

A Case of Reduplicative Paramnesia for Home

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Abstract: We present the case of a high-functioning 88-year-old woman who suddenly developed the persistent and disturbing belief that her home of 40 years was not genuine, but rather an accurate replica. Her episode was probably caused by a small stroke that left her with this single extremely specific deficit. We describe the patient in detail and link to a video interview of her 3 months after the onset of the delusion, eloquently describing her experience. We summarize some of the many reports and discussions of our patient's delusion, reduplicative paramnesia, as well as other delusional misidentification syndromes.

Key Words: reduplicative paramnesia, delusional misidentification, Capgras syndrome, Cotard delusion, Frégo delusion

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MRI = magnetic resonance imaging.

This case report describes a patient and her husband who have chosen not to maintain their anonymity. They are recognizable in the accompanying video. The authors thank them for their generosity in allowing their identities and medical information to be published for purposes of education and research.

The history of medicine details many absorbing accounts of patients with delusional misidentification syndromes. Neurologist Arnold Pick originated the term

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reduplicative paramnesia in 1903 (Pick, 1903) to describe the delusion that a place has been duplicated or moved (Table 1). As summarized by Politis and Loane (2012), Pick described an elderly woman with probable neurodegenerative disease who believed that the clinic in Prague where she was being treated was actually a duplicate of the clinic in her hometown out in the suburbs, and that both clinics were in her hometown. She assumed that Dr Pick and the other staff members worked in both clinics.

In 1923, psychiatrists Joseph Capgras and Jean Reboul-Lachaux reported a patient who believed that her husband and other people close to her had been replaced by physically identical impostors (Capgras and Reboul-Lachaux, 1923; Ellis et al, 1994). This delusion of identical-looking impostors became known as the Capgras syndrome. Capgras' patient also thought that doubles of herself were trying to impersonate her, and she suffered from delusions of grandeur. (For an English translation of Capgras and Reboul-Lachaux's paper, with commentary, see Ellis et al, 1994.)

Neurologists such as Pick and psychiatrists such as Capgras have described fascinating and puzzling cases of delusional misidentification syndrome for place or person, with highly variable features. The other-worldliness of some of these descriptions has captured our imaginations and stoked the creative fires of book and movie authors.

PRESENTATION OF PATIENTS WITH REDUPLICATIVE PARAMNESIA

Many people, such as severely ill medical patients, victims of motor vehicle accidents, and soldiers with traumatic brain injury, suffer from misidentification of place. They believe that they are being treated in a location other than where they actually are, often thinking that they are in their hometown or even in their own home (for examples, see Alexander et al, 1979; Budson et al, 2000; Feinberg and Roane, 2005; Fisher, 1982; Ruff and Volpe, 1981).

Ruff and Volpe (1981) described four patients who had either a tumor or a hematoma in the right frontal or right parietal area. All temporarily believed that they were staying in a branch or annex of a hospital within their own home. The authors called this delusion *environmental reduplication*. They described the patients as having "confusion or apathy early in the hospital course, and a strong desire to be at home."

Feinberg and Roane (2005) described a 63-year-old man with a ruptured anterior cerebral artery and bilateral

TABLE 1. Delusional Misidentification Syndromes

Reality	Patient's Perception	Syndrome
Home	Duplicated house	Reduplicative paramnesia
Hospital room	Different room, or a room at home or in a different clinic or a different geographic location	Reduplicative paramnesia
Familiar object	An imitation	Capgras syndrome
Familiar plant	A replica	Capgras syndrome
Familiar pet	An impostor	Capgras syndrome
Familiar person	An impostor	Capgras syndrome
Unknown person	Familiar person (often a persecutor)	Frégo syndrome
Self	A different known person	Reverse intermetamorphosis
Self alive	Self or part of self dead or dying	Cotard syndrome
Self in mirror	A different person who resembles self	Misidentification of reflection, mirrored-self misidentification
Other person(s)	Self as twin(s), impostor(s), clone(s)	Syndrome of subjective doubles, Doppelgänger syndrome
Person or animal	A different person, animal, or thing interchanged, or with a changing form	Intermetamorphosis
Other person(s) not present	Person(s) present in patient's home	Phantom boarder symptom

