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Delusional Misidentification

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The delusional misidentification syndrome (DMS) is a condition in which a patient consistently misidentifies persons, places, objects, or events. The most common form of misidentification is the Capgras syndrome. Originally described by Capgras and Reboul-Lachaux [1], this disorder consists of the delusional belief that a person or persons have been replaced by "doubles" or imposters. Another type of misidentification is the Frégoli syndrome [2] in which there is the belief that a person who is well known to the patient is actually pretending to take on the appearance of a relative stranger whom the patient encounters. Vié [3] characterized the Capgras syndrome as the illusion of negative doubles and Frégoli syndrome as the illusion of positive doubles. Christodoulou [4,5] suggested that Capgras syndrome is a "hypo-identification" of a person closely related to the patient, whereas Frégoli syndrome is a "hyperidentification" of a person not well known to the patient. Feinberg and Roane [6–9] concluded that different delusional misidentification syndromes can be separated on the basis of the nature of the changed personal relatedness between the self and other persons, objects, and events. When this approach is used, Capgras syndrome becomes an example of underpersonalized misidentification, and Frégoli syndrome represents overpersonalized misidentification.

Some patients who have DMS reduplicate or double the misidentified entity. For example, a patient with Capgras syndrome may deny the identity of the actual spouse and claim that there are two spouses, the actual and the imposter. Not every patient with DMS reduplicates the misidentified entity, however. For example, in the syndrome of intermetamorphosis, the patient believes that people he or she knows have exchanged identities with each other [10]. Conversely, some patients may claim the existence of a fictitious

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person or place, often a double of an actual person or place, without any actual misidentification.

The Capgras-type delusion generally has been recognized in the context of psychiatric disorders and often occurs in conjunction with paranoia, derealization, and depersonalization [11–14]. The symptom has been reported in association with a variety of diagnostic entities including schizophrenia, mood disorders, Alzheimer's disease, and other organic conditions.

Capgras syndrome is common. The frequency in schizophrenia has been reported to be as high as 15% [15], with the rate in all psychiatric inpatients ranging from 1.3% to 4.1% [16–17]. Studies in patients who have Alzheimer's disease have demonstrated prevalence between 2% and 30% [18–19].

A broad range of medical and neurologic disorders may manifest with misidentification and reduplication in general. In a review of delusional reduplication in the literature, Signer [20] found various medical comorbidities including drug intoxication and withdrawal, infectious and inflammatory disease, and endocrine conditions. Associated neurologic illnesses included seizures, cerebral infarction, and traumatic brain injury. Forty percent of patients who have identifiable organic conditions had diffuse brain syndromes such as delirium, dementia, and mental retardation. DMS has been seen in association with parkinsonism [21] and, in particular, with dementia with Lewy bodies [22]. Ballard [22] reported that DMS occurred more frequently with Lewy body dementia than with Alzheimer's dementia. Iseki and colleagues [23] found that multiple forms of DMS were common in both dementia with Lewy bodies and levodopa-induced psychosis. DMS may also present as an adverse consequence of electroconvulsive therapy [24] and has been reported in patients receiving diazepam and disulfiram [25].

This article focuses on the relationship between DMS and neurologic illness.

Delusional misidentification and brain pathology

A review of the range of DMS phenomena that has been reported in the context of localized brain pathology elucidates the neuropsychiatric aspects of this condition.

Capgras-type misidentifications

Alexander and colleagues [26] reported one of the first cases of Capgras syndrome for persons occurring in the setting of focal brain lesions. They described a 44-year-old man who sustained a traumatic brain injury and right frontotemporal encephalomalacia. This patient claimed that his wife and five children had been replaced by substitutes. Another instance of this disorder involved a 31-year-old man who, years after a traumatic brain

injury that resulted in right frontotemporal and parietal damage, claimed that his parents, siblings, and friends were not "real" but were "look-alikes" or "doubles" [27].

