

## Some Unusual Neuropsychological Syndromes: Somatoparaphrenia, Akinetopsia, Reduplicative Paramnesia, Autotopagnosia

Alfredo Ardila\*

*Department of Communication Sciences and Disorders, Florida International University, Miami, FL, USA*

\*Corresponding author at: Department of Communication Sciences and Disorders, Florida International University, Miami, 11200 SW 8th Street, AHC3-431B, FL 33199, USA. Tel.: +1-305-348-2750; fax: +1-305-348-2710.

*E-mail address:* ardilaa@fiu.edu (A. Ardila).

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### Abstract

Some unusual neuropsychological syndromes are rarely reported in the neuropsychological literature. This paper presents a review of four of these unusual clinical syndromes: (1) somatoparaphrenia (delusional belief in which a patient states that the limb contralateral to a brain pathology, does not belong to him/her); (2) akinetopsia (cortical syndrome in which patient loses the ability to perceive visual motion); (3) reduplicative paramnesia (believe that a familiar place, person, object, or body part has been duplicated); and (4) autotopagnosia (disturbance of body schema involving the loss of ability to localize, recognize, or identify the specific parts of one's body). It is concluded that regardless of their rarity, it is fundamental to take them into consideration in order to understand how the brain organizes cognition; their understanding is also crucial in the clinical analysis of patients with brain pathologies.

*Keywords:* Somatoparaphrenia; Akinetopsia; Reduplicative paramnesia; Autotopagnosia

There are some unusual neuropsychological syndromes rarely reported in the neuropsychological literature. Somatoparaphrenia, akinetopsia, reduplicative paramnesias, and autotopagnosia are just four examples of these unusual clinical syndromes. In this paper, an updated review of somatoparaphrenia, akinetopsia, reduplicative paramnesia, and autotopagnosia is presented. There were two different reasons to select these syndromes: (1) from the fundamental point of view the analysis of these unusual syndromes can significantly advance our understanding of the brain's organization of human cognition under normal and abnormal conditions; (2) from the clinical point of view potential diagnostic errors and misinterpretations can exist if we are not aware enough of these unusual syndromes. For instance, somatoparaphrenia can be interpreted as hemiasomatognosia; akinetopsia could be regarded as visual agnosia; reduplicative paramnesia as a psychotic delirium; and autotopagnosia as a Wernicke aphasia.

### Somatoparaphrenia

#### *Definition and Initial Description*

Somatoparaphrenia is a delusional belief in which a patient states that the limb, contralateral to a brain pathology, usually the left upper one, does not belong to him/her (Invernizzi et al., 2013). Somatoparaphrenia is typically associated with anosognosia, somatosensory disturbances, and unilateral spatial neglect. Gertsmann initially described this syndrome in 1942 with two patients with right hemisphere pathology. They presented anosognosia for their motor defect, significant somatosensory disturbances, and the delusional belief that the left hemi-body limbs did not belong to them. Gerstmann proposed the term "somatoparaphrenia" and defined it as the "illusions or distortions concerning the perception of confabulations or delusions referring to the affected limbs or side" (p. 895). During the following years, few cases of this syndrome were published, and it was sporadically mentioned in the neurological and neuropsychological literature (e.g., Hécaen, 1972).

Most of the cases of somatoparaphrenia correspond to single-case reports (e.g., Bottini, Bisiach, Sterzi, & Vallar, 2002; Halligan, Marshall, & Wade, 1995; Miura et al., 1996; Nightingale, 1982; Pugnaghi, Molinari, Panzetti, Nichelli, & Zamboni, 2012). This syndrome is not a fully comprehended disorder, and could be categorized as somewhere between neurology and psychiatry (Feinberg & Venneri, 2014).

