The cognitive neuropsychological understanding of persecutory delusions

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Abstract

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The cognitive neuropsychological understanding of persecutory delusions

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In considering the contribution of cognitive neuropsychology to the understanding of persecutory delusions, we shall proceed in this chapter as follows: First, we shall consider the contribution of the more conventional clinical neuropsychological approach to the study of delusions. After all, cognitive neuropsychology developed as a hybrid of clinical neuropsychology (the psychological study of brain-injured people) and cognitive psychology (the study of the mental information-processing procedures that people use to perform such activities as speaking and understanding speech, and recognizing objects). Second, we shall outline the cognitive neuropsychological approach with brief reference to its history. Third, we shall describe how this approach has been applied to the study of delusions with reference to our two-deficit cognitive neuropsychological model of monothematic delusions. Finally, we shall evaluate the applicability of this model to the explanation of other delusions that are not so obviously neuropsychological; here we shall focus on persecutory delusions.

The clinical neuropsychology of delusions
Conventional clinical neuropsychological studies proceed by administering standard batteries of tasks that are known to reliably demonstrate performance deficits in patients with identifiable lesions. The primary clinical aims include: (a) specifying which cognitive abilities have been impaired and which remain intact, consequent to the brain injury, so as to target remediation; and (b) identifying the likely sites of underlying neuropathology so as to assist in diagnosis. Standard neuropsychological batteries typically carve up cognition into relatively coarse-grained domains – e.g. executive function (linked to frontal brain regions) and spatial abilities (linked to parietal regions).

Most conventional clinical neuropsychological studies do not, however, adopt a symptom-focused approach so as to identify the neuropsychological impairments that are associated with a particular symptom - say delusions. More often, the focus will be on a particular diagnostic category, regardless of symptomatology, or, if clinical symptoms are of interest, researchers will examine the associations between neuropsychological impairments and characteristic clusters of symptoms. Delusions might be of interest but they will be grouped together with other co-occurring symptoms so as to form a symptom cluster.

If we consider first those clinical neuropsychological studies that have adopted a symptom-focused approach so as to investigate delusions, these studies are more common when the
delusions occur in the context of known neurological illness. Such delusions are termed ‘organic’ (or such patients are referred to as suffering ‘organic psychoses’) and are distinguished traditionally from ‘functional’ delusions (or the ‘functional psychoses’, including e.g. schizophrenia). In the latter case, there is less consensus concerning the nature and the role (if any, for some researchers) of underlying neuropathy. A common example of this type of work is the clinical neuropsychological study of delusions in dementia. Results from such studies generally indicate that delusions in dementia are associated with greater cognitive impairment and a more rapid cognitive decline (Haupt, Romera & Kurz, 1996). There are also indications of more specific associations with executive function and semantic memory deficits (Fischer, Ladowsky-Brooks, Millkin, Norris, Hansen & Rourke, 2006).

The focus of this volume, however, is persecutory delusions, and although persecutory delusional themes are seen in the organic psychoses, they are also very common in the functional psychoses. Clinical neuropsychological studies of psychotic conditions that focus, in some regard, on delusions, are most common in the field of schizophrenia research (see e.g. Heinrichs & Zakzanis, 1998, and Bilder, Goldman, Robinson et al., 2000, for reviews). These studies do not, however, examine associations with single symptoms (including delusions); they focus instead on symptom clusters, in particular the positive and negative symptoms of schizophrenia. Delusions are grouped together, for example, with hallucinations and positive thought disorder, all of which are considered ‘positive’ due to the presence of something which is abnormal. Negative symptoms (e.g. apathy) are instead characterized by the absence of something that should normally be present. The general finding from clinical neuropsychological studies of schizophrenia is that, whenever associations are found between neuropsychological impairment and symptoms, it is the negative symptoms and not the positive symptoms that are involved (see e.g. Addington & Addington, 1999, 2000).

