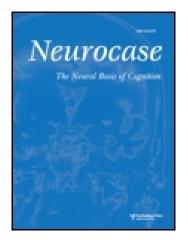
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Neurocase: The Neural Basis of Cognition Publication details, including instructions for authors and subscription information:

Publication details, including instructions for authors and subscription information: <u>http://www.tandfonline.com/loi/nncs20</u>

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To cite this article: Alberto Villarejo, Verónica Puertas Martin, Teresa Moreno-Ramos, Ana Camacho-Salas, Jesús Porta-Etessam & Félix Bermejo-Pareja (2011) Mirrored-self misidentification in a patient without dementia: evidence for right hemispheric and bifrontal damage, Neurocase: The Neural Basis of Cognition, 17:3, 276-284

To link to this article: <u>http://dx.doi.org/10.1080/13554794.2010.498427</u>

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Mirrored-self misidentification in a patient without dementia: evidence for right hemispheric and bifrontal damage

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Mirrored-self misidentification, often referred as the 'mirror sign', is a delusion characterized by the inability to recognize one's own reflected image, often associated with the intact capacity to recognize others in the mirror. It has been described mainly in moderate or severe dementia, especially Alzheimer's disease. In the few reported cases without global cognitive impairment, right hemispheric and frontal dysfunctions have been described. We report a 90-year-old man with abrupt onset of the mirror sign after a minor right hemispheric ischemic stroke. Neuropsychological testing revealed preserved cognitive capacities, except for mild to moderate impairment of visuospatial skills, suggesting right hemisphere dysfunction. Neuroimaging showed a small right dorsolateral frontal infarct, and bifrontal encephalomalacia, consistent with a past history of head trauma. Scattered ischemic torial phenomenon that usually requires right hemispheric dysfunction (perceptual abnormalities, loss of familiarity) and frontal damage (loss of judgement and inability to correct wrong beliefs). The right frontal dorsolateral prefrontal cortex seems to have a crucial role in self-recognition.

Keywords: Mirror-sign; Delusion; Misidentification; Frontal.

INTRODUCTION

Some of the most interesting disorders in neurology involve alterations of the self and self-awareness as a result of brain disease (Feinberg, 1997). One of them is the mirrored-self misidentification, the inability to recognize one's own image in the mirror, usually in the presence of an intact ability to recognize others (Foley & Breslau, 1982). Often called the 'mirror sign', it has been included among the delusional misidentification symptoms (DMS) (Joseph, 1986), a group of conditions in which a patient consistently misidentifies persons, places, objects or events. Capgras syndrome or, more correctly, symptom (Förstl et al., 1994), is the most common DMS reported. It is characterized by the delusional belief that a person, commonly a relative, has been replaced by an impostor. Other forms of DMS include the Frégoli syndrome (known person seen in the guise of others), subjective 'doubles' and reduplicative paramnesia (a place exists in two different locations). Some of them, like reduplicative paramnesia and the mirrored-self misidentification, almost always occur in patients with focal brain damage or generalized cerebral dysfunction, while others, like Capgras'

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Conflict of interest: none.

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We are very grateful to MM and his family, and to Joseph Healey for comments about the manuscript.

symptom, can be seen both in neurological or psychiatric conditions, especially schizophrenia (Cutting, 1991). It has been pointed out that the DMS share some features (Feinberg, Deluca, Giacino, Roane, & Solms, 2005; Weinstein, 1994), which allow the clinician to distinguish them from simple confabulation. According to Feinberg et al. (2005), the DMS: (1) reflect alterations in personal significance (the person, place or object misidentified is personally related to the patient); (2) they are selective and consistent; (3) the patients with DMS are usually unaware of their neurological or neuropsychological impairments; (4) they are refractory to correction; and (5) right hemisphere dysfunction is especially implicated in these disorders.