frontal infarctions who denied being a patient in a hospital. The author observed that perceiving a strange environment, such as a hospital room, to be a part of one's own home is analogous to Frégo syndrome (Courbon and Fail, 1927; translation and commentary in Ellis et al, 1994). Frégo syndrome is the belief that a misidentified stranger is a familiar person (often a persecutor) in disguise (Langdon et al, 2014). In another case of Frégo syndrome, a hospitalized woman claimed that the patient in the bed next to her was her husband, who, she noted, had stopped snoring (Feinberg and Roane, 2005).

Fisher (1982) described seven patients hospitalized for stroke who experienced disorientation for place. The central patient, who had an acute stroke, recognized his family and physicians and he retained much of his higher cortical function. Although he sometimes stated correctly that he was at Massachusetts General Hospital, however, he usually described it as being somewhere other than Boston. On different days he placed the hospital as close as Concord, Massachusetts, just a few miles distant, or as far away as California or Japan. He even reported that he was in "MGH [Massachusetts General Hospital] East....When I say east I'm thinking more of Baghdad rather than east Boston."

Fortunately, most reduplicative phenomena such as these resolve over time.

TYPES OF REDUPLICATION

A note about terminology: *Delusional misidentification syndrome* encompasses all the syndromes (Table 1). When Pick coined *reduplicative paramnesia* in 1903, he was describing his patient's delusions about duplication of events, institutions, and physicians (Fisher, 1982); since then, however, the neurology literature has come to use the term to describe reduplication of geographic location. Fisher points

out that, for sighted persons, location orientation is largely visual. Therefore, duplication of home or hospital room is reduplication paramnesia for place. Weinstein (1996) concurs with the appropriateness of the term *reduplication*, noting that many patients report more than one double.

The term *Capgras syndrome* appears more often in the psychology than the neurology literature, describing reduplicative delusions involving persons, pets, plants, and inanimate objects.

Though originating in separate fields, the terms *reduplicative paramnesia* and *Capgras syndrome* often overlap and are sometimes used interchangeably.

Patients with classic Capgras syndrome perceive a familiar person, often a close family member, as being an impostor. The patients misidentify a person, not a place. Alexander and colleagues (1979) characterized Capgras syndrome as a form of reduplicative paramnesia because of the psychiatric and neurologic parallels. They described a case of Capgras syndrome in a 44-year-old man who began to experience psychotic symptoms related to his work and finances. During a psychotic episode, he was struck by a car and left in a temporary coma. He required surgery for a large subdural hematoma and necrotic right frontal lobe. Ten months later, during his first visit home, he identified the woman and five children there as older duplicates of his "first" (real) family, and these people's house as being just like his real house. He liked the new wife, was not angry at the first wife for deserting him, and felt grateful that the first wife had found him a substitute.

People with Capgras syndrome can also have delusional beliefs about their pets and plants. Somerfield (1999) reported a 91-year-old woman who believed that her parrot had been swapped with an impostor. Islam et al (2015) described a 53-year-old woman who, within a few years after being diagnosed with Parkinson disease,

developed delusions that her pet dogs, the paintings in her house, and the cypress trees in her garden had all been replaced with fakes.

Many other delusional misidentification syndromes have been reported. For example, Politis and Loane (2012) cite Charles Bonnet's 1788 description of an elderly woman who believed not only that she was somewhere else (reduplicative paramnesia), but also that she was dead (Cotard delusion) (Förstl and Beats, 1992). Politis and Loane (2012) also describe the *Doppelgänger* (literally, “double-goer” in German) syndrome, in which people believe that they have a clone or are clones of themselves. A patient with intermetamorphosis syndrome perceives a familiar person to have exchanged identities with a stranger (Christodoulou et al, 2009); the familiar person morphs into a stranger, and may continue to morph into different strangers.

