

Reduplicative Paramnesia: Longitudinal Neurobehavioral and Neuroimaging Analysis

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ABSTRACT

Reduplicative paramnesia (RP) is a delusion in which the patient perceives familiar places, objects, or events to have been duplicated. The current case describes the development of RP in an 81-year-old male following a large right frontal lobe infarction. As the patient had been hospitalized previously with hemorrhagic contusions, neurologic, neuropsychological, and neuroimaging data were obtained both prior to and following RP onset. Psychophysiological data were obtained following the development of the delusion. Both premorbidly and at follow-up, neuropsychological functioning was characterized by significant impairments of learning and memory and frontal-executive functions. Language and visuospatial skills and motor speed were intact both before and after RP onset. The case is described within the context of preexisting theories of RP, and it is surmised that the delusion is secondary to temporal-limbic-frontal dysfunction giving rise to a distorted sense of familiarity and impaired ability to resolve the delusion via reasoning. (*J Geriatr Psychiatry Neurol* 1998; 11:174–180).

Reduplicative paramnesia (RP) is one of the most dramatic forms of delusion associated with neurologic insult. First described by Pick,¹ patients with RP hold the belief that familiar places, objects, or events have been duplicated. Subsequently, such patients may claim that objects and places with which they are confronted are identical to, but are not actually, the original entities. In cases of event duplication, the patient may claim that a single event actually occurred on two or more occasions. Capgras syndrome,² in which patients believe that familiar people have been duplicated or replaced with imposters, is likely a variant of RP.^{3,4}

Received July 28, 1998. Received revised September 22, 1998. Accepted for publication October 14, 1998.

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The patient described in this paper was seen and treated at The Miriam Hospital (TMH) and all relevant data were collected at this site. Preparation of the manuscript was also completed at TMH and at Butler and Rhode Island Hospitals. All of these hospitals are in Providence, Rhode Island and are affiliated with the Brown University School of Medicine. This work was not supported by any grant or sponsor.

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Reduplicative phenomena are relatively rare but may occur in association with a variety of neurologic, psychiatric, and metabolic disorders. Despite the considerable heterogeneity of etiologies among RP cases,⁵ the delusion appears to be most commonly observed in association with focal brain damage following head trauma and cerebral infarction.^{6,7} While the specific conditions leading to RP remain unclear, a review of case studies that examined neuroimaging, neurologic, and neuropsychological, data in patients with RP suggested that frontal lobe and right hemisphere damage are central to the disorder.⁷ Whereas most patients with RP have multiple regions of damage, the frontal cortex appears to be invariably involved, and the majority of cases include predominant right hemisphere abnormality.^{5–7}

Early models of RP characterized the delusion as a defense mechanism involving denial of illness.⁸ Since then, several more neurobehaviorally based theories have been proposed. Benson et al⁹ characterized RP as a compound symptom resulting from visuospatial impairment secondary to right hemisphere lesions, which produced alterations in spatial encoding that were not accurately recognized as such due to the coexistence of impaired frontal-executive functions. A similar model was later put forth by Hakim et al,¹⁰ and spatial impairments have also been reported in other cases of delusional misidentification.^{11,12}

Others have characterized RP as a memory-related phenomenon, proposing a disconnection of premorbid memory stores from structures responsible for new memory formation, as could theoretically occur with a lesion in the temporo-parieto-occipital junction. This disconnection would purportedly underlie the distortion of familiarity seen in RP, which would, in turn, be maintained via concomitant frontal-executive impairment.⁴ Alexander et al had earlier proposed a related temporal-limbic-frontal disconnection, resulting in a disruption of processes dealing with familiarity and object/person identity.³ Despite the plausibility of these models, none has yet adequately explained the specificity with which certain objects, events, or people are perceived as having been duplicated.

Presently, we report on a patient who developed RP following a large right frontal infarction. The functional neuroanatomic bases of this patient's syndrome were examined through neurologic, neuropsychological, psychophysiological, and neuroimaging analyses.

CASE

Pre-RP Hospitalization and Discharge

Identifying Information and Past Medical History

The patient, an 81-year-old, right-handed male with a high school education, was initially admitted to his local hospital following a fall in his home, which left him unresponsive for 10 minutes. Three days later, he was transferred to our facility for further evaluation and treatment. Past medical history was significant for hypertension, atrial fibrillation, and benign prostatic hypertrophy. By his family's report, the patient had been very independent in his activities of daily living and did not have any history of memory problems or any other cognitive impairment.