### Anatomical Correlations

In 2009, Vallar and Ronchi carried out an extensive literature review of somatoparaphrenia including PubMed database and the review works of Schilder (1935), Lhermitte (1952), Weinstein and Kahn (1955), Critchley (1953), Hécaen and Albert (1978), Bisiach (1995), and Braun, Desjardins, Gaudelet, and Guimond (2007). They found 56 cases (25 men and 31 women) with unilateral, or mainly unilateral, hemispheric lesions and somatoparaphrenic symptoms. Fifty-one of 56 patients (91%) presented somatoparaphrenia involving the left side of the body, associated with a right-sided lesion. Two such patients also had a lesion in the left hemisphere. Furthermore, five exhibited somatoparaphrenia involving the right side of the body, associated with a left-sided lesion. The patients' verbal reports varied; some patients reported a *sense of strangeness in body parts contralateral to the lesion* and they may wonder whether the affected limb belongs to another person. In other patients, body parts feel separate from the patient's body. However, the more frequent manifestations of somatoparaphrenia, observed in over two-thirds of the patients is a *sense of disownment*: the delusional belief that body parts contralateral to the brain pathology do not belong to them, but they belong to another person.

In 2012, Gandola et al. published an analysis of the anatomical correlates of somatoparaphrenia. The authors pointed out that the only three available anatomical group studies on somatoparaphrenia are contradictory, and refer to quite different locations for the syndrome. Some proposed specific lesion correlations with the right posterior insula (Baier & Karnath, 2008), the supramarginal gyrus and the posterior corona radiata (Feinberg, Haber, & Leeds, 1990), or the right medial or orbito-frontal regions (Feinberg, Venneri, Simone, Fan, & Northoff, 2010). Gandola et al. compared 11 patients with and 11 without somatoparaphrenia matched for the presence and severity of other associated symptoms (neglect, motor deficits, and anosognosia). They found a lesion pattern involving a fronto-temporo-parietal network typically associated with spatial neglect, hemiplegia, and anosognosia. Somatoparaphrenic patients showed an additional lesion pattern primarily involving white matter and subcortical grey structures (thalamus, basal ganglia, and amygdala).

Somatoparaphrenia is usually, but not necessarily, associated with anosognosia for hemiplegia. Invernizzi and colleagues (2013) described the clinical manifestations and the lesion patterns of five patients with somatoparaphrenia, but without anosognosia for hemiplegia. All patients had extra-personal and at least mild-personal neglect. The lesion localization was mainly subcortical, with a significant involvement of the right thalamus, the basal ganglia and the internal capsule. A comparison between the anatomical pattern, previously associated with anosognosia for hemiplegia, proposed by Berti et al. (2005) (viz., denial of hemiplegia is associated with lesions in areas related to the programming of motor acts, particularly Brodmann's premotor areas 6 and 44, motor area 4, and the somatosensory cortex), to the lesion distribution of each patient was done. It was found that their somatoparaphrenic patients had a sparing of most of the regions associated with anosognosia for hemiplegia. The authors suggested that motor awareness is not sufficient to build up a sense of ownership and therefore these two cognitive abilities (motor awareness and sense of ownership) are at least in part functionally independent and qualitatively different.

### Some Additional Points

Some additional points about somatoparaphrenia should be mentioned. Fotopoulou and colleagues (2011) assessed the sense of body part ownership in somatoparaphrenic patients when they saw the body in a mirror, that is, from a third-person perspective. They tested five patients with right hemisphere lesions with left hemiplegia and neglect, including two patients with this somatoparaphrenic delusion. The somatoparaphrenic patients systematically attributed the ownership of their left plegic hands to someone else in direct view, but showed a statistically significant increase in ownership of the left hand in mirror view trials, when compared with the three control patients. Depending on the view offered (mirror or direct), judgments of ownership and disownment of the left limb alternated. The patients did not seem particularly concerned by these alternations. Conditions of direct-mirror view simultaneously touching the patient's hand showed the same pattern of results as conditions without touch. The authors suggested that a neural network involving the perisylvian areas of the right hemisphere may be necessary for the integration of multiple representations of one's body, as well as for a higher order re-representation of various bodily signals into a first-person sense of body ownership. Romano, Gandola, Bottini, and Maravita (2014) found that anticipatory skin conductance responses to noxious stimuli directed to the contralesional hand are significantly reduced when compared with noxious stimuli directed to the ipsilesional hand in patients with somatoparaphrenia.