Even when we turn to the few clinical neuropsychological studies of the functional psychoses that have adopted more of a symptom-focused approach to investigate delusions, there is still little to suggest a critical contribution from neuropsychological impairment. Baddeley, Thornton, Chua and McKenna (1996), for example, compared memory (episodic and semantic) and executive function in 5 patients with schizophrenia who were currently delusional and 5 patients who were no longer delusional; these authors found few differences between the groups. Mortimer, Bentham. McKay, et al. (1996) adopted instead a correlational approach with two much larger samples (N’s of 79 and 67) yet still found no evidence that
impairments of general intellectual ability, memory, or executive function were associated with delusions in schizophrenia.

While we are aware of no clinical neuropsychological studies that have focused even more specifically on persecutory delusions (e.g. to compare persecutory deluded patients and non-persecutory deluded patients), several studies have categorised patients according to diagnostic subtypes or differing symptom profiles so as to compare ‘paranoid’ and ‘non-paranoid’ subgroups (see e.g. Hill, Ragland, Gur & Gur, 2001; Savage, Jackson & Sourathathone, 2003). Generally speaking these studies report that, even when differences are found, it is the paranoid individuals who appear less neuropsychologically impaired (see Bentall & Taylor, 2006, for discussion).

So what are we to take from these more conventional, clinical neuropsychological studies? One might conclude that neuropsychological abnormalities are only implicated in the generation of delusions when delusions arise in the context of organic psychoses; but we suggest that this conclusion is premature. It might be the case that there are also neuropsychological abnormalities associated with delusions in the psychotic conditions traditionally conceived of as functional psychoses. These have so far not been discovered, however, because the standard batteries of tasks used in conventional studies do not tap adequately the specific nature of the neuropsychological abnormalities that contribute to delusion formation. This leads us to consider the possible contribution of a cognitive neuropsychological approach to the study of delusions.

Cognitive neuropsychology
Cognitive neuropsychology developed as a blend of clinical neuropsychology and cognitive psychology. The primary aims were twofold: (1) to explain the symptoms of brain-injured patients in terms of what has been lost and what remains intact in a cognitive model (or theory) of the normal system for mental information processing; and (2) to evaluate such models in terms of how well they explain the patterns of spared and dysfunctional capacities observed in brain-injured populations (Coltheart, 1984; Ellis & Young, 1998).

Cognitive neuropsychologists attempt to develop such cognitive theories by breaking up the cognitive system into subcomponents and connections so as to model the representations, computations and transformations of information that relate to a particular domain of interest.
Up until the 1990’s, this approach was applied primarily to such ‘lower-level’ domains as reading, memory, attention and visual object recognition, in contrast to such ‘higher-level’ domains as belief formation, decision-making, theory of mind and pragmatics. Because disorders in these high-level domains often fall under the rubric of psychiatry, the application of cognitive neuropsychology to the study of these kinds of disorders came to be called *cognitive neuropsychiatry* (David & Halligan, 1996).

**The cognitive neuropsychiatry of delusions**

Hadyn Ellis and his colleagues were pioneers in the field of cognitive neuropsychiatry. These researchers were primarily interested in the misidentification delusions (e.g. Capgras delusion - the belief that someone emotionally close to you, typically a spouse, has been replaced by an impostor, and Fregoli delusion - the belief that one is being followed by familiar people in disguise). Ellis and Young (1990), for example, sought to explain these various types of misidentification delusion in terms of different patterns of breakdown in the normal cognitive system for face recognition.

Our own cognitive neuropsychological approach to the study of delusions is far more general. We advocate the adoption of a general explanatory framework in which the normal cognitive system is conceived of as subdivided into those components which, when disrupted, explain the initial generation of an implausible thought and those components which, when disrupted, account for the uncritical acceptance of the implausible thought as a belief.

