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Differential contribution of right and left temporo-occipital and anterior temporal lesions to face recognition disorders

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In the study of prosopagnosia, several issues (such as the specific or non-specific manifestations of prosopagnosia, the unitary or non-unitary nature of this syndrome and the mechanisms underlying face recognition disorders) are still controversial. Two main sources of variance partially accounting for these controversies could be the qualitative differences between the face recognition disorders observed in patients with prevalent lesions of the right or left hemisphere and in those with lesions encroaching upon the temporo-occipital (TO) or the (right) anterior temporal cortex. Results of our review seem to confirm these suggestions. Indeed, they show that (a) the most specific forms of prosopagnosia are due to lesions of a right posterior network including the occipital face area and the fusiform face area, whereas (b) the face identification defects observed in patients with left TO lesions seem due to a semantic defect impeding access to person-specific semantic information from the visual modality. Furthermore, face recognition defects resulting from right anterior temporal lesions can usually be considered as part of a multimodal people recognition disorder. The implications of our review are, therefore, the following: (1) to consider the components of visual agnosia often observed in prosopagnosic patients with bilateral TO lesions as part of a semantic defect, resulting from left-sided lesions (and not from prosopagnosia proper); (2) to systematically investigate voice recognition disorders in patients with right anterior temporal lesions to determine whether the face recognition defect should be considered a form of "associative prosopagnosia" or a form of the "multimodal people recognition disorder."

Keywords: prosopagnosia, multimodal people recognition disorders, unilateral lesions, visual object agnosia, familiarity feelings, configurational processing

INTRODUCTION

Selective analysis of faces is certainly the most powerful channel we have for recognizing familiar people and deriving important information about known or unknown persons, such as their emotional state, age, race, and gender. This is why the term "prosopagnosia," which was considered a form of visual agnosia specifically affecting face recognition, has played such a dominant role in the study of defective recognition and identification of familiar people since Bodamer's (1947) description of the defect. Nevertheless, although many studies that have tried to clarify the nature and mechanisms of acquired prosopagnosia, as well as its neuroanatomical underpinnings, many issues are still unresolved, including (a) the specific or non-specific manifestations of prosopagnosia; (b) the unitary or non-unitary nature of the disorder; and (c) the mechanisms underlying famous faces recognition disorders.

(a) Regarding the first point, some authors (e.g., Lhermitte et al., 1972; Damasio et al., 1982, 1990; Dixon et al., 1998; Gauthier et al., 1999a; Delvenne et al., 2004) have claimed that acquired prosopagnosia corresponds to a defect not only in recognizing familiar faces, but also in recognizing/discriminating among members of visually homogeneous categories. However, other authors (e.g., De Renzi, 1986a; Sergent and Signoret, 1992; McNeil and Warrington, 1993; Farah et al., 1995a; Rossion et al., 2003; Schiltz and Rossion, 2006; Busigny et al., 2010a) hold that prosopagnosia cannot be related to general difficulty in discriminating visually similar exemplars of face and non-face categories.

(b) Regarding the second point, the debate revolves around De Renzi et al.'s (1991) proposal of distinguishing an "apperceptive" from an "associative" form of prosopagnosia, extending to selective face recognition disorders the classical Lissauer's (1890) distinction between an "apperceptive" and an "associative" form of visual agnosia. According to this distinction, in apperceptive agnosias recognition fails because of a subtle defect in visual perception, whereas in associative agnosias an intact visual percept cannot be associated with data stored in memory or deriving from other perceptual modalities. De Renzi et al. (1991) applied this basic distinction to face recognition, translated it into operational terms and proposed classifying as "apperceptive" the face recognition disorders of patients who, in addition to being unable to recognize familiar faces, also had problems in treating unknown faces, and as "associative" those in which no problem was found in the treatment of unfamiliar faces. Results obtained on Benton and Van Allen's (1968) unfamiliar matching tests (Benton Face Recognition Test, BFRT) are usually considered to provide the most reliable discrimination between apperceptive and associative forms of prosopagnosia; however, some authors (e.g., Benton, 1990; Davidoff and Landis, 1990; Farah, 1990;

Sergent and Signoret, 1992; Duchaine and Weidenfeld, 2003; Duchaine and Nakayama, 2006) have argued that this test cannot be considered a good marker of the perceptual abilities involved in unfamiliar face recognition. Therefore, the distinction between "apperceptive" and "associative" forms of prosopagnosia and the most appropriate way to make this distinction are still quite controversial.

(c) Regarding the problem of the mechanisms underlying face recognition disorders, most authors agree that prosopagnosia is due to a defect in the holistic/configurational processing of faces (Yin, 1969; Sergent and Signoret, 1992; Kanwisher, 2000; Busigny et al., 2010b). But it is not clear whether this holistic process specifically concerns human faces, because of evolutionary determined innate factors, or whether it results from the acquisition of a high degree of expertise in the treatment of different classes of complex visual stimuli (Gauthier and Tarr, 1997; Gauthier et al., 1999b; Kanwisher, 2000; Gauthier and Nelson, 2001). If this were true, the holistic processing of faces would be at least partially due to the fact that faces are the visual stimuli most intensively and frequently processed by humans.

The difficulty of answering all of these questions is increased because prosopagnosia can be provoked by lesions in various parts of a bilateral network of cortical areas spanning from the inferior occipital areas [occipital face area (OFA) of Gauthier et al., 2000] to the anterior temporal cortex, with its center in the lateral portion of the mid-fusiform gyrus where the fusiform face area (FFA; Kanwisher et al., 1997) is located. Even though it is generally acknowledged that the inferior occipital areas mainly subsume the first stages of face perception and that the anterior temporal structures integrate information concerning the face, voice, and name of a familiar person, the exact functions of these different structures are only partially understood. Equally controversial is the role of disconnection mechanisms (Fox et al., 2008) or top-down processes (which allow obtaining performances that could not be obtained on the basis of simple bottom-up mechanisms) in this network (Etcoff et al., 1991; De Renzi and di Pellegrino, 1998; Barton and Cherkasova, 2003).