Christodoulou has organized the delusional misidentification syndromes into categories of “hypoidentification” (Capgras syndrome-like) and “hyper-identification” (Frégoli syndrome, intermetamorphosis, and syndrome of subjective doubles) (Christodoulou et al, 2009; Islam et al, 2015).

Delusional misidentification syndromes thus encompass a broad range of disorders in which patients misidentify themselves, another person or persons, a place, an animal, or an inanimate object (Islam et al, 2015; Somerfield, 1999). These syndromes can vary widely in their presentation, and they can overlap and even mutate (Table 1). Clear elements of accurate recognition intertwine with confusion about relation to self, identity relation, and context.

BRAIN LOCI RESPONSIBLE FOR REDUPLICATIVE PARAMNESIA

Neurologists have struggled to pinpoint the cerebral dysfunction(s) in patients with reduplicative paramnesia and to implicate a particular brain location. Aided by modern imaging, numerous case series have delineated involved brain areas. Neurologic correlates have been comprehensively detailed (see reviews by Förstl et al, 1991; Politis and Loane, 2012).

Politis and Loane (2012) note that many brain loci coincide with reduplicative paramnesia, but the most frequent lesions are in the right hemisphere and/or are bifrontal. Least often observed are lesions limited to the left hemisphere. The authors reported that their finding of more right- than left-sided lesions holds for patients labeled as having reduplicative paramnesia as well as those labeled as having Capgras syndrome.

REDUPLICATIVE PARAMNESIA FOR HOME

After a trip out of town, our patient believed that the house to which she returned was not really her home but, improbably, an accurate replica. At least three similar reports have been published of patients who believed that their own home had been duplicated and they were

living in a duplicate or “fake” house (Gerace, 2011; Kapur et al, 1988; Moser et al, 1998).

Kapur and colleagues (1988) describe a 71-year-old right-handed man who became disoriented after leaving his home. After many hours, he found his way back and questioned his daughter about why she was at “this place.” He was certain that this house was not his real home, but was amazed that the owners had furnished the house so much like his own home.

Moser and co-authors (1998) describe an 81-year-old right-handed man who was admitted to the hospital after hitting his head during a fall at home. He made a good recovery over 10 days, but showed evidence of moderate cognitive decline. Three days after returning home, he suddenly accused his family of bringing him to a “twin” of his “real home,” a place that was about 12 miles away.

Gerace, in a 2011 letter, describes a 72-year-old left-handed woman who was admitted with confusion and gait troubles. She had a normal bedside mental status exam but spoke of having discovered that she had two identical and identically furnished houses on the same street. By way of explanation, she wondered if the government had arranged it for her. Her housemaid reported that shortly before being hospitalized, the patient had asked to go “to her real house” and had spoken of having 10 dresses in each of the houses.

Kapur et al, Moser et al, and Gerace all thought that their patients’ delusions were related to organic brain disease: right frontal hemorrhage, right frontal infarct, and right temporal meningioma, respectively. Kapur et al’s patient was free of the delusion 3 months after his stroke, Moser’s had recovered partially 24 days after his fall, and Gerace’s recovered completely 1 week after her meningioma was resected.

We present the case of an elderly woman who suffered an episode of mild confusion while traveling. Over the next week, she recovered near-normal function. Upon arriving home, however, she expressed the specific and clear belief that her house was not her real home, but rather an accurate duplicate.

CASE REPORT

A generally healthy, high-functioning 88-year-old right-handed woman started to believe that her house was a duplicate rather than her real home.

Much of the following description comes from the patient’s loving and attentive husband, who is a psychiatrist. Because of his background and their close relationship, he was an astute observer of his wife, as well as a supportive caregiver. He could corroborate details given by the patient and provide details that she could not.