In addition, temporary remission through vestibular stimulation has been reported (Bisiach, Rusconi, & Vallar, 1991; Rode et al., 1992), but follow-up studies of somatoparaphrenia are rare. Cogliano, Crisci, Conson, Grossi, and Trojano (2012) described exceptionally long-lasting somatoparaphrenia in two focal brain-damaged patients. The presence and severity of somatoparaphrenia did not change in either patient during a 2-year follow-up, whereas the two patients showed different evolution of anosognosia for motor disorders, severity of extra-personal neglect, and cognitive impairments. The authors propose that somatoparaphrenia can be observed as a body-related chronic disorder and can outlast other cognitive defects, even if it arose in conjunction with them.

It is important to pinpoint the difference between somatoparaphrenia and hemiasomatognosia. Feinberg and colleagues (2010) proposes that asomatognosia refers to “unawareness of ownership of one’s arm, while somatoparaphrenia is a subtype in which patients also display delusional misidentification and confabulation” (p. 276). So, the major difference between both refers to the delusional aspect of somatoparaphrenia, and consequently, somatoparaphrenia can be interpreted as an extreme and unusual subtype of asomatognosia (Loring, 2015).

#### *How This Syndrome Provides Further Insight into the Brain*

Somatoparaphrenia can be interpreted as a syndrome in between neurology and psychiatry (Feinberg & Venneri, 2014). In this regard, the analysis of somatoparaphrenia may contribute to further our understanding about the neurological bases of psychosis.

#### *Conclusion*

Somatoparaphrenia represents an unusual delusional syndrome involving a disturbance in the body image. Up until 2009, only 56 cases had been reported (Vallar & Ronchi, 2009). This low frequency can be partially due to the fact that in some occasions this syndrome is interpreted as hemiasomatognosia. Somatoparaphrenia is usually, but not necessarily, associated with anosognosia for hemiplegia. Some disagreement about its specific location remains. Somatoparaphrenia can be particularly important to understand the neurological bases of delusional ideas.

### **Akinetopsia**

#### *Definition and Initial Description*

Akinetopsia is an unusual cortical syndrome in which patients lose the ability to perceive visual motion following an extrastriate cortical lesion (Zeki, 1991). This disorder was initially described by Zihl, Von Cramon, and Mai (1983) in a patient who presented with mild anomia due to a bilateral extrastriate vascular lesion. No other impairments were noted and no scotoma was observed. Regardless of the inability to discriminate objects in motion, her ability to perceive static forms and colors was intact. Interestingly, perception of auditory motion was relatively normal.

This syndrome was rapidly accepted and introduced into the neurological and neuropsychological literature. The term akinetopsia (or cerebral akinetopsia) was later coined by Zeki (1991) (Zihl, & Heywood, 2015). During the following years, several reports of akinetopsia were published (e.g., Cooper et al., 2012; Horton, 2000; Otsuka-Hirota, Yamamoto, Miyashita, & Nagatsuka, 2014; Zihl, Von Cramon, Mai, & Schmid, 1991), corroborating the clinical characteristics initially described by Zihl et al.

#### *Anatomical Correlations*

In addition to focal lesions, akinetopsia has been observed in different conditions, such as posterior cortical atrophy (Tsai & Mendez, 2009), associated with the antidepressant nefazodone (Horton & Trobe, 1999), and as a paroxysmal epileptic phenomenon (Sakurai, Kurita, Takeda, Shiraishi, & Kusumi, 2013). Beckers and Homberg (1992) reported that the perception of visual motion can be selectively and reversibly compromised by transcranial magnetic stimulation (TMS) of a small region of cortex, roughly 1 cm in diameter and corresponding in position to human area V5. In contrast to the complete and transient visual motion blindness, which occurs with stimulation of V5, a less-significant interference with the perception to visual motion occurred between 70 and 80 ms after the onset of the visual stimulus, when TMS was applied to V1.

