CORTEX 183 (2025) 330–348

Available online at www.sciencedirect.com

ScienceDirect

Journal homepage: www.elsevier.com/locate/cortex

Research Report

Imagery and perception in acquired prosopagnosia: Functional variants and their relation to structure

Jason J.S. Barton [a,*], *Brad Duchaine* [b] *and Andrea Albonico* [c]

[a] *Department of Medicine (Neurology), Ophthalmology and Visual Sciences, Psychology, University of British Columbia, Vancouver, Canada*

[b] *Department of Psychological and Brain Sciences, Dartmouth College, Hanover, USA*

[c] *Department of Psychology, University of the Fraser Valley, Abbotsford, Canada*

ARTICLE INFO

Article history:
Received 19 August 2024
Reviewed 07 October 2024
Revised 22 October 2024
Accepted 12 November 2024
Action editor Holger Wiese
Published online 29 November 2024

Keywords:
Face recognition
Memory
Fusiform gyrus
Anterior temporal
Associative
Imagery

ABSTRACT

Current models of face perception and the face-processing network suggest that acquired prosopagnosia may not be a single disorder but rather a family of variants differing in mechanism. It has been proposed that tests of face perception and face imagery can probe component processes to support apperceptive, associative, and amnestic distinctions. However, validating this proposal is hampered by the rarity of this condition. Here we report observations gathered over two-and-a-half decades on the perception of facial shape and the imagery for famous faces of twenty-three patients.

Patients with lesions limited to the occipitotemporal lobes had an apperceptive profile, with impaired perception of facial shape but no or mild deficits for face imagery. The apperceptive defect affected not just configuration but also feature size and external contour, especially in the upper face, and was more severe when subjects attended to multiple aspects of the face. An amnestic profile, with severely impaired imagery and minimally affected perception, was seen in two patients, one with right and one with bilateral anterior temporal damage. Four patients had an apperceptive/amnestic combination, all with bilateral occipitotemporal and right anterior temporal damage. Right anterior temporal damage alone often caused only mild imagery deficits: along with their relatively intact face perception, these subjects came closest to meeting proposed exclusionary criteria for an associative variant, i.e., relative preservation of both imagery and perception.

These results confirm a link between apperceptive prosopagnosia and occipitotemporal lesions. Damage to the right anterior temporal lobe was common to all with a severe amnestic deficit, but often requiring additional damage.

* *Corresponding author*. Neuro-Ophthalmology, Section K, VGH Eye Care Centre, 2550 Willow Street, Vancouver, British Columbia, V5Z 3N9, Canada.
E-mail address: jason.barton@ubc.ca (J.J.S. Barton).

https://doi.org/10.1016/j.cortex.2024.11.011

Recognizing faces is a seemingly effortless task performed by all humans many times a day, yet it is a complex process that involves several cognitive operations (Young et al., 2011). At a perceptual level it requires analysis of a complicated three-dimensional structure that can be viewed from different angles and has mobile elements that introduce variations in shape during emotional expression and speech. At a mnemonic level the evolving percept must be matched to a stored representation of a known face, which is not trivial given the high similarity in structure between all faces and the large number of exemplars known by each of us, which averages around five thousand faces (Jenkins et al., 2018).

Prosopagnosia is the failure of this process. The result is a loss of familiarity for the identity of faces one has seen before, which cannot be explained by more pervasive general defects in vision or memory. Given the complex nature of face recognition, it may be that different patients become prosopagnosic for different reasons. An early case with a review of 25 published cases claimed to discern a perceptual type and a second type with loss of face imagery (Hécaen et al., 1957). However, most researchers continued to see prosopagnosia as a unitary disorder, until thirty years ago the possibility of functional variants was raised again (de Renzi et al., 1991; Young et al., 1994). Eventually a review (Damasio et al., 1990) proposed three variants: an apperceptive type from right parieto-occipital damage, an associative type linked to bilateral inferior occipitotemporal lesions, and an associative/amnestic type with bilateral anterior temporal lesions.

This review did not provide criteria or data. It inferred the variant from the company it kept. Their 'apperceptive' variant was accompanied by problems with non-face visual perception, the example given being recognizing a plane from assembling its parts. Today this would be considered an integrative type of general visual agnosia (Riddoch et al., 1987) rather than prosopagnosia. In the 'amnestic/associative' type of (Damasio et al., 1990), the patient could not recognize people from other non-face cues, such as voice—i.e., 'regardless of the sensory channel' (p.92). This conforms to what is now called a multimodal person-recognition disorder, or people-specific amnesia (Gainotti, 2010). Their pure 'associative' variant had neither problems with visual perception nor difficulties recognizing people through other routes. Of these three types it is the only one that would correspond to the current definition of prosopagnosia.

The next year saw a description of two patients who could recognize non-face objects but not faces (de Renzi et al., 1991). One patient was impaired on two tests of face perception, unfamiliar face matching and age estimation from faces, while the other performed well. The authors argued that the first patient had an apperceptive variant while the second had an associative variant.

As this last report stated, instruments that probe different stages would facilitate analysis of the face processing in prosopagnosia. A common test of the perceptual stage is matching of simultaneously viewed unfamiliar faces, as with the Benton Face Recognition Test (Benton and van Allen, 1972) and the Cambridge Face Perception Test (Duchaine et al., 2007). Others have made inferences about perception from impaired judgments of other facial attributes such as age, expression or gender, or from problems on tasks such as face detection (Gainotti, 2018; Morioka et al., 2024; Sugimoto et al., 2012; Tábuas-Pereira et al., 2016; Ulrich et al., 2017). However, it is neither logical nor proven that the face information or representations used for these non-identity judgments are necessary for face identification. For instance, adaptation studies have shown that the perception of identity is primarily driven by facial structure, while the perception of age is based more on facial texture (Lai et al., 2013). An understanding of what we need to see in faces to identify them should be the foundation of perceptual tests relevant to prosopagnosia.

In typical face recognition there are contributions from the perception of facial features and their position in the face, also referred to as their 'configuration' (Barton, Keenan, & Bass, 2001; Pichler et al., 2011). In addition, holistic face perception proposes that the face is processed as an integrated whole (Tanaka et al., 2003; Young et al., 1987). These concepts may not be mutually exclusive, as configuration speaks to *what* is being processed whereas holism describes *how* they are processed (Ramon et al., 2010b). Studies of acquired or developmental prosopagnosia have found problems in perceiving facial configuration or features (Barton et al., 2002, 2003b; Grüter et al., 2009; Yovel et al., 2006). Some reported evidence of reduced holistic face processing in acquired (Busigny et al., 2010, 2011; Ramon et al., 2010a) or developmental prosopagnosia (Avidan et al., 2011; Palermo et al., 2011), though others did not (Biotti et al., 2017; Finzi et al., 2016).

How about associative or amnestic variants? These refer respectively to an inability to link perception to face representations stored in memory, or a loss of those face memories. It is not ideal to diagnose these merely by a negative inference that, if perception is intact, the problem must then be one of these two. For a start, this logic carries the assumption that the perceptual tests the investigators used have adequately assessed all elements of face perception that are necessary for recognizing faces. Second, standard face memory tests like the Warrington Recognition Memory Test (Warrington, 1984) and the Cambridge Face Memory Test (Duchaine et al., 2006) cannot probe the mnemonic operations of face recognition independently of perception, since these require subjects to respond about faces that they are seeing. To bypass perceptual stages and circumvent this problem, some researchers have turned to face imagery.