The patient’s symptoms began while she and her husband were visiting family out of state. In the middle of one night, he found her asleep sitting on the toilet. (We will refer to this as the “index event.”) She awoke groggy and he helped her back to bed. She slept very late the next

morning. For up to a week afterward, she had unusual lethargy, with occasional transient confusion.

Her husband reported that, apart from being somewhat subdued, her behavior for the remaining 8 days of the trip was mostly normal, as were her interactions with her family members. She did not voice any complaints. She occasionally needed gentle reminders or re-orientation as to place and context, but she recognized all of her relatives. Neither her husband nor her son, who is also a physician, saw any evidence of a stroke.

At the time, her husband hypothesized that she had fallen asleep on the toilet after accidentally taking an extra clonazepam tablet or two instead of a bowel stimulant. She did not recall doing that, but she said that she had occasionally been taking one clonazepam for insomnia. Her recall of these events was uncharacteristically hazy. Later, she had a very poor recollection of the entire trip. She thought she had seen more relatives than had actually been there, and she remembered little of traveling home.

By the time her husband drove her home, about 8 days after the index event, he felt that most if not all of her lethargy and mild confusion had disappeared. Upon arriving at their house, however, the patient said that she experienced the strong and unsettling sensation that her home of 40 years was not genuine, but rather an exact duplicate. She recognized the neighborhood, the street, the yard, the neighbors, and even personal objects within the house as being genuine, yet somehow she still felt that the house itself was not original, and not her home.

She felt this perception most strongly upon awakening in the mornings. Her husband's daily ritual of bringing her coffee at her bedside puzzled her. She asked him, "Did you collect it from the main kitchens?" She noted, with some surprise, her own personal belongings in the house, such as undergarments in drawers. She could identify as genuine the Danish furniture that she and her husband had bought years earlier, and a special tea set that had been handed down over generations. Although she recognized the irrationality of her belief that the house was a duplicate, she experienced this strong and disturbing perception as reality.

According to her husband, at this time she still occasionally needed slight reorientation, for example, to the correct date.

Past Medical History

Seven months before the index event, the patient had been hospitalized for exertional chest pain and was diagnosed with a small myocardial infarction without ST segment elevation. An echocardiogram showed a well-preserved ejection fraction. She was treated with metoprolol, aspirin, and pravastatin, and recovered completely.

She has a long history of mild right upper extremity weakness. In recent years she has suffered a slight decline in memory, but no significant organic cause has been found.

For at least the 2 months leading up to the index event, she did not eat any new foods or change her prescription or over-the-counter medications. Her main daily medicines were metoprolol, aspirin, omeprazole, glaucoma medications, and a laxative-stool softener. Rarely, she took clonazepam 0.5 mg to help her sleep.

She has no history of psychiatric illness.

She is a non-smoker and former light drinker; at her husband's suggestion, she quit drinking wine about 6 months before the index event because of her advancing age and mild memory loss. She is the happily married mother of two adult children, and a retired respiratory therapist.

Clinical Evaluation

The patient was already seeing author C.S.P. as her primary care physician. Over several months before the index event, she felt that her chronic right upper extremity weakness was worsening. Although the likely cause of the weakness was cervical osteoarthritis, she had worked for years with patients with polio and wondered privately whether she might have a residual of subclinical polio.

To evaluate her weakness, 2 months before the index event C.S.P. ordered a magnetic resonance imaging (MRI) scan of her brain (Figure 1, Panel A). The scan showed only age-related tissue loss and mild periventricular white matter changes presumed to be signs of chronic small vessel ischemic disease. No stroke was noted.

C.S.P. evaluated the patient 5 weeks before the index event. On physical examination, she had a temperature of 36.3°C (97.3°F), a regular pulse of 65/minute, respirations 16/minute, and blood pressure 160/80 mm Hg. She was 144 cm (57 inches) tall and weighed 48 kg (106 pounds). She had neither skin pallor nor rash, and she was normocephalic and atraumatic. Her neck was supple, with no lymphadenopathy. Her heart showed a regular rate and rhythm, with no murmurs, rubs, or gallops. Her lungs were clear to auscultation and percussion. Her abdomen was soft, non-tender, and free of masses. Her extremities were well developed, with a full range of motion and no edema. Her right upper extremity strength was 3/5, but 4/5 in her other limbs. Her gait was normal for her age and the Timed Get Up and Go Test was normal.