Patients have also developed Capgras-type misidentification for their homes. Kapur and colleagues [28] described a patient who claimed his actual home was not his "real" home, although he recognized many ornaments and bedside items as original. Moser and colleagues [29] reported an elderly man with an acute right frontal infarct who called his real home the "twin" of the original.

One further applicable condition is asomatognosia [30,31]. In this syndrome, a patient repeatedly misidentifies a part of the patient's body. Asomatognosia occurs most commonly in a patient with left hemiplegia, caused by a right hemisphere lesion, who denies ownership of the left arm. It typically co-occurs with hemispatial neglect. Early on, Jacques Vié [3,32] recognized that some syndromes, like asomatognosia, although apparently caused by neurologic illness, could not be accounted for simply by confusion. He noted that asomatognosia involved systematic and selective misidentification and resembled delusional syndromes like Capgras syndrome. More recently, Feinberg and Roane [7,9,33] have viewed asomatognosia as a form of Capgras syndrome for the arm in which there is a loss of personal relatedness to the body part. As in Capgras syndrome, in which a person is recognized, but their psychologic identity is not acknowledged, the patient with asomatognosia may be aware that the arm should be his or her arm but denies ownership of the limb.

Asomatognosia generally is seen in association with anosognosia for hemiplegia [34], in that the patient seems unaware of or persistently denies the paralysis of the arm. Both entities appear more frequently after right hemisphere lesions. Anosognosia and asomatognosia are dissociable, however, because not all anosognosic patients deny ownership of the limb, and not all asomatognosic patients deny illness or even hemiplegia.

Patients who have asomatognosia may attribute ownership of the limb to the examining doctor. This simple misattribution often can be reversed when the error is demonstrated to the patient. In other patients, the misidentifications are truly delusional, and patients maintain a fixed belief in the misidentifications when they are confronted with evidence of their errors. Patients may even develop special names for their arms that can be interpreted as personifications [35] or metaphors [36,37]. Descriptions of the arm such as "a piece of rusty machinery" or "my dead husband's hand" communicate an altered relationship between the patient and the arm.

Frégoli-type misidentification

The literature includes numerous reports of patients who have brain lesions who develop Frégoli syndrome for persons or places. Ruff and Volpe

[38] described a woman who, after the removal of a right frontal subdural hematoma, claimed that the patient in the bed next to her was her husband. She noted with satisfaction that her husband no longer snored. Feinberg and colleagues [39] described a man who developed numerous Frégoli-type misidentifications at a rehabilitation hospital after suffering a traumatic brain injury with right frontal and left temporoparietal contusions. He insisted that staff members and other patients at the facility were actually his sons, his in-laws, and his coworkers. He identified an ice skater performing on television as himself.

Patients who misidentify their current unfamiliar environment, such as a hospital room or rehabilitation hospital, for a place of close personal significance, such as a home or job site, may be said to have Frégoli syndrome for place. The syndrome was originally described by Pick [40] in 1903 as reduplicative paramnesia, because patients maintained they were in both a correct and an incorrect location, and the disorder was seen with memory impairment. Because many patients have misidentification for place without reduplication, the term Frégoli syndrome for environment is a better descriptor.

Reduplication without misidentification

Another group of delusional confabulatory patients reduplicate persons or places but do not misidentify the reduplicated entities. Weinstein and colleagues [41] described several patients who had brain lesions and believed that they were the parents of a fictitious child; the investigators termed the condition the ''phantom child syndrome.'' They noted that a recurring aspect of the delusion was that ''patients often ascribed to the 'phantom child' the same illness or disability that they themselves had.'' For example, a woman with a pituitary tumor and blindness claimed she had a child who was ''sick and blind,'' and a young soldier with a head injury and bilateral leg weakness claimed he had a 3-year-old ''daughter'' who had developed lower limb paralysis from polio. In some cases, the problems of the ''phantom child'' reflected personal issues of the patient unrelated to the neurologic illness. For instance, a woman who experienced the hospital nurses as abusive believed that she had a baby whom the staff had ''harmed and even killed.''