The mirrored-self misidentification has been mainly described in Alzheimer's disease patients, with a prevalence in epidemiological studies that varies between 2 and 22% (Burns, Jacoby, & Levy, 1990; Förstl, Osvaldo, Owen, Burns, & Howard, 1991a; Förstl et al., 1994; Mendez, Martin, Smyth, & Whilehouse, 1992; Rubin, Drevets, & Burke, 1988). These differences in prevalence may be based on different criteria for the definition of the mirror sign, and on the degree of dementia of the patients included, because the mirrored-self misidentification usually appears in patients with moderate or severe dementia, and is rare in mild Alzheimer's disease. There are only a few case reports of the mirror sign as a symptom of fixed and focal nature, independent of a global dementia (Spangenberg, 2005). These cases suggest that the mirrored-self misidentification is not only a manifestation of global cognitive deterioration, but a distinct delusion with neuroanatomical and neuropsychological correlates. We report the case of a patient with a past history of head trauma with full recovery, who experienced the mirrored-self misidentification shortly after a mild right hemispheric stroke. Evidence of right hemispheric dysfunction and asymmetrical bifrontal damage were found in this patient without dementia.

CASE REPORT

Patient MM

MM is a 90-year-old right-handed patient, a widower with three children. He attended primary school until 14 years of age, with 8 years of formal education, and worked most of his life as a farmer. He lived alone in a rural area of central Spain, but in the last 10 years he has spent the winter months in Madrid with his daughters. His past medical history included mild arterial hypertension and a radical nephrectomy for renal carcinoma when he was 60 years old. He served as a soldier during the Spanish Civil War, and had a politraumatism with head trauma in a battle action. We lack any medical record of that time, and do not know the type of head trauma or the duration of the loss of consciousness. He was in a hospital for 6 months and apparently he made a full recovery, but he was discharged from duty, resuming his civil life. There was no other history of neurological or psychiatric disease.

One year before the initial consultation, he had an episode of sudden onset of clumsiness of the left hand and paresthesias in the left extremities that lasted for 3-4 days. A few days later, the family observed that MM started to believe that his own reflection in the mirror was his father and, 5 days later, his father-in-law, who had died 60 years ago, and would be the object of the misidentification for the following months. He could see him in any reflecting surface, and believed that he was following him around. He tried to talk to him, and recognized that the presence did not answer because 'he is mute'. When his family tried to reason with him, saying that his father-in-law was long dead, he would answer: 'That is true, but he has resuscitated like Jesus Christ'. The mirror sign appeared continuously when he was in Madrid, but only sporadically (about once a week) when he was in his village. He had preserved knowledge of what a mirror was and provided examples of different uses of mirrors. He continued to shave using a mirror.

The first 6 months, MM was calm and even joked about the presence of his father-in-law in the mirror, saying 'something must be wrong in my head when nobody else sees him'. Later, he developed paranoid ideas about the mirror presence, believing that he wanted to harm him or his family, arguing with the mirror, or asking his family to 'feed the father-in-law'. Finally, his daughters decided to cover all the mirrors and reflecting surfaces in the house and asked for a medical evaluation. Neither the patient nor his family had other cognitive or behavioural complaints. He had no other misidentifications, illusions or hallucinations. He never incorporated the reflection of other persons in the mirror delusion. He was independent for all basic and instrumental activities of daily living, and continued living alone in his village for part of the year. Looking for a psychological explanation, we asked his daughters about his relationship with his father-in-law, and they did not remember any conflict between them. The father-in-law had died years before the patient's wedding, but MM was angry with him now because 'he did not come'.

When he was first evaluated in our memory clinic, the patient had been experiencing the mirrored-self misidentification for 1 year. The general and neurological examinations were unremarkable. He was a pleasant man, looking younger than his age. Bedside language, orientation and memory testing were normal. Fundoscopy, visual fields and acuity were normal. He had a bilateral hyperreflexia with down-going toes. Sensation, coordination and gait showed no abnormality.

Neuropsychological study

Hability

Neuropsychological tests included the Wechsler Adult Intelligence Scale - revised (Wechsler, 1987a), the Wechsler Memory Scale - revised (Wechsler,

1987b), Test Barcelona - revised (Peña-Casanova, 2005), the Boston Naming Test (Kaplan et al., 1983), the Sunnybrook Neglect Battery (Leibovitch et al., 1998), and a test for phonemic verbal fluency (Ramier & Hècaen, 1977). Results are shown in Table 1. The MMSE score (Folstein, Folstein, & McHung, 1975) was 29/30. The patient had a normal intelligence and had preserved attention, executive function, verbal memory, praxis and perceptual skills. He presented mild problems with naming and category fluency. Visuoconstructional tasks were mild to moderately impaired, suggesting right hemispheric damage. There were no signs of prosopagnosia in a face recognition test, and he showed no problems in identifying famous persons, family members or himself in different photographs provided by his daughter.