We believe that two main sources of variance at least partially account for these controversies. The first concerns the hemispheric side of the lesion in patients with posterior temporo-occipital (TO) injuries. In patients with left TO lesions, face recognition disorders can be part of a general visual recognition defect (or "visual object agnosia") in which the ability to access conceptual and person-specific semantic information from the visual modality is impaired; instead, in patients with right TO lesions the visual recognition disorder selectively concerns faces and seems due to a defect of a specific (configurational) form of visual processing.

Because both forms of visual agnosia can coexist in patients with bilateral lesions, there is the risk of attributing visual recognition disorders due to the concomitant left TO lesion to the mechanism responsible for prosopagnosia.

The second source of variance concerns the intrahemispheric locus of lesion and the nature of the people recognition defect in patients with right hemisphere damage. In fact, if disorders are circumscribed to the visual modality in patients with lesions encroaching upon the TO areas of this hemisphere, in those with lesions involving the right anterior temporal structures, people recognition disorders are multimodal, because they consistently affect familiar faces, voices, and names (see Gainotti, 2007a for a recent review). But, as these patients are often only aware of their difficulty in recognizing people by their faces and not of a similar defect for voices (Gainotti, 2010) and as the attention of students of prosopagnosia is often focused on subtle visual defects (sometimes neglecting the presence of similar defects in other modalities), the risk of incorrectly diagnosing prosopagnosic patients who have a multimodal person recognition disorder must be acknowledged.

THE MAIN DIFFERENCES BETWEEN FACE RECOGNITION DISORDERS RESULTING FROM LEFT AND RIGHT TO LESIONS

There are several important differences between "prosopagnosia" proper, resulting from right TO lesions, and the face recognition disorders that are sometimes observed in a context of "visual object agnosia" in patients with left TO damage. On one hand, these differences concern the impact of associative or semantic disturbances on the pathophysiology of visual recognition disorders resulting from left and right TO lesions and, on the other hand, the mode of stimulus processing disrupted in patients with left and right TO lesions. Regarding the first point, several authors (e.g., McCarthy and Warrington, 1986; De Renzi et al., 1987b; Damasio et al., 1988; De Renzi, 2000) have exhaustively demonstrated that visual recognition disorders resulting from left TO lesions are mainly due to associative or semantic disturbances and usually spare (but can sometimes include) face recognition. Regarding the second point, it is usually claimed that in left brain-damaged patients the defect mainly concerns analytical feature-based processing (Peretz, 1990; Bever and Chiarello, 2009), whereas in right brain-damaged patients the lesion disrupts a more holistic, configurational process operating on the spatial relationships among the face components (Sergent and Signoret, 1992; Kanwisher, 2000; Busigny et al., 2010b).

A final difference between the recognition disorders observed in patients with left and right TO lesions is that, due to the social relevance of distinguishing well known from unfamiliar people, an early step in the recognition of a known person concerns the emergence of familiarity feelings. These feelings are usually absent or very mild in the process of object recognition (with the possible exception of personal owning), but contribute in the process of person recognition by focusing attention on known faces and gathering additional, confirmatory information about them (Atkinson and Juola, 1974). Therefore, in the present review we will take into account each of these points and will try to disentangle their individual contributions to controversies concerning prosopagnosia.

VISUAL RECOGNITION DISORDERS DUE TO GENERAL SEMANTIC DEFECTS OR SELECTIVELY AFFECTING FACES IN PATIENTS WITH LEFT AND RIGHT TO LESIONS

Several pioneering authors (e.g., Albert et al., 1975; Pillon et al., 1981; Ferro and Santos, 1984; McCarthy and Warrington, 1986; Feinberg et al., 1994) showed that the recognition of familiar faces is often spared in patients with visual object agnosia provoked by unilateral left TO lesions. Results of investigations that have confirmed this clinical impression are reported in **Table 1**, in which each patient's reference authors, lesion anatomy, and clinical symptomatology are summarized.

Table 1 | Patients with unilateral left temporo-occipital lesions who show visual object agnosia with spared recognition of faces.

Patient	Anatomical lesion	Symptomatology
Vid. (Pillon et al., 1981)	Hemorrhagic infarct in the territory of the left PCA	Right homonimous hemianopia, pure alexia, normal matching of objects and pictures but inability to categorize and show the use of visually presented objects. *Visual associative agnosia* without prosopagnosia
CARJ (Ferro and Santos, 1984)	Infarct in the territory of the left PCA, involving the lower temporo-occipital areas	Right homonimous hemianopia, severe alexia, normal drawing by copy. Inability to categorize pictures of objects and to mime their use. *Visual associative agnosia*, but no prosopagnosia
Patient of Feinberg et al. (1986)	Infarct of the left PCA, involving the left occipital lobe, the inferior posterior mesial temporal lobe, and the splenium	Right homonimous hemianopia, alexia, and combined visual and tactile agnosia. Could not name, describe, or show the use of objects presented visually or tactually, but could perfectly copy figures. *Visual associative agnosia* without prosopagnosia
FRA (McCarthy and Warrington, 1986)	Infarct in the territory of the left PCA, involving the lower part of the occipital lobe	Right homonimous hemianopia, alexia, spared high level visual abilities but impaired categorization and visual matching of objects and pictures taken from a different view. *Visual associative agnosia* without prosopagnosia
Patient of Gallois et al. (1988)	Infarct of the left PCA, involving the mesial part of the occipital and temporal lobes, and the splenium	Right homonimous hemianopia, alexia, and sparing of high level visual abilities but impaired categorization and visual matching of pictures of objects taken from different views. *Visual associative agnosia* without prosopagnosia
Patient 1 (Feinberg et al., 1994)	Infarct of the left PCA, involving the mesial part of the occipital and temporal lobes	Right homonimous hemianopia, alexia, and impairment of visual object recognition (naming and demonstrating their use) in spite of spared high level visual abilities. *Visual associative agnosia* without prosopagnosia
Patient 2 (Feinberg et al., 1994)	Infarct of the left PCA, involving the mesial part of the occipital and temporal lobes, and the splenium	Right homonimous hemianopia, alexia, and impairment of visual object recognition with inability to name and pantomime. Sparing of high level visual abilities. *Visual associative agnosia* without prosopagnosia
Patient 3 (Feinberg et al., 1994)	Infarct of the left PCA, involving the mesial part of the occipital and temporal lobes	Right homonimous hemianopia, alexia, and impairment of visual object recognition with inability to name and pantomime objects. Sparing of high level visual abilities. *Visual associative agnosia* without prosopagnosia