Enquiries about the ability to visualize faces have a long history. Indeed, the report that coined the term "prosopagnosia" reported that the two patients S and A were still able to imagine specific faces (Bodamer, 1947, 1949). Two other early patients with probable occipital lesions reported that they could imagine specific faces (Ellis et al., 1990; Pallis, 1955) but others had problems with face 're-visualization' (Lhermitte et al., 1975; Rondot et al., 1967; Shuttleworth et al., 1982). To move beyond a subject's own impression, initial imagery probes asked subjects to describe known faces verbally, which invariably emphasized features and were difficult to quantify (Levine et al., 1985; Ogden, 1993; Shuttleworth et al., 1982; Whiteley et al., 1977). Studies then created questionnaires that asked subjects to imagine the faces of celebrities and make forced-choice responses about them (Bartolomeo et al., 1998; Barton et al., 2003a; Tree et al., 2010; Young et al., 1994).

On the basis of observations about configural perception and imagery in a series of ten patients, it was proposed that there were variants of prosopagnosia that correlated with the locus of damage (Barton, 2008). An apperceptive variant occurred with inferior occipitotemporal damage, as this impaired the ability to perceive facial structure accurately. An amnestic variant was caused by damage including but not limited to the right anterior temporal lobe, which impacted perception less but severely degraded face memories, as indexed by imagery for famous faces.

How has this proposal fared with time? The concept of variants is not universally shared. There are still claims that acquired prosopagnosia is a single disorder whose mechanism is the same regardless of lesion location (Busigny et al., 2014), a conclusion that was reached from comparisons between three patients. This underscores a key obstacle to clarifying the situation: the rarity of acquired prosopagnosia. Even the observation above was based on a small sample of ten patients, only two of whom had damage limited to the anterior temporal lobe (Barton, 2008).

In the sixteen years since that publication (Barton, 2008) we have studied a second cohort of thirteen patients in Vancouver, including five with only anterior temporal damage. We have reported many studies with various members of this second cohort, concerning their colour perception (Moroz et al., 2016), reading (Hills et al., 2015), topographic orientation (Corrow et al., 2016), scanpaths with faces (Pancaroglu et al., 2016), response to face-training (Davies-Thompson et al., 2017), etc. Here we focus on their data for face imagery and face perception, comparing and combining the two cohorts to re-examine this issue in one of the larger samples of acquired prosopagnosia. The primary goal is to compared those with anterior temporal and those with occipitotemporal damage, first in their perception of structural differences between faces, and second in their ability to imagine known faces. As secondary objectives we examined the pattern of perceptual deficits in the occipitotemporal group to determine if there are selective vulnerabilties for certain types of facial structure, and the pattern of imagery deficits to determine if there is greater impairment of global than feature imagery.

1. Methods

1.1. *Participants*

Patients from the Boston cohort reported in 2008 consisted of 10 subjects, 2 of whom were female, with a mean age of 44.8 years (SD 7.4, range 37–54). These patients were local to the New England region and tested at the Beth Israel Deaconess Medical Center. The Vancouver cohort was recruited locally and from across North America using the website www.faceblind.org. It consisted of 13 subjects, 5 female, of mean age 45.4 years, (SD 15 .9, range 23–71 years). The average duration of prosopagnosia was 13.1 years (SD 12.4, range .25–36) for the Boston cohort and 12.5 years (SD 11.9, range .25–34) for the Vancouver cohort. These demographic parameters did not differ significantly between the two groups. Sample size calculations are not relevant as this is a rare disorder and the sample is simply determined by how many of these unusual patients we can find and/or contact us, over the 25 years of study.

Prior reports have described our diagnostic criteria (Barton et al., 2002; Fox et al., 2011; Hills et al., 2015). Both groups had relatively preserved performance on a battery of tests of general memory and visual perception. In the Boston cohort we assessed face perception and recognition with a Famous Faces test (Albert et al., 1979), the Warrington Recognition Memory Test (Warrington, 1984), the Benton Face Recognition Test (Benton et al., 1972), and the BIDMC Famous Faces Test, which assessed the ability to discriminate famous from anonymous faces (Barton, Cherkasova, & O'Connor, 2001). The Vancouver cohort was assessed with these as well as the Cambridge Face Memory Test (Duchaine et al., 2006).

Patients in the Vancouver cohort had functional MR imaging showing preservation of the fusiform and occipital face areas in those with anterior temporal lesions alone, and loss of one or both of these regions in those with occipitotemporal damage (Hills et al., 2015; Pancaroglu et al., 2016). As the Boston cohort had been tested prior to the development of reliable single-subject functional imaging of the face network (Fox et al., 2009), involvement or sparing of the fusiform face area was deemed probable by comparing their structural MRI scans to functional imaging in control subjects (Barton, 2008).

For ease of reference we retained the subject designations of those prior reports. Patients of the Boston cohort (Barton, 2008) were identified by a three-digit number (e.g., 005, 013, etc.). Patients of the Vancouver cohort (Hills et al., 2015; Pancaroglu et al., 2016) had labels conveying anatomic information. R, L and B indicated whether they had right, left or bilateral lesions, IOT indicated inferior occipitotemporal damage, AT indicated anterior temporal damage, and ATOT a combination of the two. One patient, 008/BAT2, travelled from Boston to Vancouver to participate in further studies and has two labels. For this study she is considered part of the Boston cohort.

The total group thus consists of 23 subjects with acquired prosopagnosia (Table 1). Twelve had inferior occipitotemporal lesions, seven right and five bilateral, the latter including LIOT2, whose left fusiform resection was accompanied by right fusiform atrophy on MRI. Seven had anterior temporal lesions, five right and two bilateral.

Four patients had complex damage to right anterior temporal and bilateral inferior occipitotemporal cortex, the latter worse on the right (Fig. 1). Our grouping of these complex patients reflected the two specific questions we are asking, which are, a) does occipitotemporal damage impair face perception and b) does anterior temporal damage impair face imagery? Hence the perceptual analysis sought to compare patients with and without occipitotemporal damage. In this first analysis the complex patients belonged to the occipitotemporal group. On the other hand, the aim of imagery analysis was to compare patients with and without anterior temporal damage. Here the complex patients were assigned to the anterior temporal group.

Control groups were described in the original reports. The controls for the *A-Z test* of configuration used with the Boston cohort were 14 subjects, 4 female, aged 16–43 years (Barton et al., 2002), and for the *George test* used with the Vancouver cohort 12 subjects, 8 female, aged 25–55 years (Pancaroglu

Table 1 – Demographic data of the two cohorts.

Subject	Age	Duration	Gender	Lesion	Fields
Right occipitotemporal					
RIOT1	56	19	M	Hemorrhage, vascular malformation	L-UQ
RIOT3	71	2	M	Infarct	L-HH
RIOT4	62	1	M	Infarct	L-UQ
005	59	1	M	Infarct	L-HH
006	52	.5	M	Tumour, resection	L-HH
009	49	.25	M	Infarct	L-HH
012	55	.5	M	Infarct	L-HH
Bilateral occipitotemporal					
LIOT2	59	18	M	Seizures, left resection, right atrophy	Full
BIOT1	41	.25	M	Infarcts	B-UQ
BIOT2	60	34	M	Subdural hematoma	B-complex
004	38	18	M	Gunshot	B-UQ
010	41	21	M	Subdural hematoma	R–HH
Bilateral occipitotemporal and right anterior temporal					
BATOT1	46	32	F	Herpes encephalitis	L-UQ
BATOT2	23	13	F	Herpes encephalitis	Full
007	37	20	F	Viral encephalitis	Full
011	54	36	M	Trauma, temporal resection	B-complex
Right anterior temporal					
RAT1	24	1	F	Seizures, lobectomy	Full
RAT2	34	9	F	Herpes encephalitis	Full
RAT3	37	7	M	Herpes encephalitis	Full
RAT5	60	28	F	Tumour, resection	Full
013	41	11	M	Seizures, lobectomy	L-UQ
Bilateral anterior temporal					
BAT1	25	4	M	Herpes encephalitis	Full
008/BAT2	47	23	F	Trauma, temporal resection	Full

Numerical designations of 00_ indicate a member of the Boston cohort.
Designations beginning with R, B or L are from the Vancouver cohort.
L = left, R = right, B = bilateral.
IOT = occipitotemporal, AT = anterior temporal, ATOT = anterior temporal and occipitotemporal.
M = male, F = female.
UQ = superior quadrantanopia, HH = hemifield defect, complex = complex hemifield defects.

et al., 2016). The imagery test had 31 controls, 18 female, aged 22–60 years (Barton et al., 2003a).

