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Reduplicative paramnesia for places: A comprehensive review of the literature and a new case report

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ABSTRACT

Reduplicative paramnesia for places (i.e., the delusional belief that a place has been duplicated or exists in two different locations) is a rare disorder observed in neurological patients. We review the existing literature on the topic, highlighting commonalities and differences among the 51 cases published since the first report in 1903.

Our results highlight the combination of multiple factors in the pathogenesis of this monothematic spatial delusion. From a neurological perspective, a crucial role is played by damage to the right frontal and temporal lobe. Deficits of non-verbal memory and executive functions, along with topographical disorientation, appear to be the most common (but, not systematic) cognitive impairments. The clinical picture of the disorder is further complicated by often overlooked psychological and motivational factors. Consequently, the precise neuro-cognitive substrate of this disorder is yet to be described in detail.

We stress the need for a more detailed and systematic approach exploiting neurological, neuroimaging, neuropsychological and psychopathological methods. To guide future investigations, we provide clinical- and research-oriented recommendations. Finally, we illustrate the interplay of all above-mentioned factors with a new case report.

1. Introduction

Orientation in time and space is a key feature of the fully conscious and responsive human being. Indeed, asking current date and location is the initial step of most neurological and neuropsychological bedside examinations. Disorientation in time and/or space is a frequent sign of acquired brain injury (traumatic, vascular, neurodegenerative, neoplastic, etc.). Generally speaking, the lack of orientation in space is characterized by the inability to correctly locate the current environment. When asked, the patient is unable to identify his/her whereabouts, or states that s/he is in a different place. In exceptional cases, a peculiar symptom has been described in subjects who strongly believe that an environment (e.g., their house) has been duplicated and/or exists in two different places simultaneously. Even though the first description of the phenomenon may be attributed to Charles Bonnet in 1788 (cited in [1]), the first scientific report was provided by the Czech neurologist and psychiatrist Arnold Pick. An elderly woman admitted to his ward asserted that the entire hospital had been replicated and moved from Prague (the "town clinic") to her birthplace (the "suburb clinic"). Pick named this symptom *reduplicative paramnesia* [2]. Another typical manifestation of the disorder is that of a patient who believes that he is in two (or more) different places at the same time. The novelist Henry James suffered from this latter condition and masterly described the feeling of disorientation associated with it [3].

To date, no consensus has been reached on the core neurological and neuropsychological features of this peculiar syndrome. Since the disorder is extremely rare, group studies are not a viable approach, and thorough neurological and neuropsychological investigations of single cases are still the main data source. A first attempt at describing the different manifestations of the syndrome has been made by Politis and Loane [4]. We update and expand their results by reviewing all published cases. Notwithstanding the variability of clinical pictures and the heterogeneity of the neuropsychological evaluations conducted in each case, data allows an outline of the prototypical patient in terms of

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demographic features, neural correlates and neuropsychological profile. A finer-grained description of the phenomenology can provide clinicians with helpful information for diagnosis, prognosis and treatment of the disorder, and cognitive neuroscientists with insights on its neuro-functional substrate.

Following a review of the literature and the presentation of a new case, we discuss the relation between neurological and psychiatric cases (Conclusions, Sect. 1) and propose a new classification (Conclusions, Sect. 2). We also provide clinical recommendations (Conclusions, Sect. 3) and list the core and accessory features (Conclusions, Sect. 4) of this peculiar disorder. Finally, we discuss the theoretical implications of the findings (Conclusions, Sect. 5).

2. The review

Our search was carried out through two on-line databases (i.e. PubMed/Medline and WebOfScience). We implemented the keywords: “reduplicative paramnesia” and “environmental reduplication”. We then used the references in each paper to expand the selection of studies considered for the review. We included case reports that: (1) described one or more cases showing a monothematic delusion that had a place as its main content; (2) did not present a psychiatric disorder. Following these criteria, we identified 53 published cases (see Fig. 1). Two of these received a clinically confirmed diagnosis of schizophrenia [5,6]. A critical analysis of these two psychiatric cases, in the light of the subjects with verified neurological diagnoses, is included in the Conclusions. All cases included in the review are briefly described in Suppl. Mat. 1, Commented bibliography.

It must be stressed at the outset that the papers included in the present review span over 100 years, during which the tools and techniques available for neurological and neuropsychological diagnoses have changed substantially, along with our understanding of the brain structures and functions involved in this delusion. This has two implications. First, since anatomical correlates were sought in each study with the neuroimaging technologies available at the time, failure to find damage to a given brain structure could be due to the use of a tool that was not sensitive enough (e.g., a metabolic and functional alteration would be detected by PET or fMRI, but not by CT-scan). Second, given that case reports focus on impairments, when no explicit mention is made of a particular cognitive skill, it is not clear whether that function was preserved or never tested. For these reasons, in this review, for each variable we report the number of subject presenting (or not presenting) a given feature, out of the number of subjects in whom that feature was explicitly taken into consideration (rather than out of the entire sample of 51 cases).

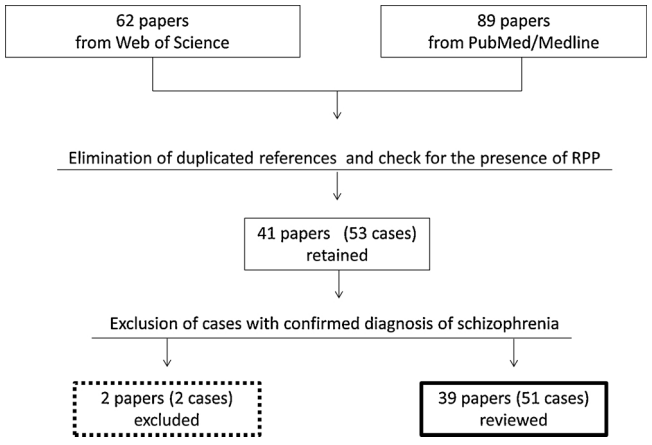

Fig. 1. Schematic representation of the selection process.
We aimed at including all cases of monothematic spatial delusion described in literature, excluding only those presenting neuropsychiatric symptoms.

3. Definition

Generally speaking, *reduplicative paramnesia*, recognized as one of the so-called *delusional syndromes of misidentification and/or reduplication* (DSMRs), entails the delusion (i.e. a fixed belief not amenable to change in light of conflicting evidence) that a place, an object, or an event has been duplicated or exists in two different places at the same time [7]. In most reported cases, the content of the delusion is a place (e.g., the patient’s house or the hospital). This variant is generally referred to as *reduplicative paramnesia for places*. Contrary to other DSMRs such as Cotard’s syndrome (i.e. the patient holds the delusion that s/he is dead [1]) and Capgras’s syndrome (i.e. the patient holds the delusion that some close friend or family member has been replaced by an impostor [8]), *reduplicative paramnesia* has been observed predominantly in neurological (as opposed to psychiatric) cases. This overwhelmingly neurological origin, as opposed to the mainly psychiatric nature of the other DSMRs, has two implications.

Firstly, patients manifesting *reduplicative paramnesia* fail to meet criteria for delusional disorder according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-V). In order to receive this diagnosis, a patient (1) should present a delusion lasting over a month, but (2) should not meet the diagnostic criteria for schizophrenia; (3) should function relatively well, apart from the impact of the delusion; (4) should suffer from minor (if any) mood episodes; (5) should not present the disorder as the consequence of substance abuse or of a general medical condition [9]. This last criterion separates “secondary”, organically-based, delusional disorders from “primary”, psychiatric disorders – a distinction introduced by the DSM-III in 1980. Such subdivision appears to be increasingly problematic in light of the fact that improvement in neuroimaging techniques is leading to reports of functional and structural brain pathology even in classical psychiatric delusions (e.g., schizophrenia). However, the distinction still holds and not without consequences: depending on the diagnosis, a patient will be assigned to different health professionals, thus radically changing the way the case is going to be treated and studied.

Secondly, the study of patients with this peculiar monothematic delusion for space can help elucidate the neurobiological substrate of delusions and related cognitive dysfunctions, in particular disorders of memory and spatial cognition. Different theories have been proposed to account for the symptoms observed in these individuals. Initially, psychodynamic and psychological perspectives dominated, stressing the role of motivational, emotional and environmental factors [10,11]. Subsequently, largely due to the development of brain imaging techniques and to improved neuropsychological practice, new theories have been put forward, highlighting the neurofunctonal underpinnings of the disorder [12–15]. However, given the paucity of data, to date there is no consensus on the neurobiological substrate of this exceptional symptom.