An additional case described by Feinberg [6,8] involved a 63-year-old man with a ruptured anterior cerebral artery aneurysm and bilateral frontal infarctions. On neuropsychologic testing, the patient displayed evidence of dysfunction of attention, memory, and executive functioning. He was anosognosic for his impairments, and he denied being a patient in the hospital, declaring that he was ''a guest'' with the ''optimists club.'' The patient had fathered three children. While in the hospital he developed the delusion that he had an adopted child who was undergoing an evaluation and was being judged unfairly.

Neuroanatomic pathology

A number of investigators have studied the neuroanatomic correlates of DMS. Joseph [42] reported that 16 of 29 personally examined patients with misidentification for person had abnormal CT scans with bilateral cortical atrophy, including bifrontal atrophy (in 88%), bitemporal atrophy (in 73%), and biparietal atrophy (in 60%). Weinstein and Burnham [43] found that bilateral and diffuse brain involvement, with right hemispheric predominance, were the most typical lesion patterns. Feinberg and Shapiro [44] reviewed the anatomic correlates in a selected series of case reports of patients with misidentification-reduplication. They found that bilateral cortical involvement occurred frequently (in 62% of patients who had Capgras syndrome and in 41% of patients who had reduplication). In considering cases in which cerebral dysfunction was unilateral, they found that right hemispheric predominance in reduplication was highly significant (52% right hemisphere versus 7% left hemisphere), with a statistical trend for more frequent right hemispheric damage in the smaller number of patients who have Capgras syndrome (32% right versus 7% left). Förstl and coworkers [45] reviewed a diverse group of misidentification cases and found right-sided abnormalities in 19 of 20 patients with focal lesions on brain CT scans. Based on three cases of head trauma, Benson and associates [46] proposed a relationship between reduplicative paramnesia and bifrontal impairment in concert with damage to the posterior portion of the right hemisphere. In a prospective study of 50 alcoholic patients, Hakim and coworkers [47] found that three of four reduplicators had acute right hemispheric lesions. They assumed all patients to have chronic bifrontal damage based on chronic alcohol use and neuropsychologic test results. Fleminger and Burns [48] compared right versus left hemispheric asymmetries in CT scans of patients who had misidentification. In one selected group, asymmetry was found, with greater right hemispheric damage in the occipitoparietal area. In the analysis of a second group of patients, greater right hemisphere damage could be detected in frontal, temporal, and parietal lobes.

Forstl and colleagues [49] found that patients who had Alzheimer's disease and misidentification had greater atrophy in the right frontal lobe than did demented controls. Another study involving patients who had Alzheimer's disease demonstrated that those who had DMS had significantly greater hypometabolism in bilateral orbitofrontal and cingulated regions on positron emission tomography than those who did not have DMS [50].

Case review

To understand further the central aspects of delusional misidentification after focal brain lesions and to clarify the underlying neuropathology of these disorders, the authors recently conducted an analysis of a series of previously published DMS cases plus one unpublished case [51].

Case reports included in this analysis involved patients who demonstrated persistent misidentifications or reduplications of either the Capgras or Frégoli type in the context of focal brain illness. Case descriptions had to provide sufficient clinical and neuroanatomic information to permit analysis. There were 29 applicable reports of DMS involving 27 different individuals (two patients had more than one form of misidentification). In general, all cases were easily divided into Capgras- and Frégoli-type misidentifications, and virtually all delusions involved the patients themselves or highly significant others.

The authors examined the main effects for hemisphere and brain regions. In all 29 cases, the lesion affected the right hemisphere; only 15 (51.7%) involved left hemisphere damage. When cases with bilateral lesions were excluded, there were 14 (48.3%) cases with right hemisphere damage only and no cases with left hemisphere damage only. These data indicate that DMS is strongly associated with right hemisphere damage.