Neurologic Examination

At the time of transfer, neurologic examination revealed the patient to be somnolent but easily aroused. His speech was mildly dysarthric and he was inattentive, disoriented to place, but able to follow one-step commands requiring facial, axial, and appendicular movements. Muscle power, tone, and deep tendon reflexes were normal. Plantar responses were flexor and nociception was intact in that he grimaced when a pin was touched to any extremity or to his face or torso. The visual fields were full to visual threat testing, and cranial nerve function was intact.

Brain Imaging and Electroencephalography

Magnetic resonance imaging (MRI) and computed tomography (CT) revealed two small hemorrhagic contusions in the left frontal lobe. One of these was located in the deep white matter near the supplementary motor area,

just anterior to the sulcus marginalis. The other was in the centrum semiovale, immediately subcortical near the superior frontal sulcus. A subdural hematoma lay over the left tentorium and blood was also visible in the intrahemispheric fissure adjacent to the left mesial occipital lobe. Small-vessel ischemic changes were noted subcortically and in the periventricular region, as was a moderate degree of atrophy, primarily in the frontotemporal regions (Fig. 1). No evidence of aneurysm, arteriovenous malformation, or vasculitis was visible on a selective arteriogram. Electroencephalography revealed intermittent background slowing that occurred bilaterally and occasionally unilaterally. Occasional mild suppression was noted in the left hemisphere. There was no evidence of paroxysmal activity in the temporal lobes.

Neuropsychological Evaluation

Neuropsychological testing conducted 10 days after his fall revealed that the patient was alert and oriented, with grossly intact language, visuoperceptual, and attentional skills. He performed within normal limits on informal tests of naming, phrase repetition, comprehension, and reading and was able to match abstract designs to copies. However, moderate cognitive decline was evident (Dementia Rating Scale¹³ = 121), with greatest impairments in the areas of verbal memory and frontal-executive functioning. There was also a very mild impairment of visuoconstructive skills. The patient's affect was appropriate to the situation (Table 1).

Following this 12-day hospitalization, the patient was discharged and returned home without incident. Three days later, however, he suddenly began to claim that his family had brought him to a "twin" of his "real home," which he believed to be approximately 12 miles away. He became quite agitated and mildly aggressive as he demanded to be brought "home" and was subsequently rehospitalized.

Post-RP Hospitalization and Discharge

Description of Delusion

Upon readmission, the patient was significantly distressed by the perception that he had been brought to a duplicate house. He stated, "When I walked into that house I got the shock of my life...It was identical in every way...the twin of twins...[to my first house]." Within the context of this delusion, he became further distressed regarding financial issues related to having owned a house for many years without having paid any taxes or mortgage. He was similarly upset by his belief that he must have secretly left his family during many nights in order to have built the "duplicate house," and the belief that his family was betraying him by not admitting that the other house existed. In addition to these feelings of distress, the patient also expressed feelings of deep pride in his belief that he had been able to renovate two houses. He explained that he had com-