4. Terminology

Various terms have been used to refer to this disorder (Table 1). *Reduplicative paramnesia* is the most widely used (in 23 papers, 46.9%). Alternatives include: *environmental reduplication* [16–18], *topographical delusion* ([19,20]), *misidentification* [21,22], *spatial disorientation* [11], *partial orientation* [23], *reduplication for place* [10], *disorientation for place* [24], *spatial delirium* [25], *delusion for place* [26]. The choice of terminology is linked to the theoretical interpretation of the disorder: when the term ‘reduplicative paramnesia’ is selected, the phenomenon is interpreted primarily as a distortion or disconnection of memories [7,12]; when the term ‘disorientation’ is preferred, the problem is framed in the larger context of visuo-spatial disorders [24]. However, often the distinction does not correspond to clear differences across subjects, and patients to whom different labels are applied in different reports share more clinical features than patients described under the same label in the same report. Moreover, none of these terms fully

Table 1
Single cases reviewed: demographic and neurological characteristic of the patients.

ID	Reference	Term used	Gender	Handness	Age	Etiology	Lesion site	Hemisphere
1	[2]	reduplicative paramnesia	F		67	dementia senilis		
2	[11]	spatial disorientation	M	R	22	TBI	T	R
3			M	R	25	TBI	dilated ventricles	B
4	[23]	partial orientation	M			TBI	movable foreign body in third ventricle	
5	[10]	reduplication for place	F		53	metastatic neoplasm	cerebellum	R
6	[7]	reduplicative paramnesia	M		49	TBI	F	R > L
7			M		28	TBI	spread	R
8			M		25	TBI	spread	R > L
9	[19]	topographical delusion	M		72	probable stroke	PT	R
10	[16]	environmental reduplication	F	R	64	glioblastoma	P	R
11			F	R	23	haematoma	FP	R
12			M	R	29	meningioma	F	R
13			F		60	subdural haematoma	F	R
14	[12]	reduplicative paramnesia	M		23	TBI	widespread	R > L
15	[24]	disorientation for place	M		72	stroke	PO	R
16	[17]	environmental reduplication	M	R	74	haemorrhage	thalamus	R
17	[14]	reduplicative paramnesia	M		66	stroke		R
18	Ewert et al. 1985	reduplicative paramnesia				trauma	F	R > L
19	[27]	reduplicative paramnesia	M	R	25	subdural hygroma following TBI		R
20	[28]	reduplicative paramnesia	M	R	71	haemorrhage	F	R
21	[29]	reduplicative paramnesia	M		64	nr	no focal	
22			M		63	stroke		R
23			M		23	contusion		R
24			M		56	stroke		R
25	[20]	topographical delusion	F	L (corrected)	61	stroke	FT	R
26	[25]	spatial delirium	M	R	85	subcortical ischemia	corona radiata and internal capsule	R
27	[30]	environmental reduplication	M	ambidextrous	71	haemorrhage	thalamus, corona radiata and internal capsule	R
28	[31]	reduplicative paramnesia	M		69	recurrent migraine	no focal	
29	[26]	delusion for place	F	R	64	HSE + haemorrhage	T	R > L
30	Luzzatti and Verga 1996	reduplicative paramnesia	M		64	subdural haematoma	FTP	R
31	[32]	reduplicative paramnesia	M	R	52	ACA aneurysm	F	R
32			M	R	52	TBI	F	B
33	[33]	reduplicative paramnesia	M	R	81	ischemic stroke	F	R
34	[21]	misidentification/reduplication	M	R	51	trauma	TF	R > L
35	[34]	reduplicative paramnesia	M	L	45	stroke	T-P-O junction	L
36	[22]	misidentification	F		71	ischemic stroke	T	R
37	[35]	reduplicative paramnesia	M	R	67	subaranchnoid haemorrhage	FTP	R
38	[36]	reduplicative paramnesia	F	R	73	meningioma	FT	R
39	[18]	environmental reduplication	F	R	66	ischemic stroke	PT	R
40	[37]	environmental reduplicative paramnesia	M		75	AD	FT	R > L
41	[38]	reduplicative paramnesia	F		71	glioblastoma	genu corpus callosum	
42	[39]	reduplicative paramnesia	M		64	Morvan syndrome		
43	[40]	reduplicative paramnesia	F	L	72	meningioma	T	R
44	[41]	reduplicative paramnesia	M	R	69	stroke	F cortical and subcortical	R
45	Gerace and Blundo	reduplicative paramnesia	M		63	ischemic stroke	FT	R
46	2012		M		80	atrophy	FT	R > L
47			F		80	atrophy	F	
48	[42]	environmental reduplicative paramnesia	M	R	53	trauma	FT	R
49	[43]	reduplicative paramnesia	M		66	CVA	FTP	R
50	[44]	reduplicative paramnesia	F	R	88	stroke	thalamus	R
51	[45]	reduplicative paramnesia	M	R	55	glioblastoma	FTP	R

F female, M male, R right-hander, L left-hander, Amb ambidextrous, B bilateral,* corrected left-hander, F frontal, T temporal, P parietal A blank entry illustrates the failure to identify a given data in a paper.

captures the complexity of the disorder. They typically stress only one of its components (e.g. memory) whereas combinations of spatial, executive and memory disorders underpin the spatial delusion. For these reasons, our review includes all the patients presenting the symptom, irrespective of the label used to describe them. While being fully aware of the above-mentioned terminological limitations and ambiguities, for exposition purposes we opted for the label most commonly used to refer to the disorder (*reduplicative paramnesia for places,* henceforth RPP).

5. Epidemiology

Our search of the literature revealed 53 patients presenting RPP from 1903 to October 2017. Two of these received a diagnosis of schizophrenia and were therefore excluded from further analyses. Few authors have attempted to characterize the prevalence of RPP in specific neurological or psychiatric populations ([24,29,46–48,32,10]). From these group studies, we included only single patients described in sufficient detail.

Hakim et al. [29] described RPP in 4 out of 50 consecutive patients

Table 2
Summary report of the patients' delusions.

ID	Copy	Displacement	Length	Resolution	Anosognosia	Confabulation	Description of the RPP
1	Yes	Yes	2			Yes	She believed there was a "town clinic" and a "suburb clinic", and that there was a third floor in the "clinic with another room n.118 (the patient's room)
2	Yes	Yes	2	letter from wife	Yes		"Grimsby and Scotland are the same place. […] It's Scotland on the map but it's Grimsby nearer home"
3	No	Yes	4				He believed the hospital, C, was in Somerset normally, but had moved to Scotland.
4	No	Yes					He believed he was in San Diego and Long Beach at the same time.
5	Yes	Yes	4	died			[there are] "Two hospitals" [one is a] "sort of branch, although they don't charge you any less."
6	Yes	Yes	6	general improvement			Hospital is copied from Boston to Taunton
7	No	Yes	2	general improvement			Hospital is in Great Falls, not in Boston
8	No	Yes	2	general improvement			Hospital is in Sault St Marie, not in Boston
9	Yes	Yes	52	neuroleptic	Yes	No	He believed the hospital to be in Villafranche de Rouerghe, not in Lyon, and that there was a second floor in the apartment.
10	Yes	Yes	4	general improvement			"Doctors had constructed a duplicate Memorial Hospital" in her home
11	Yes	Yes	1	general improvement			She stated that it was kind of the New York Hospital to have constructed an annex in her home.
12	Yes	Yes	4	general improvement			He claimed he was at home, in a branch of the New York Hospital.
13	Yes	Yes	4	general improvement			She stated that it was very convenient to have the New York Hospital in her home.
14	Yes	No	416	antipsycotic		Yes	"They didn't have this kind of hospital in Fargo, it must be somewhere else"
15	Yes	Yes	4	improvement visuo-spatial	Yes		He believed he was in "a branch of the MGH"
16	No	Yes					"It is a nice clinic like the one you have mentioned but it isn't in Buenos Aires, it is in Finland".
17	Yes	Yes	28		No		He provided a descriptions of different hospitals, all with the same name, usually located in northeast Ohio and northwest Pennsylvania.
18				improvement memory			nr
19	Yes	Yes	12	general improvement			He stated that there were two hospitals, one in Denver and one in El Paso.
20	Yes	Yes	12	improvement memory and visuospatial deficits			He claimed that the house he was living in was not his "real" house, and that he had been transferred to Southampton General Hospital from another hospital nearby.
21							nr
22							nr
23							nr
24							nr
25	No	Yes	2		No		"During the night, you transformed my house in a hospital"
26	Yes	Yes	16	antipsychotic	Yes		He thought he was in Prague one day, in Brussels, Denmark or Morocco on other days.
27	Yes	Yes			No	No	He insisted he had been moved daily to identical hospitals (all with the same name, and located in neighbouring cities).
28	Yes	Yes	1				He believed his original house has been replaced by a cleverly produced copy.
29	No	Yes			No	Yes	"I know I am in the hospital, but it is nevertheless my room at home".
30	Yes	Yes	32	moved back to Brazil			He belived that there is a "Milan-in-Brazil" and a "Milan-Milan".
31	Yes	Yes	12	improvement topographical disorientation		No	He belived in the existance of another rehabilitation center from which he had been transferred.
32	Yes	No	24			No	"I was hospitalized at two K Hospitals consecutively"
33	Yes	Yes	4				He believed he had been taken to a " twin" of his "real home".
34	Yes	Yes	208			Yes	"This house is not my house"
35	Yes	Yes					He believed that he was sometimes in a room at the hospital, sometimes at home.
36	Yes	No	104				She referred to her house as a rented replica of her original home.
37	Yes	Yes	12	general improvement			"I am in some town in California, I know it's not Illinois."
38	Yes	Yes	48	antipsychotic			She insisted on going back to her real house.
39	Yes	No	4	antipsychotic			She believed her car, her furniture and her house had been duplicated.
40	Yes	Yes	48	cognitive decline			He claimed he was in another city about 120 km away, in an apartment identical to his own dwellings in the city of Lausanne.
41	No	Yes					She believed that she was in Bursa and in Istanbul at the same time.
42	Yes	Yes		IVIG			He believed his home had been reduplicated by a stranger, and that the replica existed 40 miles away.
43	Yes	No	4	tumor removal			"Two identical houses on the same street, same floor, same furniture."
44	Yes	Yes	12		Yes		He claimed he had been taken to hospital branches located on the second floor of a convenience store and inside his neighbor's home.
45	Yes	No					"There are the same doctors, the same beds, but this is not my room"