Evaluation of the mirror sign

Direct Score

Formal assessment of recognition of objects, family members and different examiners reflected in

Percentile Score

TABLE 1	
Neuropsychological	study

SubTest

Cognitive status	MMSE (1)	29	
Visual inattention	Sunnybrook neglect battery (2)	1	50
Verbal Attention	Forward span digits (3)	5	63
Visual Attention	Forward block span (4)	3	16
Verbal Working memory	Reversed span digits (3)	3	47
Visual working memory	Reversed block span (4)	2	45
Concept formation	Similarities (3)	14	91
Verbal fluency	FAS (5)	21	9
Verbal memory	Words list, learning slope (4)	1-3-4-7	25
5	First Delayed recall. 5'min (4)	1	9
	Second delayed recall, 20'min. (4)	3	63
	Recognition (4)	18	15
Language	Orders comprehension (6)	16	55
	Naming (7)	26	10
Category fluency	Animals (6)	8	15
Visuoconstruction	Clock drawing test (8)	1	1
Copying tasks	Copy house-cube (6)	0	1
Gestural praxis	Bilateral Praxias (6)	4	1
Dimensional construction	Block desing (3)	6	9
Visual recognition	Faces (6)	6	50
	Overlapping line-drawn (6)	19	63
	Family photo test	10	
	Famous people photo test	8	

Vechsler Memory scale (' Tea Ediciones, 2004. (5) FAS (Ramier AM et al, 1977), spanish data: Rami L, 2007. (6) Barcelona Test (Peña-Casanova J, 1991). (7) BNT: Boston Naming Test (Kaplan E et al, 1983), spanish data: García-Albea JE, 1986. (8) Clock drawing test (Freedman M et al, 1994), spanish data: Del Ser T, 2004.

the mirror showed no abnormality. Many times during the examination, he could point to his own image in the mirror and recognize it as himself, but at times he would also identify his father-in-law, who the patient said did not particularly resemble himself. Surprisingly, when asked to describe the facial features of the father-in-law, he made an accurate description of himself, but said that the mirror presence 'is much younger than me'. When asked to provide more details about the presence, the delusion became gradually more elaborated. We can provide an example of a videotaped conversation:

- EXAMINER: What kind of clothes does your father-in-law wear?
 - MM: My clothes: my shirt, my pants, my jacket.
- EXAMINER: Why is he wearing your clothes? MM: Because I wear them myself.
- EXAMINER: And what happens if you are not in front of the mirror?
 - MM: He stays there.
- EXAMINER: What about his clothes?
- MM: He stays there, naked.
- EXAMINER: And what is he doing there if nobody invited him?
 - MM: The moment I arrive, he arrives. He lives in our apartment building and he has a house like our house. I can see it through the mirror, and he can see us through his windows.

This reduplicative paramnesia had not been described by the family and was elicited during the examination, but remained as a feature of the delusion during follow-up, saying that the house was 'exactly like ours'. This delusion of a house in the back of the mirror suggested some degree of mirror agnosia, and we decided to examine this possibility. With a test based on that by Ramachandran, Altschuler, and Hillyer (1997), we placed four objects, one at a time, behind his right or left shoulder, when the patient was standing in front of the mirror. When asked to grasp the object with his own hand, the patient appropriately reached for the object behind his shoulders, showing no sign of mirror agnosia.

Neurorradiological and other ancillary tests

A Magnetic Resonance Imaging (MRI) of the brain showed bilateral frontal postraumatic encephalomalacia, with more involvement of the right frontal lobe, and a small right dorsolateral frontal infarct, with a triangular shape and cortical involvement. Scattered periventricular white matter T2 hyperintensities were seen, especially around the posterior horns of the lateral ventricules (Figure 1). Hemogram, blood biochemistries, vitamin B12, folic acid, TSH, electrocardiogram and chest X-ray were normal. EEG showed mild symmetric slowing in cerebral activity and a carotid ultrasound study revealed mild ateromathosis without stenosis.