Eight days after the index event and just after the couple returned from their trip, the husband called C.S.P. to report the patient's delusion about the duplicated house. Because she seemed stable, they agreed that she could wait to be seen at a neurologic consultation with author E.R.W. that had been scheduled coincidentally for 10 days later.

E.R.W. first saw the patient 18 days after the index event, and again 4 weeks, 6 weeks, 10 weeks, and 7 months into her course. C.S.P. saw her at 6 weeks, 10 weeks, 14 weeks, and 8 months. At each of the visits with C.S.P., the physical exam remained essentially normal and unchanged from the exam 5 weeks before the index event.

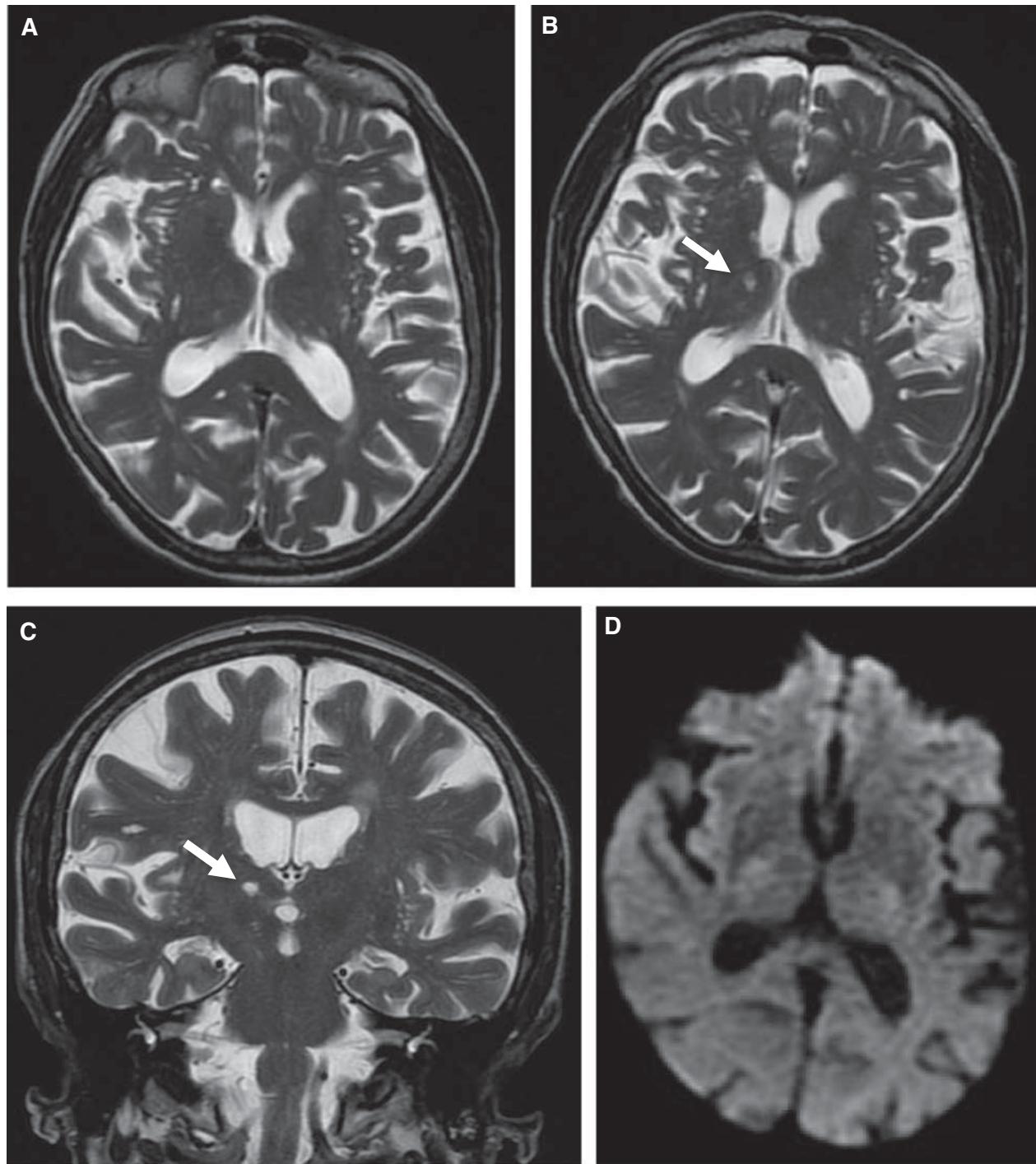


FIGURE 1. This patient's magnetic resonance imaging scans.

Two months before the onset of her delusion (Panel A): An axial T2-weighted image shows only age-related tissue loss and mild periventricular white matter changes presumed to be signs of chronic small vessel ischemic disease. There is no evidence of a stroke.

Seven months after the onset of her delusion (Panels B–D): Arrows in T2-weighted axial (Panel B) and coronal (Panel C) slices point to a focus of encephalomalacia in the medial right thalamus, consistent with a lacunar stroke. The overall prominence of the sulci reflect age-related atrophy. A diffusion-weighted image (Panel D) does not show restricted diffusion corresponding to the focus of T2 signal hyperintensity. The normal appearance of the image indicates that the infarct is old.

As was the couple's custom, the patient's husband respectfully took part in all of her medical visits.

On day 18, E.R.W. found the patient to be alert, pleasant, and in a good mood, with facile communication skills. She performed serial 7s quickly and with no errors. She correctly named her state's new senator. Her carotid arteries were full and without bruits. Her extra-ocular movements were intact. Her visual fields were full by confrontation, and without nystagmus. Her smell and hearing were within normal limits. Optokinetic nystagmus was symmetric. Her facial sensation and movement were normal. She had no