The data reported in **Table 1** are clear, homogeneous, and consistent from both clinical and neuropathological viewpoints. All patients showed complete right homonymous hemianopia and were unable to name visually presented objects, which contrasted with their spared ability to name the same objects upon verbal definition or (less systematically) after tactile exploration. Furthermore, they were unable to pantomime the use of seen objects, categorize pictures, or match different pictures of the same object, but were able to copy figures and match identical versions of the same picture. Although they easily recognized familiar people, they were not always able to give their names because of their inability to name visual stimuli.

From a neuropathological point of view, all of these patients had suffered an infarct in the territory of the left posterior cerebral artery (PCA), which involved the inferior and mesial parts of the occipital and temporal lobes and, in some cases, the splenium of the corpus callosum.

These findings were also confirmed by results of systematic investigations (e.g., De Renzi et al., 1987b), which showed that patients with a left PCA infarct often have associative visual object agnosia with alexia but without prosopagnosia. There are, however, some exceptions to this rule. In fact, an association between prosopagnosia (with or without visual object agnosia) and left TO lesions has been observed in a limited number of patients. The data of these patients are reported in **Table 2**; they include reference authors, lesion anatomy, clinical symptomatology, presence of familiarity feelings and, when reported, type (configurational/holistic or based on a feature-by-feature analysis) of face processing.

Three main observations can be made from the data presented in **Table 2**: (1) First, a high proportion of the few left brain-damaged patients with a face recognition (or identification) defect reported in the literature were left-handed. This was the case for patients AC and DN, reported respectively by Tzavaras et al. (1973) and Mattson et al. (2000) and for patient 015 reported by Barton (2008). (2) Second, with the exception of patient DN (Mattson et al., 2000), who had very low visual acuity, familiarity feelings were relatively or completely spared in these patients. This is surprising, because [as rightly stressed by Gross and Sergent (1992), and acknowledged by almost all authors] inability to experience a feeling of familiarity when viewing known faces is a hallmark of prosopagnosia. (3) Third, the observation that in patients with left TO lesions face recognition disorders often coexist with severe signs of visual object agnosia suggests (in agreement with Damasio et al.'s (1988) construct of "deep prosopagnosia") that these recognition disorders are part of a general inability to access conceptual and person-specific semantic information from the visual modality. Very few authors have analyzed the type of face processing used by their patients and in these cases the configurational processing defect seems less frequent than the feature analysis defect.

On the other hand, since the advent of neuroimaging, several studies have shown that when prosopagnosia is due to unilateral lesions these lesions usually encroach upon the right TO areas. Therefore, in **Table 3** we tried to summarize the characteristics of the prosopagnosic patients we found in the literature with a lesion restricted to the right hemisphere; for each patient, we summarized

Table 2 | Patients we found in the neuropsychological literature with face recognition disorders resulting from left temporo-occipital lesions.

Patient	Anatomical lesion	Symptomatology	Face processing	Familiarity feelings
AC (Tzavaras et al., 1973)	*Left-handed*. Resection of the left temporal lobe	Fluent aphasia, severe memory disorders. Associative prosopagnosia	Not studied	Spared
DN (Mattson et al., 2000)	*Left-handed*. Traumatic BI with left occipital lesion	Visual acuity 20/70. Integrative prosopagnosia. Moderate visual disorders. Correct but slow unfamiliar faces matching	Feature-by-feature analysis?	Impaired
Subject 015 (Barton, 2008)	*Left-handed*. Neonatal anoxia with left temporo-occipital infarct	Impairment in recognizing line drawings of objects. Prosopagnosia with severe defect on the Benton FRT. Poor familiarity and recall of person-specific semantic information from names	Global processing less impaired than feature processing	Mildly impaired (as with names)
Two patients (Damasio et al., 1988)	Left occipital lesions	"Deep prosopagnosia," "Semantic errors" in face recognition (subjects with semantic features in common with the targets)	Not studied	Spared
RC (Carlesimo et al., 1998)	Left occipital (+lower half splenium)	Associative visual agnosia. Associative prosopagnosia with impaired identification (3/20 = 15%) from faces	Not studied	Relatively spared (15/20 = 75%)
EY (Verstichel and Chia, 1999)	Left occipital infarct	Right superior hemianopia. Alexia without visual agnosia. Impaired people identification from face (11/38 = 30%)	Not studied	Spared (20/20 = 100%)
DBO (Anaki et al., 2007)	Left occipital infarct	Associative visual agnosia. Associative prosopagnosia with inability (9/24 = 37%) to access semantic information about known faces	Configural processing unimpaired	Spared (19/24 = 80%)

BI, brain injury; FRT, Face Recognition Test.

the same data reported in **Table 2**, to characterize patients with face recognition or identification disorders resulting from left TO lesions.