Akinetopsia, in patients after lesions such as strokes, usually do not persist, probably because diverse cortical areas participate in human perception (Shipp, Jong, Zihl, Frackowiak, & Zeki, 1994). However, at least one case has been reported of an acute transient akinetopsia following a stroke, where a patient showed evidence of abnormal motion vision for 23 years after his stroke (Cooper et al., 2012).

### Some Additional Studies

Akinetopsia has some similarities with the so-called *Zeitraffer* phenomenon; this phenomenon refers to an altered perception of the speed of moving objects, described in a series of German-language case reports since the early 20th century (Ovsiew, 2014; Tonkonogy & Puente, 2009). Recently, Ovsiew (2014) reported the case of a man with the illusory experience of decelerated motion, following a large right temporal hematoma. The author proposed that the *Zeitraffer* phenomenon probably arises from certain dysfunction of brain networks responsible for the visual perception of speed. This dysfunction shares the characteristics of visual impairment to perceive motion, with akinetopsia.

### How This Syndrome Provides Further Insight into the Brain

Understanding the neurological bases of motion perception has not been easy (Gibson, 2014). The description of akinetopsia represents a crucial step in the comprehension of how motion perception is organized in the brain. Noteworthy, the perception of biological motion (visual phenomenon of a moving, animate object) stimuli may also be impaired by focal brain damage. Biological motion blindness has been proposed to be associated with parietal and temporal lobe damage (Blanke et al., 2007).

### Conclusion

Akinetopsia represents an unusual cortical syndrome described relatively recently (Zihl et al. 1983). Very few cases have been reported up to date. However, it has been critical in furthering our understanding of the brain organization of visual perception.

## Reduplicative Paramnesias

### Definition and Initial Description

Reduplicative paramnesia was first described by Pick (1903). It is characterized by the belief that a familiar place, person, object, or body part has been duplicated (Hakim, Verma, & Greiffenstein, 1988). Most reported cases refer to duplication of places; that is, the hospital, the city, the patient's house, or other geographical points are duplicated (Benson, Gardner, & Meadows, 1976; Filley & Jarvis, 1987; Patterson & Mack, 1985).

### Anatomical Correlations

Murai, Toichi, Sengoku, Miyoshi, and Morimune (1997) selected 77 patients with focal brain damage (47 with left hemispheric, 21 with right hemispheric, and 9 with bilateral damage) and were assessed for the presence of reduplicative paramnesia using a questionnaire. Two patients showed typical reduplicative paramnesia for place, and four patients showed atypical reduplicative paramnesia (three for place and one for person). In three patients, the lesions were situated in the right hemisphere; in two, the lesions were bilateral (right dominant); and in one, the lesions were in the left hemisphere. Other reports clearly support the finding that reduplicative paramnesia is very specially associated with right hemisphere (e.g., Hakim et al., 1988; Kapur, Turner, & King, 1988; Lee, Shinbo, Kanai, & Nagumo, 2011; Moser, Cohen, Malloy, Stone, & Rogg, 1998) or bilateral (Joseph, O'Leary, Kurland, & Ellis, 1999; Pisani, Marra, & Silveri, 2000) lesions.

### Some Additional Studies

It is important to bear in mind that there is a whole group of disorders known as "delusional misidentification syndromes," where patients think that a particular familiar person is someone else or a certain familiar place is a duplicate (Atta, Forlenza, Gujski, Hashmi, & Isaac, 2006; Ellis, Luauté, & Retterstøl, 1994; Hudson & Grace, 2000). Delusional misidentification syndromes are found in various psychotic and organic brain diseases. In addition to the *reduplicative paramnesia*, they include the *Capgras syndrome* (Ardila & Rosseli, 1988; Capgras & Reboul-Lachaux, 1923) consisting of the belief that one or more people (usually close relatives) have been substituted by imposters or doubles; the *Frégoli syndrome* (or "the delusion of doubles") characterized the delusional belief that one or more familiar persons, usually persecutors following the patient, repeatedly change their appearance; that is, different people are in fact a single person who changes appearance (Courbon & Fail, 1927; Mojtabai, 1994). Additionally, "intermetamorphosis" is characterized by the belief that the patient can perceive that an individual has been transformed both psychologically and physically into another person (Courbon & Tusques, 1932; Silva, Leong, Shaner, & Chang, 1989). Therefore, there exists the "doubles of the self" or "subjective doubles" syndrome consisting of the belief that the patient has a double