(*continued on next page*)

Table 2 (*continued*)

ID	Copy	Displacement	Length	Resolution	Anosognosia	Confabulation	Description of the RPP
46	Yes	No					He had the feeling that he was in another house, identical to his, but at same time different.
47	Yes	No	52				"It's no longer the house you knew; it's another one, even if it looks the same."
48	Yes	Yes	5	visual confutation		No	He insisted that he was in his hometown in Portugal, in a hospital also called University Hospital of Geneva
49	No	Yes	2				"Everybody is telling me that I am in the hospital, but this is my house."
50	Yes	No	24				"Most of my brain thinks it is not my house. And yet it is."
51	Yes	Yes	1	tumor removal			He maintained that he was in a version of the Mount Sinai Hospital located in San Jose instead of Huston

IVIG Intravenous immunoglobulin. A blank entry illustrates the failure to identify a given data in a paper.

(8% of the total) with a history of alcoholism, admitted to the hospital after an acute neurological event [32]. tested 77 patients with focal brain damage and no communication disorders, documenting RPP in 5 cases (6.5%). Levine and Grek [48] restricted the analysis to an unselected sample of subjects with right hemisphere stroke, finding only 9 cases in a 2-year period. Similarly, Weinstein et al. [10] reported observing 16 patients with RPP. Since these last two studies did not report the total number of patients tested for RPP, the prevalence of the disorder cannot be estimated. Likewise, Fisher [24] reports 1 case of RPP and refers to 6 other patients, less well documented, encountered in his professional life. In a dementia population, a low incidence of RPP was reported [46]: in a total sample of 724 patients, only 7 cases of RPP were identified (0,96%). In this study, the disorder was observed in 6/392 subjects with Alzheimer's Disease (1.5%) and 1/24 patients with Semantic Dementia (4.2%), while no cases of RPP were observed in patients with Lewy Body Dementia (n = 36), behavioral variant of Fronto-Temporal Dementia (n = 119), Primary Progressive Aphasia (n = 101), Progressive Supranuclear Palsy (n = 8), Corticobasal Degeneration (n = 18), and Parkinson's Disease (n = 26). The prevalence of DSMRs (including RPP) was assessed also in a psychiatric population [47]. In a sample of 195 patients, they observed DSMRs (Capgras's delusion and/or subjective doubles syndromes) in 8 cases, none of whom showed RPP. Taken together, these findings confirm the rarity of the symptom while stressing that neurological damage is by far its most frequent cause.

6. Demographic profile and characteristics of the spatial delusion

Reported RPP patients are more frequently male (70.5%) and right-handed (84%). Mean age in the sample retained for discussion was 58.4 years (SD 18.2). The place that is the object of the RPP is the hospital in most cases (Table 2). Such place can be duplicated (i.e. there is an explicit mention of a copy) and/or displaced (i.e. it is re-located elsewhere). The location of the copy is usually someplace near the patient's home (e.g., [16]) or the house of someone related to him/her (e.g. [41]), but it can also be in a completely different city (e.g. [11]), at times far away (e.g. [24],). Occasionally the house of the patient is duplicated (as in [40]). Some cases present a simultaneous reduplication of people [36,38] or body parts [29,30]. For instance, the patient described by Yamada et al. [36] believed she had two husbands labeled 'this husband' and 'that husband', while the hemiplegic patient described by Berthier et al. [30] reported having three left legs. The location of the duplicated place often appears to be of emotional value for the patient, frequently bringing him/her closer to loved ones (e.g., Hinkebein et al. [35]).

The possibility that the disorder is caused solely by the fact that the patient is under medical care can be ruled out. Although many patients show the delusion while in a hospital, in some cases RPP either was the reason for seeking neurological advice or appeared only after discharge from the hospital. For instance, Kapur et al. [28] describe a patient who, after having gone missing for about 5 h, arrived home and started claiming that the house he had just returned to was not his own. He was later admitted to the hospital, and a CT-scan confirmed an otherwise asymptomatic hemorrhage in the right frontal lobe. On the other hand [33], described the case of a man who manifested RPP for his own house three days after discharge from the hospital, where small hemorrhagic contusions in the left frontal lobe had been found. After the onset of RPP, MRI showed a right frontal ischemic infarct as well. Finally, in the patient described by Yamada et al. [36] the symptom appeared 3 months after removal of a right inferior frontotemporal meningioma.

Familiarity with a given place, as well as the desire to be somewhere else, appear to be key factors in RPP. However, while these emotional factors can determine content and mood of the RPP, they do not suffice to trigger it. The affective tone is negative in many cases, with the patient expressing paranoid feelings ([27,41]) or the desire to go back

to the “real” house [22,28]. In an emblematic case [12] the reduplication involved all relatives and friends, along with the hospital and the city itself, and was associated with strongly negative feelings. This patient was described as being suspicious but not paranoid, manifesting a reactive depression and expressing low self-esteem and pessimism about the future. Few patients express positive feelings, mainly happiness for “being” closer to their relatives [16,20]. In some cases, the emotional tone is detached, resulting in a sort of anosodiaphoria (i.e. lack of emotional reaction to the sequelae of brain injury [49]) or even anosognosia (observed in 5 of the cases reviewed here ([11,19,24,25,41]). By definition, delusional beliefs are held strongly, resisting any physical evidence provided by hospital personnel and/or relatives. To date, only one case of RPP responded positively to confrontation by unequivocal visual information [42]. In some cases, patients spontaneously explain and justify their delusion, volunteering details and, at times, even acting it out. Examples include: assuming that the changes in location are a mandatory tour meant to help during rehabilitation [25]; complaining that relatives are trying to steal the patient’s possessions [18]; accusing the hospital of kidnapping [41]; and claiming to be the victim of a Mafia strategy [27]. In three cases, patients provided details on concomitant delusions concerning a person, but not on place delusions [10,16,21].

7. Neurological profile

Regarding etiology and mode of onset, RPP can follow an acute neurological event – i.e. traumatic brain injury (e.g., [29]) or stroke (e.g. [41]) – as well as a slowly developing condition such as a neurodegenerative disease ([2,37]). In few cases, no abnormalities were detected by CT scan (e.g [29,31]); or MRI [39].

A lateralized lesion – focal or diffuse – was found in 34 cases. Damage affected the right hemisphere in all cases, with the exception of one left-handed patient with probable reversed hemispheric dominance [34]. Bilateral lesions were documented in 10 patients. In 8 of these, damage was more extensive in the right hemisphere.

Information on the localization of the lesion is present in almost all cases (n = 47, with the exception of [29,31,39], and [2]). Damage involved exclusively the frontal lobe in 10 subjects, while a fronto-temporal lesion was observed in 7 cases, a fronto-parietal lesion in 1 and a fronto-temporo-parietal lesion in 3. Damage was restricted to the temporal lobe in 4 cases and to the parietal lobe in 1. Lesions were parieto-occipital in 2 cases, parieto-temporal in 2 and temporo-parieto-occipital in 1. Finally, four patients suffered from a subcortical lesion involving the thalamus and/or the corona radiata and the internal capsule. Overall, the frontal lobe was damaged in 21 cases, the temporal lobe in 15, the parietal lobe in 8, and the occipital lobe in 2. In general, lesions circumscribed to a small portion of the cerebral cortex or to a specific subcortical structure are rare while large lesions, probably affecting multiple networks prevail. It is worth stressing that most neurological reports include only CT-scans, and that the finer-grained structural, metabolic and connectivity data necessary to better characterize the neurofunctional substrate of the disorders are missing.