The data reported in **Table 3** allow us to make several observations. First, contrary to what happens in patients with face recognition disorders resulting from left TO lesions, face familiarity feelings are more or less severely impaired in all right prosopagnosic patients. This defect usually consists of a lack of subjective feelings alerting the patient that he is looking at a friend or a family member, but sometimes also includes the tendency to feel the faces of familiar people as "stranger" (Michel et al., 1986; Wada and Yamamoto, 2001; Uttner et al., 2002) or the tendency to feel well known and unknown faces as equally familiar (e.g., Barton et al., 2001, 2002). The second observation is that, when face processing is studied with methods that allow distinguishing global configurational encoding from local feature-by-feature analysis, a defective configurational process is observed in most patients. The severity of this defective configurational process can, however, vary from patient to patient. For instance, it was very clear in patients LH (Levine and Calvanio, 1989; Etcoff et al., 1991; Farah et al., 1995b), BM (Sergent and Villemure, 1989), PM and RM (Sergent and Signoret, 1992), CR (Gauthier et al., 1999a; Marotta et al., 2001), and in some patients intensively studied by Barton et al. (2001, 2002) and Barton and Cherkasova (2003), whereas it was only partial in patient FB (Riddoch et al., 2008) and was absent in patient PC (Sergent and Signoret, 1992). It should be noted that PC is the only patient reported in **Table 3** who had been considered as having a type of "associative prosopagnosia," in contrast with the very high frequency of patients affected by left PCA (and reported in **Tables 1 and 2**), whose visual recognition defect had been attributed to an associative rather than an apperceptive mechanism. The third observation is that visual object agnosia, which was very often observed in patients with left TO lesions with or without face identification disorders, was rarely observed in right prosopagnosic patients; and in some of these patients, it seemed to specifically affect the categories of animals and other living beings [e.g., patients LH (Etcoff et al., 1991) and CR (Gauthier et al., 1999a)].

Some authors (e.g., Sergent and Signoret, 1992; Wada and Yamamoto, 2001; Uttner et al., 2002) also noticed that objects were usually well recognized from a canonical but not from a non-canonical perspective; but this result is not specific to prosopagnosia. Indeed, since the publication of Warrington and Taylor's (1973, 1978) papers, it is known that difficulty in identifying an object from an unusual perspective is generically linked to damage in the posterior parts of the right hemisphere and is not specific to prosopagnosia.

DISRUPTION OF ANALYTICAL FEATURE-BASED AND HOLISTIC-CONFIGURATIONAL PROCESSES IN PROSOPAGNOSIA

One of the most striking differences that emerged from this review (and from many previous studies) between the face identification disorders of patients with left TO lesions and of right prosopagnosic patients was the disruption of face processing based on a local, feature-by-feature analysis in patients with left TO lesions and of global, configurational encoding in those with right-sided lesions. This is consistent with the documented importance (e.g., Bradshaw and Nettleton, 1981; Grill-Spector et al., 1998) of the left occipital

Table 3 | Patients we found in the neuropsychological literature with face recognition disorders resulting from right temporo-occipital lesions.

Patient	Anatomical lesion	Symptomatology	Face processing	Familiarity feelings
Michel et al. (1986)	Right occipital hematoma	Left hemianopia. Moderate prosopagnosia. No visual agnosia	Not studied	Clinically impaired (stranger)
Patient 1 (Charnallet et al., 1986)	Large right occipital hemorrhagic infarct	Left hemianopia. Moderate prosopagnosia without visual agnosia	Not studied	Moderately impaired
Patient 1 (De Renzi, 1986b)	Right PCA infarct	Left hemianopia. Severe prosopagnosia. No visual agnosia	Not studied	Impaired
Patient 2 (De Renzi, 1986b)	Right PCA infarct	Left hemianopia. Severe prosopagnosia with visual agnosia	Not studied	Impaired
BM (Sergent and Villemure, 1989)	Right hemispherectomy	Severe prosopagnosia. No visual agnosia	Defective configural processing	Impaired
LH (Levine and Calvanio, 1989; Etcoff et al., 1991; Farah et al., 1995b)	Right temporal lobectomy with subcortical lesions in the parieto-occipital white matter	Severe prosopagnosia. Relatively spared visual perception, but impaired recognition of living beings. Person recognition spared through other modalities	Defective configural processing	Very impaired
PC (Sergent and Signoret, 1992)	Right occipito-temporal hemorrhage	Associative prosopagnosia with relatively spared visual perception	Unimpaired configural processing	Impaired
PM (Lhermitte and Pillon, 1975; Sergent and Signoret, 1992)	Right occipital resection for AVM	Severe prosopagnosia. No signs of visual agnosia	Defective configural processing	Impaired
RM (Sergent and Signoret, 1992)	Ruptured temporo-occipital aneurysm	Severe prosopagnosia with very impaired visual perception	Defective configural processing	Impaired
PA (De Renzi et al., 1994)	Right PCA infarct	Left upper quadrantanopsia. Severe apperceptive prosopagnosia (7/32 = 22%)	Not studied	Impaired (13/36 = 36%)
OR (De Renzi et al., 1994)	Right temporo-parieto-occipital infarct	Left visual field defects. Severe prosopagnosia (12/32 = 37%) without visual object agnosia	Not studied	Impaired (17/36 = 47%)
LM (De Renzi et al., 1994)	Right PCA infarct	Left upper quadrantanopsia. Moderate prosopagnosia (8/20 = 40%)	Not studied	Impaired (9/20 = 45%)
Patient 3 (Takahashi et al., 1995)	Right temporo-occipital infarct	Lack of identification and memory from faces. Good recognition from voice. No visual perceptual defects	Not studied	Impaired
CR (Gauthier et al., 1999a; Marotta et al., 2001)	Micro-abscesses of the right temporal and occipital lobes	Prosopagnosia with impaired recognition of living beings. Prevalent left fusiform activation while viewing faces	Local over global processing advantage	Not tested
Patient 5 (Barton et al., 2001), patient 4 (Barton et al., 2002), patient 5 (Barton and Cherkasova, 2003)	Right medial occipital stroke	Left hemianopia. Moderate perceptual face defects. Moderate prosopagnosia. Impaired imagery for facial shape	Moderate configural processing defect	Impaired (many false alarms)
Patient 6 (Barton et al., 2001), Patient 5 (Barton et al., 2002), Patient 6 (Barton and Cherkasova, 2003)	Right occipital lobectomy for tumor resection	Left hemianopia. Severe perceptual face defects. Moderate prosopagnosia. Impaired imagery for facial shape	Severe defect of configural processing	Impaired (many false alarms)
DE (Verstichel, 2001)	Right occipital hematoma	Visual agnosia + severe prosopagnosia (8/28 = 29%)	Not studied	Impaired (5/11 = 45%)
Wada and Yamamoto (2001)	Right occipital hematoma	Left hemianopia. Severe prosopagnosia. No visual agnosia.	Not studied	Impaired (stranger)
Patient 2 (Uttner et al., 2002)	Right PCA infarct	Left hemianopia. Severe apperceptive Prosopagnosia. No visual agnosia	Not studied	Impaired (stranger)
FB (Riddoch et al., 2008)	Embolization of a right temporo-occipital AVM	Severe prosopagnosia with a perceptual defect limited to face processing and intact subordinate object recognition	Partial defect of configural processing	Very impaired