with the same appearance, but usually with different character traits, who is leading a life of its own (Christodoulou, 1991). Important to note, Capgras syndrome can be interpreted as a reduplicative paramnesia (Alexander, Stuss, & Benson, 1979; Staton, Brumback, & Wilson, 1982).

These syndromes have been reported both in psychiatric and neurological literature, and each one of them can appear in either psychiatric or neurological pathologies (Cipriani et al., 2013). Forstl, Almeida, Owen, Burns, and Howard (1991) analyzed 260 case reports of misidentification syndromes. One hundred and seventy-four patients had a Capgras syndrome misidentifying another person, 18 had a Fregoli syndrome, 11 had intermetamorphosis, 17 had reduplicative paramnesia, and the rest had other forms or combinations of mistaken identification. Schizophrenia (127 cases), mostly of paranoid type, affective disorder (29), and organic mental syndromes including dementia (46) were the most common diagnoses in patients who misidentified others or themselves. The patients with reduplicative paramnesia frequently suffered from head trauma or cerebral infarction and showed more features of right hemisphere lesions on neuropsychological testing or CT scan, than the patients with other misidentification syndromes. The authors concluded that the misidentification of persons can be a manifestation of any organic or functional psychosis, but the misidentification of place, usually found in reduplicative paramnesia, is often associated with neurological diseases, predominantly in the right hemisphere damage.

Fleminger and Burns (1993) reviewed two series, each of 50 cases of delusional misidentification reported in the literature. The first series of cases included delusional misidentification either of place, and/or of persons; the second series was limited to cases, published since 1977, with delusional misidentification of person, who had had an EEG and/or CT scans. Paranoid delusions, preceding the onset of the delusional misidentifications, were more common in cases without evidence of organic cerebral disorder. In the second series, there was good evidence of an inverse relationship between the presence of paranoid delusions preceding the delusional misidentification, and the intensity of organic cerebral disorder. While in the first series, delusional misidentification of place (reduplicative paramnesia) was more common in cases with evidence of organic brain disorder, whereas delusional misidentification of person was more common with functional mental disorder.

#### *How This Syndrome Provides Further Insight into the Brain*

Reduplicative paramnesia as well as other “delusional misidentification syndromes” can provide clues about the brain organization, not only of visual perception and memory but also delusional misperception and psychotic thought.

#### *Conclusion*

Reduplicative paramnesia is a delusional memory disorders most frequently characterized by reduplication for place, and often associated with right hemisphere or bilateral pathology. It can be concluded that reduplicative paramnesia usually represents condition associated with brain pathology and not a psychiatric condition, even though its interpretation is still controversial (Politis & Loane, 2012).

### **Autotopagnosia**

#### *Definition and Initial Description*

Autotopagnosia, initially described by Pick (1908), is usually defined as the disturbance of body schema involving the loss of ability to localize, recognize, or identify the specific parts of one's body (Mendoza, 2011). Patients are able to point to parts of objects, plants and animals on command; however, they face difficulty when asked to locate their body parts in relation to the whole body (Ogden, 1985). Patients' difficulties seem to be body specific; trouble at localizing single parts of objects, suggesting a disturbance in the conceptual representation of individual body parts (Semenza, 1988). Patients can also have difficulty codifying/identifying the position of body parts relative to each other and fail to update the position of body parts after passive movements (Schwoebel, Branch Coslett, & Buxbaum, 2001). Interesting to note, autotopagnosia is ameliorated by looking at the image reflected in a mirror (Tobita, Hasegawa, Nagatomo, Yamaguchi, & Kurita, 1995).