Evolution of the disease

The initial sudden episode of left hand clumsiness and paresthesia was considered a right hemispheric ischemic stroke, and treatment with clopidogrel was begun. In the following year, no episodes of focal neurological findings recurred. The mirror sign remained stable, and the tendency to appear in the big city persisted, and not in his village. Two offers of treatment with atypical neuroleptics were declined by the patient and his family because of concerns about side-effects. Two years after the onset of the mirrored-self misidentification, the patient remains stable and no dementia symptoms have appeared.

DISCUSSION

Summary of findings

We describe the case of a patient with mirrored-self misidentification and associated reduplicative paramnesia after a minor right frontal ischemic stroke. The delusion was strikingly modified by changes in the patient's surroundings, being more frequent in unfamiliar places. Neuropsychological evaluation revealed mild to moderate visuospatial impairment, suggesting right hemispheric dysfunction, with no evidence of prosopagnosia or mirror agnosia. Neuroimaging showed a small right frontal hemispheric infarct, bilateral asymmetrical encephalomalacia of presumed traumatic origin, and sparse white matter ischemic damage in posterior periventicular regions.

Findings in the context of the literature

The ability to recognize oneself in the mirror has acquired theoretical importance as a potential way

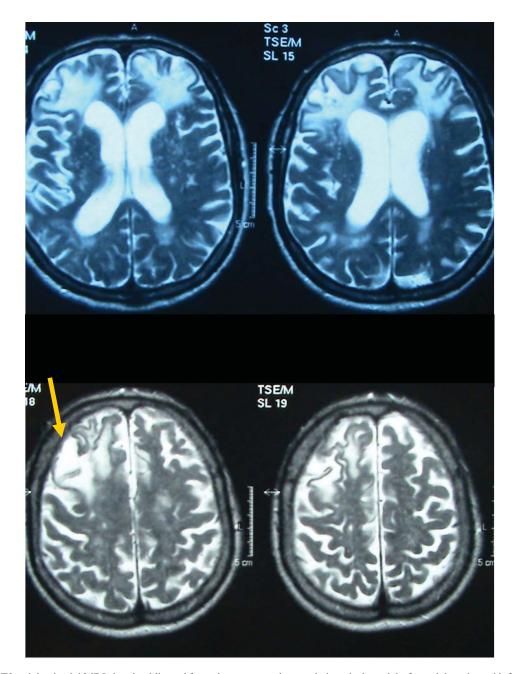


Figure 1. T2 weighted axial MRI showing bilateral frontal posttraumatic encephalomalacia, a right frontal dorsolateral infarct (yellow arrow) and scattered periventricular white matter hyperintensities.

of testing self-awareness (Gallup, 1970), yet little is known about how the brain achieves it. Apart from humans, mirrored-self recognition is a stable trait only in chimpanzees and a few other species, and may be subject to decline with age (De Veer, Gallup, Theall, van den Vos, & Povinelli, 2003). In comparison with recognition of other people, selfrecognition in humans has received little attention (Brédart & Young, 2004). Infants begin to show mirror self-recognition between 18 and 24 months of age (Amsterdam, 1972). Several lines of evidence suggest that self-recognition forms a separate process, accomplished by a 'self-network' within the brain, and it is not simply a part of the 'facenetwork' (Bredart & Young, 2004). Although studies suggesting left hemisphere involvement in selfrecognition also exists (Kircher et al., 2001; Turk et al. 2002, 2003), a growing body of literature points towards selective activation of a right fronto-parietal network, with a bias in the right prefrontal cortex, during tasks of self-recognition or self-other discrimination. Consistent data supporting this idea has been obtained with the Wada Test (Keenan, Nelson, O'Connor, & Pascual-Leone, 2001a), transcranial magnetic stimulation (Keenan et al., 2001a) and functional neuroimaging (Devue et al., 2007; Kaplan, Aziz-Zadeh, Uddin, & Iacoboni, 2008; Keenan, McCutcheon, & Pascual-Leone 2001b; Platek et al., 2006; Sugiura et al., 2005; Uddin, Kaplan, Molnar-Szakacs, Zaidel, & Iacoboni, 2005). Studies with functional MRI have shown that viewing one's own face leads to greater signal changes in the right inferior frontal gyrus, the right inferior occipital gyrus and the right inferior parietal lobe (Uddin et al., 2005). In addition, the right inferior frontal gyrus also shows greater signal change when hearing one's own voice compared to hearing a friend's voice. Thus, the preference for self-related stimuli in the right inferior frontal gyrus is not restricted to visual stimuli, and this result supports the theory that the right prefrontal cortex supports an abstract self representation (Kaplan et al., 2008). Likewise, evidence from patients with frontotemporal dementia suggests that those with asymmetric loss of function in the right hemisphere show dramatic changes in selfconcept (Miller et al., 2001).