PCA, posterior cerebral artery; AVM, arterio-venous malformation.

areas for local analysis and of right TO structures for configurational processing. It should be noted, however, that in the right prosopagnosic patients included in our review the severity of this defective configurational process varied from patient to patient and this variability was only in part due to the intrahemispheric locus of lesion. The three patients reported by Sergent and Signoret (1992), for instance, showed different levels of configurational impairment, although the distribution of their lesions was very similar, that is, in all cases encroaching on the right inferior TO cortices and involving the fusiform gyrus. One of the reasons for this reported variability probably stems from the fact that the term "configurational processing" does not denote a unitary mechanism, but refers to a family of more or less related models of normal face recognition. These models agree that perceiving a whole face is more than perceiving its parts and that face inversion disrupts the coding of relational features more than isolated features; however, they disagree as to what is exactly meant by "*whole* face" and how face inversion disrupts this configurational processing. In fact, Rhodes (1988) and Rhodes et al. (1993) assumed that recognizing a face initially consists of perceiving separate parts or primary features, which are then integrated and give rise to second order features. By contrast, Farah and colleagues (Farah, 1990; Tanaka and Farah, 1993) maintained that face recognition does not start from the encoding of separate parts, but that the face is represented holistically from the onset of visual processing and its parts are only represented in the whole context. Other sources of variability can be found in task-related attentional and temporal factors (Barton et al., 2002) and in the level of expertise attained by the patient (Diamond and Carey, 1986; Gauthier and Tarr, 1997). The influence of attentional and temporal factors is due to the fact that distributed spatial attention and a short response time favor holistic treatment, whereas the instruction to focus on a specific spatial relationship, allowing more time, permits perceiving faces in a more serial manner. On the other hand, the influence of expertise could derive from the fact that the mechanism suggested to mediate the acquisition of expertise is configurational processing. All of the above help clarify why it is equally difficult to find universally accepted criteria to distinguish the "apperceptive" from the "associative" forms of prosopagnosia and a general agreement about the severity of the configurational impairment in prosopagnosia.

LOSS OF FAMILIARITY FEELINGS IN PATIENTS WITH FACE IDENTIFICATION DISORDERS RESULTING FROM UNILATERAL RIGHT AND LEFT TO LESIONS

One of the most striking results of our comparison between the characteristics of patients with face recognition or identification disorders resulting from right and left TO lesions concerns the study of face familiarity feelings. In fact, these feelings were relatively or completely spared in patients with left TO lesions and systematically impaired in right prosopagnosic patients. It is interesting that the loss of face familiarity feelings is not only considered a hallmark of prosopagnosia by authorities such as Ellis and Young (1988) or Gross and Sergent (1992), but was shown to be the main marker of prosopagnosia in a well designed study by Carlesimo and Caltagirone (1995). In that study, groups of patients with right and left, anterior and posterior brain lesions, with and without prosopagnosia were given tests that required age attribution, unfamiliar faces matching, pointing to familiar faces, and retrieving person-specific semantic information about people whose faces had been judged as familiar. Loss of familiarity feelings was found to be a much more specific marker of right posterior lesions (and of prosopagnosia) than the other tests of apperceptive prosopagnosia or of retrieval of person-specific semantic information. Furthermore, it must be stressed that, just as the expression "configurational processing," also the expression "face familiarity feelings" denotes a family of related phenomena rather than a unitary mechanism. This claim is based on the tendency shown by some patients reviewed in **Table 3** (e.g., Barton et al., 2001, 2002) to feel that well known and unknown faces are equally familiar and is confirmed by results obtained in a group study by Rapcsak et al. (1996). These authors showed: (a) that in patients with focal right hemisphere damage, false recognitions, and prosopagnosia can be associated in some patients and dissociated in others; and (b) that the frequency of false recognition errors increases when the lesion extends toward the right frontal areas.

THE DISTINCTION BETWEEN "PROSOPAGNOSIA" AND "MULTIMODAL PEOPLE RECOGNITION DISORDERS"

In the last part of the Section "Introduction," we stated that even though some patients, in particular those affected by (right) anterior temporal lesions, are unable to recognize familiar people by their faces, they cannot be considered as having "prosopagnosia" because their inability to recognize familiar people is not restricted to faces, but also extends to voices and, to a lesser extent, to names. In order to document this claim, in **Table 4** we included all patients found in the literature with face recognition disorders labeled as "prosopagnosia" in the title of their case report and affected by anterior temporal lesions. Our aim was to see whether analogous recognition disorders had been investigated in other modalities and what the outcomes were.

