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Face-specific impairment in holistic perception following focal lesion of the right anterior temporal lobe

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ABSTRACT

Recent studies have provided solid evidence for pure cases of prosopagnosia following brain damage. The patients reported so far have posterior lesions encompassing either or both the right inferior occipital cortex and fusiform gyrus, and exhibit a critical impairment in generating a sufficiently detailed holistic percept to individualize faces. Here, we extended these observations to include the prosopagnosic patient LR (Bukach, Bub, Gauthier, & Tarr, 2006), whose damage is restricted to the anterior region of the right temporal lobe. First, we report that LR is able to discriminate parametrically defined individual exemplars of nonface object categories as accurately and quickly as typical observers, which suggests that the visual similarity account of prosopagnosia does not explain his impairments. Then, we show that LR does not present with the typical face inversion effect, whole-part advantage, or composite face effect and, therefore, has impaired holistic perception of individual faces. Moreover, the patient is more impaired at matching faces when the facial part he fixates is masked than when it is selectively revealed by means of gaze contingency. Altogether these observations support the view that the nature of the critical face impairment does not differ qualitatively across patients with acquired prosopagnosia, regardless of the localization of brain damage: all these patients appear to be impaired to some extent at what constitutes the heart of our visual expertise with faces, namely holistic perception at a sufficiently fine-grained level of resolution to discriminate exemplars of the face class efficiently. This conclusion raises issues regarding the existing criteria for diagnosis/classification of patients as cases of apperceptive or associative prosopagnosia.

1. Introduction

1.1. Apperceptive and associative prosopagnosia

For most of us, recognizing a friend by his/her face is so easy and natural that we are usually unaware of the complexity of the operations at play during face recognition, a function that has been the topic of many investigations in experimental (neuro)psychology over the past decades (Calder, Rhodes, Johnson, & Haxby, 2011; Bruce & Young, 2012). Traditionally, experimental psychologists and cognitive neuropsychologists have divided the process of face recognition into several sub-functions or sub-processes (Bruce & Young, 1986). These processes are thought to be carried out by distinct brain structures, organized in a network (e.g., Sergent, Otha, & MacDonald, 1992; Allison, Puce, Spencer, & McCarthy, 1999; Haxby, Hoffman, & Gobbini, 2000; Gobbini & Haxby, 2007; Fox, Iaria, & Barton, 2009; Weiner & Grill-Spector, 2010; Rossion, Hanseeuw, & Dricot, 2012; Pyles, Verstynen, Schneider, & Tarr, 2013).

A major distinction in the field is made between the perceptual and mnesic aspects of face recognition. For instance, several authors have applied the apperceptive/associative classification of visual agnosia, called "psychic blindness" by Lissauer (1890), to the domain of faces (De Renzi, 1986; De Renzi, Faglioni, Grossi, & Nichelli, 1991; Sergent & Signoret, 1992a, 1992b; Barton, 2003). These authors argued that there are at least two separate forms of the inability to recognize faces following brain damage: an apperceptive and an associative form of prosopagnosia. Patients with apperceptive prosopagnosia are unable to perceive faces properly, while patients with associative prosopagnosia are unable to associate a correct percept of a face with stored memories about this face. Apperceptive prosopagnosia has been linked to posterior occipito-temporal lesions, whereas associative prosopagnosia has

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been associated with lesions of the anterior part of the temporal lobe, in particular in the right hemisphere (Gross & Sergent, 1992; Sergent & Signoret, 1992b; Barton, Cherkasova, & Hefter, 2004; Pancaroglu et al. 2011).

The vast majority of impairments in face recognition following damage to the anterior temporal lobe has been reported in the context of neurodegenerative diseases, such as the right temporal variant of frontotemporal dementia (Tyrrell, Warrington, Frackowiak, & Rossor, 1990; Barbarotto, Capitani, Spinnler, & Trivelli, 1995; Evans, Heggs, Antoun, & Hodges, 1995; Gentileschi, Sperber, & Spinnler, 1999, 2001; Gainotti, Barbier, & Marra, 2003; Gainotti, Ferraccioli, Quaranta, & Marra, 2008; Gorno-Tempini et al., 2004; Thompson et al., 2004; Joubert et al., 2006; Busigny, Robaye, Dricot, & Rossion, 2009). However, these patients are usually also impaired in recognizing individuals by their names and voices. Since face recognition impairment is only one symptom of (somewhat diffuse) damage in the anterior temporal lobe, it might be more appropriate to characterize these patients as having "multimodal person recognition disorder" rather than "associative prosopagnosia" (Gainotti, 2013).

More rarely, sudden focal brain damage to the anterior temporal lobe due to herpes simplex encephalitis (Warrington & McCarthy, 1988; Hanley, Young, & Pearson, 1989; Sergent & Poncet, 1990; Haslam, Cook, & Coltheart, 2001; Barton, Hanif, & Ashraf, 2009; Dalrymple et al., 2011; Pancaroglu et al., 2011), closed head injury (Kapur, Ellison, Smith, McLellan, & Burrows, 1992; Barton, Zhao, & Keenan, 2003), or following anterior temporal lobe resection in the context of epileptic seizures resistant to medication (Ellis, Young, & Critchley, 1989; Tippett, Miller, & Farah, 2000; Glosser, Salvucci, & Chiaravalloti, 2003; Chiaravalloti & Glosser, 2004; Drane et al., 2008; Pancaroglu, Johnston, Sekunova, Duchaine, & Barton, 2012) (see Table 1) can all result in impairments in face recognition. However, the majority of these patients are also impaired at recognizing individuals by their names and voices.