dysarthria or drift. Her deep tendon reflexes were 2+ except for absent ankle jerks, and her toes were downgoing bilaterally. Pin sensation was decreased almost to her ankles. She did not feel the tuning fork vibration in her right toes, and she barely sensed it in her left toes. There was wasting of her right thenar area and loss of sensation over the ventral aspect of the lateral three fingers of her right hand. She had a slight snout reflex, a 2+ grasp in the right hand, and a 1+ grasp in the left hand.

Her diagnostic workup included a basic metabolic panel, complete blood counts, and a vitamin B12 level, all of which were largely normal. She did have a mild anemia and a borderline-low vitamin B12 level that we felt to be likely unrelated to the delusion. She was started on a B12 supplement.

Four weeks after the index event, she underwent a non-contrast computed tomogram of the head. Beyond age-appropriate atrophy, it showed only scattered subcortical and periventricular areas of low attenuation not significantly changed from a scan performed 20 months earlier.

About 7 months after the index event, a brain MRI (Figure 1, Panels B–D) revealed a focus of encephalomalacia in the medial right thalamus, consistent with a lacunar stroke. Diffusion-weighted images did not show restriction of diffusion correlating with the focus of T2 signal hyperintensity. The normal appearance of the diffusion-weighted images indicated that the infarct was old.

Course and Outcome

The patient's nonsensical but vivid belief frightened her. She felt terror at realizing that, in her husband's words, "she could not trust her own thinking." Her husband stayed at home, close by her side, for nearly a month after they returned from their trip, continually giving her gentle reassurance and reorientation. With his help, she gradually became less afraid and better accommodated to her seemingly unbelievable circumstance. She reported (and her husband confirmed) that she was improving emotionally, was better oriented, had more energy, and was engaging in more activities. Still, however, she maintained that her home was somehow a replica of itself.

