address Heilman's feedforward hypothesis and there does not seem to be any consensus that damage to intentional-preparatory systems is a factor in every case of anosognosia for hemiplegia.

4. Levine's Discovery Theory

No feedback or feedforward problem has been shown to be either necessary or sufficient for anosognosia for hemiplegia. But consider, for a moment, patients in

whom some such problem produces concurrent unawareness of particular failures to move their paralysed limbs. In a vivid passage, Marcel and colleagues argue that such concurrent unawareness of motoric failure does not provide an adequate explanation of denial of paralysis in response to interview questions (2004, p. 35):

... it is not just that they fail *motorically*. The consequence of such [motoric] failures is that, in trying to get out of bed to go to the toilet or to lift an object, they fall over or incur a similar accident, often lying helpless or hurting themselves. Unless such patients have some other problem, it is unlikely that they are unaware of these incidents, ... or that they rapidly forget them, or that they hallucinate the success of the intended *action* (as opposed to the movement).

Even if, as a result of loss of proprioceptive feedback, unilateral neglect, or failure of a comparator to generate a mismatch signal, a patient is not concurrently aware of failure to move an arm or leg still, in many or most cases, patients have available to them a mass of evidence of their paralysis. Something more than concurrent unawareness of motoric failure is needed in order to explain why patients with anosognosia for their hemiplegia do not make appropriate use of this mass of evidence.

4.1. Degrees of Difficulty of Discovery

Levine (1990) argues that sensory impairments, such as blindness or visual field defects, are 'never phenomenally immediate' (1990, p. 234) but need to be discovered 'by observation and inference' (*ibid.*). Thus, for example, there is no canonical experience of blindness. It is true that people who are suddenly struck blind during daylight commonly report visual blackness. But visual blackness could have many explanations and recognition that one is blind requires adoption of one explanation of the experience rather than another. Discovery that one is blind is primarily a cognitive rather than a sensory matter. But the functional consequences of blindness are so severe that the testing, observation and inference that are required are not very demanding. So cognitively normal subjects do not remain ignorant of their blindness.

As in the case of blindness, so also and a fortiori there is no canonical experience of having a visual field defect. Furthermore, a host of automatic mechanisms, including perceptual completion and eye movements, reduce the functional consequences of visual field defects and make discovery of the impairment more difficult than discovery of blindness. As a result, according to Levine's discovery theory, anosognosia for hemianopia can occur even in cognitively normal individuals.

4.2. Discovering Hemiplegia: Sensory and Cognitive Factors

The idea of different degrees of difficulty of discovery can also be applied to motor impairments. Levine argues that, given a somatosensory loss, and particularly given a

loss of proprioceptive feedback, paralysis is not phenomenally immediate. Knowledge of paralysis requires a process of discovery. The observation and inference that are required are not especially demanding for cognitively intact individuals (1990, p. 252): 'Loss of the effective use of even one limb creates major functional limitations that are not quickly and automatically compensated'. Anosognosia for hemiplegia arises when somatosensory loss is accompanied by additional defects that impair observation and inference.

Levine, Calvanio and Rinn (1991) compared two groups of patients following a right-hemisphere stroke, six with severe anosognosia for their hemiplegia lasting for at least a month and seven whose anosognosia had lasted for a few days at most. The findings were consistent with the claim that somatosensory loss is *necessary* but *not sufficient* for anosognosia. For all the patients with anosognosia and two patients without anosognosia had severe somatosensory deficits of both touch and proprioception. In this study, all the patients with anosognosia for their hemiplegia also had severe unilateral neglect. But Levine and colleagues argue that sensory loss and unilateral neglect together were still *not sufficient* to account for anosognosia, since one of the patients without anosognosia had both severe somatosensory deficits and severe neglect.

Considered as a group, the patients with anosognosia performed significantly worse than the patients without anosognosia on a number of tests of attention, memory, intelligence, and mental flexibility. For example, only one of the patients with anosognosia was able to add by 3s from 1 to 40 within 30 seconds. Overall, these results were broadly consistent with the claim that *both* somatosensory loss and cognitive impairments are required for anosognosia for hemiplegia. But no particular cognitive impairment emerged as pivotal and Levine and colleagues (1991) report that the most striking aspect of the neuropsychological profiles of the patients with anosognosia for hemiplegia was their poor mental organisation.