Before discussing the appropriateness of the term "prosopagnosia" to classify the patients reported in **Table 4**, we will analyze their main characteristics, considering separately: (1) etiology and side of lesion; (2) status of perceptual processes; (3) frequency with which people recognition had been studied through voice and name; (4) outcomes of these investigations; and (5) status of familiarity feelings.

(1) Regarding *etiology of the lesion*, almost all patients reported in **Table 4** suffered from diseases [herpes simple encephalitis (HSE), closed head injury (CHI), or the temporal variant of fronto-temporal degeneration] that preferentially damage the anterior parts of a temporal lobe and almost always involve the "unimpaired" contralateral one. Therefore, from the etiological point of view these patients differed from typical cases of prosopagnosia due to unilateral (right) or bilateral infarcts in the territory of the PCAs. On the other hand, *side of lesion* was similar in patients with prosopagnosia due to unilateral vascular lesions and in patients with multimodal people recognition disorders, because the lesion affected the right temporal lobe in 8 of the 10 patients reported in **Table 4**, was bilateral in one (patient 8 of Barton et al., 2001) and left-sided in patient LP (De Renzi, 1986a; De Renzi et al., 1991). Note that in the latter patient prosopagnosia was

Table 4 | Patients we found in the literature with face recognition disorders labeled as "prosopagnosia" and associated with anterior temporal lesions.

Patient	Anatomical lesion	Symptomatology	Person recognition through		Familiarity feelings
			voice	name	
M.me V. (Boudouresques et al., 1979), PV (Sergent and Poncet, 1988, 1990)	HSE with massive damage of the anterior parts of the right temporal lobe	Selective defect of familiar people recognition. Unaware or poor voice recognition	Impaired	Not tested	Not tested
LP: Patient 5 (De Renzi, 1986a; De Renzi et al., 1987a)	HSE with lesion of the anterior and inferior parts of the *left* temporal lobe	Severe defect of familiar people recognition and semantic memory disorders (mainly for vegetables)	Impaired	Impaired	More impaired for faces than for names
VA: Case 3 (De Renzi et al., 1991)	HSE with lesion confined to the right temporal lobe	Moderate defect of familiar people recognition when seen out of context	Not tested	Not tested	Very impaired for faces
MT (Schweinberger et al., 1995; Henke et al., 1998)	Right temporo-parietal infarct	Severe defect of familiar people recognition from faces (1/18), without signs of visual agnosia. Poor memory for faces, but not for words	Not tested	Impaired (13/18)	Impaired
VH (Evans et al., 1995)	Diffuse atrophy of the right antero-inferior temporal lobe	Progressive defect recognizing familiar people with mild memory disorders	Initially clinically unimpaired	Normal retrieval of semantic information	Moderate impairment for faces
Patient 8 (Barton et al., 2001), patient 1 (Barton et al., 2002), patient 8 (Barton and Cherkasova, 2003)	Bilateral anterior temporal lobe damage from CHI and right temporal lobe resection	Severe defect of familiar people recognition with impairment on the Benton FRT	Not tested	Not tested	Very impaired for faces
FG (Joubert et al., 2003)	Prevalent atrophy of the right fusiform gyrus; relative sparing of the anterior temporal cortex	Progressive defect recognizing familiar people with impaired configurational processing	Initially the voice of the person helped recognition	Normal retrieval of semantic information from name	Mildly impaired for faces
BD (Williams et al., 2006)	Right anterior temporal lobe atrophy	Progressive defect recognizing familiar people, with impaired configurational processing but spared semantic information	Not tested	Normal retrieval of semantic information from name	Moderately impaired for faces
LR (Bukach et al., 2006)	Traumatic lesion of the right antero-inferior temporal lobe; sparing the fusiform gyrus	Moderate defect of familiar people recognition when seen out of context	Not tested	Not tested	Impaired (many false alarms)
MT (Nakachi and Muramatsu, 2007)	Atrophy of the right anterior temporal lobe	Selective form of associative prosopagnosia	Spared	Spared	Impaired for faces

associated with severe semantic disorders, like the patients with face recognition disorders resulting from left TO lesions we reported in **Table 2**.

(2) *Perceptual processes* were intact in most patients reported in **Table 4**, but an impairment of configurational processing was described in patients FG (Joubert et al., 2003) and BD (Williams et al., 2006). In patient FG, this unexpected finding was probably due to atypical distribution of the temporal lobe atrophy, which involved the right fusiform gyrus and the parahippocampal cortex, and relatively spared the temporo-polar cortex; however, patient BD's impaired configurational processes could not be explained, because of the atypical method used to assess them and the poor description of atrophy.

(3) Regarding the frequency with which *voice recognition and retrieval of person-specific semantic information from names* had been studied, they had not been systematically investigated (or in any case had not been reported) in about half of these patients (it must be acknowledged, however, that usually this information was also lacking for the patients reported in **Tables 2 and 3**).

(4) With respect to outcomes of the investigations concerning voice and name recognition, *voice recognition* was impaired in two (M.me V and LP) and spared in three (VH, FG, and MT) of the five patients in whom it had been studied. This last result must, however, be evaluated with caution because voice recognition had been considered as intact on the basis

of a purely clinical judgment in patients VH (Evans et al., 1995) and FG (Joubert et al., 2003) and of a short clinical task in patient MT (Nakachi and Muramatsu, 2007).