#### *Anatomical Correlations*

Autotopagnosia is usually associated with left posterior parietal lesions (Corradi-Dell'Acqua, Hesse, Rumiati, & Fink, 2008; Laiacina, Allamano, Lorenzi, & Capitani, 2006; Ogden, 1985), but at least one case was associated with a left subcortical vascular accident (Guariglia, Piccardi, Allegra, & Traballese, 2002). Although a case of autotopagnosia was noted following a right hemisphere lesion, this patient was right hemisphere dominant for language (Denes, Cappelletti, Zilli, Dalla Porta, & Gallana, 2000).

Autotopagnosia has also been reported in Alzheimer's disease (Tariska & Urbanics, 1995). Using functional magnetic resonance imaging to identify the neural mechanisms underlying the body structural description, Corradi-Dell'Acqua and colleagues (2008) suggested that damage in the left posterior intraparietal sulcus may underlie autotopagnosia.

Gainotti, Caltagirone, Carecchi, and Ibba (1975) selected 120 patients with unilateral cerebral hemisphere damage (54 aphasics, 21 non-aphasic left and 45 right brain-damaged patients and 57 control subjects without cerebral lesions; and administered a battery of verbal and non-verbal tests of autotopagnosia, with the aim of studying the relationships between disorders of body schema and the side of the hemispheric lesion. Results showed that (1) the aphasic patients were significantly more impaired than the non-aphasic patients on all the tests, non-verbal as well as verbal; (2) when results obtained on tests of naming and of pointing to parts of the human body were matched with results obtained on tests of naming and of pointing to parts of objects other than the human body, no difference could be demonstrated. The authors concluded that autotopagnosia is a nonspecific symptom.

### *Some Additional Studies*

The explanation of autotopagnosia has been polemic. Sirigu, Grafman, Bressler, and Sunderland (1991) that multiple, partially independent systems are involved in body knowledge; they suggest deficits in visual structural descriptions of the human body and its parts, in the context of spared semantic and proprioceptive-spatio-motor body representations. Buxbaum and Branch Coslett (2001) suggest that human body part localization depends upon structural descriptions of human (but not animal) bodies that enable viewpoint-independent body part recognition and participate in the calculation of equivalence between the body parts of self and others across transformations in orientation. Felician, Ceccaldi, Didic, Thinus-Blanc, and Poncet (2003) suggest that left superior and inferior parietal regions are parts of networks involved in the respective processing of somatosensory and visuospatial representations of bodies.

Moreover, Degos and Bachoud-Levi (1998) described a disorder similar to autotopagnosia characterized by the inability to designate targets situated outside the body. The authors named this disorder "allotopagnosia"; it occurs exclusively in subjects with a lesion involving the posterior region of the left parietal lobe. The most common manifestation is the designation of parts of the body of another person as being part of the patients' own body (heterotopagnosia with self-designation). Patient's expression of the confusion between their own body with that of others and the gestures they use to indicate a relationship with reality (non-self and target) was confined to the human species. They hypothesized that allotopagnosia results from a deficit or dysfunction of the left parietal lobe, where outside elements are attributed to situations and identities independent of self.

### *How This Syndrome Provides Further Insight into the Brain*

The analysis of autotopagnosia can significantly advance our understanding of the brain organization of cognition not only by illustrating the brain representation of own body image (Cash & Pruzinsky, 2004) but also by advancing the understanding of the organization of language semantics. Contemporary clinical and neuroimaging studies have corroborated that different semantic categories are differentially impaired in cases of brain pathology. For instance, in anomia it has been traditionally recognized that naming body parts, external objects, and colors depend (and are altered) on the activity of different brain areas (e.g., Hécaen & Albert, 1978). It has also been found that naming impairments can be limited to a rather specific semantic category (e.g., people's names, living things, tools, geographical names, etc.) (e.g., Goodglass, Wingfield, Hyde, & Theurkauf, 1986; Harris & Kay, 1995; Lyons, Hanley, & Kay, 2002; Warrington & Shallice, 1984), and even as specific as "medical terms" (Crosson, Moberg, Boone, Rothi, & Raymer, 1997). Autotopagnosia includes not only a defect in naming body parts but also localizing, recognizing, or identifying the specific parts of one's body. It is a naming defect and it is also a body-schema impairment.