4.3. Challenges to the Discovery Theory of Anosognosia

Neither sensory loss nor cognitive impairment has been shown to be necessary for the occurrence of anosognosia for hemiplegia. As we have seen (section 3.1.), the findings from many studies challenge Levine's claim that impaired feedback from the paralysed side of the body is essential for anosognosia for hemiplegia. The suggestion that cognitive impairments always play a role in the aetiology of anosognosia has also been challenged (section 2). Indeed, as against Levine's discovery theory, Small and Ellis (1996) note that only four of twenty patients with anosognosia showed both proprioceptive loss (assessed by a test of joint position sense) and defective cognition (assessed by the MMSE). Furthermore, five of the twenty patients showed neither sensory loss nor cognitive impairment on these measures and two of these patients were also assessed as not having extrapersonal neglect.

5. The Generic Two-Factor Theory of Delusions

We have described anosognosia for hemiplegia and reviewed some of the existing theories that try to explain it. In recent papers on anosognosia for hemiplegia, a recurrent theme is that none of the existing theories is fully adequate: 'No theory appears to account for the phenomenon in all its forms' (Venneri and Shanks, 2004, p. 231); 'no single-factor or combined-factor explanation is adequate to account for anosognosia for plegia in all the patients in the sample' (Marcel, Tegnér and Nimmo-Smith, 2004, p. 31). Our proposal is to approach anosognosia for hemiplegia by way of a generic two-factor theory of delusions. For, as we mentioned at the outset, a patient's belief that he or she is able to move a limb that is, in reality, paralysed seems to count as a delusion by the usual definitions.

The two-factor theory is offered, in the first instance, as a theory of monothematic delusions of neuropsychological origin. A somewhat familiar version of the theory agrees in part, but also disagrees in part, with Maher's (1988, 1999) claim that delusions are false beliefs that arise as normal responses to *unusual experiences*.³ The theory's point of agreement with Maher is that unusual experiences figure in the aetiology of delusions. The point of disagreement is over Maher's claim that the unusual experience is normally sufficient to produce the delusion. According to this familiar version of the two-factor theory an unusual experience is the first factor in the aetiology of a delusion, but there must also be a second factor.

The generic version of the two-factor theory, in contrast, does not involve any commitment to Maher's claim that unusual experiences always figure in the aetiology of delusions. Maher himself says (1999, p. 551): 'The origins of anomalous experience may lie in a broad band of neuropsychological anomalies'. Since the generic two-factor theory is offered as a theory of delusions of neuropsychological origin, we focus on neuropsychological anomalies themselves, rather than on the unusual experiences to which they may or may not give rise.

5.1. The First Factor

According to the generic two-factor theory, the first factor in the aetiology of a delusion is a neuropsychological anomaly. The example of a monothematic delusion that is most frequently discussed in the literature is the *Capgras delusion*, in which the patient claims that someone, often the patient's spouse or another close relative, has been replaced by an impostor. In this case, there is a widely accepted account of the neuropsychological anomaly that is supposed to be operative (Ellis and Young, 1990); namely, a disruption of the connection between the face recognition system and the autonomic nervous system. This account could be correct whether or not the Capgras patient's experience of the spouse differs

³ See Davies and Coltheart, 2000; Langdon and Coltheart, 2000; Davies, Coltheart, Langdon and Breen, 2001. We consider experiences to be events in the *conscious* mental life of the subject.

affectively from a neuropsychologically intact subject's experience of his or her spouse.

Breen and colleagues describe two cases of *mirrored-self misidentification*, in which the patient claims that the person that he sees in the mirror is not him (Breen, Caine, Coltheart, Hendy and Roberts, 2000; Breen, Caine and Coltheart, 2001). One of these patients, FE, had a disorder of face processing evidenced by poor performance on a face-matching task. The other patient, TH, did not. His performance on face-matching tasks was in the normal range and he was able to identify both famous faces and family faces.