As tabulated elsewhere (Corrow et al., 2016; Hills et al., 2015), all subjects of the Vancouver cohort had an extensive neuropsychological evaluation, including tests of executive function, verbal and visual memory, attention, visual perception and language. These included two standardized tests requiring non-face imagery: the mental rotation test (Grossi, 1991) and the road map test of direction sense (Money et al., 1965). These visuospatial imagery tests were chosen to probe the general ability to form visual imagery. Imagery tests for objects and their properties may not be suitable for that purpose: many prosopagnosic subjects have additional colour or object perceptual deficits, as was the case in our cohort (Barton et al., 2019; Moroz et al., 2016) and in some this is accompanied by parallel imagery deficits, as shown previously for colour processing in subject 011, also known as LH (Levine et al., 1985). On our visuospatial imagery tests, all scored in the normal range according to published criteria (Morganti, 2018; Trojano et al., 2015). In fact, most subjects made either no errors or a single error on these tests (Supplementary Table 1).

Testing protocols were approved for the Boston Cohort by the institutional review board of the Beth Israel Deaconess Medical Center, and for the Vancouver cohort by those of the Vancouver Coastal Health Research Foundation and the University of British Columbia. All subjects gave written consent in accordance with the Code of Ethics of the World Medical Association, Declaration of Helsinki.

1.2. *Procedures*

These have been described in detail elsewhere (Barton et al., 2002, 2003a; Malcolm et al., 2005). We present brief summaries here. For the perceptual tests, subjects sat 57 cm away from a screen in standard dim room lighting. For the imagery test, subjects read and circled answers on a paper questionnaire.

1.2.1. *The A-Z face perception test*

This was used to test configural perception in the Boston cohort (Barton et al., 2001b, 2002). It presented faces of two people as stimuli, one male (person A) and one female (person Z). We used these as base faces and to create target faces, which were altered in one of three ways: decreasing the interocular distance, decreasing the nose-to-mouth distance, or lightning eye colour. A trial presented three faces in a triangular arrangement, two being the base face and one being a target. The target face occurred with equal probability at any of the three positions. The subject's task was to indicate which

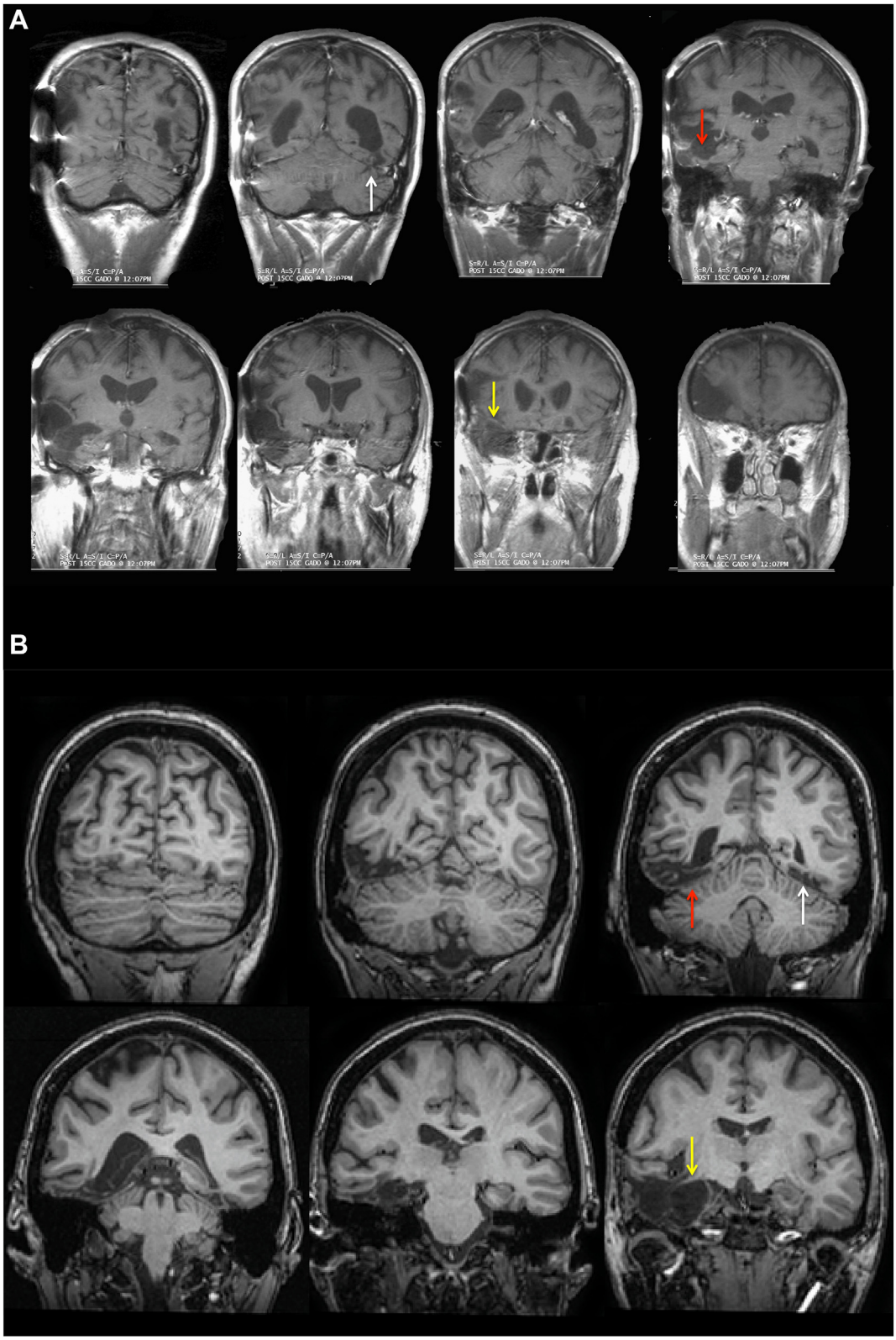

Fig. 1 – Coronal T1-weighted MRI scans showing examples of complex lesions in the group combining right anterior temporal and bilateral occipitotemporal lesions (ATOT). Each sequence marches anteriorly from occipital (top left) to temporal (bottom right) regions. Dark regions indicate damaged cortex. A. Subject 011, also known as LH in many reports e.g., (Etcoff et al., 1991; Farah et al., 1991, 1995; Levine et al., 1985, 1989). There is substantial traumatic damage to the right anterior temporal (yellow arrow) and inferior occipitotemporal regions (red arrow), some damage to the left occipitotemporal region (white arrow), as well as to the right occipitoparietal and right lateral frontal cortex. B. Subject

of the three faces was the altered target face, with chance performance being 33% correct.