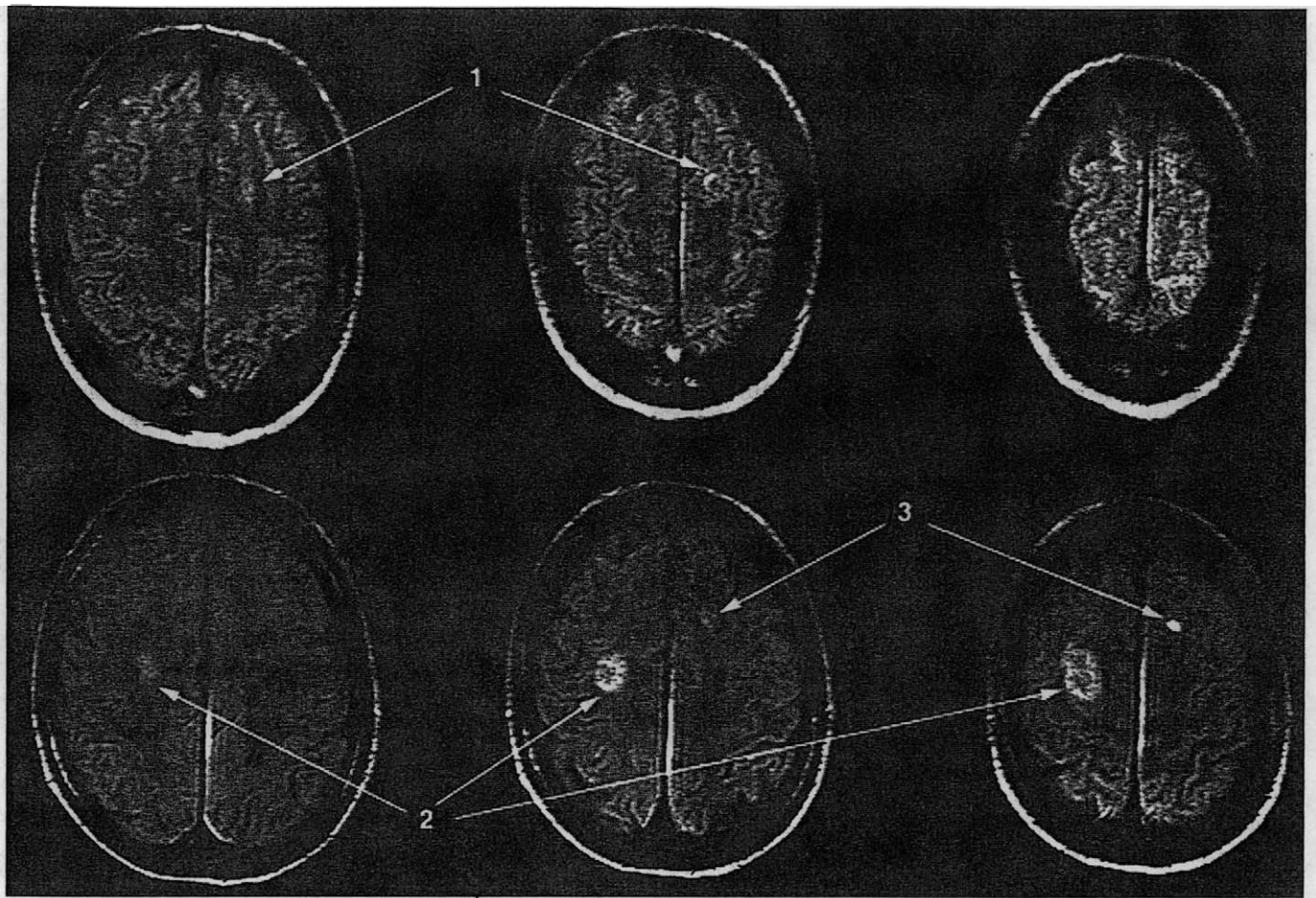


Figure 1. Proton density axial MRI brain images. Top: Pre-RP MRI shows hemorrhagic contusions in the left frontal lobe (1). Bottom: Post-RP MRI shows new large right frontal ischemic infarct (2) and remaining hemorrhagic contusions (3).

pletely remodeled the interior of his home many years ago, and that he had done so quite meticulously and with a great amount of skill. He was moved to tears as he described his handiwork and his belief that he had done this not only in one house but in two. When asked about the neighbors' houses, the patient indicated that they appeared to be identical to the ones in his original neighborhood. With further discussion, it became clear that the patient did not perceive them as having been duplicated when he considered them separately. Rather, he was so certain that his own house was a duplicate, and was 10 to 15 miles from his "real house," that the presence of familiar-looking surrounding houses forced him to conclude inaccurately that these houses must also have been duplicated.

When the fantastic nature of his account was challenged, the patient admitted that it was quite strange but became no less resolute in his statements. He was also asked repeatedly how he could tell that the house to which he was taken was a duplicate if, in fact, the two were perfect copies of one another. In response to this, he typically became involved in lengthy descriptions of his skilled handiwork without addressing the conundrum.

Neurologic Examination

At the time of his second hospitalization, neurologic examination revealed the patient to be alert and oriented to location but not time. He was easily distracted, but speech and language were intact as he waxed eloquently about his reduplicated house. Both immediate and delayed memory were impaired, but he was able to do basic calculations. The patient had a mild left hemiparesis involving the arm more than the leg. Sensation and coordination were preserved. Cranial nerve function was abnormal only in that he had a mild left lower facial weakness. The visual fields were full to confrontation testing.