Consider Capgras delusion, for example. It has been shown that sufferers from this delusion do not exhibit the autonomic response to familiar faces that is normally found in non-deluded individuals (Ellis, Young, Quayle, & de Pauw, 1997; Hirstein & Ramachandran, 1997; Brighetti, Bonifacci, Borlimi, & Ottaviani, in press). Note that face recognition itself is not seriously impaired in people with Capgras: if it were, they would not say “This woman looks exactly like my wife (but it is not her)”. The cognitive abnormality here is instead a disconnection between intact face recognition and the autonomic response which recognition of a familiar face should normally trigger. It is the viewing of a face that matches the loved one’s face in the absence of an expected autonomic sense of familiarity which explains the generation of the impostor content – the initial thought that the person being seen is a stranger who looks like the spouse.
But the presence of this particular deficit, even if it is necessary for the occurrence of Capgras delusion, is not sufficient. We know, for example, that there are patients with damage to bilateral ventromedial regions of frontal cortex who also show evidence of a disconnection between intact face recognition and autonomic responses, yet are not delusional (Tranel, Damasio, & Damasio, 1995). And we know that those patients who do develop Capgras delusion are provided with ample evidence (e.g. from the spouse and other family members) that their belief is false. We have therefore argued that a second deficit must also be present in people with Capgras delusion that explains these people’s failure to reject the implausible impostor-thought after it has come to mind.

In considering what this second deficit might be, we shall turn to other monothematic delusions with thematic content that also appears (at first sight) just as incomprehensible as the impostor-content of Capgras. Patients with mirrored-self misidentification, for example, believe that the person they see when they look in the mirror is not them, but some stranger. How could such an implausible thought come to mind? In one such case (see Breen, Caine, & Coltheart, 2001), the answer was an impaired appreciation of mirror spatial relations and a consequent inability to interact appropriately with mirrors, elsewhere termed ‘mirror agnosia’ (Ramachandran, Altschuler, & Hillyer, 1997). For this patient, a mirror was effectively a window or a hole in the wall. Any person seen through a window or a hole in the wall is occupying a different region of space from you, and therefore can’t be you: this is what we suggest prompted the initial implausible thought in our patient with mirrored-self misidentification delusion.

Take another example: people with sufficiently large right temporoparietal lesions will suffer left-sided paralysis. Some of these people will exhibit anosognosia for their hemiplegia (i.e., will deny the paralysis of the left limbs), and some of these anosognosic patients will attribute ownership of the paralysed limbs to other people. This is an example of somatoparaphrenia, the belief that some part of your body – say, your left arm – is not yours but belongs to some other person (often your neurological examiner). If your examiner places your left arm in your intact visual field and asks you to move your paralysed arm, the arm will not move. This is an arm that you cannot accept as paralysed due to your anosognosia. Therefore you must find some explanation of the fact that it did not move other than that it is paralysed. If it were not your arm but instead some one else's, that would provide such an explanation. That, we suggest, is what generates this thought about the arm in patients with somatoparaphrenia who
have anosognosia for their hemiplegia.

What we are building up to here is that the left hemisphere is typically intact in these cases of Capgras, mirrored-self misidentification and somatoparaphrenia. So, if we want to pursue, as we do, the hypothesis that the second deficit of belief evaluation is the same across a variety of monothematic delusions, we are led to conclude that the second deficit has something to do with the right hemisphere: some region of the right hemisphere is critically involved in the evaluation and rejection of implausible beliefs.