There were five gradations for each of the three types of change. Each was presented nine times, for both the male and female face, giving a total of 270 trials per block. Subjects performed one block with unlimited viewing time, and several blocks with limited viewing times ranging from 1 to 4 s.

For comparison with the Vancouver cohort, whose testing focused on the perception of facial shape, we analyzed the data for targets with changes in inter-ocular or nose-to-mouth distances, from the blocks with unlimited viewing duration.

1.2.2. *The George face perception test*

Named after its creator (Malcolm et al., 2005), this probe assessed the Vancouver cohort. This was intended as an improved version of the *A-Z test*, focusing only on shape (i.e., not eye colour) and using more than two faces. The set of base faces was increased to six, with frontal images of three young males and three young females. Target faces were altered in either the upper or the lower face, in internal feature position, feature shape, or external contour (Fig. 2). Changes in internal feature position were the same as in the *A-Z test:* a decrease in inter-ocular distance or a decrease in the nose-mouth distance. Changes in feature shape were either an increase in the vertical width of both eyes or an increase in the vertical width of the mouth. Changes in external contour were either elevation of the hairline or narrowing of the chin. As in the *A-Z test*, each trial presented two base faces and one target face in a triangular arrangement. To reduce low-level image matching, the faces differed slightly in size. The target face occurred with equal probability at any of the three positions. Again, the subject's task was to indicate which of the three faces was the altered target face, with chance performance being 33% correct.

We present data for two different types of blocks. In the '*6-change*' block, any of the six changes was possible on any trial: hence subjects had to attend to the entire face. Each trial stimulus was presented three times in a random order for a total of 108 trials. In the '*1-change only*' blocks, each of the six types of changes was separated into six smaller blocks of 18 trials, again with a total of 108 trials. In the *1-change only* blocks, subjects knew what type of change they were looking for. All trials had unlimited viewing time.

1.2.3. *Famous face imagery test*

This test asked subjects to imagine the faces of two famous people and answer a question comparing their facial appearance (Barton et al., 2003a). The 37 questions included 18 about facial features and 19 about the global face shape, given in random order. Subjects omitted questions if they did not know one or both of the celebrities, or if they did not recall having seen their faces. A question was included in the battery if at least 70% of the 31 control subjects chose to respond to it, and at least 80% gave the correct answer. Controls had a similar accuracy for both feature (.93, SD .04), and global shape (.94, SD .06) items.

Subjects omitted a question if they had never heard of one member of the pair or did not recall seeing their face. The control subjects omitted a mean of 3.6 (SD 3.7) questions. In the Boston cohort the mean number of omitted items was 3.0 (SD 2.5), while the Vancouver cohort omitted a mean of 1.3 items (SD 2.6), despite the fact that these subjects were tested later than the Boston cohort. Hence most of the celebrities used had remained familiar over time.

Evidence that this face imagery test probes stored visual representations of faces comes from three subjects with congenital blindness (Dietz et al., 2022). As such subjects have never had vision, their ability to answer items must rely on semantic, not visual knowledge. All three were severely impaired on the face imagery questionnaire, two no better than chance.

1.3. *Analysis*

1.3.1. *Perceptual tests*

As these had unlimited viewing duration, we measured both reaction times and accuracy. While our prior reports of the Boston (Barton, 2008) and Vancouver (Pancaroglu et al., 2016) cohorts analyzed accuracy alone, here we report on inverse efficiency, which is the mean reaction time divided by accuracy (Townsend et al., 1983). This variable mitigates against speed-accuracy trade-offs and addresses concerns that assessments of prosopagnosic performance should include both accuracy and reaction time (Geskin et al., 2018), concerns that have a long history in prosopagnosia research (Lhermitte et al., 1975). When needed for comparisons across cohorts or different stimulus types, we normalized these inverse efficiencies, by subtracting a score from the mean for controls, and dividing this difference by the standard deviation of the control group. Hence this is a z-score, corresponding to the number of standard deviations away from the control mean that a subject's score lies.

For the *George test* used with the Vancouver cohort we calculated an overall structural perception score, averaging across all three types of changes (configural, feature shape, contour) in both the upper and lower face. We had two questions. First, we asked whether the inverse efficiency for shape perception differed between the occipitotemporal and anterior temporal groups. Second, we asked if group differences were affected by whether a subject could focus on one change at a time or had to monitor for any of the six. We used a repeated measures ANOVA on inverse efficiency scores, with Group (occipitotemporal, anterior temporal) as a between-subject factor, and Block (*6-change*, *1-change only*) as a within-subject factor. The overall perception scores for the *6-change* blocks are shown in Table 2.

To analyze configural perception in both cohorts, we extracted from the *A-Z test* used with Boston patients and the *George test* used with Vancouver patients the inverse efficiency for perceiving eye position and mouth position changes (reaction times and accuracy are provided in Supplementary Table 2). We averaged the data for eye and mouth position

BATOT1 has right anterior temporal (yellow arrow) and right (red arrow) and left (white arrow) inferior occipitotemporal damage, worse on the right, from herpes encephalitis.

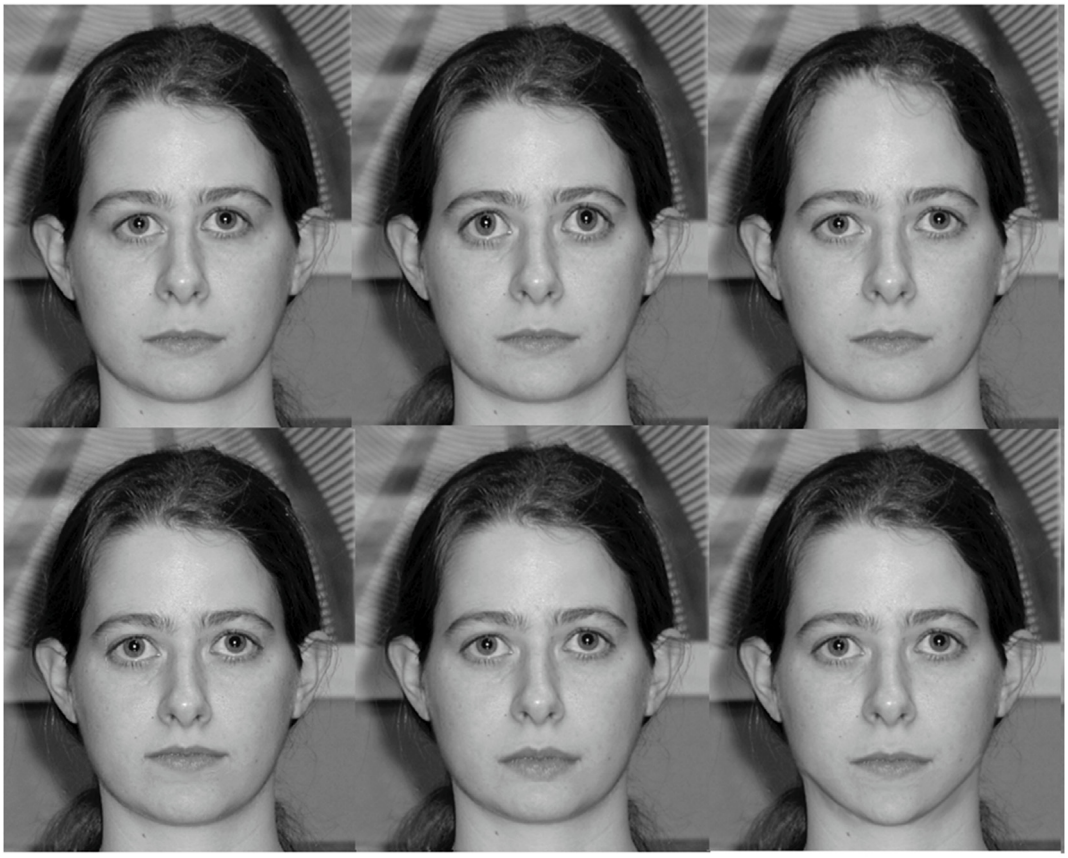

Fig. 2 – Example stimuli from the *George test*. Top row shows alterations in the upper face, bottom row changes in the lower face. From left to right are changes in configuration, feature shape, and external contour. Modified with permission from (Malcolm et al., 2005).

to give a single configural score, also shown in Table 2. To facilitate comparisons between the cohorts we used the normalized inverse efficiency, subjected to an ANOVA with Group (occipitotemporal, anterior temporal) and Cohort (Boston, Vancouver) as between-subject factors.