The injury was limited to the frontal lobe in 34.5% of cases. In no case did parietal or temporal damage alone produce DMS. The association between frontal lobe damage and DMS was statistically significant. Consistent with the finding of predominantly right hemisphere pathology, almost all patients who had neuropsychologic testing evidenced a pattern of memory (primarily nonverbal), perceptual, and executive impairments.

Features of delusional misidentification syndrome

Cases of delusional misidentification associated with brain disease share a number of characteristic features that distinguish these conditions from simple unawareness, confusion, and confabulation in general.

Alterations in entities of personal significance

Misidentifications and reduplications, almost universally, involve delusions concerning entities of great personal significance such as one's body, family, current location, and job situation.

Selectivity and consistency

Patients persistently misidentify the same particular aspects of the self and environment. This persistence makes it unlikely that most cases of DMS can be attributed to general impairment in memory or perception.

Lack of awareness or minimization of illness, functional loss, or personal problem

Many cases of delusional misidentification or reduplication manifest with denial or anosognosia. For patients who have Frégoli-type DMS, the delusions themselves often allow patients to view their situation as being

better than it actually is. Thus, patients preferentially locate themselves in pleasant, familiar surroundings such as at home or at work, when actually they are confined in a hospital or rehabilitation setting. If they acknowledge being in a hospital, they tend to believe that the hospital is close to home. Thus, Ruff and Volpe [38] have identified possible wish-fulfilling aspects of the delusions of several patients who have DMS.

Resistance to correction

Misidentifications are fixed, false beliefs and, therefore, represent true delusions. Even when patients are confronted repeatedly with the illogical nature of the delusion, they maintain their position. Indeed, patients may demonstrate implicit or explicit awareness of their true situation [6,7,9, 38,52,53]. This feature distinguishes DMS from other forms of confabulation (eg, amnestic confabulation) and from unawareness in general. Clearly any causal explanation of DMS must consider the delusional nature of this condition.

Right hemisphere dysfunction

Right frontal dysfunction predominates in focal and diffuse cases of DMS regardless of the type of delusional misidentification or reduplication.

Explanations for delusional misidentification syndrome

Spatial disorientation

Spatial disorientation has been noted to be a causative factor in the production of reduplicative paramnesia [36,38] and is probably associated with virtually any case of delusional misidentification for place. Indeed, visuospatial disorders including hemispatial neglect were noted in nearly all cases in the authors' series, including misidentification for persons. Visuospatial disorientation alone cannot explain the selectivity and delusional quality of DMS, however.

Anatomic disconnection

Some theories have attributed DMS, especially Capgras syndrome for persons, to anatomic disconnection. Alexander and colleagues [26] argued that a deep right frontal lesion could disconnect temporal and limbic regions functionally from the damaged frontal lobe. This disconnection could result in a disturbance in familiarity of people and places and, in the presence of frontal pathology, could lead to an inability to resolve the cognitive conflict. Staton and colleagues [27] proposed that disconnection of the hippocampus from other parts of the brain important for memory storage could prevent association of new information with previous memories, leading to reduplication.

Some investigators have suggested that Capgras syndrome is caused by visuoanatomic disconnection. Ellis and Young [54], based on the finding that some patients who have prosopognosia have covert (emotional) but not overt recognition of faces [55,56], suggested that patients who had Capgras syndrome have overt recognition of faces without the appropriate emotional reaction. The covert, emotion-laden form of recognition is subserved by a "dorsal route" that runs between the visual cortex and the limbic system through the inferior parietal lobule. A lesion in the dorsal system would allow explicit recognition without the feeling of familiarity and, they suggest, could produce Capgras syndrome. Similarly, Hirstein and Ramachandran [57,58] suggested that a disconnection between the inferotemporal cortex and the amygdala could allow the patient to identify faces correctly but not experience the appropriate emotion connected to familiar faces, leading to delusional misidentification.