Our current thinking is that this region lies in the right frontal lobe (see Coltheart, in press, and Coltheart, Langdon & McKay, 2007, for detailed discussion). For example, Staff, Shanks, Macintosh, Pestell, Gemmell, and Venneri (1999) conducted neuropsychological testing and SPET imaging of deluded and nondeluded patients with Alzheimer’s Disease. The two groups were matched on general severity of cognitive deterioration. While the conventional neuropsychological testing revealed no significant differences between groups, the imaging results revealed a consistent pattern of hypoperfusion in the right frontal (and limbic) brain regions in the deluded group compared to the nondeluded group. Consider also Papageorgiou, Ventouras, Lykouras, Uzunoglu and Christodoulou (2003) who conducted an event-related potential (ERP) study of 9 patients experiencing misidentification delusions (a mixed group of Capgras and/or Fregoli sufferers) and 11 healthy controls. The deluded patients showed a significant reduction in the P300 amplitude at the right frontal brain region compared to the healthy controls. Papageorgiou and colleagues described the P300 ERP component as “the physiological correlate of updating a cognitive hypothesis, or the working memory (WM) update of what is expected in the environment” (p. 366). The process of updating a cognitive hypothesis of what is occurring in the environment is clearly a major component of belief evaluation.

We have therefore proposed that two distinct neuropsychological deficits need both be present to explain monothematic delusions with thematic content that defies everyday commonsense: Deficit-1 is responsible for the theme of the delusional belief and will necessarily differ from delusion to delusion, while Deficit-2, conceptualized as a right-frontal impairment of the capacity to reject the implausible thought triggered by Deficit-1, may be the same across many delusions.
Thus far we have focused on monothematic delusions with apparent nonsensical content. There are many delusions associated with schizophrenia, however - including persecutory delusions, grandiose delusions, erotomaniac delusions and delusional jealousy - that seem less bizarre and so perhaps are not so well explained by our two-deficit model. In the next section we shall evaluate the applicability of the two-deficit account to the explanation of persecutory delusions.

**A two-deficit cognitive neuropsychological account of persecutory delusions?**

We begin by considering neuropsychological impairments of the first type (Deficit-1) which might contribute to the generation of persecutory delusional themes. Appropriate candidates are not difficult to find – e.g., hearing loss consequent to a failing auditory nerve. Claims of a connection between paranoia and deafness have long been made (e.g. Piker, 1937; Houston & Royse, 1954; Cooper, 1976) and, despite some inconsistent findings (e.g. Thomas, 1981; Blazer, Hays, & Salive, 1996; Thewissen, Myin-Germeys, Bentall, de Graaf, Vollebergh, & Os, 2005; Stefanis, Thewissen, Bakoula, Os, & Myin-Germeys, 2006), it is nevertheless plausible that the experience of surrounding voices at lower than expected volume will prompt the initial thought, “People are whispering.” If a deficit in belief evaluation is also present, this initial thought, which should be deemed implausible in the light of evidence of hearing loss (including that provided by doctors), will be accepted as true and will persist. This false belief will itself prompt further thoughts, “Why do they whisper? It must be because they don't want me to hear what they are saying. Why don't they want me to hear what they are saying? It must be that they are plotting against me.”

Memory impairment is another likely candidate. Consider delusions of theft, common in dementia and associated with persecutory elaborations (Hwang, Yang, Tsai & Liu, 1997). Likely scenarios are not difficult to imagine: A patient opens his top drawer expecting to find his wallet, only to discover it gone. Having forgotten that he had earlier moved his wallet, the patient will naturally think, “Someone has moved my wallet”. Now imagine what might happen if this initial thought is accepted uncritically as true and never revised in light of evidence of memory loss. This patient will question others about moving his wallet, only to be met with general denials. Similar incidents, each triggered by an initial thought that should be rejected as implausible yet is not despite the evidence of memory loss, will likely promote persecutory themes.
These rather convoluted chains of thought seem, however, quite different to the more direct links between neuropsychological impairments and the themes of Capgras, mirrored-self misidentification and somatoparaphrenia suggested above. Perhaps we can get a bit closer to a Capgras-like explanation of persecutory delusional themes if we consider the involvement of hallucinations. Hallucinations and delusions reliably co-occur in factor-analytic studies of symptom ratings in schizophrenia (see e.g. Liddle, 1987) and hallucinatory experiences are also strongly associated with delusional ideation in the non-clinical population (see e.g. van Os, Hanssen, Bijl & Ravelli, 2000). We shall focus on auditory verbal hallucinations. These are the most common type of hallucination in schizophrenia and are typically unpleasant experiences which lend themselves rather naturally to persecutory elaborations. Auditory verbal hallucinations are associated with disruption to left-side language areas of the brain (David, 2004). The cognitive impairment here is conceived of as an impaired capacity to monitor the source of inner speech (see e.g. Johns, Rossell, Frith et al, 2001). The initial thought of a person who first experiences unpleasant voices, however, is likely to be, “Someone is saying nasty things to me”. It is only after this thought is accepted uncritically as true (and never revised) and further reflection follows (“Hearing such nasty things upsets me; Why should the speaker want to upset me in this way? They must be trying to hurt me”), that a persecutory delusional theme emerges.