In summary, RPP most commonly follows an acute neurological event. The frontal and temporal lobe of the right hemisphere play a crucial role. Damage may affect different brain regions either directly or indirectly (i.e. perturbing subcortical connections).

8. Neuropsychological profile

Trying to describe the cognitive impairments observed in subjects with RPP as if each deficit was independent from the other is an artifice, given the heterogeneous and composite nature of the syndrome. Furthermore, case reports cover a very long-time span, and neuropsychological assessments vary to a large extent in breadth and depth. With these important caveats, we report in Fig. 2 the main cognitive impairments observed in 51 individuals with RPP. For each functional domain, we indicate the number of individuals showing/not showing difficulties (Fig. 2a), and the number of subjects for whom no data are available as the domain was not examined (Fig. 2b). Individual results are also presented in Suppl. Mat. 2, where the full neuropsychological profile of each patient is reported. In most cases N’s are too small to allow reliable statistical analyses. Therefore, only the relative incidence of each disorder is reported.

8.1. Attention and executive functions

These abilities were impaired in the majority of the RPP cases in which they were investigated. Attentional skills were evaluated in 15 subjects, and found to be impaired in 13 (86.7%). The most common deficit concerned attentional shifting (i.e. the ability to shift attention between two or more tasks or types of stimuli), consistent with the possibility that in these patients a crucial deficit involves the supervisor attentional system (executive in nature). Tasks requiring abstraction and categorization were administered to 17 subjects, 14 of whom obtained pathological scores (82.4%). Similarly, planning and problem solving were impaired in 9 out of 11 subjects (81.8%). Inhibition of behavior was tested in 8 subjects, and found to be impaired in 7 (87.5%).

8.2. Visuo-spatial functions

Unilateral spatial neglect (i.e. the inability to orient, attend and/or respond to controlesional stimuli) was found in 10/14 cases (71.4%). Constructional apraxia (i.e. the inability to build, assemble, or draw objects) was also observed in a majority of cases (17/27, or 62.9%). By contrast, visual agnosia (i.e. the inability to recognize visually presented objects) was reported in only 3/11 patients (27.3%).

8.3. Memory

Verbal and non-verbal long-term memory (vLTM, nvLTM) were tested in 37 and 25 subjects, respectively. Performance was disrupted more often on non-verbal (22/25, or 88%) than on verbal tasks (17/37, or 46%). Verbal and non-verbal Short-Term Memory (vSTM and nvSTM) were tested less frequently (in 26 and 18 studies, respectively). Short-Term memory was impaired less often than Long-term Memory, but also in this case non-verbal tasks were impaired more often than verbal tasks (nvSTM: 11/18, or 61.1%, vs vSTM: 4/26, or 15.4%, respectively). Working memory (WM) was tested, only with verbal material, in 5 patients and was impaired in 4 (80%).

8.4. Temporal and spatial orientation

Orientation in time was investigated in 24 patients, and was poor only in 8 (33.3%). As regards orientation in space, retrograde topographic amnesia (i.e. the inability to remember familiar paths, learned before the onset of the brain lesion) was unimpaired in the 3 subjects in whom it was tested (100%). By contrast, anterograde topographic amnesia (i.e. the inability to learn new paths) was present in 3 cases out of 4 in which it was evaluated (75%). The ability to draw and use a map was impaired in 9/12 cases (75%). Finally, the ability to mentally visualize and describe familiar paths was damaged in 2/10 patients (20%). Thus, it would appear that the key features of topographical disorientation in RPP are ‘heading disorientation’ and anterograde disorientation, which have been associated with lesions to posterior cingulate and parahippocampal cortex respectively [50].

8.5. Anosognosia

Anosognosia is a disorder in which someone affected by a disease does not recognize or acknowledge his/her deficits. Unawareness can affect sensory or motor neurological disorders such as hemiplegia,

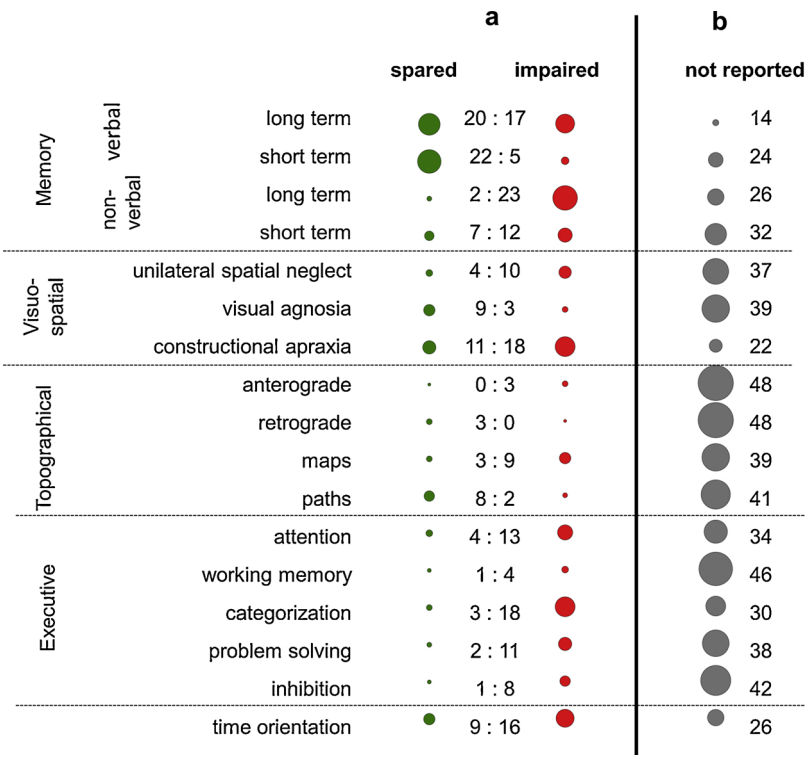

Fig. 2. Neuropsychological profile.
For all the cognitive functions considered, we report: (a) the number of patients in whom a function was spared (green dots) or impaired (red dots) and (b) the number of patients for whom date are not provided. Dots represent the normalized number of patients in each category.

hemianestesia or hemianopia. The most studied variant is anosognosia for hemiplegia, which has been associated with impaired motor planning [51]. Patients with anosognosia for hemiplegia may also manifest delusional beliefs concerning the ownership of contralesional body parts, denying ownership (asomatognosia) or attributing ownership to someone else (somatoparaphrenia) [52]. Anosognosia is also considered a key component of the unilateral spatial neglect syndrome [53]. Similar to RPP, anosognosia occurs more frequently after right hemisphere damage [52], and cannot be explained by sensory or motor deficits per se, or by general cognitive impairments (Ronchi and Vallar, 2006). Clinical manifestations can vary and dissociations have been reported. For example, anosognosia for left hemiplegia may occur in the absence of anosognosia for spatial neglect or hemianesthesia (Bisiach, 1986 [54–56];).

In our review anosognosia was found in 5 out of 8 cases (62.5%, [11,19,24,25,41]). Thus, even though anosognosia and RPP appear related, dissociations are observed. Nevertheless, RPP and anosognosia may share a common origin as both are associated with right-hemisphere damage and can be caused by a combination of lower level (sensory) and higher level (orientation, memory, executive functions) factors, with additional influences of emotional and motivational elements.

8.6. Confabulations

Confabulations are defined as false statements stemming from fabrications or distortions of memories (and not from the conscious intent of fooling the examiner) [57]. They can be provoked (i.e. produced only in response to challenges to memory), or may occur spontaneously [58]. They differ from DSMRs under some key respects: monothematic delusions are virtually always unamenable to changes by confrontation and rather invariable in time (at least as regards their main content); in contrast, most confabulations change frequently and can be stopped by verbal confrontation. When confabulations and RPP co-occur in the same patient, they tend to have a different time course, as confabulations improve more rapidly [21]. In the sample reviewed here, confabulations were explicitly sought only in 9 RPP cases, and detected in 5 (55.6%). One might be tempted to argue that RPP falls into the definition of behaviorally spontaneous confabulation (as reported in [59]). This possibility should be evaluated in future investigations, e.g., by considering performance in reality filtering tasks.