We (E.R.W. and C.S.P.) realized that the patient had a particularly clear and discrete case of reduplicative paramnesia for home, with no superimposed psychiatric disease. We also appreciated how articulate she was and

how clearly she described her experiences. We thought that it would be valuable and potentially instructive to videorecord an interview with her and her husband. Despite knowing that they would be identifiable to viewers, they gave their enthusiastic consent for the video because they wanted their story to benefit others.

E.R.W. conducted the 45-minute interview with the patient and her husband when the patient was about 3 months into her delusion. Our goals were to understand what had happened and when, and to learn the patient's current perceptions of her home and of what was happening to her. We excerpted 15 minutes of the interview to include with this article (Supplemental Digital Content 1, <http://links.lww.com/CBN/A64>). The patient and her husband approved the final version of the video.

About 6½ months after the index event, the patient realized that her delusion had disappeared. She again understood her home to be genuine, and she had no discernible residual deficit. Three years later she remains entirely well, with no recurrence of the delusion.

DISCUSSION

The cause of delusional misidentification syndromes is not known. Both psychological and neurologic hypotheses have been proposed. Some patients have multiple concurrent psychotic symptoms, while others have no abnormalities besides the delusion.

Capgras himself initially suggested that *l'illusion des "sosies"* ["the illusion of doubles"] was an agnosia of identification as opposed to perception, emanating from dementia, confusion, or depression (Ellis et al, 1994). Later, in keeping with the thinking of the times, Capgras proposed Freudian explanations, such as the use of denial as a defense mechanism for Oedipal feelings (Ellis et al, 1994; Ramachandran, 1998).

In an analogous fashion, reduplicative paramnesia for place, such as a hospital room, has been considered a possible form of denial of illness as a defense mechanism. Patients themselves have wondered whether their own strong desire to be at home was partially responsible for their apparently irrational beliefs. The four patients described by Ruff and Volpe (1981) "each claimed that their confabulated stories resulted from a desire to be at home." Strikingly, the patient hospitalized in Feinberg and Roane's (2005) case denied being in the hospital altogether, and described himself as being "a guest" with the 'optimists club.'

The original patient with Capgras syndrome, and many more since her, were relatively young, had obvious psychiatric illness, and manifested a host of additional delusions and hallucinations. Other people with delusional misidentification syndromes, including our patient, have been older people with some age-appropriate cortical atrophy but no pre-existing psychiatric illness; presumably, they suffered a structural insult that, because they had lost compensatory ability, initiated the delusional state.

These cases without psychiatric predisposition are likely related to specific damage from stroke, traumatic brain injury, focal tumor, or dementias such as Alzheimer disease, Lewy body dementia, or Parkinson disease (Alexander et al, 1979; Gerace, 2011; Islam et al, 2015; Kapur et al, 1988; Kyrtos et al, 2015; Moser et al, 1998; Ruff and Volpe, 1981; Sottile et al, 2015; Thaipisuttikul et al, 2013). The numerous brain loci involved and the great clinical variability among cases have made it difficult to generate an explanatory hypothesis (Forstl et al, 1991). Recent patients have been analyzed based on neuroanatomy, neurophysiologic testing, neuropsychological testing, and functional brain imaging.

Ramachandran (1998) used galvanic skin response to assess emotional arousal in a patient with Capgras syndrome. The patient's lack of response to a photograph of his mother suggested a disconnection between the visual and the emotional. Ramachandran postulated that the visual recognition area(s) of the temporal lobe, including the fusiform gyrus, are interrupted from the amygdala and limbic system, leaving patients unable to recognize emotional significance.