Patient TH showed a severely impaired ability to interpret reflected space. While he was facing a mirror, objects were held up behind his left or right shoulder. He was able to identify each of these objects. But when he was asked to take the object in his hand, he unflinchingly (in twenty trials) reached towards the mirror, scratching on its surface or trying to reach behind the glass. Patient TH suffered from mirror agnosia. He had not lost his encyclopaedic knowledge about mirrors but he had lost the ability to interact fluently with mirrors. Presumably, TH's neuropsychological anomaly was impaired access to a complex set of visuo-motor transformations that normally allow subjects to reach and grasp appropriately in reflected space. Thus, the neuropsychological anomaly that is the first factor varies, not only from delusion to delusion, but also from patient to patient with the same delusion.

5.2. From Neuropsychological Anomaly to Delusional Candidate Belief

The first factor is but one step along a path that leads to the delusional hypothesis or candidate belief. There are many questions to be settled before we can give a detailed account of the role of the first factor in generating the delusional hypothesis and lending it salience and credibility—and it may be that this role varies from case to case. One question is whether an unusual experience figures early on the path to the delusional candidate belief, or late, very late, or not at all.

In the case of mirrored-self misidentification, when the first factor is a disorder of face processing (patient FE), it seems plausible that the patient's experience of his own face seen in a mirror was unusual to the extent that the way that the person in the mirror looked now was different from the way he remembered himself looking in earlier years. When the first factor is mirror agnosia (patient TH) it seems plausible that when the patient looked in the mirror he saw a man as being in the space behind the glass. But it is not obvious that the patient's experience was unusual since this seems to be an equally correct description of a neuropsychologically intact subject's experience of looking at himself in a mirror.

However, even if TH's mirror agnosia did not give rise to an unusual experience at an early stage of the route towards the delusional candidate belief, 'The man I see in the mirror is not me', it is still reasonable to suppose that the neuropsychological anomaly figured in the aetiology of that candidate belief. That is, the neuropsychological anomaly was a factor in generating the delusional hypothesis and in

lending it salience and credibility. Although we offer no detailed account of the processes involved, we suggest that patient TH, as a result of his mirror agnosia, interpreted his mirrored-self not as himself, but as another man in a space behind the glass. This hypothesis, generated and rendered salient as a result of the neuropsychological anomaly, suffused TH's experience of looking in the mirror so that it became the experience of *seeing* the man in the mirror *as* someone other than himself—a 'dead ringer' for himself, as TH put it (Breen, Caine and Coltheart, 2001, p. 241).

A second, and related, question about the role of the first factor concerns the extent to which the first factor *determines the content* of the delusional hypothesis and the extent to which other influences may be at work. In the case of patient FE, it is plausible that his strategies for explaining his unusual experience when looking in the mirror played some role in the aetiology of the delusional candidate belief, 'The man I see in the mirror is not me'. In the case of patient TH, it seems more plausible that the form of the delusional candidate belief was mainly determined by unconscious processes—though we have not offered any detailed account of this.

In general, the path from the first factor to the delusional candidate belief might lie mainly at the personal level or mainly at the subpersonal level. If the path lies mainly at the personal level, the typical pattern would be that the neuropsychological anomaly itself contributes relatively little to the specific form of the delusional hypothesis. The individual subject's beliefs, preferences, concerns, reasoning biases and explanatory strategies would play a substantial role in shaping the content of the hypothesis and the hypothesis would achieve a measure of *prima facie* credibility because of its explanatory potential. If, on the other hand, the path lies mainly at the subpersonal level, then the typical pattern would be that the neuropsychological anomaly itself is the primary determinant of the specific form of the delusional hypothesis. The hypothesis would achieve *prima facie* credibility because it would be presented as the content of, or as suffusing, a perceptual experience.

We shall not attempt to choose between these options here and the generic two-factor theory is officially neutral between them.

5.3. The Second Factor

The argument for a second factor in the aetiology of delusions is that the neuropsychological literature provides many examples of subjects who have the kind of neuropsychological anomaly that plausibly figures in the aetiology of a delusion yet who are not delusional.