For one of our secondary objectives, we examined the data from occipitotemporal patients alone to determine if certain types of structural change were especially difficult for them to perceive. Our previous report on accuracy on the *George test* for a subset of the Vancouver cohort concluded that such patients had greater difficulty perceiving eye shape and eye position than the other four changes, especially in the *6-change* blocks (Pancaroglu et al., 2016). Since the performance of controls varied with the type of structural change, we used normalized inverse efficiency scores from the *6-change* blocks, which were a greater challenge for these subjects than the *1-change only* blocks. We performed a repeated measures ANOVA, with Change-type (configuration, feature shape, contour) and Location (upper face, lower face) as within-subject factors.

A contrast between perception for the upper versus the lower face had not been reported before for the Boston cohort. We also compared the normalized inverse efficiency scores for configural perception of the eyes versus the mouth, in the occipitotemporal groups of both cohorts. We used a repeated measures ANOVA with Cohort (Boston, Vancouver) as a between-subject factor and Location (eye, mouth) as a within-subject factor.

1.4. *Imagery test*

As the same famous-face imagery test was used for both cohorts, we analyzed the combined result for the entire group, for 22 of whom we have imagery data. BAT1 was not included because he claimed not to follow any politics or entertainment and did not know any of the people in our test. Of the remaining 22, ten had lesions including one or both anterior temporal lobes and twelve had occipitotemporal lesions that spared the anterior temporal lobes. We used a repeated measures ANOVA to assess accuracy, with Group (occipitotemporal, anterior temporal) as a between-subject factor and Imagery-Type (feature, global) as a within-subject factor. For specific post-hoc comparisons of subgroups, we used paired *t*-tests.

Finally, we examined individual subjects for putative classical dissociations (Gerlach et al., 2018) between feature and global shape imagery. An individual's score had to meet three criteria. First, it had to be in the normal range for one

Table 2 – Selected face perception test results, including overall inverse efficiency for the AZ and George tests, and overall imagery accuracy.

	Famous Faces	Faces WRMT	Perception		Imagery Overall
			AZ test eye + mouth	*George test* all 6 changes	
	d'	/50	Inverse efficiency		Accuracy
Control Mean	2.78		2.14	3.97	.93
SD	.37		1.4	1.28	.04
95% limit	2.01		5.27	7.02	.85
Right occipitotemporal					
RIOT1	1.96	33		9.63	.82
RIOT3	.29	33		40.56	.73
RIOT4	1.29	**39**		37.10	.84
005	.67	33	27.66		**.90**
006	1.12	32	7.94		.82
009	.88	33	44.65		**.86**
012	.00	–	47.73		.73
Bilateral occipitotemporal					
LIOT2	.00	27		22.89	.41
BIOT1	**2.21**	28		12.29	**.89**
BIOT2	1.31	21		15.23	**.86**
004	−.14	33	42.30		.78
010	−.22	24	29.57		.78
Bilateral occipito- and anterior temporal					
BATOT1	0	27		21.71	.61
BATOT2	.15	19		27.51	.48
007	1.04	29	35.10		.67
011	−.18	33	43.22		.64
Right anterior temporal					
RAT1	.97	17		11.33	**.93**
RAT2	.65	27		**6.76**	.72
RAT3	.9	31		9.09	.49
RAT5	1.52	28		13.84	.81
013	1.29	32	**4.88**		.81
Bilateral anterior temporal					
BAT1	.36	27		9.44	–
008/BAT2	.68	31	**4.86**		.5

Numerical designations of 00_ indicate a member of the Boston cohort.
Designations beginning with R, B or L are from the Vancouver cohort.
L = left, R = right, B = bilateral.
IOT = occipitotemporal, AT = anterior temporal, ATOT = anterior temporal/occipitotemporal.
Bold = normal score.

test. Second, it had to fall below the 95% prediction limit for the other. Third, the difference between the two scores had to exceed the 95% prediction limit for this subtraction in controls.

2. Results

2.1. *Perception of facial structure*

a. Structure perception in the Vancouver cohort (Fig. 3A)

The ANOVA showed, as expected, a main effect of Block [$F_{(1,11)} = 21.1$, $p < .001$, $\eta_p^2 = .66$]. Performance was better in the *1-change-only* condition (M = 5.5, SE = 1.1) than in the *6-change* condition (M = 16.7, SE = 2.6). The main effect of Group was significant [$F_{(1,11)} = 6.2$, $p = .03$, $\eta_p^2 = .36$]. The occipitotemporal group (M = 15.0, SE = 1.9) had more difficulty than the anterior temporal group (M = 7.2, SE = 2.5). The interaction between group and block was also significant [$F_{(1,11)} = 5.0$, $p = .047$, $\eta_p^2 = .31$]. Pair-wise contrasts with Bonferroni correction showed that the anterior temporal and occipitotemporal groups differed only in the *6-change* condition ($p < .03$). Conversely, the decline in performance when moving from the *1-change-only* to the *6-change* condition was significant only in the occipitotemporal group ($p < .001$).

Individual subjects showed some overlap between the scores of the occipitotemporal and anterior temporal groups. However, none of the anterior temporal group had the markedly severe inefficiencies (e.g., inverse efficiency scores of more than 15sec for the *6-change* block) seen in most of the occipitotemporal group. Conversely, while some of the anterior temporal patients had normal scores, none of the occipitotemporal group did.

b. Configural perception in both cohorts (Fig. 3B)