Coltheart and colleagues (2011) have suggested a two-factor hypothesis for many cases of reduplicative paramnesia: Patients have simultaneous right hemispheric and bifrontal lesions that create a necessary "dual hit" to maintain a particular delusional misidentification syndrome. This hypothesis suggests a disconnection between the cortical sensory areas and the limbic emotional identification function, left uncorrected because of compromised frontal cortical function. The sensory areas identify the perception, and the limbic areas provide the contextual relation to self. Not only must this connection be disrupted, the theory postulates, but a normal compensatory capacity must also be compromised (the second hit) in the form of frontal cortical damage. Young, healthy people would be expected to have excellent compensatory capacity, while older people with cortical atrophy, varying degrees of dementia, or other secondary or pre-existing insults would not be able to compensate.

Budson and colleagues (2000) described a 45-year-old man with a small left temporo-parieto-occipital junction stroke who developed reduplicative paramnesia for his hospital room. Based on MRI findings, the authors suspected a disruption of the occipito-temporal projection system of the ventral visual stream. Interestingly, because the patient was left-handed, it is possible that the left-sided lesion affected his non-dominant hemisphere much as many right-handed patients have right-sided lesions postulated to cause their symptoms. This patient also had evidence of bifrontal dysfunction, presumed to be from alcoholism. This may have been the second factor allowing the delusion to persist.

The brain area most often identified as compromised in patients with delusional misidentification syndromes is the right hemisphere. This hemisphere is non-dominant in the 70% to 95% of all persons who are right-handed; further, most left-handed people have the same left-hemisphere specialization for language typical

of right-handers (Holder, 2015). Therefore, one would expect most cases to involve the right hemisphere.

Thiel and colleagues (2014) studied a 70-year-old right-handed woman who developed Capgras syndrome for her husband after she suffered a right frontal hemorrhage. The authors used functional MRI to assess her neurologic activity in response to familiar and unfamiliar faces. They concluded that she had "impaired coupling of the left posterior superior temporal and left superior frontal cortex when viewing the partner's face," indicating a deficiency in the extended face processing system, a deficiency that began coincident with her right frontal lesion. Thiel et al presented this conclusion as support for the two-factor theory of Coltheart et al (2011). The defect in the left side of the patient's extended face processing system became evident after her right frontal injury.

A 63-year-old man with a history of alcohol dependence developed phantom boarder symptoms after traumatic brain injury (Thode et al, 2012). He thought that his brother, who lived far away, was now staying with him. Brain MRI showed only diffuse cortical atrophy, but single-photon emission computed tomography demonstrated relative hypoperfusion in the frontal and superior parietal lobes of the right hemisphere.

Nomura and colleagues (2012) used neuropsychiatric testing to evaluate delusions in 87 consecutive outpatients with Alzheimer disease, and single-photon emission computed tomography in 25 of the patients to identify specific areas of hypoperfusion and hyperperfusion. Using factor analysis, the authors classified eight types of delusions into three factors. Their Factor 1 delusions included those like Capgras, phantom boarder, and reduplicative paramnesia for home. The Factor 1 delusions were "related to hypoperfusion in the right temporal pole and hyperperfusion in the bilateral medial frontal regions and the precentral gyrus" (Nomura et al, 2012).

A 70-year-old right-handed woman with Capgras syndrome for her husband and children had MRI findings very similar to those in our patient (Peña-Salazar et al, 2014). Peña-Salazar's patient had a lacunar infarct in the left thalamus as well as a left frontal subcortical density correlating with the onset of her delusion. On MRI, T2-weighted and fluid-attenuated inversion recovery sequences showed affected areas in the left thalamus. A cranial single-photon emission computed tomogram indicated hypofunction in the left thalamic region.

Our patient had a right thalamic lesion near the massa intermedia. This lesion may represent a limbic disconnection such as that postulated in other case reports. Her age, diffuse cerebral atrophy, and/or a small unseen frontal defect may have provided the second hit hypothesized to be needed to maintain her delusion. She would have benefited—and future patients with delusional misidentification syndromes will benefit—from neuropsychological testing and functional imaging to delineate better the anatomy and physiology of their brain function. Because most patients recover, follow-up

testing and scans would be helpful to demonstrate the reversibility of the defects.

We hope that the video interview of our patient will help other clinicians better evaluate and manage their patients.

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