In the authors' series of reviewed cases, within the right hemisphere both the temporal and parietal lobes were intact in a majority of the cases of Capgras syndrome for person or place. On the other hand, all eight cases of Capgras syndrome for persons or places had nondominant frontal lesions. Therefore, if anatomic disconnection is important, it seems that a disconnection of nondominant frontal structures, as suggested by Alexander and colleagues [26], is most likely to play a role in the origin of Capgras syndrome.

Theories such as that of Ellis and Young [54] and Hirstein and Ramachandran [57] emphasizing visuoanatomic disconnection do not account for most cases of Capgras syndrome, because this disorder generally is not limited to the visual modality. In fact, Dietl and colleagues [59] described a case in which a mother developed Capgras syndrome for her daughter during a time that the two had no direct contact. Shah and colleagues [60] have shown through functional imaging that the retrosplenial cortex may be a single anatomic locus for both visual and auditory recognition of familiarity. Still, as Ellis and Lewis [61] note, faulty perceptual experience does not account for the delusional nature of the Capgras syndrome.

When the range of DMS phenomena is considered, explanations that emphasize anatomic disconnection would seem to apply best to the Capgras-type delusions. In these cases, an inability to match current experience with autobiographical memories could result in the underidentification of people and places. Frégoli syndrome, however, is a disorder of overidentification characterized by the confabulation of imaginary resemblances between the misidentified entity and the original. How could a disconnection of current experience from premorbid memory explain overrelatedness to one's environment?

A partial answer to this latter question may come from Rapcsak and colleagues [62] who described a patient without prosopagnosia who displayed false recognition (overidentification) of faces following the

surgical removal of a right prefrontal lesion. They attributed the patient's pattern of impairment to an intact reflexive face-recognition system but an impaired reflective or strategic face-processing system, leading this patient to mistake an unknown face for one in memory. This kind of defect might explain some instances of visual overidentification of faces. This account still does not explain selectivity, refractoriness, delusional nature, or multimodality.

Memory, executive impairment, and confabulation

In addition to the perceptual disturbance seen in the case review, memory and executive impairments were found in nearly all cases in which these functions were assessed. In two other cases of misidentification for the mirrored image reported by Breen and colleagues [63], only one patient had significant face-processing deficits, but both had visual memory and executive dysfunction. Because many of the current cognitive and neuropsychologic theories of confabulation also involve memory impairment and executive dysfunction [64–67], the possibility of a relationship between DMS and confabulation must be considered. Because patients who have Frégoli-type DMS claim familiarity with unfamiliar persons or places, and patients who confabulate may mistake past for current environmental stimuli [68], it is relevant to consider whether explanations of confabulation account for aspects of DMS.

Confabulation has been defined as the production of erroneous statements made without a conscious effort to deceive [69] or "statements or actions that involve unintentional but obvious distortions" [70]. Korsakoff [71] first discovered that amnesia and confabulation tend to co-occur ("pseudoreminiscences"). Numerous subsequent authors have reported the presence of confabulation in patients who have Korsakoff syndrome [72–77].

Korsakoff [71] observed that, in the course of confabulating, "patients confused old recollections with the present impressions. Thus, they may believe themselves to be in the setting (or circumstances) in which they were 30 years ago, and mistake persons who are around them now for people who were around them at that time" [78]. Kraeplin [79–81] distinguished two subtypes of confabulation. One, which he called simple confabulation, was caused in part by errors in the temporal ordering of real memories. Another form, which he called fantastic confabulation, consisted of bizarre, patently impossible statements unrelated to actual memories. Van Der Horst [74] and Williams and Rupp [75] also noted that many confabulations were based on intact past memories. As an example of this phenomenon, Talland [76] observed that amnestic confabulators tended to misidentify their doctors as old acquaintances.

These observations are consistent with a current theory that confabulation results from temporal context confusion caused by frontal-executive

dysfunction. By this explanation, confabulation results from inability to maintain an accurate temporal order of memories [82–86]. Thus, spontaneous confabulation occurs when patients cannot establish the contrast between current experience and memories of past events because of an inability to suppress irrelevant memory traces.