8.7. Additional features

The analysis of I.Q. scores confirms that visual-spatial abilities are more severely affected than verbal skills. Performance I.Q. was lower than verbal I.Q. in 28/29 cases (96.5%), and both measures were similarly affected in the remaining patient. Some disorders were described only in few cases: apraxia [16,24], prosopagnosia ([22,26,37,28]), autotopoagnosia – i.e. the inability to localize and orient different parts of the body [34], left-right disorientation [19], and hallucinations [31,38].

In summary, patients presenting with RPP perform worse in visuo-spatial tasks than in verbal tasks. Coherently, performance I.Q. is lower than verbal I.Q. in an overwhelming majority of cases and non-verbal LTM and STM are more frequently and severely impaired than verbal LTM and STM. Anterograde topographic amnesia occurs more frequently than retrograde topographic amnesia and, accordingly, topographic visual imagery is less severely affected than the ability to draw and use a map. In addition, LTM is more frequently impaired than STM, in both verbal and non-verbal contexts. Executive functions (i.e. abstraction, categorization, planning, monitoring and inhibition) are usually impaired, and neglect and anosognosia co-occurs frequently. The relations between RPP and confabulations require further scrutiny.

9. Psychological profile

Albeit early accounts suggested psychological factors (e.g., personal

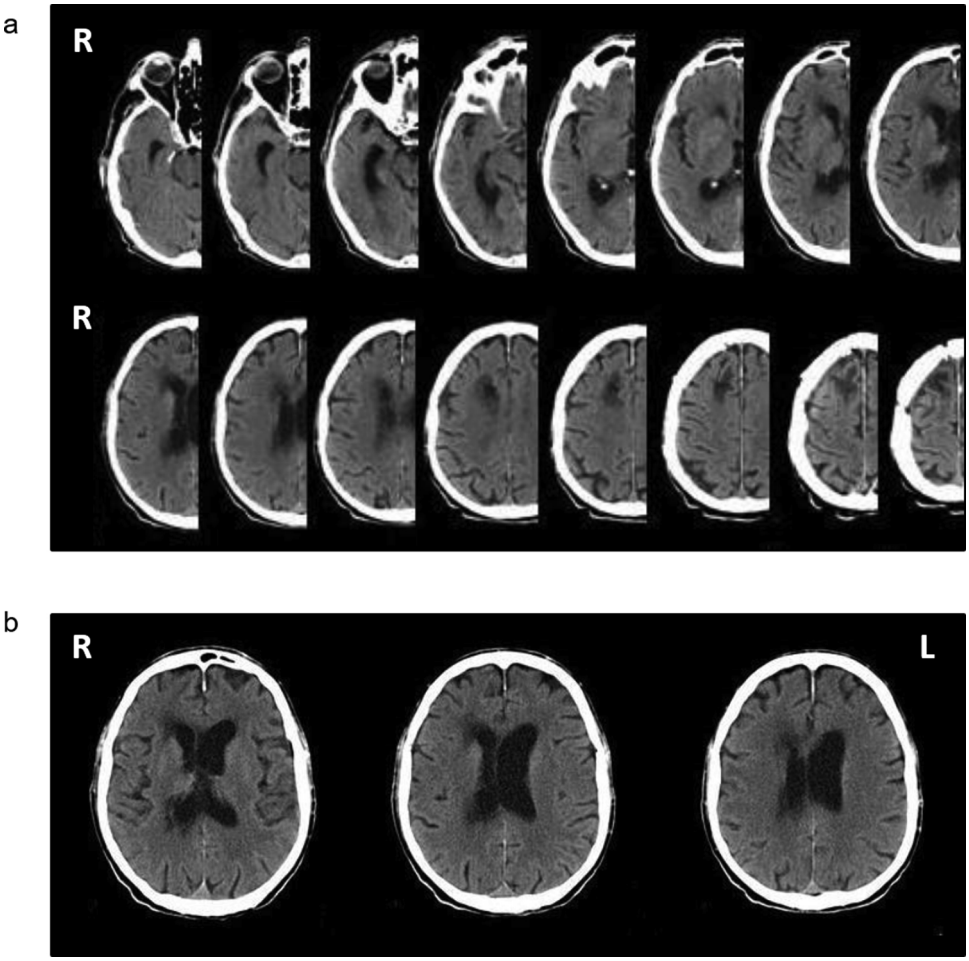

Fig. 3. Neuroimaging findings.
The results of the CT-scan performed 3 months post-onset showing (a) details of the right frontal lobe lesion, and (b) asymmetric ventricular dilatation.

motivations and premorbid personality traits) as key elements to explain the development and the content of RPP, insufficient data are available in published reports to fully appreciate their influence. However, careful reading of case descriptions highlights some crucial emotional and psychological features:

- strong desire to be home [11,16];
- inclination to deny psychological concerns [14];
- emotional dependency [14].

Paranoid tendencies are often observed once RPP is established ([18,27,36,41]), and are usually associated with negative emotions, such as anger, sadness, and fear. However, it is not easy to establish whether such feelings were already present premorbidly, albeit to a lesser extent. Only von Gunten and colleagues (2005) had the opportunity to assess the personality of their patient before the onset of RPP and described him as a narcissistic, controlling and critical person.

In future studies, attention should be paid to these aspects of the patients' global profile, to better understand their role in determining onset, content, maintenance and resolution of RPP.

10. Resolution

It is not easy to assess if and when a delusion has ended. In fact, even though patients may stop expressing distress and/or acting out their delusion, when directly questioned they frequently manifest long-lasting delusional beliefs with respect to the reduplication/displacement experienced in the past (e.g., [37,35]). For instance [33], described a patient who, during the active stage of the delusion, became mildly aggressive and demanded to be brought back to "*his real house*". The delusion was subsequently considered to have resolved, and the patient returned to his home, where he did not show overt delusional beliefs. However, if questioned he would clarify that he was "*now living in the correct home*" and that, to prove his point, he could take the examiner to the second home.

Attempts to treat RPP have met with inconsistent results. Medical treatment with neuroleptic/ antipsychotic drugs was successful in four cases (*risperidone*: [43] and [36]; *quetiapine*: [18]; *haloperidol*: [25]), but unsuccessful in three (*risperidone*: [38]; *haloperidol*: [60] and [37]). Other drug treatments were administered only occasionally. *Intravenous immunoglobulin* and *prednisone* successfully alleviated RPP in a patient with an immunological disease – Morvan's syndrome [39]; but *L-DOPA/carbidopa*, administered to stabilize patient's feeling of "*unreality*", worsened RPP in another subject [12].

Similarly, inconsistent results were observed following neurosurgical treatment. RPP was resolved in two patients following tumor removal [40,45], but was unaffected by partial resection of a right parietal glioblastoma in another case [16].

Verbal and visual confrontations with a therapist have been generally ineffective, except in one case [42]. This patient rejected verbal confrontations with logical arguments, but repeated exposure to unequivocal perceptual evidence restored spatial orientation (researchers took the patient twice a month to see salient landmarks in Geneva, which he was relocating in Portugal). In most cases, patients are impermeable to contrary evidence: a very poignant example is described by Gazzaniga [61]. His patient, prompted to justify the presence of

elevators in what she considered to be her house, replied: "*Doctor, do you know how much it cost me to have those put in?*".

Improvement of RPP occurs more consistently following the amelioration of associated cognitive impairments (e.g., [16]), and especially of disorders of memory (e.g. [28]) visuo-spatial cognition and spatial orientation (e.g. [32]).

11. A new case report: D.A

D.A., a right-handed man of 75, retired and with 8 years of education, suffered from a right capsulo-thalamic hemorrhage with effusion in the ventricles and subsequent acute hydrocephalus (confirmed by CT scan) in February 2012. MRI could not be performed because of a prosthetic heart valve in place since 1997. The hemorrhage was initially treated with a left frontal external ventricular shunt. Due to a persistent intraventricular hematoma, a right frontal craniotomy was subsequently performed. In March, a repeat-CT scan (Fig. 3) showed absence of hydrocephalus, almost complete reabsorption of the hematoma, and asymmetric ventricular enlargement (greater on the right in the temporal section, and on the left in the other sections). In addition, a hypodense area was observed in right prefrontal regions (superior frontal gyrus, possibly extending to adjacent portions of the anterior cingulate cortex). A similar area was present in the periventricular temporal white matter; the temporal horn was enlarged. D.A. was then admitted to a rehabilitation unit, where he showed signs of unilateral spatial neglect for both personal space (e.g., he wouldn't shave the left side of his face) and extra-personal space (e.g., while walking in the ward he would always turn right). When prompted, he demonstrated orientation in space, but not in time, and began to manifest reduplicative delusions concerning his room in the care center. He claimed that every now and then the hospital staff moved him from his room to another exact replica. After discharge, the delusional belief re-emerged only sporadically. On two occasions, while momentarily hosted by his daughter, D.A. asked to be brought to her "*real house*", as opposed to the copy where he felt he had been confined to: very similar to the original, with the same furniture, but not the real one. Neither reduplication nor displacement where manifested with regards to the patient's own home.