Brain Imaging

MRI and CT revealed a new large infarct in the right superior frontal gyrus, with definite gyriform enhancement, believed to be the result of vasospasm and subsequent ischemia. This infarct extended subcortically almost to the ventricle. A smaller enhancing infarct was noted in the mesial and inferior portion of the left cerebellar hemisphere, and a very small infarct was visible in the left calcarine cortex. Regarding previous imaging,

Table 1. Neuropsychological Functioning Before and After Onset of RP

Domain/Instrument	Pre RP	Post RP	Follow-Up	Normal Values
General Cognitive Functioning				
DRS Total Score	121*	101*	110*	137.30 (6.90)
WAIS-R Full Scale IQ	—	—	96	100.00 (15.00)
Verbal IQ	—	—	101	100.00 (15.00)
Performance IQ	—	—	90	100.00 (15.00)
Memory				
WMS-R Logical Memory I	—	7*	12*	19.00 (5.50)
Logical Memory II	—	2*	2*	13.50 (5.50)
Visual Reproduction I	—	17*	21	24.50 (5.50)
Visual Reproduction II	—	0*	0*	15.50 (8.50)
DRS Memory	17*	15*	14*	23.30 (2.10)
Language				
Boston Naming Test	50	—	55	51.50 (7.00)
WAIS-R Vocabulary	—	8	8	10.00 (3.00)
Visual-Spatial				
WAIS-R Block Design	—	—	8	10.00 (3.00)
DRS Construction	4*	5	6	5.80 (.60)
Executive Functions				
DRS category fluency	12*	6*	9*	≥ 13
DRS attention	36	36	36	35.50 (1.60)
DRS initiation/perseveration	25*	18*	25*	35.50 (3.00)
DRS conceptualization	37	27*	29*	37.20 (2.60)
Contrasting motor programs	18*	7*	3*	Not normed**
Go/no-go	18*	15*	3*	Not normed**
Trails A	—	156*	69*	48.00 (13.50)
Trails B	—	>300*	>300*	128.00 (39.50)
Motor				
Finger tapping dominant	—	54	57	48.47 (7.90)
Finger tapping nondominant	—	51	64	44.04 (5.65)

*Significant impairment; DRS = Dementia Rating Scale¹³; WAIS-R = Wechsler Adult Intelligence Scale—Revised¹⁴; WMS-R = Wechsler Memory Scale—Revised¹⁷; Boston Naming Test¹⁸; WAIS-R Vocabulary and Block Design scores are age-corrected scaled scores; contrasting motor programs and go/no-go = number of errors across 20 trials in each paradigm.

**These measures are not normed, but most healthy subjects do not make errors. Trails A and B¹⁹ scores represent seconds to completion; finger tapping²⁰ scores represent mean number of taps across four 10-second trials with the dominant and nondominant hands.

the aforementioned left frontal hemorrhagic contusions remained visible, as did blood in the left mesial occipital lobe (see Fig. 1).

Neuropsychological Evaluation

On neuropsychological testing, the patient demonstrated further cognitive decline (Dementia Rating Scale¹³ = 101) and continued to demonstrate significant impairment of verbal memory and frontal-executive functioning. Visuoconstructive skills, which had been very mildly impaired, were within normal limits. On nonverbal memory testing, his immediate recall was in the low average range, although he forgot all of this information across a delay. Language, visuo-perceptual, and attentional abilities remained intact. Furthermore, the patient performed above the mean on a test of motor speed (see Table 1). Regarding affect, he presented with pseudo-bulbar characteristics in that he would occasionally grimace and appear to be crying; however, no tears were evident and he was easily redirected.

Psychophysiological Testing

Additionally, psychophysiological recording was performed as the patient engaged in imagery, viewed pictures, and engaged in cognitive tasks. He did not exhibit significant differences in skin conductance level (SCL) or skin conductance reactivity (SCR) in response to pictures of familiar versus unfamiliar stimuli. His values across these autonomic modalities did not increase in response to pictures of family members, whom he recognized, or to pictures of his house or its interior. In contrast, the patient exhibited a marked increase in SCR during a mental arithmetic task involving serial seven subtractions, indicating that psychophysiological reactivity accompanied effortful demands for focused attention and working memory. He also responded significantly during an imagery task during which he was asked to imagine his feelings upon realizing that he had a duplicate house. The patient exhibited his largest SCR response during the final condition (5 minutes), when he was asked to discuss these feelings with the examiners. Dur-

Table 2. Skin Conductance Levels and Responses to Various Stimuli

Condition	Mean SCL (μ Siemens)	Change in SCR (μ Siemens)
Resting	2.40	<.10
Mental arithmetic	4.30	2.68
Imagery of duplicated house	4.65	2.52
Pictures	3.10	<.10
Familiar people/objects		<.10
Unfamiliar people/objects		<.10
Patient's house		<.10
Describing feelings re: RP	3.75	1.65
Brief emotional release	5.70	3.48

Mean SCL = mean skin conductance level for that condition; change in SCR = maximum change in skin conductance response relative to baseline within that condition.

ing this condition, he exhibited pseudobulbar characteristics with rapid affective change, facial grimacing, and an expression of intense sadness (Table 2).