Ideas of reference fall into a similar category (i.e. we can get closer, but not quite close enough, to a Capgras-like explanation). Ideas of reference and persecutory delusions are reported to be associated in factor-analytic and multidimensional scaling studies which use ratings of individual symptoms rather than global symptom ratings (see e.g. Kitamura, Okazaki, Fujinawa, Takayanagi, & Kasahara, 1998; Minas, Stuart, Klimidis et al, 1992). These co-occurrences may reflect the self-referential quality of both phenomena (see Freeman & Garety, 2000, for discussion of key characteristics of persecutory ideation – e.g. that harm is being done to oneself, and Startup & Startup, 2005, for discussion of the self-referential quality of referential ideas). Ideas of reference include experiences of innocuous events (e.g. the location of a crumpled brown paper bag on the pavement) appearing to have special significance uniquely for the patient. Kapur (2003) suggests that such experiences reflect aberrant states of salience that attach inappropriately to events which ought normally to be screened from attention rather than becoming the focus of attention. The underlying cause, Kapur further suggests, is dopamine hyperactivity. But why should experiences of this type prompt thoughts of harm, also characteristic of persecutory ideation (Freeman & Garety,
2000, McKay, Langdon & Coltheart, 2006)? When, for example, an aberrant state of salience attaches to the image of Kylie Minogue performing on the TV, might not the affected viewer think, “She’s secretly sending a message to me. Why? It must be because she secretly loves me.” – in other words, an example of Clérambault syndrome?

Perhaps a complicating factor here is in assuming that referential ideas are unitary. Some referential ideas concern the inappropriate perception of meaningful contingencies between coincidental events. Empirical support for a link between referential thoughts of this type and persecutory delusions was provided by Blakemore, Sarfati, Bazin and Decety (2003) who found that persecutory-deluded patients perceived contingencies between shapes (e.g. one shape seen as launching another shape) when non-persecutory deluded patients and healthy controls did not. Startup and Startup (2005) have also suggested a distinction between referential ideas of communication (e.g., thinking that others say things with double meanings) and referential ideas of observation (e.g. thinking that one is under surveillance). These authors found that only the referential ideas of observation were associated with persecutory delusions. What type of neuropsychological impairment, if any, could prompt ideas of observation? One intriguing possibility, we suggest, is disruption to the cognitive system for monitoring other people’s gaze. This system is sustained by a neural network linking the superior temporal sulcus, amygdala and orbitofrontal cortex which is reported to be disrupted in schizophrenia (see, e.g., Emery, 2000). People with schizophrenia have also been found to consciously misjudge the averted gaze of others as directed towards themselves (Rosse, Kendrick, Wyatt, Isaac & Deutsch, 1994). At the same time, these individuals also show some evidence of an unconscious hypersensitivity to signals of intentionality from other people’s gaze-direction (Langdon, Corner, McLaren, Coltheart & Ward, 2006).

We think these findings intriguing, if complex. But even if future work were to tease apart the multifaceted nature of ideas of reference and identify the various types (and combinations) of neuropsychological impairment that might be involved, we suspect that an explanatory gap would remain. If, for example, neuropsychological impairment(s) were to prompt the initial thought, “Others are intentionally observing me”, why should a threat-related persecutory interpretation be favoured over say a grandiose interpretation?