A comprehensive neurological and neuropsychological evaluation was conducted about 12 weeks after the stroke (June 2012), in the clinical center to which he had been referred.

Neurological examination revealed a mild left hemiparesis with increased deep tendon reflexes and a Babinski sign. Moderate spastic hypertonus and pareto-spastic gait were present. Grip strength was reduced in the left arm, and the patient could not stand on his left leg. A grasp reflex was elicited on the left; the palmomental reflex was elicited bilaterally (brisker on the right). On double simultaneous stimulation, left-lateralized extinctions were observed in all modalities (tactile, auditory and visual).

Neuropsychological testing documented a cognitive profile closely matching that derived from the literature review. Whenever possible, scores were corrected for age and education based on normative data. D.A. performed within normal limits in a scale measuring functional autonomy in daily life (Catherine Bergego Scale, Azouvi et al., 2003), in which he did not show signs of neglect. However, formal testing revealed both attention and executive impairments. In particular, while selective attention was within normal limits (Trail Making Test, part A), task switching was impaired (similar observations were reported in 86.7% of the reviewed cases, Trail Making Test, part B). Moreover, perseverations and difficulties with abstraction and categorization were detected (as was the case in more than 80% of RPP cases). Executive dysfunction appeared to be at the origin of the poor performance on [62], where mild impairment was observed. When judging the emotion expressed by the different faces, D.A. adopted the unsuccessful strategy of focusing only on the eyes. He was able to recognize sadness only in 2/10 faces, fear in 3/10, and happiness in 9/10. At the time of the formal evaluation D.A. did not present with unilateral spatial neglect (observed in 10 previous cases) nor visual agnosia (observed in 3 RPP cases). There were indications of constructional apraxia: like 62.9% of the patients reviewed here, D.A. had difficulties copying complex drawings. Working memory, verbal and non-verbal short-term memory were spared, whereas in the literature deficits are found respectively in 80%, 15.4% and 61,1% of the cases. As expected from our review, verbal long-term memory was normal (as in 64% of the cases) while non-verbal long-term memory was impaired (as in 88% of the cases). Also unsurprisingly, performance I.Q. was lower than verbal I.Q. (72 vs 84). On formal testing, D.A. presented with anosognosia (previously demonstrated in 5/8 reviewed cases) for motor difficulties, which was resolved by observing movement execution. Finally, no spontaneous confabulations were noticed during the neuropsychological assessment nor could they be elicited by formal testing [63].

We designed ad hoc clinical tasks to further explore the key features of RPP: the interplay between dysexecutive symptoms, temporal and topographic disorientation and memory deficits. Normative data are not available for the tasks that follow, whose results should be taken as qualitative measures.

D.A. manifested persistent temporal disorientation, observed in 33.3% of the RPP patients in which it was tested. To study whether time disorientation extended to retrospective temporal order judgment impairment, we asked him to collocate events on timelines spanning different periods of his life (childhood, early adulthood, late adulthood) and tapping autobiographic (4 lines, 5 events each) as well as semantic (4 lines, 5 events each) knowledge. He had difficulties with both autobiographic and semantic events, with worse performance in the latter (14/20 and 9/20 correct, respectively).

We then assessed D.A.'s ability to memorize visually presented items with and without the support of verbal encoding. To evaluate memory for visual objects with verbal support, D.A. was brought in a naturalistic environment (the kitchen of the Center where testing was taking place) and asked to name 10 items (e.g. fridge, cup, plate, etc.). His ability to recall from memory the same 10 items was tested in an adjacent room 2 min later (8/10 correct), and subsequently after 10 min (6/10 correct). Finally, we assessed his ability to recognize the items among 10 semantically related distractors (10/10 correct). Needless to say, when asked to name the 10 items in the kitchen, D.A. was not forewarned that he would be asked to recall them on a later occasion. To evaluate memory for visual objects in the absence of overt verbal support, D.A. was guided through another naturalistic environment (the gym of the Center) and asked to pay attention to the objects displayed in the room. Once brought back to the testing room, he was first asked to recognize the items observed in the previous room among 10 distractors (4/5 correct), and then to place a photo of each item in the correct position on a picture replica of the original room (3/5 correct). Finally, he correctly selected the picture representing the global configuration of the room among three pictures in whom objects had been moved to different places. These observations indicate preserved abilities to memorize visually presented material, even when verbal encoding was not explicitly prompted.

Signs of retrograde topographic amnesia (found in 3 previous cases) were reported by the caregiver during the first weeks following the vascular accident, but were no longer present at the time of the formal evaluation. By contrast, the patient showed anterograde topographic amnesia and a clear inability to use maps in a real-world scenario (both observed in 75% of the cases in which they were investigated). It should be noted that premorbidly D.A. was a mountain guide with great orienteering skills, teaching adolescents how to navigate through the Alps with the help of cartographic maps. Thus, additional testing was conducted to investigate topographical disorientation in detail. The ability to recognize topographical landmarks was tested by presenting color photographs of famous buildings. D.A. correctly named and located 24/30 national landmarks and 9/11 regional landmarks. Moreover, he correctly placed 7/12 cities on a map of the region. He was then asked to describe verbally 3 familiar paths (e.g. from his house to that of his

daughter), and a square in the center of his town. He successfully described 2 of the 3 paths and retrieved 13 out of 13 key details of the square. When shown a map of the Center and asked to navigate the rooms looking for specific landmarks, he failed the task. He recalled the appearance of the landmarks (e.g., "*there is a red fire extinguisher between two doors*"), but was unable to use the map to guide his steps and to positively recognize landmarks when taken to the correct place.

Overall, performance in these tasks suggests sparing of semantic memory for known landmarks and rules out retrograde topographic amnesia, while confirming difficulties in learning new paths and navigating through a new environment with the help of a map.

A separate interview with the caregiver allowed assessing D.A.'s premorbid personality as well as emotional and behavioral changes. Three different questionnaires were administered to the caregiver, his daughter: the NeuroPsychiatric Inventory (NPI, Cummings et al., 1994), the Frontal Behavioural Inventory (FBI, Kertesz, 1987), and the Big Five Observer (Caprara, Barbaranelli, Borgogni, 1994). The caregiver described D.A. as a very energetic and talkative person, with sense of humor and stubborn tendencies. After the event, his family noticed a stiffening of some personality traits – notably stubbornness and a rather inflexible, rigid thinking. Moreover, he became less friendly and tended to withdraw from social circumstances. This combination led to the emergence of paranoid tendencies and an external locus of control for negative events (i.e., tendency to attribute bad outcomes to causes out of his control). Based on his daughter's report, some pre-existing behavioral traits had worsened during his hospital stay, namely severe apathy and dysthymia with tendency to isolation. While these symptoms greatly improved upon discharge, the caregiver noticed persisting executive difficulties that affected planning, attention maintenance and decision-making. Furthermore, she reported impulsivity, irritability, emotive lability, restlessness, verbosity, and a fair degree of disinhibition (in the form of excessive jocularity).

D.A. underwent cognitive rehabilitation in two phases: first, an intensive training (i.e., 5 sessions per week, for a total of 35 sessions), then an extensive training (i.e. 1 session every 2 weeks, for a total of 24 sessions). The program targeted the improvement of visuo-spatial deficits and dysexecutive symptoms. Three follow-up examinations were conducted in November 2012, August 2013, and January 2014. To describe the evolution of the key neuropsychological features investigated, Table 3 reports the scores in the test administered at the four time points. Overall, the rehabilitation program was effective in partially restoring the executive functions.