Results obtained by analyzing *the retrieval of person-specific semantic information from names* are rather different, because this ability was severely impaired only in patient LP (De Renzi, 1986; De Renzi et al., 1991), who was affected by a left temporal lesion and showed severe semantic disorders, and mildly impaired in patient MT (Schweinberger et al., 1995). Retrieval of person-specific semantic information from names was, on the contrary, unimpaired in most right anterior temporal patients in whom it had been studied, namely in patients VA (Evans et al., 1995), FG (Joubert et al., 2003), BD (Williams et al., 2006), and MT (Nakachi and Muramatsu, 2007). These findings are consistent with results of a recent review (Gainotti, 2007a) of the patterns of famous people recognition in patients with right and left anterior temporal lesions.

(5) Regarding *face familiarity feelings*, they were more or less severely impaired in all patients (9 out of 10) in whom they had been investigated. This result is quite similar to that obtained in the prosopagnosic patients with right TO lesions reported in **Table 3** and confirms the importance of the loss of familiarity feelings in defects of known faces recognition disorders resulting from right hemisphere lesions.

Taken together, the data reported in **Table 4** suggest the need for caution in using the term "prosopagnosia" to denote the face recognition disorders of patients with right anterior temporal lesions. In fact, in a few patients (e.g., VH, FG, and MT) this term seems appropriate, whereas in other patients (e.g., M.me V, LP, and MT) the term "multimodal people recognition disorders" is more appropriate, and in others we lack data about voice and name recognition that would allow distinguishing a form of "prosopagnosia" from a form of "multimodal people recognition disorders."

GENERAL DISCUSSION

The main results of this review concern, on one hand, the qualitative differences between defects of visual recognition observed in patients with lesions involving the ventral TO structures of the right and left hemisphere and, on the other hand, the distinction between "prosopagnosia" and "multimodal people recognition disorders." This second point of our review has led us to take into account aspects of familiar people recognition (such as the voice or the name) that clearly exceed the specific issue of prosopagnosia. This was made with two aims in mind: (a) to place the discussion of prosopagnosia in the more general context of familiar people recognition disorders; (b) to stress the need of investigating the other modalities of people recognition before considering a patients with face recognition disorders as an instance of prosopagnosia. In order to clarify the various parts of this discussion, we have reported in **Figure 1** the critical areas of the right and left temporal and occipital lobes that could play a critical role in different varieties of prosopagnosia and of multimodal familiar people recognition disorders:

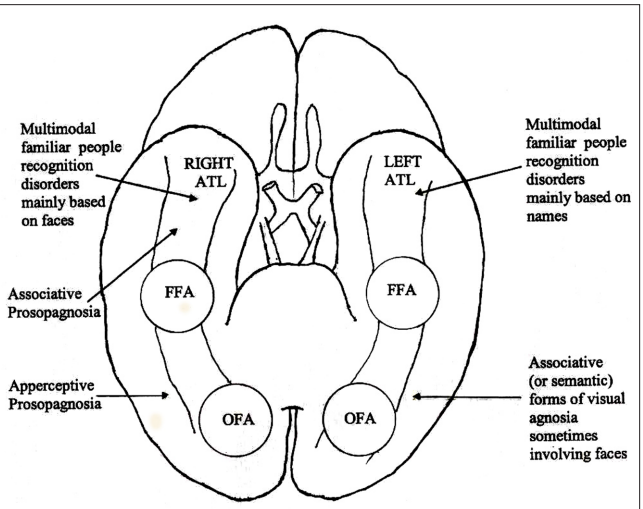

FIGURE 1 | Critical areas of the right and left, temporal and occipital lobes that could play a critical role in different varieties of prosopagnosia and of multimodal people recognition disorders.

QUALITATIVE DIFFERENCES BETWEEN THE VISUAL RECOGNITION DISORDERS OF PATIENTS WITH LESIONS INVOLVING THE VENTRAL TO STRUCTURES OF THE RIGHT AND LEFT HEMISPHERE

Although important qualitative differences and a different clinical context were observed in face recognition and identification disorders associated with right and left TO lesions, only those resulting from right-sided lesions fully satisfied the criteria of a modality-specific recognition disorder selectively affecting familiar faces (prosopagnosia). This claim is based on the following: (a) the *type of perceptual processing disrupted in these patients* is the holistic-configurational one, which is considered most appropriate for the perceptual treatment of faces (Bradshaw and Nettleton, 1981; Rhodes, 1988; Grill-Spector et al., 1998); (b) right brain-damaged patients show a systematic *loss of face familiarity feelings*, which can be considered the main mechanism through which we automatically orient attention toward socially relevant known people and disregard unknown ones.

Patients with left TO lesions can also rarely show defects of familiar people identification, but these defects are usually included in a context of associative visual object agnosia or of more general semantic disorders, as shown by data reported in **Tables 1 and 2**.

This interpretation might also explain why these patients, in whom faces are processed normally in a configurational manner by the right hemisphere, do not show the loss of face familiarity feelings, which is a hallmark of true prosopagnosia.

More generally, the fact that the two markers of prosopagnosia resulting from disruption of the TO structures of the right hemisphere consist of (a) a defect in face configurational processing and (b) a loss of face familiarity feelings, raises the issue about the relationships that might exist between these two right hemisphere processing mechanisms. One hypothesis that might be advanced on this subject could consist in assuming that a sort of functional integration may exist between the coarse holistic treatment of faces

performed by the right hemisphere and the emergence of familiarity feelings, aiming to check that the person who has raised these feelings really corresponds to the tentatively identified people. The hypothesis of a link between defective face configurational processing and impaired face familiarity feelings could explain the strong correlation observed in patients with a lesion involving the ventral TO areas of the right hemisphere (reported in **Table 3**), between defective configurational processing and lack of familiarity feelings.