1.2. Perceptual impairment in associative prosopagnosia

The studies listed above suggest that patients with right anterior temporal lobe damage are impaired at memorizing new faces, identifying familiar faces and retrieving semantic information about familiar faces. In general, these patients have been reported as being unimpaired in face *perception,* i.e., the ability to build a proper visual representation – an internal image – of a face. For instance, most patients were able to match simultaneously presented pictures of unfamiliar faces, as in the Benton Face Recognition Test (BFRT, Benton & van Allen, 1968) or similar tests (Warrington & McCarthy, 1988; Ellis et al., 1989; Hanley et al., 1989; Sergent & Poncet, 1990; Kapur et al., 1992; Tippet et al., 2000; Haslam et al., 2001; Glosser et al., 2003; Chiaravalloti & Glosser, 2004; Barton et al., 2009; Dalrymple et al., 2011; see Table 1). However, importantly, with the exception of a single-patient study (Sergent & Poncet, 1990), response times were never reported. Consequently, whether these patients relied on a slow feature-by-feature strategy to match faces, as reported in many cases of prosopagnosia with face perception impairment (e.g. Newcombe, 1979; McNeil & Warrington, 1991; Young, Flude, Hay, & Ellis, 1993; Mattson, Levin, & Grafman, 2000; Rossion et al., 2003; Delvenne, Seron, Coyette, & Rossion, 2004), remains unknown. Moreover, there are good reasons to doubt that perception of faces is intact in these patients. In a review of 99 cases of associative visual agnosia, Farah (1990/2004) reported that most patients characterized as being of the 'associative' form were abnormally sensitive to the visual quality of the stimuli, and performed poorly when recognizing line drawing stimuli or stimuli presented tachistoscopically. Most of these patients' recognition errors were visual in nature. When patients did copy drawings reasonably well, they were described as using a "slavish, line-by-line, and piecemeal" strategy (Farah, 1990/2004, p. 74). Although these observations concern object rather than face recognition, they raise issues around the diagnosis criteria of a purely associative form of prosopagnosia.

1.3. Nature of the perceptual impairment in apperceptive prosopagnosia

There is now converging evidence supporting the view that patients with acquired apperceptive prosopagnosia present with impaired configural/holistic face perception (for a review see Busigny, Joubert, Felician, Ceccaldi, & Rossion, 2010a). While their perception of face parts is relatively well preserved, these patients seem unable to integrate these different parts into a single, unified

Table 1
Face perception results of patients reported with anterior temporal lesions and presenting with face recognition difficulties.

Patient	Etiology	Face perception	Results
RFR (Warrington & McCarthy, 1988)	HSE	OK	18/20 (same/diff matching task)
BD (Hanley et al., 1989)	HSE	OK	46/54 (BFRT)
PV (Sergent & Poncet, 1990)	HSE	OK	46/54, 6min21 (BFRT)
TG (Haslam et al., 2001)	HSE	OK	43/54 (BFRT)
B-AT1 (Barton et al., 2009; Dalrymple et al., 2011)	HSE	OK	45/54 (BFRT) 100% (same/diff discrimination, FAB) 48% of errors (CFPT, mean=36.7; SD=12.2)
R-AT2 (Barton et al., 2009)	HSE	OK	47/54 (BFRT) 90% (same/diff discrimination, FAB) 40% of errors (CFPT, mean=36.7; SD=12.2)
LT (Kapur et al., 1992)	CHI	OK	Preserved (BFRT)
TS/008 (Barton et al., 2003; Barton, 2008)	CHI	impaired	24/54 (BFRT) No face geometry effect
KS (Ellis et al., 1989)	RATL	OK	45/54 (BFRT)
CT (Tippett et al., 2000)	RATL	OK	43/54 (BFRT) 91% (1581 ms) upright faces 78% (1641 ms) inverted faces Normal performance+normal FIE
R-AT1 (Barton et al., 2009)	RATL	OK	41/54 (BFRT) 58% of errors (CFPT, mean=36.7; SD=12.2)
26 patients (Glosser et al., 2003)	RATL	OK	$Z=-0.71$ (0,22) (BFRT) compared to controls
38 patients (Chiaravalloti & Glosser, 2004)	RATL	OK	$\pm$44/54 (before surgery) (BFRT) $\pm$43/54 (after surgery) (BFRT)

HSE=Herpes Simplex Encephalitis.
CHI=Closed Head Injury.
ATL=Right Anterior Temporal Lobectomy.
BFRT=Benton Face Recognition Test (Benton & Van Allen, 1968).
FAB=Florida Affect Battery (Bowers et al., 1991).
CFPT=California Face Perception Test (Duchaine et al., 2007).