Having followed D.A. for about two years, we can examine the evolution of his RPP through his own words. During the first assessment (June 2012), the examiner asked about his staying at the rehabilitation center and at his daughter's house:

Exp. "*Have you been always in the same room?*"

D.A. "*Well, everyone says I have…sometimes I say I haven't. At times, I have slept in another room number 3.*"

D.A. "*I don't know how it happened… They used to bring me the newspaper, and I always found the newspaper in every room number 3 I went to.*"

Exp. "*Do you mean you have been in different rooms number 3?*"

D.A. "*Yes!*"

Exp. "*Where they visually different?*"

D.A. "*No, they looked exactly the same*"

Exp. "*Then, why do you think they were different rooms?*"

D.A. "*Because the path to get there was different. Maybe one was at the mezzanine, one was on the first floor, one was on the second floor.*"

Exp. "*Now you are living with your daughter, in a new apartment, is that so?*

D.A. "*I don't know if she took this apartment for me, because I'm there too, or if it is her old apartment, perhaps it is the one we renovated two years ago…*"

Exp. "*Is it the apartment of your daughter, the one you renovated or…*"

D.A. "*Yes, yes*"

Exp. "*..or is it another house?*"

D.A. "*No, it is another house!*"

Exp. "*So, are there two identical houses?*"

D.A. "*No, they are not identical.*"

Exp. "*Is it the same apartment?*"

D.A. "*Yes, yes.*"

Exp. "*Why were you unsure before?*"

D.A. "*I don't know how she did it… It looks bigger that the one I remembered. I don't know how she did it, this is what I wanted to say.*"

When questioned few months later (November 2012) he would provide very contradictory answers:

D.A. "*There was a bit of confusion. I, as I can see it now, I'm sure the rooms were not just one. Because I thought about it over and over, I revised all my theories, the feelings I had, and I can say that there was not only one room number 3, there were at least two or three. In different buildings, on different floors. Something happened that I cannot figure out, but I was likely to be wrong, indeed, it's sure… yet even today I still have some doubts.*"

More than one year after the first assessment (August 2013), the RPP was still present:

D.A. "*That story of the 3 is long gone. I had to accept that.*"

Exp. "*Do you think about it sometimes?*"

D.A. "*Sometimes, and I laugh to myself. Because I did check, I checked how that could have happened, and someone always brought me the newspaper. That's why I lost it, because they always moved my stuff.*"

Even two years later (January 2014), the mention of room number 3 sufficed to trigger the same memories and the doubts on how to interpret them:

Exp. "*And in that Center, you were in the room number 3…*"

D.A. "*Ah! Terrible! I see it as if it was real, eh. Because, it seemed like they took my things and moved them around. I would enter in any room 3 and I would find my things. That's why I thought it was always the same. They had a system I could not understand…*"

Exp. "*So they moved you to another room?*"

D.A. "*No no, it was me, I was off. I don't think they were allowed to do such a thing. Surely they didn't help me out.*"

D.A. "*I went as far as to think that there was a room number 3 in all pavilions.*"

Exp. "*How could your belongings be in another pavilion?*"

D.A. "*Eh, I don't know how my things could follow me! This is what I couldn't figure out!*"

12. Conclusions

The present review focused on 51 published cases of pure *reduplicative paramnesia for places* (RPP) – the false belief that a place has been duplicated, or exists in two different places at the same time, or has been moved to a different location. Commonalities and differences related to the demographic, neurological and neuropsychological profiles of RPP patients were considered, and a new case report presented.

12.1. A neurological delusion?

Both epidemiological data and the literature search confirm that RPP occurs far more frequently in neurological than in psychiatric patients. Out of 53 cases retrieved from our search, only 2 (3.8%) received a diagnosis of schizophrenia. The first patient [6] manifested an extremely complex clinical picture, including Fregoli syndrome (i.e., he believed that different people were a single individual who changed appearance), intermetamorphosis syndrome (i.e., he believed that he could see others change into someone else) and RPP. Based on DSM-III criteria, he was diagnosed with paranoid schizophrenia. No neuropsychological data are provided, but CT-scan showed bilateral temporal lobe atrophy. In a 17-year old girl with similarly complex manifestations, Arisoy et al. [5] described a Capgras syndrome (e.g., her family had been replaced by impostors), persecutory delusions (e.g., her father was trying to harm her), intermetamorphosis (e.g., people

Table 3
Key aspects of D.A. neuropsychological profile.

	Cut-off	First Visit		First Follow-up		Second Follow-up		Third Follow-up	
		Jun-12		Nov-12		Aug-13		Jan-14	
		raw		raw		raw		raw	
Attention and executive functions									
Trail Making Test [64]									
- Part A	(n.d. < 93")	54		61		44		37	
- Part B	(n.d. < 282")	int.	*	190		int.	*	172	
- Part (B-A)	(n.d. < 186)	n.c.		129		n.c.		135	
Five Point Test [65]									
- cumulative number of unique designs	23.83	14	*	9	*	17	*	19	^
- cumulative strategies	< 1.89	0	*	0	*	0	*	2	
- pervasive errors	16%	64.30%	*	22%	*	35.29%	*	0%	
Modified Wisconsin Card Sorting Test [66]									
- perseveration	6.41	15	*						
- number of categories (0-6)	2	2	*						
Visuo-spatial functions									
Rey-Osterrieth's figure copy (0-36) [67]	28.87	23	*	27		27		31	
Star cancellation test (0-56) (BIT, [68])	51	50	*	50	*	52		n.c.	
Memory									
Recognition Memory Test (RMT, [69])									
- faces (0-30)	≤ 21.58	15	*	15	*	11	*	13	*
- buildings (0-30)	≤ 21.41	13	*	13	*	14	*	12	*
- words (0-30)	≤ 22.47	8	*	23		22		25	
Implicit visual-spatial learning (supra-block-span) [70]	5.75	7.81		3.92		7.54	^	14.71	
Rey-Osterrieth's figure delayed recall [67]	9.46	5.5	*	6.5		9		11.5	

v.n. normative data; * impaired range score; ^ borderline range score; int. testing interrupted; n.c. not computable.

around her were switching places among themselves and with objects), mirrored-self misidentification (e.g., her reflection in the mirror was that of another girl) and RPP (e.g., the hospital was located in another city). From the neuropsychological perspective, this latter patient had problems with figure copy, calculation and immediate recall. Structural MRI and EEG were normal and she was diagnosed with schizoaffective disorder of manic type according to DSM-IV-TR criteria. Antipsychotic treatment (risperidone and valproate) improved visuospatial functions and psychotic symptoms, including the delusional misidentification syndromes.

Two interesting findings emerge from these two reports. The first is that in psychiatric cases RPP was associated with other monothematic delusions, while in neurological patients it is usually the only delusional symptom. In addition, the first case suggests that a neurological substrate can be identified even in some so-called psychiatric cases and the second that visuo-spatial functions might be a common neuropsychological feature of neurologically- and psychiatrically-based RPP. Further studies contrasting neurological and psychiatric patients at different levels (e.g., neurological, neuropsychological, psychopathological) are needed before stronger conclusions can be reached. Crucially, in the future, even cases diagnosed with a psychiatric disorder should undergo neuroimaging and neuropsychological exams, aimed at detecting subtle structural anomalies in areas known to be affected in neurological RPP cases.

12.2. Classification

As regards the content of RPP, delusions can be classified on the basis of two cardinal concepts: copy and displacement. Patients may show: (1) reduplication with displacement - i.e. the reduplicated place X, originally located in A, was copied and moved to B [42]; (2) reduplication without displacement - i.e. the target place X was replaced in the same location by a copy X_1 more or less faithful to the original, [18]; (3) displacement without reduplication - i.e. the place X (or the patient himself), originally located in A, was moved to B (no copying mentioned), [26]. Politis and Loane [4] proposed a similar classification, based on the distinction among reduplication of place (i.e. the belief that two identical places exist in two different locations), extravagant spatial localization (i.e. the belief that one place was moved to a completely different location), and chimeric assimilation (i.e. the belief that two different places were merged into one, combining the features of both). However, we believe that the notions of copy and displacement capture the symptoms observed in all RPP patients more precisely, and that a finer-grained connotation can be achieved by reporting also the affective tone (positive or negative) and the presence of chimeric details (if applicable). Based on these considerations, *reduplicative paramnesia* might not be the most appropriate label for the phenomena discussed here. It emphasizes duplication, which is observed inconsistently, and memory problems, which are indeed present, but are not the only (and not even the main) neuropsychological feature of the disorder.

Regardless of terminological issues, we believe that focusing on the two key features of reduplication and displacement can facilitate the development of working hypotheses. For instance, differences between patients showing only reduplication and those showing only displacement could shed light on the underlying functional mechanisms (see below), as the former might be associated with worse memory deficits, and the latter with impaired visuo-spatial processing.

12.3. Clinical recommendations

Based on the heterogeneity of neurological and neuropsychological profiles observed in RPP patients, some suggestions can be given to clinicians and researchers dealing with this rare symptom. Whenever possible, routine structural examinations (CT and MRI) should be integrated with tractography and neurofunctional and neurometabolic studies (fMRI, PET, MEG or EEG). The relative variability in lesion location suggests that the key mechanism might lie in the disruption of a functional network encompassing right parietal and frontal areas. The exact site of damage would then determine the array of concomitant symptoms. Virtual dissection of white matter tracts would inform on the status of structural connections, while functional and metabolic data would help identify affected cortical networks, beyond strict anatomical changes.