Both normally, and normatively, the first factor—even though it gives rise to an apparently salient and *prima facie* credible hypothesis or candidate belief—is not sufficient, by itself, to account for the delusion. To arrive at a delusional candidate belief is not yet to suffer from a delusion because the delusional hypothesis normally can be, and normatively should be, rejected. A salient but implausible hypothesis—'My wife has been replaced by an impostor', 'The man I see in the

mirror is not me'—may gather some prima facie credibility because of its explanatory potential or because it suffuses or is encoded in one's perceptual experience. But that does not explain why it is adopted and tenaciously maintained as a belief. There must be a second factor in the aetiology of a delusion.

According to the generic two-factor theory of delusions, the second factor is an impairment in belief evaluation or assessment and so it does its work *after* the generation of the delusional hypothesis or candidate belief (and not along the route from the first factor to the candidate belief). Without a delusional candidate belief, the second factor would not, by itself, produce a delusion. Beyond this, the generic two-factor theory is officially non-committal about the nature of the second factor.

6. Anosognosia and the Two-Factor Theory

Our proposal to treat anosognosia for hemiplegia as a delusion falling within the scope of the generic two-factor theory of delusions faces two immediate problems. The first problem is that the intuitive structure of anosognosia is rather different from the structure of more familiar delusions such as the Capgras delusion. The second problem is that the two-factor theory of delusions, as applied to anosognosia for hemiplegia, bears some resemblance to Levine's discovery theory and so it faces some of the same challenges as Levine's theory.

6.1. Two Structures Compared

In familiar delusions, a bizarrely false hypothesis achieves some salience and prima facie credibility as the result of a neuropsychological anomaly (the first factor). Despite the *DSM-IV* definition of delusion, it is not clear that there is always evidence—in the ordinary sense of that term—that goes against the hypothesis. The Capgras hypothesis, for example, might be regarded as similar to sceptical hypotheses, such as Descartes's evil demon hypothesis, in being 'unfalsifiable'. But even if there is no evidence that incontrovertibly goes against it, the Capgras hypothesis is still intuitively exotic and is implausible in the light of the subject's other beliefs. So adopting the hypothesis as a belief would call for substantial revision to the subject's antecedent web of beliefs. Yet, despite the prima facie credibility that is attached to the hypothesis as a result of the first factor, the antecedent web of beliefs is not updated. The hypothesis, even when it is adopted as a belief, remains quarantined; the delusion is circumscribed. Since the hypothesis is implausible in the light of the antecedent web of beliefs and those beliefs are not updated, the remaining way for the subject to maintain overall coherence and consistency would be to reject the hypothesis. But, because of the second factor, the hypothesis is not rejected; instead, it is adopted and maintained as a belief.

In the case of anosognosia for hemiplegia, the structure is different. The belief that there is no impairment is false—newly false as a result of the subject's recent

paralysis. But it is not bizarre or implausible in the light of the subject's antecedent web of beliefs. On the contrary, it is part of that web. The subject has always believed that he could use his left arm and leg. However, even though the belief that there is no impairment is not implausible in the light of the subject's antecedent beliefs, there is evidence that goes against it—evidence newly acquired, since the onset of the paralysis. This evidence calls for substantial revision to the subject's antecedent web of beliefs and, in particular, it calls for the rejection of the old, now false, belief. The subject ought to acknowledge his impairment and accept that he is paralysed. But the old belief is not rejected; instead it is maintained in the face of all the evidence.

In summary, the structure of anosognosia for hemiplegia is different from the structure of more familiar delusions in two striking ways. First, the delusional belief is not new and exotic but old and commonplace. Second, the reason why the delusional belief ought to be rejected is not because it is implausible but because a mass of evidence goes against it. These two differences impose constraints on the project of bringing anosognosia within the scope of the two-factor theory of delusions. If a neuropsychological anomaly is the first factor in the aetiology of anosognosia for hemiplegia, the primary role of this factor is not to render a bizarre hypothesis salient and somewhat credible, but to allow a familiar hypothesis to retain a measure of credibility in changed circumstances. And if there is a second factor in the aetiology of anosognosia for hemiplegia then its role is to explain why a belief is maintained, not just in the face of implausibility, but in the face of experienced evidence that clearly goes against it.

6.2. A Common Structure

The constraints that are imposed by the differences between anosognosia for hemiplegia and more familiar delusions are not trivial. Nevertheless, our proposal is that anosognosia for hemiplegia can be brought within the scope of the generic two-factor theory of delusions.