This hypothesis could also explain why in patients with a lesion involving the territory of the left PCA (**Table 2**) face recognition disorders often satisfied the criteria of associative prosopagnosia, whereas in those with a lesion involving the ventral TO areas of the right hemisphere (**Table 3**) the defect was not limited to the recognition of familiar faces but also extended to the discrimination of unfamiliar faces. This was probably because in patients with right hemisphere lesions the defect of configurational processing, typical of the right ventral TO areas, concerned both familiar and unfamiliar faces and therefore made the generation of the associative form of prosopagnosia (**Table 3**) very difficult, whereas in patients with homologous lesions of the left hemisphere the associative nature of visual agnosias concerning objects and faces (**Tables 1 and 2**) could have been due to the mnesic/semantic nature of their recognition disorders.

DISTINCTION BETWEEN "PROSOPAGNOSIA" AND "MULTIMODAL PEOPLE RECOGNITION DISORDERS"

The distinction between "prosopagnosia" and "multimodal people recognition disorders" is probably based on the different functions accomplished in the earliest and in the last stages of face processing by the posterior (TO) and the anterior parts of the right temporal lobe.

According to a classical feed-forward model (Haxby et al., 2000), the more posterior face-sensitive regions of the visual cortex (OFA) could be involved in basic analysis of facial features and could project to more anterior regions (FFA), which could encode the structural face properties related to identity processing (Fox et al., 2008). Other more dynamic models (e.g., Rossion et al., 2003; Sorger et al., 2007) suggest that not only feed-forward, but also re-entrant interactions between right and left OFA and FFA could play a role in normal face perception; in any case, disruption of these TO structures selectively impairs visual perception, leading to an apperceptive form of prosopagnosia.

In the anterior temporal structures, on the contrary, visual processing output is associated with the output of other sensory modalities and with the mechanisms of episodic and semantic memory.

Impairment of these structures should, therefore, rarely provoke a modality-specific face recognition defect, but rather a multimodal person recognition disorder in which defects of face recognition are sometimes prominent. According to some authors (e.g., Mohedano-Moriano et al., 2008; Joassin et al., 2011) the right hippocampus could play a particular role in the integration of face and voice information, because this structure has an enhanced connectivity with both visual (the FFA) and auditory (the superior temporal gyrus) unimodal cortical areas. Anyway, irrespectively of the role that hippocampus and peri-rhinal cortex could play in the multimodal integration of face and voice information and in the generation of face familiarity feelings (see Yonelinas, 2002; Gainotti, 2007b for reviews), it remains possible that some patients with right anterior temporal lesions may show a form of associative prosopagnosia. This variety of prosopagnosia could result from a disconnection (Fox et al., 2008) between anterior temporal structures and the FFA, or from the earliest cortical lesions, marking the onset of a right temporal variant of fronto-temporal degeneration. The hypothesis that "associative prosopagnosia" may be the first manifestation of a right temporal variant of fronto-temporal degeneration is, at first glance, supported by the data reported in **Table 4**. If we look at these data, we see that two patients (VA and MT) could satisfy the criteria for associative prosopagnosia, because they have a famous face recognition defect, but no visual perceptual defects, voice recognition disorders or defects in the retrieval of person-specific semantic information from names. Nevertheless, caution is required in concluding that these are true cases of associative prosopagnosia, because in patient MT voice recognition was considered unimpaired on the basis of a very short clinical task, and in patient VA the integrity of voice recognition was based on her statement that difficulty in recognizing familiar people lessened when she heard their voices. However, both M.me V of Boudouresques et al. (1979) and our patient CO (Gainotti et al., 2003) repeatedly claimed they were able to identify familiar people by hearing their voices but performed very poorly when voice identification was systematically investigated. And voice recognition was as impaired as face recognition in other patients with face recognition disorders associated with right anterior temporal lesions (e.g., BD, Hanley et al., 1989; KS, Ellis et al., 1989; Maria, Gentileschi et al., 1999; Emma, Gentileschi et al., 2001; CD, Gainotti et al., 2008; MD, Busigny et al., 2009; KL, Hailstone et al., 2010) who were not reported in **Table 4** because they had not been labeled as prosopagnosia in the title of their case report. Taken together, these data indicate that in patients with anterior temporal lesions and familiar face recognition disorders a systematic investigation of voice recognition and retrieval of person-specific semantic information from names is necessary to decide whether the patient should be considered as having a form of "associative prosopagnosia" or a form of "multimodal people recognition disorder."

CONCLUDING REMARKS

In the introductory part of this review, we listed some issues (such as the specific or non-specific manifestations of prosopagnosia, the unitary or non-unitary nature of this syndrome and the mechanisms underlying face recognition disorders) that are still controversial in the study of prosopagnosia. We also suggested that two main sources of variance might be the qualitative differences between the face recognition disorders observed in patients with a prevalent lesion of the right or left hemisphere and in those with a lesion encroaching upon the TO or the (right) anterior temporal cortical areas.

Results of our review confirm that different kinds of face recognition disorders can be observed as a function of the right vs left and of the TO vs (right) anterior temporal lesion location. In fact, they show that the most specific forms of prosopagnosia are due to lesions of a right posterior network, including the OFA and the FFA, whereas (a) the face identification defects observed in patients with left TO lesions seem due to a semantic defect impeding access to the person-specific semantic information from the visual modality

and (b) the face recognition defects resulting from right anterior temporal lesions can usually be considered as part of a multimodal people recognition disorder.

From the operational point of view, our results suggest: (a) considering the components of visual agnosia often observed in prosopagnosic patients with bilateral TO lesions as part of a semantic defect resulting from the left-sided lesion (and not of prosopagnosia proper); (b) systematically investigating voice recognition disorders in patients with right anterior temporal lesions to determine whether the patient's face recognition defect should be considered a form of "associative prosopagnosia" or a form of "multimodal people recognition disorder."

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