Another theory identifies deficient strategic retrieval as the primary deficit in confabulation [70,87–88]. Strategic retrieval refers to a conscious, effortful, and self-directed mechanism of memory recovery, and it requires frontal functions. According to this theory, confabulation results from a breakdown in strategic retrieval processes involved in memory search, temporal ordering, and output monitoring. Confabulation can occur when a particular cognitive subsystem (eg, memory) is damaged and produces faulty output (eg, failure to remember correctly) in the context of impaired output monitoring (eg, unawareness of response discrepancies).

Cognitively based theories of confabulation could help explain some features of DMS. Temporal context confusion or retrieval defects might allow remote memories to be confused with recent memories, explaining in part why patients who have had DMS believe they are located in previously known locations or are performing well-known social roles. These explanations, however, do not explain why only certain, select entities are misidentified or why persons or places of personal significance are typically the subject of the DMS. General theories of confabulation also fail to explain why most confabulators after anterior communicating artery aneurysms do not display a delusional fabric to their confabulations [67], whereas patients who have DMS are fixed in their delusions. For instance, Paterson and Zangwill [89] noted that their patients who had reduplicative paramnesia, who misidentified their current location as a geographical location closer to their actual homes, were refractory to correction, and their errors could not be explained solely by memory loss. Their patients might accept the correct orientation in an ''abstract geographical'' sense, such as knowing the correct locale ''according to the map,'' but they still maintained that they ''felt''' they were located closer to home. Subsequent authors also emphasized the delusional quality of the statements of some patients' misidentifications. Thus, Weinstein [90] referred to instances in which patients adamantly maintained an incorrect orientation in spite of correction as examples of ''symbolic or delusional environmental disorientation.'' These cases suggest that the delusional aspects are an important distinguishing feature between DMS and confabulation in general.

Additional observations support the notion that delusional misidentification is partially dissociated from confabulation in general. Box and coworkers [91] described a woman who developed Frégoli-type misidentifications after a traumatic brain injury. Initially the patient had inconsistent and short-lived confabulations that resolved. Only later did the patient demonstrate more stable Frégoli-type misidentification. Capgras-type DMS can also be dissociated from confabulation. Mattioli and colleagues [92]

reported a man with right frontopolar, right temporal, and bilateral frontobasal hypodensities after traumatic brain injury. The patient developed confabulation in both personal recollections and formal long-term verbal memory testing, along with Capgras-like misidentifications for his wife, daughters, and house. One year later, the confabulations were restricted to verbal memory tasks. The delusional misidentification of the wife persisted and remained refractory to correction, however.

Numerous studies have demonstrated that ventromedial frontal damage is critical for the occurrence of spontaneous confabulation [64,67,70,84, 93–98]. There do not seem to be strongly lateralized effects when a broad range of confabulatory patients is considered and when cases with confabulation manifesting as Capgras syndrome, reduplicative paramnesia, and Anton's syndrome are excluded, however [98]. Therefore, the finding of strong right frontal hemisphere predominance in the authors' series indicates that delusional misidentification and reduplication can be distinguished both clinically and neuroanatomically from confabulation.

All these approaches constitute "negative" explanations in a Jacksonian sense [53]. That is, they view neurologic or neuropsychologic deficits as the causal agents for DMS. Many of the negative features discussed are seen commonly in patients who have significant right hemisphere pathology. The question remains why most patients who have right hemisphere lesions, including many with right frontal lesions, do not demonstrate delusional misidentification or reduplication. Following is a discussion of the "positive" mechanisms that may better account for the features of these conditions not explained by the deficits alone. These positive neurologic features result from the functions of the remaining intact brain [53].