### *Conclusion*

Although autotopagnosia was described over a century ago, research has been limited. Autotopagnosia is generally associated with left posterior parietal lesions and can be regarded as a syndrome between a body agnosia (asomatognosia) and a language (anomia) defect.

### **General Conclusions**

In neuropsychology—as in any clinical area—there are some unusual syndromes that are only sporadically found. But their rarity does not diminish their importance in the fundamental understanding about the brain organization of cognition; and in

the clinical analysis of patients with brain pathologies. The four clinical syndromes analyzed before illustrate these types of unusual neuropsychological syndromes.

Somatoparaphrenia and reduplicative paramnesia are particularly interesting not only because of their rarity but also because they represent two borderline syndromes between neurology and psychiatry. They could be regarded as “neuropsychiatric syndromes.” Somatoparaphrenia supposes the belief of something impossible and absurd (that—usually—the right arm does not belong to the patient and has been replaced by an artificial or somebody else’s arm), or in other words a delusional belief. It could be regarded as a kind of acquired psychotic thought resulting from a specific brain pathology. It is probably the neuropsychological syndrome most alike to a psychiatric condition; and consequently, it represents the neuropsychological syndrome that could potentially best contribute to the understanding of the neurological bases of psychosis.

Reduplicative paramnesia is just one of the several “delusional misidentification syndromes” characterized by the belief that a particular familiar person is someone else or a certain familiar place is a duplicate (Atta et al., 2006; Ellis et al., 1994; Hudson & Grace, 2000). Delusional misidentification syndromes are found in various psychotic and organic brain diseases, and hence, can also provide clues about the neurological bases of psychosis. As mentioned above, it has been suggested that some right hemisphere brain abnormalities could represent the neurological bases of psychosis (see Feinberg & Roane, 2005; Ramachandran 1995). Interestingly, these two psychotic-like neuropsychological syndromes (somatoparaphrenia and reduplicative paramnesia) are both found in cases of right hemisphere pathology, especially right parietal pathology.

Akinetopsia has a very special place among the neuropsychological syndromes. First, it was described very late in history (most of the neuropsychological syndromes were described during the 19th century or early 20th century). Second, despite of its rarity, it was rapidly accepted and integrated in the neurological and neuropsychological literature. Frequently, when a clinical syndrome is initially described, it takes some time to be accepted; and even after decades controversies can remain, as it is observed with the Gerstmann syndrome (Ardila, 2014; Benton, 1961; Gerstmann, 1940). The description of akinetopsia significantly contributed to advance the understanding of visual perception.

Furthermore, autotopagnosia is unique and special because it represents a borderline syndrome between a language disorder (aphasia) and body perception impairment (asomatognosia). Autotopagnosia is not the only borderline syndrome in neuropsychology. For instance, conduction (afferent motor) aphasia could be interpreted as a borderline syndrome between aphasia and apraxia (Luria, 1976); constructional apraxia could be regarded as a borderline syndrome between apraxia and agnosia (“apractognosia” syndrome; Lange, 1936); transcortical (extrasyllvian) motor aphasia could be interpreted as a borderline syndrome between aphasia and a dysexecutive disorder (Ardila, 2010); and so forth. It is clear that the analysis of autotopagnosia can positively contribute to further understanding not only the brain organization of body schema but also the semantic organization of language.

In conclusion, the analysis of unusual syndromes in neuropsychology can significantly contribute not only to having more precise neuropsychological diagnoses but also to significantly advancing our understanding of the brain organization of cognition in normal and abnormal conditions.

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