To see that this is at least somewhat plausible, consider anosognosia for hemiplegia alongside mirrored-self misidentification. In each case there is a proposition *P* that the subject normatively ought to believe—in the case of mirrored-self misidentification, 'The man I see in the mirror is me', and in the case of anosognosia, 'My left arm is paralysed'. In each case the subject actually believes the opposite, the false proposition not-*P*—in the case of mirrored-self misidentification, 'The man I see in the mirror is not me', and in the case of anosognosia, 'My left arm is not paralysed; I can move it just as I have always been able to'. In one case not-*P* is new and exotic and in the other case it is old and commonplace. But in both cases it is plausible that a neuropsychological anomaly is one factor in the aetiology of the false belief. In the case of mirrored-self misidentification, the neuropsychological anomaly might be impaired face processing (as for patient FE) or mirror agnosia (as for patient TH). In the case of anosognosia for hemiplegia, the neuropsychological anomaly might be impaired proprioceptive

feedback so that the patient is not concurrently aware of his or her failure to move. Furthermore, the familiar form of argument for a second factor is available in the case of anosognosia for hemiplegia just as in the case of more familiar delusions. There are cases in which the first factor is present but the delusion is absent.⁴

Our proposal for bringing cases of anosognosia for hemiplegia within the scope of the generic two-factor theory of delusions is that the first factor would be a neuropsychological anomaly impairing the patient's awareness of his or her paralysis while the second factor would impair the patient's ability to make appropriate use of evidence and other information to update his or her beliefs. The second factor would somehow prevent the patient from rejecting the old and now false belief and acknowledging his or her impairment.

6.3. Similarity to Levine's Discovery Theory

Levine's discovery theory of anosognosia for hemiplegia (Levine, 1990; Levine, Calvanio and Rinn, 1991) is a kind of two-factor theory. The first factor is supposed to be a somatosensory deficit whose role is to prevent a kind of experience that would provide the patient with immediate or concurrent awareness of his or her paralysis.

Levine actually groups together all factors other than sensory loss and assigns them the role of making discovery more difficult by impairing processes of observation and inference. So he does not conceive the second factor as specifically an impairment in belief evaluation. But he does predict that 'cognitive defects impairing the ability to observe and infer will distinguish those individuals with sensory loss and paralysis who are anosognosic from those who discover and become aware of their paralysis' (1990, p. 254). And he remarks that intellectual impairments will play an important role because '[t]he anosognosic is unable to assimilate information from a variety of sources to form a consistent and accurate judgement of the reality of his paralysis' (*ibid.*).

In summary, Levine's discovery theory is similar to a version of the two-factor theory of delusions, as applied to anosognosia for hemiplegia, but the discovery theory takes on a specific commitment concerning the nature of the first factor: it is a somatosensory deficit. The results of studies reviewed earlier show that anosognosia for hemiplegia can occur without somatosensory loss (section 3.1.) and without disorientation, confusion, or general intellectual impairment (section 2.1.) and that some patients with anosognosia for their hemiplegia show *neither* proprioceptive loss *nor* defective cognition. These challenges to the discovery theory have to be addressed by anyone proposing to apply the two-factor theory of delusions to anosognosia for hemiplegia.

⁴ Marcel and colleagues (2004) report patients who are concurrently unaware of their failure to move a paralysed limb yet who acknowledge their paralysis in an interview. Of course, the argument for a second factor would have to be considered anew if a different neuropsychological anomaly were proposed as the first factor.

6.4. The Challenges Addressed

So far as the first factor is concerned, the generic two-factor theory of delusions is less open to challenge than the discovery theory because it does not take on any specific commitment as to the nature of the first factor. The two-factor theory allows that the first factor may vary from patient to patient with the same delusion. All that is required of the first factor is that it should be a neuro-psychological anomaly that impairs the patient's awareness of his or her paralysis. Somatosensory loss and unilateral neglect would be candidates, as would damage to intentional-preparatory systems (Heilman, 1991) or even a specific memory deficit that may be associated with damage to the right temporal lobe (Carpenter *et al.*, 1995).