In some instances, it is not entirely clear whether a characteristic is a negative or positive feature of a disorder. For example, is the delusional nature of these disorders a positive or negative feature of DMS? Delusions in general have been reported to occur with increased frequency in the presence of right hemisphere pathology [99]. Malloy and Richardson [100], in a review of content-specific delusions that included delusional misidentification, sexual delusions, and somatic delusions, found a high incidence of lesions of the frontal lobes and right hemisphere. Kumral and Özturk [101] found an association between right posterior temporoparietal lesions and delusional ideation, and Sultzer and colleagues [102] found that the severity of delusional thinking in Alzheimer's disease was associated with right prefrontal hypometabolism on positron emission tomographic scans.

Alexander and colleagues [26] attributed the delusional ideation of a patient who had Capgras syndrome to the presence of bilateral frontal deficits leading to a failure to resolve conflicting or competing information. One striking features in the authors' subgroup of Capgras-type misidentification involving persons, however, was that a majority had prior or current paranoia, suspiciousness, or depression. Indeed, in the case reported

by Alexander and coworkers [26], the patient suffered from "grandiose and paranoid delusions, and had auditory hallucinations" before the brain injury that led to his Capgras-type delusions. This observation raises the possibility that premorbid psychopathology subserved by brain areas left undamaged by cortical lesions, including paranoia, may play a positive role in the production of DMS. Several of the cases with Frégoli-type delusions for persons in the authors' review had paranoia, but this association was not as strong as with Capgras-type delusions for persons.

An entirely different picture emerges in the cases with Frégoli-type delusions for place. In this group, no patients were reported to be paranoid or to demonstrate other evidence of psychopathology. In every instance, however, the patients' conviction that they were close to or actually in their homes could not be corrected. Patterson and Zangwill [89] hypothesized that the failure of patients to accept evidence that conflicted with their delusional disorientation was related to the patients' desire to return home. They argued that "a strong desire was actively inhibiting the cognitive mechanisms which normally subserve orientation," and that these patients were oblivious to the conflict presented by their dual orientation and would confabulate explanations when confronted with the disparity. The authors attributed the delusional disorientation to the negative features of anterograde and retrograde amnesia, restriction of perception, and defective judgment in which there is a failure to correct incompatible interpretations, as well as to the positive features of motivation.

Ruff and Volpe [38] also suggested that motivation might play a role in the maintenance of delusional disorientation. These authors described four patients who misidentified the location of their hospital rooms and claimed the hospital was located within their homes or that the hospital had been moved into their house. These authors suggested that a multiplicity of neurologic and psychologic factors created the delusional beliefs; these factors included "right parietal or frontal cerebral lesion, impaired spatial perception and visual memory, confusion or apathy early in the hospital course, and a strong desire to be at home." Therefore, within the Frégoli group, there is evidence that motivation or wish fulfillment is important in the creation and maintenance of the delusion.

Turk and colleagues [103] proposed that the functioning left hemisphere's attempt to make sense of or to interpret faulty information might account for DMS. That is, when the injured right hemisphere cannot produce the appropriate emotional response to a patient's spouse, the left hemisphere concludes the spouse has been replaced by an imposter.

Finally, a disturbance in ego functions may be involved in the creation of delusional misidentification. For instance, in the delusional asomatognosia cases, the arm is not simply misidentified, it is projected onto another person close to the patient. Further, in the cases with delusional reduplication without misidentification, the patients' own disabilities often are projected onto external fictitious or reduplicated persons. Thus, these cases

demonstrate the potential role of psychologic projection in DMS, often in association with anosognosia.

Delusional misidentification and the self

To account for the various features of delusional misidentification, the authors propose the following hypothesis. In addition to perceptual, memory, and executive impairments, patients who have delusional misidentification and reduplication suffer from a disturbance of self and self-related functions. That is, the right hemisphere may be dominant for the self, and right hemisphere damage may result in a disorder of ego boundaries and ego functions. This disorder could explain why delusional misidentifications are almost universally and selectively about aspects of the self or others of personal significance.