The study of patients with the mirrored-self misidentification, in addition to its clinical relevance, offers the possibility to analyze a lesional model of self-recognition in humans. Instances of selfmisrecognition can occur in normal people, but they are usually quickly corrected (Bredart & Young, 2004). Self-misrecognition in the mirror also appears in patients with prosopagnosia, but using gestures and mimicking they readily infer that the reflected face is their own, even though they do not recognize the facial pattern itself (Damasio, Tranel, & Damasio, 1990). In contrast, a patient with mirrored-self misidentification may deny that the reflected image is him or herself, although overt recognition of faces is well preserved (Breen, Caine, & Colheart, 2000). Mirrored-self misidentification has rarely been described in the literature (Foley & Breslau 1982; Burns et al., 1990). Most of the reported patients have global dementia of the Alzheimer type, making it difficult to infer the anatomical basis of this disorder. The only study with a significant number of patients was perfored by Förstl, Burns, Jacoby, and Levy (1991b), using brain computed tomography planimetric measurements in Alzheimer's disease patients, with and without misidentification symptoms, including misidentification of the mirrored-self. They found that patients with misidentification symptoms had significantly larger left frontal lobes, and larger right anterior horns, indicating a right frontal atrophy. The authors 'hypothesize' that misidentification in Alzheimer's disease may arise from a combination of right hemisphere atrophy and a relatively wellpreserved left hemisphere that would allow patients to verbalize their 'sense of lost familiarity'.

To our knowledge, only four patients with mirrored-self misidentification as a focal phenomenon have been reported before (Breen, Caine, & Colheart, 2001; Feinberg & Shapiro, 1989; Spanenberg, Wagner, & Bachman, 1998). The most important clinical, neuropsychological and radiological features are summarized in Table 2. All patients were old and had neuropsychological and/or radiological evidence of multifocal cerebral involvement. The description of the mirrored-self misidentification was somewhat different in every case, with two patients claiming to see themselves as an 'other self' (Feinberg & Shapiro, 1989) or as a young girl (Spanenberg et al., 1998), two patients describing a stranger in the mirror (Breen et al., 2001) and our patient seeing a relative. The influence of the environment on the appearance of the delusion is not detailed in every patient, but we can infer from the descriptions that it usually occurs in unfamiliar surroundings, like a hospital (Patient MH in Spanenberg et al., 1998) or moving to a new house (present report and Patient FE in Breen et al., 2001). Neuropsychological evaluation of the five patients disclosed signs of right hemispheric dysfuntion and, interestingly, two patients had facial processing deficits (Breen et al., 2001; Feinberg & Shapiro, 1989), and one patient mirror agnosia (Breen et al., 2001). Thus, both mirror agnosia and mild facial recognition deficits have been considered contributing factors for the mirror sign, but they are not found in every patient. In neuroradiological studies, only the patient described by Feinberg and Shapiro (1989) had an isolated finding like right temporoparietal atrophy, although they also described 'some generalized atrophy of the brain'. All the other patients had multiple lesions, most of them of vascular origin and with preferential involvement of the right hemisphere. Interestingly, our patient and patient TH described by Breen et al. (2001) had small cortical infarcts in the right frontal lobe, a region that has been involved in self-recognition in different studies (Uddin et al., 2005). Both patients had an associated reduplicative paramnesia,