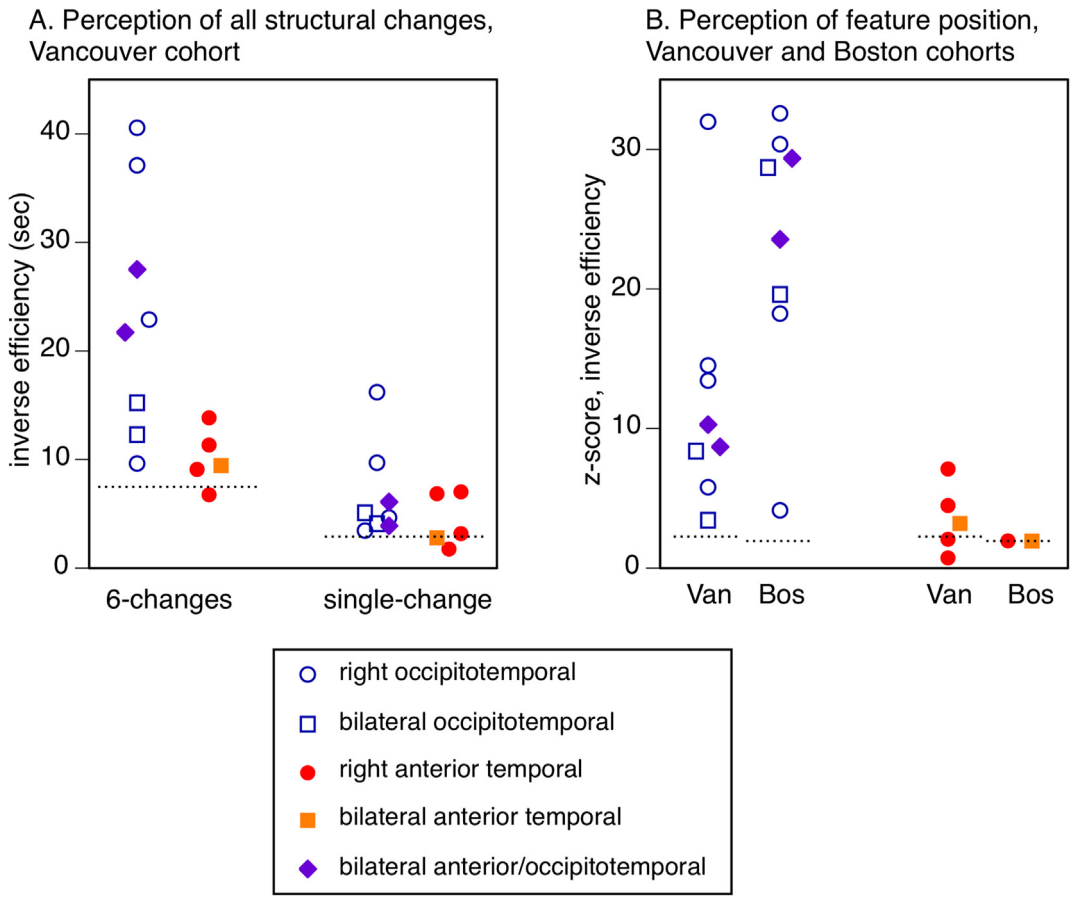

Fig. 3 – Results for shape perception. A. Combined results for inverse efficiency, across all stimuli in the *George Test* for the Vancouver cohort, comparing the 6-change (left) and the 1-change only conditions (right) in patients with and without occipitotemporal damage. B. Combined results for normalized inverse efficiency of perception of eye and mouth configuration changes in either the *AZ-test* used in the Boston cohort (Bos) or the *George test* used in the Vancouver cohort (Van). Dotted lines indicate the upper 95% prediction limits for single subject performance, obtained from controls. Note that patients with combined anterior temporal and occipitotemporal damage (purple diamonds) are grouped with those with occipitotemporal lesions alone.

The ANOVA showed a significant main effect of Group [$F_{(1,19)} = 15.2$, $p < .001$, $\eta_p^2 = .44$]. The anterior temporal group ($M = 2.7$, $SE = 3.3$) was more efficient in processing configuration than the occipitotemporal group ($M = 17.7$, $SE = 2.0$). The main effect of Cohort was not significant [$F_{(1,19)} = 1.6$, $p = .22$, $\eta_p^2 = .08$], and neither was the interaction between the two factors [$F_{(1,19)} = 2.8$, $p = .11$, $\eta_p^2 = .13$]. Again, at the individual level 13 of 16 occipitotemporal patients had abnormal scores beyond any seen in the anterior temporal group, and none had a normal score.

c. Perception of specific structural changes in subjects with occipitotemporal lesions.

With the *George test* in the Vancouver cohort, the first point is that individual scores were abnormal for most structural changes, with only a few scores for the lower face falling in the normal range (Table 3). Our analysis here asks whether there was a *relatively* greater impairment for a location or a type of change. We used normalized inverse efficiency scores to compensate for the variability in the control group's performance for each of the six different changes, and focused our analysis on the *6-change* data, since that is where the occipitotemporal and anterior temporal groups diverged (Fig. 4A). The ANOVA showed a main effect of Location [$F_{(1,7)} = 13.0$, $p = .009$, $\eta_p^2 = .651$], as performance was more efficient for lower ($M = 8.4$, $SE = 2.2$) than the upper face ($M = 20.1$, $SE = 4.1$). There was also a main effect of Change-Type [$F_{(2,14)} = 5.5$, $p = .017$, $\eta_p^2 = .442$]. Pair-wise contrasts showed that the perception of external contour was less affected than that of feature shape ($p < .04$). The interaction between the two factors was not significant [$F_{(2,14)} = 2.98$, $p = .083$, $\eta_p^2 = .299$], which indicated that all types of change had a pattern of greater difficulty in the upper face.

We also examined the effect of location in the configuration data of the occipitotemporal groups of both cohorts (Fig. 4B). The ANOVA showed a significant effect of Location [$F_{(1,14)} = 20.3$, $p < .001$, $\eta_p^2 = .59$], due to better performance with

Table 3 – Inverse efficiency for the 6-change condition of the George test, for the occipitotemporal group of the Vancouver cohort.

	Upper face			Lower face		
	Configuration	Feature shape	Contour	Configuration	Feature shape	Contour
Controls						
Mean	3.66	3.32	3.23	5.17	4.29	4.15
SD	1.53	.90	1.31	2.20	1.48	2.08
95% limit	7.29	5.46	6.34	10.39	7.79	9.10
Occipitotemporal group						
R-IOT1	18.72	6.25	7.98	**9.00**	9.90	5.95
R-IOT3	29.03	42.26	53.79	27.71	41.52	49.03
R-IOT4	83.41	46.93	19.49	30.97	20.90	20.87
L-IOT1	38.37	31.20	23.21	19.05	16.85	8.68
B-IOT1	10.70	9.21	11.47	**10.12**	18.35	13.88
B-IOT2	26.85	23.38	15.46	**8.62**	11.25	**5.85**
B-ATOT1	30.19	46.88	7.92	12.16	25.38	**7.71**
B-ATOT2	23.98	29.86	16.65	14.11	43.62	36.84
Anterior temporal group						
R-AT1	11.33	9.37	9.19	13.86	16.66	**7.57**
R-AT2	7.84	**4.14**	7.24	**8.27**	**5.41**	**7.68**
R-AT3	**4.36**	12.22	20.59	**7.39**	**6.65**	**3.30**
R-AT5	16.87	15.50	11.04	17.39	11.10	11.15
B-AT1	8.22	7.92	15.94	12.68	8.94	**2.92**

Bold = normal scores.

the mouth (M = 9.4, SE = 1.6) than with the eyes (M = 26.1, SE = 3.8). The main effect of Cohort was also significant [$F_{(1,14)}$ = 6.2, p = .026, η_p^2 = .307]: the Vancouver cohort (M = 12.0, SE = 3.2) was less impaired than the Boston one (M = 23.5, SE = 3.2). Importantly, though, the interaction between the two factors was not significant [$F_{(1,14)}$ = .6, p = .457, η_p^2 = .040], indicating that both cohorts had more trouble perceiving configural changes in the eyes than in the mouth.