After his 5-day hospitalization, the patient was transferred to a nursing home where he stayed for approximately 1 week before returning home without incident.

Follow-Up

Description of Delusion

The patient was seen for another neuropsychological evaluation 24 days following the onset of RP. According to his wife, there had been no evidence of his delusion upon his second return home, and he had been functioning well relative to recent weeks. We asked the patient about his feeling that he owned two duplicate houses, to which he replied, "I don't feel it. I know it." He calmly added that he was now living in the correct home and that he was not going to worry about the other one until he was feeling stronger physically. Furthermore, he suggested that he and the examiner go for a ride in the car sometime soon thereafter, in order to provide the patient with a chance to prove the existence of the two houses.

Neuropsychological Examination

During this evaluation, we had the opportunity to administer a more comprehensive neuropsychological battery. Overall intellectual functioning was found to be in the average range (Wechsler Adult Intelligence Scale—Revised¹⁴: Full Scale IQ = 96, Verbal IQ = 101, Performance IQ = 90). Cognitive functioning, although still below that observed during his initial assessment, had improved somewhat relative to his most recent efforts (Dementia Rating Scale¹³ = 110). His verbal memory impairment was similar to that observed during previous testing. Regarding nonverbal memory, the patient was able to recall an average amount of information immediately, but forgot all of this material across a delay. Performance on tests of frontal-executive abilities had

Table 3. Visual Assessment Battery Performance at Follow-Up

Discrimination Tested	% Correct	Expected % Correct
Frequency	92	90 (9)
Location	92	90 (10)
Orientation	80	68 (16)
Pattern	88	91 (10)
Trajectory	75	67 (19)
Velocity	88	90 (10)

Visual Assessment Battery¹⁵; % correct values show our patient's performance; expected % correct values are mean values for individuals above the age of 65. Values in parentheses are standard deviations for these means.

improved relative to his most recent testing session but was still in the impaired range. As observed previously, the patient demonstrated intact language and visuo-constructive skills and motor speed. In order to assess his visuospatial skills more thoroughly, he was administered a computer-generated test battery that required him to discriminate among stimuli based on the following visuospatial parameters: spatial frequency, location, orientation, pattern, trajectory, and velocity.¹⁵ As is shown in Table 3, he performed within normal limits in all of these domains. The patient's affect during this assessment was marked by exaggerated joviality, and he made mildly disinhibited comments regarding many of the test materials that were placed before him.

DISCUSSION

The present case demonstrates several potentially important issues related to the development of RP. The patient experienced RP following an ischemic stroke that produced a large infarction in the right frontal cortex and associated subcortical white matter. Because he had been hospitalized only 2 weeks before this event, we had the unique opportunity to obtain brain imaging, neurologic, and neuropsychological data just prior to and shortly after the development of RP. This enabled us to examine several mechanisms that have been theorized to explain this delusion.

Prior to his right frontal stroke, the patient had no significant psychiatric history and exhibited no evidence of delusional ideation. There is no reason to believe that his delusional symptoms were related to defense mechanisms associated with denial of illness or other psychological phenomena that have been previously proposed.⁸

Prior to his development of RP, the patient had exhibited cognitive impairment limited to problems with learning and memory and executive functions. These were observed at the time that he was hospitalized secondary to a fall that produced two small left frontal hemorrhagic contusions. The fact that he showed no RP at this point in time suggests that these hemorrhages, along with moderate atrophy and small-vessel ischemic

changes, were not sufficient to produce his subsequent delusion. Therefore, our subsequent data suggest that the development of a right frontal lesion was a necessary condition for RP to occur.

Interestingly, there was little change in pattern of neuropsychological impairment between the patient's first and second neuropsychological assessments. He showed a worsening of his already impaired executive functioning but otherwise was unchanged. In fact, attentional, language, visual, and motor functions were intact. Therefore, it does not appear that RP resulted from factors associated with verbal or visual processing capacity.