Reference	Name Sex Age	Mirror sign	Neuropsychological findings	Neuroradiological findings	Diagnosis
Feinberg and Shapiro, 1989	SM / F / 77 y	Other version of herself (Capgras for her mirror image)	Focal RH impairment and mild diffuse cerebral dysfunction. Borderline performance in a task of facial recognition	MRI: mild atrophy and right temporoparietal atrophy	None provided
Spanenberg et al, 1998	MH /F / 82 y	Young girl who looked like her	RH findings	CT: Watershed infarction, particularly R posterior parietal; MRI: R basal ganglia infarcts, bilateral thalami	Mild vascular dementia
Breen et al, 2001	FE/ M/87	Stranger in the mirror after moving to a new house	RH findings; facial pro- cessing deficits	CT: Multiple periventricular lacunar infarcts. MRI: atrophy and scattererd white matter high signal intensity	Mild vascular dementia
	TH/M/77	"Dead ringer" for himself. Associated reduplicative paramnesia	RH findings; mirror agnosia	CT: normal. MRI: diffuse atrophy, ischemic white matter changes, small cortical infarct posterior right frontal lobe	Atypical AD vs. dementia with lewy bodies.
Present report	MM / M /90	Father (5 days); Father-in-law. Associated reduplicative paramnesia	RH findings	MRI: Bilateral frontal postramatic encephalomalacia. Right Dorsolateral frontal infarct	Right frontal ischemic stroke. Postraumatic brain injury

 TABLE 2

 Case reports of mirror-self misidentification in patients without global dementia

F: female; M: male. RH: Right Hemispheric; CT: computed tomography; MRI: magnetic resonance imaging; AD: Alzheimer's disease. Modified from: Spanenberg K. The mirror sign delusional misidentification symptom. In: Feinberg T, Keenan JP: The lost self. Pathologies of the brain and identity. Oxford University Press; New York. 2005.

another delusional misidentification symptom usually described in right hemispheric lesions (Benson, Gardner, & Meadows, 1976).

Interpretation

Delusions are false, sustained beliefs based on incorrect inferences about the self or environment that cannot be overturned by refuting evidence. The right hemisphere and frontal lobes are considered critical lesion sites for delusions (Devinsky, 2000). In the peculiar delusion of the mirrored-self misidentification, different mechanisms seems to be involved, but we can summarize them in four ways: (1) abnormalities in perception; (2) loss of familiarity; (3) lesion of a putative center for selfrecognition; and (4) loss of judgement and inability to correct wrong beliefs. Abnormalities in perception, like mild or moderate deficit in facial recognition, loss of visuospatial skills or mirror agnosia have been described in patients with the mirror sign as a focal symptom, and they are very common in Alzheimer's disease, the condition most frequently associated with it. These perceptual abnormalities are probably related to right hemispheric dysfunction, described in every patient with the mirroredself misidentification.

Loss of familiarity plays a crucial role in other DMS like the Capgras symptom (Devinsky, 2009). In three out of five patients the with mirror sign as a focal symptom, an unfamiliar environment was a frequent trigger, and this is also common in Alzheimer's disease delusions. It is not clear how the brain encodes familiarity, but it seems that the right hemisphere dominates in analyzing whether a stimulus is familiar or not. Impairment of this function could contribute to deficits in autobiographical memory, source memory or false recognition of faces or information (Rapcsak, Polster, Comer, & Rubens, 1994; Schacter, Curran, Galluccio, Milberg, & Bates, 1996; Tulving, Kapur, Craik, Moskovitch, & Houle, 1994), as well as delusional reduplication syndromes associated with right hemisphere lesions. In addition, the ictal hallucination of familiarity (déjà vu) is much more common with right than left temporal lobe seizure foci (Gupta, Jeavons, Hughes, & Covanis, 1983; Mullan & Penfield, 1959).

The existence of a center for self-recognition in the right prefrontal cortex is supported by different studies in healthy volunteers (Uddin et al., 2005). In our patient, the mirror sign appeared immediately after a right hemispheric stroke. There was radiological evidence of a small infarct in the right prefrontal cortex, a feature already described in another patient (Breen et al., 2001). These two cases provide further evidence for a self-recognition center in the right frontal lobe, and although we consider that a lesion in this area is not enough to elicit the mirrored-self misidentification, it probably impairs the ability to recognize one's own image in the mirror, playing a crucial role in the genesis of the delusion.

Finally, a delusion cannot be fixed and sustained in the presence of preserved judgement and ability to correct wrong perceptions or beliefs. DMS have been linked with clinical syndromes that appear in right hemisphere-injured patients, like neglect or asomatognosia (Feinberg, 1997). For example, in these patients, the wrong beliefs of the own left arm not belonging to the body become fixed despite rational arguments to the contrary. Something similar happens in patients with the mirror sign. Thus, right hemisphere and bifrontal damage could lead to the inability to revise a distorted perception or interpretation of one's own face reflected in the mirror, producing one of the most bizarre and fascinating symptoms in neurology.

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