To draw the appropriate conclusions on the functional neuroanatomy of the disorder, a careful neuropsychological assessment is mandatory. It should focus on the functions that play a pivotal role in the syndrome: memory, visuo-spatial and executive functions, and topographical orientation. The following cognitive areas should be tapped systematically:

- Short-term memory (verbal and non-verbal);
- Long-term memory (anterograde and retrograde; verbal and non-verbal);
- Working memory (verbal and visuo-spatial);
- Retrograde and anterograde topographic memory;
- Attention, in particular visuo-spatial attention (hemispatial neglect);
- Topographical orientation, including the ability to use ad generate maps and mental paths;
- Autobiographic memory, for a complete assessment of awareness of the three dimensions: time, place and person (i.e. personal information);
- Categorization and abstraction;
- Problem solving and planning;
- Inhibition;

When selecting materials for testing, special attention should be devoted to the evaluation of visual-spatial skills. Subjects with RPP frequently suffer from damage to anterior portions of the right hemisphere, and typically score poorly on visual and visuo-spatial tasks. Therefore, neuropsychological testing should focus on visual and perceptual skills, ranging from vision (e.g., visual field defects) to high-level visual cognition, in order to establish the extent in which co-occurring disorders like spatial neglect, visual agnosia, loss of navigation skills, etc. may constrain the clinical manifestations of the disorder. A psychiatric consultation should be sought in order to exclude a diagnosis of schizophrenia, and appropriate testing for confabulations should be performed. Whenever standardized tests are unavailable, further investigations tailored to the in-depth investigation of temporal and spatial orientation should be carried out by means of experimental tasks or semi-structured interviews/questionnaires. For instance, ad hoc tests allowed us to appreciate the extent of D.A.'s temporal disorientation and to ascribe his topographical disorientation to a deficit in implementing navigation (as opposed to a deficit in landmark identification).

Finally, a detailed psychological history and/or a neuropsychiatric assessment should be obtained. Premorbid personality traits should be investigated with the help of family members. Follow-up assessments should focus on the ways in which patient and family adjust to the neurological accident and its ensuing symptoms. The case of D.A. highlights how premorbid personality and experiences can play an important role. He used to be a mountain guide, and was greatly frustrated when he lost his orienteering skills. His generalized mistrust of others can explain the paranoid tendencies, such as the belief that the staff of the Center was actively trying to fool him: "*The people were different, the things were the same. I noticed the differences because of the people, otherwise they would have fooled me easily*" (August 2012), and "*Sometimes I would find myself in another pavilion, I would enter in the room number 3 and I would find all my things, someone had brought them along*" (August 2013).

In light of the fact that in most cases delusions seems to cease as cognitive deficits improve, treatment options will depend on concomitant symptoms. No specific neuropsychological rehabilitation protocol for RPP has been proposed yet. Focusing on topographical orientation, non-verbal memory and executive functions should facilitate the resolution of delusional symptoms. Ideally, the patient should be followed longitudinally and the persistence of the delusion should be investigated systematically, not only by eliciting spontaneous reports, but also by direct questioning. As seen in the evolution of D.A.'s answers, the patient might attempt to rationalize at first (e.g., "*It was me, I was off*"), only to then let the everlasting doubts emerge (e.g. "*I went as far as to think that there was a room number 3 in all pavilions […] I don't know how my things could follow me! This is what I couldn't figure out.*").

Ultimately, the neuropsychological evaluation in these patients should aim at testing specific working hypotheses. For the time being, and based on available evidence, only some speculations are allowed. For instance, one could expect partially distinct neural underpinnings in cases yielding only reduplicative symptoms vs. those resulting only in displacement symptoms. Worse performance on memory tasks could correlate with more extensive damage to medio-temporal structures in the first case, while worse performance on visuo-spatial tasks with more severe lesions of the parietal lobe in the second.

12.4. Core and accessory features

The paucity of reported cases suggests that RPP might arise only in the presence of a combination of factors (i.e. neurological damage, personality traits, etc.), that add up differently in each patient, yielding heterogeneous neuropsychological profiles. In addition, the few published cases have been studied with very different imaging techniques and neuropsychological tests. This diversity adds to the difficulty of comparing and generalizing across cases.

The constellations of disorders documented in subjects with RPP vary across subjects. None of the cognitive signs and symptoms reported in the literature, not even those evaluated and/or reported on more frequently, were observed in all cases. Therefore, proposals on the relevance of each cognitive disorder in determining the observed phenotypes must be very cautious. Tentatively, a distinction can be made between core features (i.e. traits that are described very frequently and are more likely to play a causative role in RPP) and accessory features (i.e. traits that contribute to the different nuances of the psychological and cognitive profile, but are probably not directly responsible for the disorder). Core features resulting in a monothematic delusion involving a duplicated and/or displaced place – the defining feature of RPP – appear to be: damage to right frontal (and temporal) lobe; dysexecutive symptoms; visuo-spatial deficits; non-verbal memory impairment. Accessory traits associated to the above features could be confabulations; anosognosia; time disorientation; paranoid personality traits.

Our patient is a prototypical case. He shows all the core features (i.e., deficits of non-verbal long-term memory, topographical orientation, attentional shifting, and cognitive flexibility), as well as two accessory features (temporal disorientation and paranoid personality traits). Moreover, his profile corroborates the evidence that these deficits can dissociate from a wide range of verbal and non-verbal skills. In D.A. language, verbal reasoning, non-verbal logical deductions, visual selective attention, problem solving in non-ecological setting, short-term memory (both verbal and non-verbal) and verbal long-term memory were spared.

12.5. Theoretical implications

Given the heterogeneity of the cognitive impairments associated with reported cases of RPP, the accounts offered for the disorder are diverse. Early theories assigned a key role to emotional and psychological factors. Thus, RPP was seen as a rationalization generated by a disoriented subject, and therefore treated as a hysterical reaction [11]. Other authors assigned a crucial role to psychodynamic factors, seeing RPP as a psychological ploy to relieve patient's issues, with personality traits becoming more rigid and stereotyped as a consequence of brain damage [10]. Afterwards, RPP was linked to cognitive deficits and neuropsychological mechanisms and interpreted as a disconnection of memories [12], a spatial disorientation [24], or a triad of cognitive deficits affecting memory, visuo-spatial skills and executive functions [14]. Similarities in the neurological and neuropsychological profiles observed in RPP, and in other monothematic delusions, anosognosia and confabulations led to propose unitary accounts for all false beliefs,

stemming from either psychological/psychodynamic [71] or neurocognitive considerations [72]. Similarly, other authors addressed DSMRs as a whole, and attempted to account for the entire range of clinical manifestations based on a failed sense of familiarity [13], identity [73] or uniqueness [74].

A more comprehensive framework is provided by the two-factor theory of monothematic delusion [15]. On this account, two elements are necessary and sufficient to generate a DSMR: (1) a perceptual or affective deficit (required to generate the content of the delusion) which differs widely from patient to patient, accounting for the observed variability; (2) an impairment of the belief evaluation system (crucial to allow the delusion to develop and persist) resulting from damage to the right hemisphere, and especially to the lateral prefrontal cortex. This theory implies that a right frontal lesion should be observed in virtually all cases, a hypothesis partially supported by the cases here reviewed.

It has also been suggested that RPP might be due to left-hemisphere hyperactivity, subsequent to right hemisphere damage. On this account [75,76], delusions would result from the hyper-inferentiality of the left hemisphere, associated with the inability to detect illogical inferences due to right hemisphere damage. Even acknowledging this possibility, the biological and psychological mechanisms by which only a very small minority of patients with right (frontal) lesions develop a monothematic delusion for places are yet to be understood, and need careful neuroimaging and neuropsychological investigation [77] have recently stressed that RPP, contrary to other space-related confabulations and delusions, does not relate to a particular portion of extrapersonal space (e.g., the contralesional space in unilateral spatial neglect), while still being highly correlated with right hemisphere damage. Further investigations of the differences and commonalities between RPP and other right-hemisphere space-related confabulations and delusions will be instrumental in improving our understanding of their neural substrate. For instance, attentional networks appear to be right lateralized: in a framework assigning to right hemisphere structures the role of continuously integrating sensory evidence, RPP might be seen as a faulty update of information relative to one's current location [78].

In conclusion, a theory capable of explaining all the facets of RPP should elucidate the specific contribution of neurological, psychological (emotional, motivational) and environmental factors. As regards the cognitive mechanisms underlying the disorder, a careful investigation of mnestic, visuo-spatial and executive functions, as well as of their interactions, will be critical for the development of such comprehensive theory. This would enable to predict specific associations and dissociations of symptoms in RPP patients, and to distinguish them from those observed in other DSMRs, thereby laying the foundations for reliable and detailed hypotheses on the neural underpinnings of these disorders.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.clineuro.2019.03.022.

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