The fact that the patient showed intact object recognition, visuospatial and visuomotor performance is particularly important, as some past theories of RP have postulated that this delusion results from an interaction of spatial and frontal-executive impairments. The patient's right frontal lesion was dorsally located, raising the possibility that dorsal visual pathways involved in spatial perception were affected. Yet, he performed very well on a highly controlled visual assessment battery. Regarding the lesion in the left calcarine cortex, it appeared to be so small as to have been clinically insignificant. Although it is certainly possible that visuospatial impairment may give rise to RP via distortions of familiarity, this does not appear to have been the mechanism underlying RP in this case.

Regarding the possibility that the patient became delusional secondary to a disconnection of new memory registration from preexisting memory stores,⁴ the present findings are somewhat less conclusive. Staton's hypothesis requires the presence of posterior lesions that disconnect right temporal-parietal areas involved in memory.⁴ The patient did not have such lesions and had memory problems prior to his RP that did not worsen substantially with RP onset. Therefore, it does not appear that memory dysfunction is a sufficient catalyst for the development of RP, although it is quite possible that having a memory disorder is necessary to its genesis.

The hypothesis that fits most closely with the present case is that RP results from a disconnection of temporal-limbic and frontal regions resulting in distortions of familiarity.³ The most striking aspect of the patient's delusion and behavior was the fact that he was certain that he was in a duplicate house, based not on any physical characteristics of the environment but on his subjective experience that it did "not feel like home." Furthermore, he exhibited altered emotional reactivity when discussing his home, with evidence of pseudobulbar affect. On psychophysiologic assessment, the patient showed a lack of autonomic reactivity to pictures of the very house associated with his delusion, did not show differential reactivity to pictures of familiar and unfamiliar people, and yet showed a dramatic response when he was asked to imagine and describe his feelings when he realized he was in the duplicate house. While

limited to a single case, these data are generally consistent with those of Ellis et al, who found that patients with Capgras syndrome demonstrated uniform autonomic hyporesponsivity to familiar and unfamiliar faces but had normal orienting responses to auditory tones.¹⁶ Our findings of distorted sense of familiarity, pseudobulbar affect, and altered autonomic reactivity strongly suggest that the patient's delusion is closely tied to changes in the quality of his emotional processing and subjective experience.

Despite the fact that this hypothesis seems to provide the most plausible explanation of RP in this case, several issues remain unresolved. The right frontal lesion observed on MRI was rather posterior and dorsal relative to what one might expect if there is to be a frontal-temporal-limbic disconnection. While there was evidence of white matter involvement, there was no evidence of obvious involvement of the basal forebrain or subcortical pathways involved in limbic processing. It is noteworthy that the patient's brain imaging revealed moderate frontotemporal atrophy prior to the onset of his delusion. Furthermore, it is possible that his fall led to some form of trauma-related frontotemporal dysfunction. Perhaps his right frontal infarction, superimposed on these preexisting conditions, led to the development of RP.

With respect to the delusion itself, alteration in the patient's sense of familiarity does not fully explain its persistence. Although he recognized that his account of a duplicate house must sound outlandish, he was unable to dismiss the delusion as simply his brain playing a trick on him. Instead, he insisted that it was reality and offered to take the examiner to the duplicate house. Therefore, beyond the phenomenologic aspects of this case, it appears likely that, independent of the alteration in his sense of familiarity associated with the frontal lesion, executive impairments caused problems with abstraction and reasoning that contributed to the maintenance of the delusion. Similar explanations of the role of frontal functions in RP have been proposed.^{3,4,9,10}

Perhaps the most puzzling aspect of this and other RP cases is the specificity of the delusion. For the patient, the only entity that truly appeared to have been duplicated was the inside of his home. We can only speculate as to the reason for this. The home is obviously an emotionally laden object for most people, as it represents our territory and in some sense defines who we are. This appears to have been particularly true for the patient, as he had personally renovated his home's interior and had reportedly done so with great care and meticulous craftsmanship. Furthermore, patients with RP do not typically perceive emotionally neutral or random objects to have been duplicated. On the contrary, it appears that RP is most likely to occur in response to events or objects in which the patient has a particularly strong emotional and personal investment. It is possible that when the

patient with RP encounters such an object, the subjective sense of distorted familiarity is at its most striking.

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