So far as the second factor is concerned, there are issues of assessment to be raised. The claim that anosognosia for hemiplegia can occur without cognitive impairments is often based on the results of a very small number of tests and there is a clear risk that cognitive impairments that could impact on belief evaluation may be underestimated. For example, patients may suffer from impairments in components of memory despite performance in the normal range on tests such as Digit Span and Story Recall. And the use of a test like MMSE that screens for dementia does not provide any detailed assessment of cognitive functions such as sustained attention or working memory that seem especially relevant in cases where a subject 'is unable to assimilate information from a variety of sources to form a consistent and accurate judgement' (Levine, 1990, p. 254).

7. Cognitive Impairments and the Second Factor

The impairment in belief evaluation that is the second factor in the aetiology of delusions can be described—albeit rather uninformatively—as a loss of the ability to reject a candidate for belief on the grounds of its implausibility and its inconsistency with everything else that the person knows. A delusional patient adopts and maintains as a belief a hypothesis that should have been rejected. In short, the patient makes an error. But, according to the two-factor theory, adoption of a delusional belief is not only a departure from what is normatively correct but also a departure from what is normally the case.

When we apply the two-factor theory of delusions to the case of anosognosia for hemiplegia, this aspect of the theory is challenged by the results of studies that purport to demonstrate that anosognosia for hemiplegia can occur without any departure from cognitive normality. As we have just seen, we can respond to that challenge by pointing out that these studies may very well underestimate cognitive impairments. But it would be useful to be able to substantiate that response by demonstrating relevant cognitive impairments in subjects who are not disoriented or confused, who are not grossly intellectually impaired, and who score in the normal range on MMSE, Digit Span and Story Recall, for example.

7.1. A Study of Nine Patients with Persisting Unilateral Neglect

In order to indicate the kinds of results that a detailed investigation of cognitive functioning in a single case or a small group might yield, we very briefly describe some of the findings of a study conducted by Anne Aimola Davies (Aimola, 1999). The subjects were nine patients (age 41 to 63 years) with unilateral neglect persisting at least three months following a unilateral CVA. Eight of the patients had right-hemisphere damage and one patient had left-hemisphere damage. All of the patients demonstrated severe motor impairments in the acute phase following their CVA. Anosognosia was assessed by interview and scored on the four-point scale that was described earlier (section 1). Five patients demonstrated moderate or severe anosognosia for their hemiplegia. Two patients, including the patient with left-hemisphere damage, frankly acknowledged their hemiplegia and two other patients demonstrated only mild anosognosia, acknowledging their hemiplegia when asked specifically about it. All the patients showed both extrapersonal neglect and personal neglect but the severity of their neglect (ranging from mild to severe) was not a good predictor of whether or not they would have anosognosia for their hemiplegia.

A detailed neuropsychological assessment of the nine patients was conducted, including tests of orientation, autobiographical, visual and verbal memory, sustained attention, and working memory.⁵ There was no evidence of disorientation or confusion. All the patients passed the Information and Orientation subtest of the Wechsler Memory Scale–Revised (WMS-R; Wechsler, 1987) without difficulty (range of 12 to 14; maximum = 14).

The patients did not demonstrate difficulties with memory for people and incidents in their past and they all scored in the normal range on the Digit Span Forwards and Logical Memory (Story Recall) subtests of the WMS-R. However, four patients demonstrated serious problems on less structured tests of verbal memory or tests of visual memory (Doors and People Test; Baddeley, Emslie and Nimmo-Smith, 1994), although these memory problems were not a good predictor of whether the patients would have anosognosia for their hemiplegia. A different but overlapping group of five patients demonstrated problems on tests of sustained attention (Test of Everyday Attention; Robertson, Ward, Ridgeway and Nimmo-Smith, 1994).⁶ But once again, these problems did not distinguish the patients with moderate or severe anosognosia for their hemiplegia from the others.

While memory involves the preservation of information, working memory involves both storage and manipulation of information. In many

⁵ For details of the neuropsychological assessment and structural MRI, see Aimola, 1999; Aimola Davies, Davies and Ogden, forthcoming; Maguire and Ogden, 2002.