2.2. *Imagery* (*Fig. 5*)

ANOVA showed a significant main effect of Group [$F_{(1,19)}$ = 6.6, p = .018, η_p^2 = .259], only now it was the occipitotemporal group that did better (M = .78, SE = .04) than the anterior temporal group (M = .64, SE = .04) (Fig. 5A). The main effect of Imagery-Type was not significant [$F_{(1,19)}$ = .2, p = .693, η_p^2 .008]: performance was similar for feature

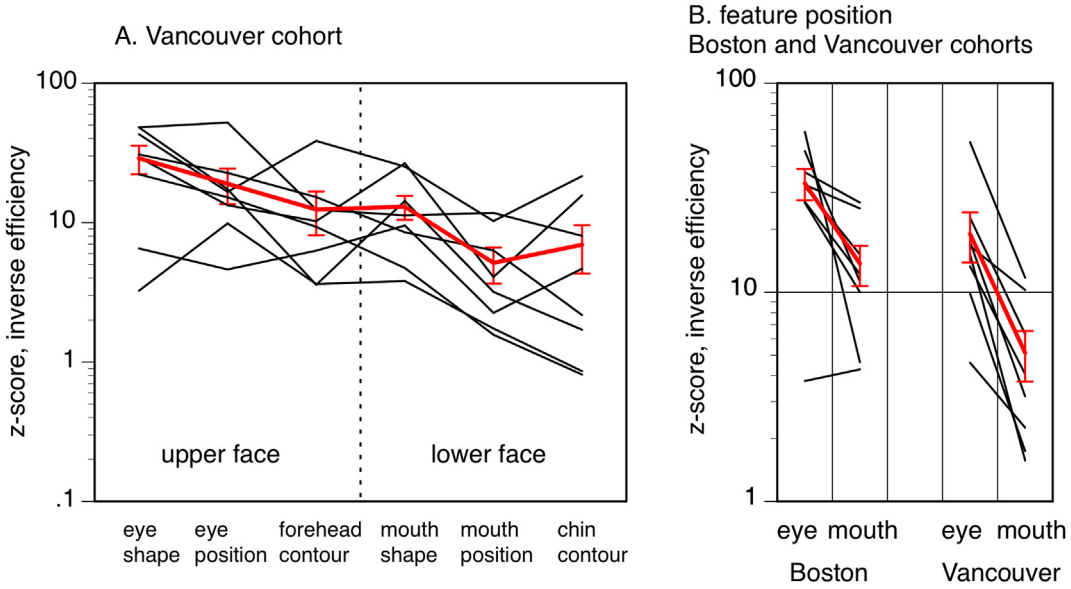

Fig. 4 – Normalized (z-scores) inverse efficiency for different types of changes, in the Occipitotemporal group. In both graphs, thin black lines are for individual subjects, with thick red line showing the mean, with error bars for 1 standard error. A. The 6-change condition in the Vancouver cohort, with upper face changes on the left side and lower face changes on the right side, showing in both cases, from left to right, the data for feature shape, feature position and external contour. Given that the upper 95% prediction limit for a single subject is 2.37, there are extreme inefficiencies—hence the logarithmic scale to the Y-axis. B. Comparison of perception of configuration of eyes versus mouth in the Boston cohort on the AZ test and the Vancouver cohort on the George test.

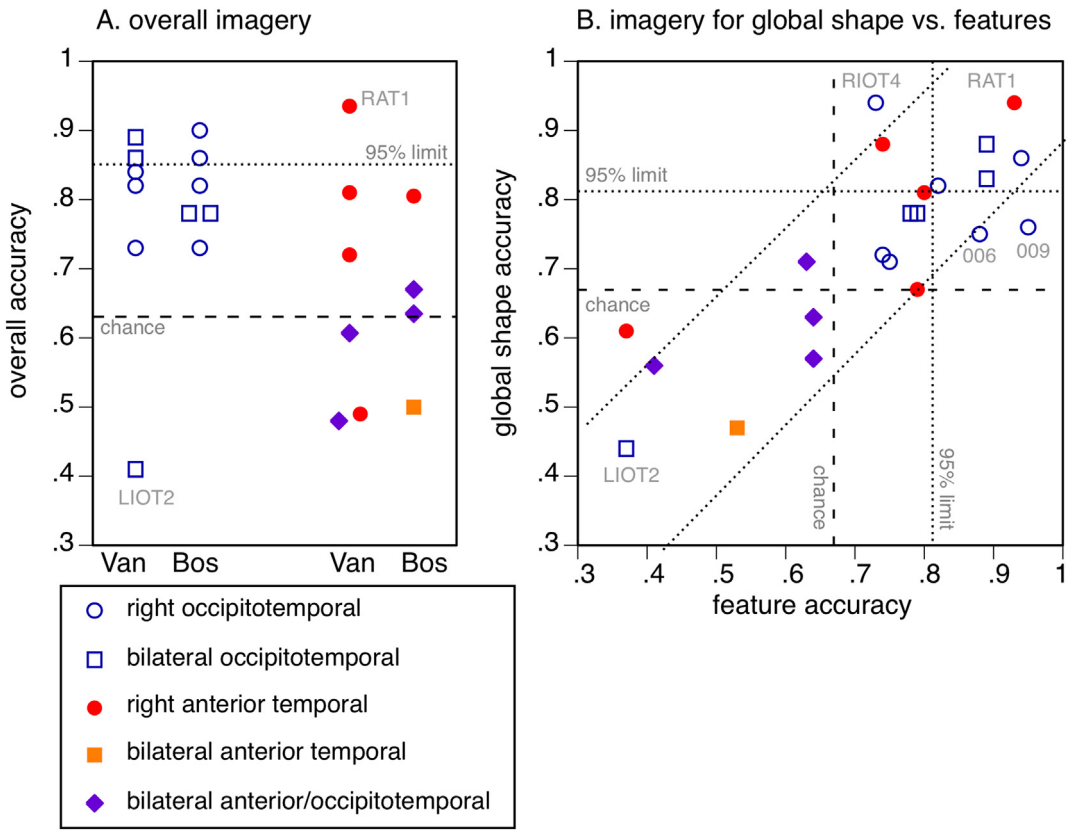

Fig. 5 – Imagery accuracy. A. Overall (combined feature and global shape) imagery for the Vancouver (Van) and Boston (Bos) cohorts, shown for those with sparing of the anterior temporal lobes (hollow symbols, left) and those with anterior temporal damage (filled symbols, right). Outlying results for LIOT2 and RAT1 are labeled. B. Imagery for global shape versus features. In both graphs the dotted lines show the lower 95% prediction limits for single subject performance, while the dashed lines show the upper limits for chance performance of .5. In B, the diagonal dotted lines indicate the 95% prediction limits for the difference between feature and global shape imagery. RIOT4, 006 and 009 meet criteria for a putative classical dissociation.

(M = .71, SE = .03) and global shape imagery (M = .72, SE = .03). The interaction between Group and Imagery-Type was not significant either [$F_{(1,19)} = 1.6$, $p = .221$, $\eta_p^2 = .078$].

This overall analysis did not show a difference between feature and global shape imagery: in fact, the two were highly correlated across the entire sample [$r = .79$, $F_{(1,21)} = 35.8$, $p < .0001$, Fig. 5B]. Nevertheless, we examined whether a difference might be found in specific types of patients. We performed two post-hoc paired *t*-tests between feature and global shape imagery scores. First, we looked at only those with anterior temporal lesions, since they have the more pronounced imagery deficits. These subjects showed if anything a small 3% advantage for global shape (mean .68, SD .15) over feature (mean .65, SD .18) imagery, which was not significant [$t_{(9)} = 1.02$ $p = .33$]. Second, we examined only those with right-sided lesions, given hypotheses that holistic face processing is lateralized to the right hemisphere (Schiltz et al., 2010). Their imagery for features (mean .79, SD .16) also did not differ from that for global shape [mean .78, SD .10, $t_{(11)} = .14$, $p = .89$].