What our examples serve to illustrate is that, while neuropsychological impairments like those listed above might precipitate a train of thought leading (more or less directly) to a persecutory
delusion, the tendency to threat-related persecutory elaboration exists prior to and independently of these neuropsychological events. Is a neuropsychological impairment then necessary to trigger the persecutory train of thought? Elsewhere we have allowed that a neuropsychological impairment of the first type (Deficit-1) might not be necessary when the delusional content does not have the same apparent nonsensical quality of say Capgras or somatoparaphrenia (Langdon & Coltheart, 2000). In cases of ‘everyday’ persecutory content (e.g. the neighbours are spying on me), attentional biases to threat-related material in the environment might be sufficient to set the persecutory train of thought rolling.

But what causes the attentional bias to threat? It may have something to do with latent negative self-beliefs. Bentall and colleagues (Bentall & Kaney, 1996; Kinderman & Bentall, 1996, 1997) have argued that persecutory delusions are purposive constructions that serve to avoid the activation of negative self-beliefs, thus maintaining self-esteem. A prediction of their model is that persecutory delusions will be associated with a discrepancy between relatively high measures of conscious, overt self-esteem and relatively low measures of unconscious, covert self-esteem. This prediction has received empirical support (Kinderman, 1994; Lyon, Kaney, & Bentall, 1994; McKay, Langdon, & Coltheart, 2007; Moritz, Werner, & von Collani, in press). Freeman and colleagues (e.g. Freeman & Garety, 1999; 2003) have highlighted the role of emotional factors, in particular social anxiety, in the explanation of threat anticipation in persecutory-deluded individuals. Their view has also received considerable support (see Freeman, in press, for a review). However, the contribution of neuropsychological factors ought not to be ignored even here. For example, neuropsychological and neuroimaging studies have implicated the amygdala and pre-frontal circuits in the interpretation of social signals of threat. These neural networks are disrupted in schizophrenia and such disruptions might explain the abnormally heightened perception of social threat that is associated with persecutory delusions in this disorder (Green & Phillips, 2004).

Let’s now consider why the persecutory-deluded patient clings so tenaciously to his or her delusional belief when there is so much pressure to reject it. Might disruption to the normal cognitive system for belief evaluation (caused by right-frontal brain dysfunction: Deficit-2) contribute to the failure to reject a persecutory thought, even when that thought has been prompted by threat-related attentional biases that are unrelated to neuropsychological deficits?

We noted earlier that delusions in Alzheimer’s disease are associated with greater hypoperfusion
in right frontal (and limbic) brain regions. Persecutory themes are common in these delusions (Cook, Miyahara, Bacanu et al., 2003; Heinik, Solomesh, Shein, Mester, Bleich, & Becker, 2001). It therefore follows that right-frontal brain damage might contribute to the maintenance of persecutory delusions in cases of dementia. Persecutory themes are also common in the delusions that can arise after traumatic brain injury (TBI: Sachdev, Smith & Cathcart, 2001; Zhang & Sachdev, 2003). Frontal brain regions are frequently damaged in TBI, although the brain damage is typically quite diffuse. Nevertheless, it is plausible that right frontal damage also contributes to the failure to reject persecutory thoughts in TBI patients.