⁶ Tasks that assess sustained attention or vigilance typically require the patient to monitor the presentation of intermittent sensory targets (visual or auditory) that may occur amongst a greater number of non-targets (distractors). Sustained attention has been associated with persisting unilateral neglect (Robertson, 2001) and with poor recovery from hemiplegia (Robertson, Ridgeway, Greenfield and Parr, 1997).

cases, performing the correct manipulation requires retaining information about serial order. Thus, a simple test of working memory is Digit Span Backwards, in which the subject is required to reverse the order of a series of digits presented by the examiner. Only one of the nine patients demonstrated a working memory problem on this test. The formal assessment of working memory also included more demanding tasks (Test of Everyday Attention) and the results indicated that four patients had working memory problems. These four patients all had moderate or severe anosognosia for their hemiplegia. The remaining four patients tested showed no working memory problems and either no anosognosia or only mild anosognosia for their hemiplegia. (One patient with severe anosognosia was not administered the full set of working memory tasks.)

The findings of this study clearly demonstrate that patients who, by the standards of many published studies, would be classified as having no cognitive impairments may, in reality, depart from cognitive normality in respect of their memory, sustained attention, and working memory. It remains to show, of course, that cognitive impairments of these kinds could be implicated in the second factor in the aetiology of anosognosia for hemiplegia. But it is surely plausible that working memory, for example, which involves the retention and manipulation of information, should have a role in 'assimilat[ing] information from a variety of sources to form a consistent and accurate judgement' (Levine, 1990, p. 254).

7.2. A Case Study: Amnesia as a Second Factor?

We have suggested that, in some cases of anosognosia for hemiplegia, a specific memory deficit that may be associated with damage to the right temporal lobe could play the role of the first factor. It would not, strictly speaking, impair the patient's concurrent awareness of failure to move a paralysed limb. But it would prevent concurrent awareness of paralysis from influencing the patient's more lasting beliefs about his or her motor abilities and would, to that extent, allow the familiar hypothesis that the patient can move his or her limbs to retain a measure of credibility.

A first factor is not adequate, by itself, to explain denial of paralysis in response to interview questions. For a mass of other evidence of paralysis is usually available to the patient. But more general memory impairments might enter the picture as the second factor. For, in order to make appropriate use of evidence to evaluate and update beliefs, the patient needs to be able to remember that evidence. Such a role for memory problems in the explanation of some cases of anosognosia for hemiplegia is suggested by a single-case study reported by Cocchini and colleagues (2002).

The patient, NS, was a 27-year-old male with left hemiplegia and anosognosia for his hemiplegia persisting a year after a severe closed head injury. The following is a short extract from an interview in which his anosognosia is clearly expressed (2002, p. 2031):

Neuropsychologist: How are you?

NS: I'm fine, really fine.

N: Why are you in a wheelchair?

NS: Perhaps I fell from my bicycle and bruised my leg. [touching his *right* leg]

N: Which leg did you hurt?

NS: [touching his right leg] This one is fine.

N: Could you go surfing on the sea should you wish to do so?

NS: Why not, if the wind is strong enough.

Patient NS showed clear extrapersonal neglect and also personal neglect on tests in which he was asked to simulate combing his hair and shaving. In addition, he moved his right limbs when asked to move his left limbs. If this mis-routing of motor instructions—which may have been a consequence of unilateral neglect—also influenced the construction of the forward model then no mismatch between predicted state and actual state would be detected. It is plausible, then, that unilateral neglect and mis-routing of motor instructions combined to function as a first factor that usually prevented patient NS from detecting his paralysis.

But these neuropsychological anomalies are not adequate to explain NS's persisting anosognosia. When NS's left limbs were moved to his right side and his attention was directed towards them, he discovered his hemiplegia. He was taken aback by his failure to move his left arm and leg and became so upset that the experiment had to be halted.

Patient NS was presented with evidence of his paralysis—not only evidence of failure to move his paralysed left limbs but also evidence of his inability to carry out bi-manual tasks. He appreciated the significance of this evidence and was dismayed by it. Yet his anosognosia persisted. Despite incontrovertible evidence to the contrary, he maintained his false belief that he could move his left arm and leg and was able to go surfing. His reasoning abilities were well preserved and he was not disoriented or confused. But he showed retrograde amnesia for autobiographical events that occurred about a year before his accident and anterograde amnesia for new information related to himself. Cocchini and colleagues suggest, in effect, that these memory problems function as the second factor in NS's persisting anosognosia for his hemiplegia (p. 2036).