This hypothesis is consistent with the idea that delusional misidentification syndromes should be viewed as disorders of personal relatedness and the self [6–8,33,104]. This explanation also may account for disorders involving either under-relatedness or over-relatedness. Consistent with the finding of the frequent presence of right frontal damage in DMS, a growing body of research indicates that the right hemisphere is integral to the function of self-representation [105,106]. In a study of self-face presentation using functional MRI, Kircher and coworkers [107] found that self-faces activated almost twice as much area in the right hemisphere compared with unfamiliar faces and 1.3 times greater activation when compared with familiar faces. Another study [108] employed patients who had epilepsy anesthetized during a presurgical Wada test. These subjects were presented with a morphed face generated from a composite formed from their own face and that of a famous person. Under the condition of left hemisphere inactivation, with the right hemisphere functioning, subjects tended to identify the morphed face as their own. In contrast, with right hemisphere inactivation, they identified the face as a famous person. Right hemisphere dominance also has been demonstrated for self-face recognition [106,109] and for other functions related to the self such as autobiographical memories [110–114].

In the authors' view, right frontal hemisphere damage creates a deficit in the ego functions that mediate the relationship between the self and the world for personally significant incoming afferent information and for self-generated affects and drives. Thus, there is a two-way relationship between the self and the environment with regard to personal relatedness that, when disturbed, can result in disorders of both under- and over-relatedness to the environment. Without the intact functioning of right frontal regions that subserve certain self-related functions, personally significant incoming information may be disconnected from a feeling of familiarity [26] or personal relatedness [6,8]. Conversely, when internal motives, such as the desire to be home, are not monitored appropriately by the ego functions of

the right frontal regions, the patient may view the wish as an externalized reality. Similarly, in delusional asomatognosia, when the right frontal regions fail to establish appropriate ego boundaries, the feelings of alienation from the limb can result in an actual denial of ownership of the limb. Finally, in the case of delusional reduplication without misidentification, as occurs in the "phantom child syndrome," personal affects are projected onto fictitious others in the environment.

Spatial cognition may play a special role in linking right hemisphere pathology to disturbed ego boundaries. An accurate representation of the self–nonself boundary requires intact spatial cognition, because this boundary depends fundamentally on a concrete spatial distinction. Right hemisphere damage that impairs spatial cognition may therefore lead to disturbances of ego boundaries. These disturbances consist of deficits of veridical self–nonself space representation and in the release of more primitive (affectively driven) representations of ego boundaries whereby the self represents space according to wishes rather than to unwelcome current reality [115].

Treatment considerations

The treatment of the specific form of delusion discussed in this article has not been studied systematically. For patients manifesting any psychotic disorder in the context of a neurologic illness, atypical antipsychotics are generally recommended because of the decreased risk of adverse neurologic effects. With patients who have progressive dementia, such as dementia with Lewy bodies, in which DMS is common, cholinesterase inhibitors have demonstrated some ability to reduce psychiatric symptoms [116]. Wells and Whitehouse [117] have emphasized the importance of distinguishing true delusions such as DMS from confabulations, arguing that the latter do not respond to pharmacotherapy. Pihan and coworkers [118], however, described patient with an anterior communicating artery aneurysm patient who had both DMS and spontaneous confabulation whose delusions and confabulations both improved with risperidone treatment.

Summary

The Capgras syndrome and other forms of delusional misidentification may be encountered frequently in neuropsychiatric settings. DMS can occur in the presence of idiopathic psychiatric illness, in diffuse brain illness such as dementia, and in focal neurologic disease. In patients who have focal lesions, there is evidence that right hemisphere damage is necessary for the production of DMS. Although DMS is associated with a pattern of neuropsychologic impairments in the domains of memory, perception, and executive function, these impairments alone do not account for the selectivity and delusional nature of DMS. Therefore, other factors such as

premorbid psychopathology, motivation, and loss of ego functions may be important in determining which vulnerable patients develop DMS and which do not.

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