When we turn to the functional psychoses, however, the findings are less convincing. In support of the involvement of right frontal dysfunction is the historical view that paranoid schizophrenia (identified according to traditional diagnostic subtypes) is associated with right hemisphere dysfunction while non-paranoid schizophrenia is characterised by left hemisphere deficits (see eg Magaro & Page, 1983). Current evidence also suggests right frontal abnormalities in schizophrenia (see e.g. Coltheart, Langdon & McKay, 2007, for details). For example, structural magnetic resonance imaging (MRI) work has indicated right but not left frontal hypergyria (Narr, Bilder, Kim et al, 2004; Vogeley, Tepest, Pfeiffer et al, 2001). Regional cerebral blood flow (rCBF) studies, which employ neuroimaging techniques to measure the blood supply to specific brain regions at given times, have also reported abnormal metabolism in right but not left frontal lobe in schizophrenia patients (Hook, Gordon, Lazzaro et al., 1995; Malaspina, Bruder, Furman, Gorman, Berman, & Van Heertum, 2000). But despite such findings of right frontal abnormalities in schizophrenia and despite the prominence of persecutory delusions in this disorder there is little consistent evidence of a direct link between right frontal dysfunction and persecutory delusions. Some studies, for example, report disturbances of right hemisphere function in patients with paranoid (and not non-paranoid) schizophrenia (Magaro & Chamrad, 1983; Romney, Mosley & Addington, 2000). At the same time, other studies report evidence of left hemispheric abnormalities in relation to paranoid ideation. Sallet, Elkis, Alves et al. (2003), for example, investigated cortical folding in different subgroups of schizophrenia patients. They found that the paranoid subtype showed reduced cortical folding that was restricted to the left hemisphere. Kohno, Shiga, Kusumi et al. (2006) evaluated the relationship between regional cerebral blood flow (rCBF) and clinical symptoms in schizophrenia. These authors found the suspiciousness score on the Brief Psychiatric Rating Scale was positively correlated with rCBF in the left inferior temporal gyrus; no other associations reached statistical significance.
Conclusions

While our two-deficit model has proven useful for the explanation of monothematic delusions with themes that defy everyday commonsense (Davies, Coltheart, Langdon, & Breen, 2001; Langdon & Coltheart, 2000), the applicability of this model to the explanation of ordinary persecutory delusions appears somewhat limited in contrast. Threat-related attentional biases might be sufficient to trigger a persecutory train of thought in the absence of a precipitating neuropsychological event. And, although a heightened anticipation of social threat might be related to deregulation of a neural network linking amygdale and pre-frontal circuits, the chicken-and-egg question remains; Which comes first, the neural disturbance or the experiences of harm from others? As for the involvement of right frontal disruption to the normal cognitive system for belief evaluation, the findings concerning persecutory delusions in the functional psychoses are equivocal. In light of such considerations, we have recently explored the need to incorporate motivational factors into a model of the normal system for belief generation and evaluation so as to explain those delusions that appear somewhat resistant to a strictly neuropsychological account (e.g. Reverse Othello—see McKay, Langdon & Coltheart, 2005). This work has led us to advocate a more general two-factor account of delusions in which we propose that, regardless of the delusional theme and regardless of the etiology, one needs to answer two questions in order to explain the presence of any delusion:

(a) What gave rise to the belief in the first place – what caused the patient to first entertain a thought with this particular content?

(b) Having once entertained this particular thought, why does the patient cling to it rather than rejecting it?

For some patients, the answers to these two questions will be wholly neuropsychological. For other patients they will not. We suggest that both questions are best addressed on a case-by-case basis. Neither motivational nor neuropsychological factors should be ruled out automatically because of either the etiology or the thematic content of a delusion. The fact that certain delusions are conceived of traditionally as functional (e.g. persecutory delusions) does not mean

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1 This was a patient reported by Butler (2000) who had sustained severe head injuries in a car accident. The accident left him a quadriplegic, unable to speak without reliance on an electronic communicator. One year after his injury, the patient developed a delusion concerning the continuing fidelity of his partner (who had in fact severed all contact with him soon after his accident). The patient became convinced that he and his former partner had recently married, and he was eager to persuade others that he now felt sexually fulfilled.
that neuropsychological impairment will not feature in the explanation of such delusions in all patients. And, likewise, the fact that a delusion is conceived of traditionally as organic does not mean that motivational factors should be ruled out as significant explanatory forces.
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