In this case, the patient understood that the evidence that he experienced as the result of the neuropsychologist's intervention incontrovertibly went against his belief that he could move his left arm and leg just as he had always been able to do. He made a correct evaluation or assessment of his old, but now false, belief in the light of his newly acquired evidence. But he never updated his antecedent web of beliefs because, as a result of his amnesia, he kept forgetting the evidence.

7.3. Anosognosia Without a Second Factor?

According to Levine (1990), different impairments present different degrees of difficulty of discovery. Blindness is relatively easy to discover while visual field

defects are relatively difficult to discover (section 4.1.); hemiplegia falls somewhere in between (section 4.2.).

In patients with unilateral neglect and hemiplegia, their neglect may be more difficult to discover than their paralysis. For example, a patient with personal neglect who fails to comb the left side of his hair will not immediately become aware of this failure. And if he also has extrapersonal neglect then looking in a mirror may still not provide the feedback that is needed. In the case of hemiplegia, in contrast (Levine, 1990, p. 252): 'Loss of the effective use of even one limb creates major functional limitations that are not quickly and automatically compensated'. It would thus be consistent with the discovery theory to suppose that anosognosia for extrapersonal or personal neglect, like anosognosia for a visual field defect, might occur in the absence of a second factor, while anosognosia for hemiplegia would always require a second factor.

This difference between anosognosia for neglect and anosognosia for hemiplegia need not present a problem for the generic two-factor theory of delusions if we allow that, at least in some cases, anosognosia for neglect is not a delusion. This claim would not be *ad hoc*. A patient with anosognosia for neglect denies, in answer to an interview question, that she fails to detect stimuli on her left side. But she might not be going against any incontrovertible evidence that is genuinely available to her, even though she is going against incontrovertible evidence that is available to the examiner. In contrast, under ordinary conditions of daily living, paralysis has serious and inescapable functional consequences that are evident to the patient, even if the patient's concurrent awareness of failure to move his or her limbs is impaired. This is why anosognosia for hemiplegia is a delusion.

However, suppose that paralysis were to occur in a patient who was lying in bed and was not engaged in everyday activities and that the paralysis were to last only for a very short time. Under such informationally and temporally restricted conditions, the paralysis might be much more difficult to discover. In such a case, even anosognosia for hemiplegia might not be a delusion and might occur in the absence of a second factor.

This point is of some significance for our proposed application of the generic two-factor theory of delusions to anosognosia for hemiplegia. As we have seen (sections 2.2., 2.3.), there is a body of research in which experiments conducted as part of the Wada procedure are used to investigate anosognosia for hemiplegia. But, in the Wada procedure, paralysis of one side of the patient's body is induced for just a few minutes and during that time the patient is not engaged in everyday activities. So it may be that, for explanatory purposes, denial of weakness resulting from an intracarotid barbiturate injection belongs in a different psychological category from cases of anosognosia for hemiplegia to which the two-factor theory of delusions applies. It may be that experiments conducted as part of the Wada procedure can contribute to our understanding of the first factor, but not the second factor, in the aetiology of anosognosia for hemiplegia, considered as a delusion.

8. Conclusion

Beginning from the point that anosognosia for hemiplegia seems to count as a delusion by the usual definitions, we have described examples (section 1) and then reviewed existing theories of anosognosia that appeal to cognitive impairments (section 2), impaired feedback from the paralysed side of the body (section 3), and combinations of these (section 4). None of the existing theories is reckoned to be fully adequate as an explanation of anosognosia for hemiplegia.

Our central proposal is to approach anosognosia for hemiplegia by way of a generic two-factor theory of monothematic delusions such as the Capgras delusion (section 5). This proposal faces two immediate problems, but we have argued that they can be overcome (section 6). In the final section, we have addressed some issues about cognitive impairments and the second factor in the aetiology of anosognosia for hemiplegia, considered as a delusion.

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