Looking at individual subjects, there were two outliers. First, unlike the rest of the anterior temporal group, RAT1—also reported elsewhere as Florence (Rezlescu et al., 2014)—had normal imagery. Notably she had the least amount of right anterior temporal cortical damage, her causal lesion being an amygdalohippocampectomy—Fig. 6, also see Fig. 4 in (Fox et al., 2011). Second, unlike the rest of the occipitotemporal group, LIOT2 had severely impaired imagery. LIOT2's story is unusual because he became prosopagnosic after a resection of his left fusiform gyrus (Fig. 6). A meta-analysis suggests that this structure may be a key interface in the process of generating visual mental imagery (Spagna et al., 2021). LIOT2 did make the most errors on our two tests of visuospatial imagery, and so one might question if he has a broader imagery problem than just faces. However, we emphasize that he was still scoring in the normal range for visuospatial imagery, while his face imagery deficits were severe.

Another potential factor in these outliers is the nature of their lesions. Along with subject 013, LIOT2 and RAT1 differed from the others in the type of their neurologic dysfunction: before surgery all had had refractory epilepsy since their teens. One cannot exclude the possibility of anomalous cerebral re-organization of function, as has been demonstrated for

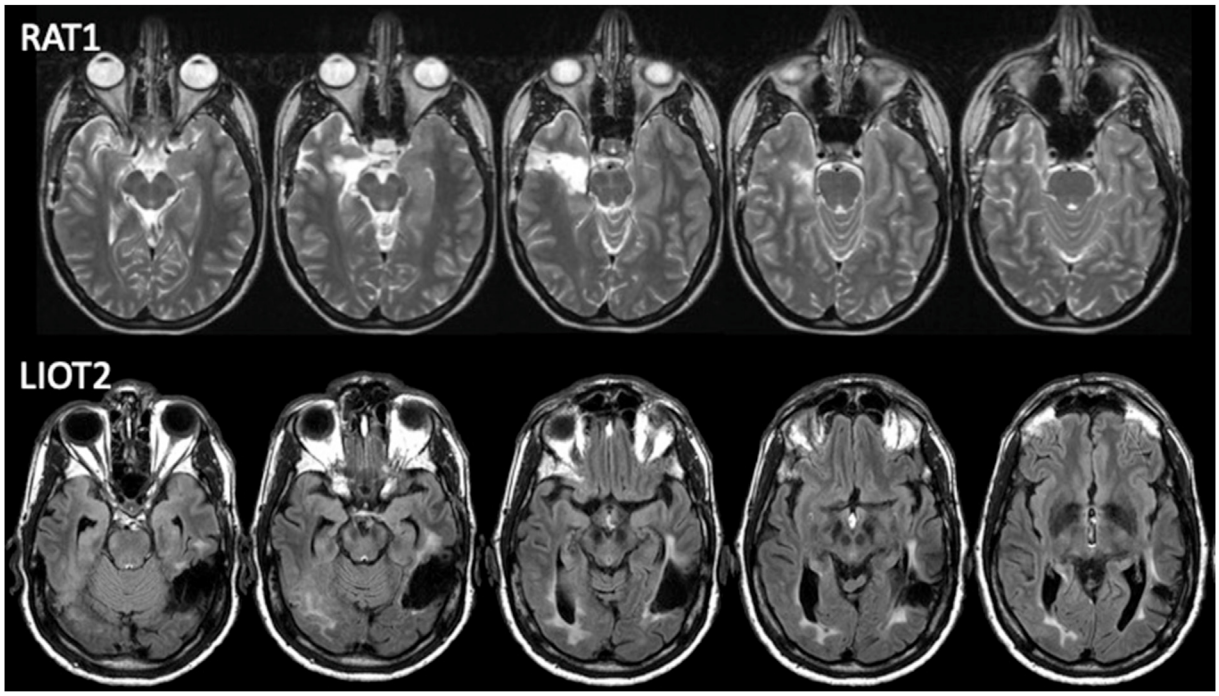

Fig. 6 – Axial MRI images of imagery outlier subjects. Top: T2-weighted images of subject RAT1, with right amygdalohippocampectomy sparing inferior aspects of anterior temporal lobe. Bottom: T1-weighted images of subject LIOT2 showing the extensive left fusiform resection in this man who also had right fusiform atrophy.

both the lateralization and localization of language networks (Hamberger et al., 2011). This may confound structure–function inferences from their data.

A second observation from the individual data is that, with the exception of RAT3, who performed at chance, subjects with right anterior temporal lesions had moderate imagery impairments that were similar to some with occipitotemporal damage. More severe impairments were seen in those with bilateral lesions, i.e., including either left occipitotemporal or left anterior temporal regions. However, bilaterality in itself is not the factor driving severe imagery impairments: two of the four with bilateral lesions confined to the occipitotemporal cortex scored normally (BIOT1 and BIOT2) and two were only mildly impaired (004 and 010) (Table 1, Fig. 5A). Rather, severe impairments in imagery required a combination of right anterior temporal damage with either a left anterior temporal lesion (BAT2) or bilateral occipitotemporal lesions (BATOT1, BATOT2, 007, 011).

Of note, BATOT1, BATOT2, and RAT3 had nearly perfect scores on our visuospatial imagery tests, and a prior study had shown normal visuospatial imagery in subject 011 (Levine et al., 1985), so in these four subjects severe face imagery deficits cannot be attributed to a loss of general imagery mechanisms.

We examined individual subjects for putative classical dissociations between feature and global imagery (Fig. 5B). Minor dissociations were seen in three subjects. Two, 006 and 009, showed the hypothesized putative classical dissociation between impaired global and spared feature imagery, but one, RIOT4, showed the opposite.

3. Discussion

3.1. *Perception*

Occipitotemporal lesions had a greater impact on the perception of facial shape than anterior temporal lesions, consistent with our prior finding that damage to the fusiform area impaired perception of facial configuration (Barton, 2008; Barton et al., 2002). The configuration results for the Vancouver cohort were consistent with those for the Boston cohort. Most occipitotemporal patients had perceptual impairments that exceeded any seen in the anterior temporal group. Thus, despite overlap between the two groups at the milder end of the spectrum, apperceptive deficits are more strongly linked to occipitotemporal lesions, with loss of the fusiform and/or occipital face areas as shown in prior functional imaging of the Vancouver cohort (Hills et al., 2015; Pancaroglu et al., 2016). These deficits were equally severe after right or bilateral lesions.

Our results also speak to the nature of this perceptual deficit. First, while the Boston cohort was studied specifically for the perception of configuration, given evidence of its role in face perception (Barton, Keenan, & Bass, 2001; Searcy et al., 1996), the data for the Vancouver cohort showed that the

perception of contour or feature shape is not spared. This is consistent with prior reports of cases of acquired prosopagnosia who were impaired in the perception of both feature shape and feature position (Bukach et al., 2006, 2008; Busigny et al., 2010; Ramon et al., 2010b). Such results support the conjecture that configuration only indexes one aspect of facial structure that is not processed well in apperceptive prosopagnosia (Barton, 2008), much as others have also concluded for developmental prosopagnosia (Russell et al., 2012; Yovel et al., 2006).

Second, these perceptual deficits are emphasized in the upper face for all three aspects of shape. Our prior analysis of accuracy in a subset of these patients showed a relative vulnerability of the eye region (Pancaroglu et al., 2016). This followed case reports of greater perceptual impairment in the eye region in other patients with occipitotemporal damage, namely PS (Caldara et al., 2005; Rossion et al., 2009; Ramon and Rossion, 2010b) and GG (Busigny et al., 2010). This has also been reported for patient LR, with right anterior temporal damage, and HH, with uncertain anatomic damage (Bukach et al., 2006, 2008). An eye processing defect is likely to have a pronounced effect on recognizing face identity, since the eyes are the most useful region for face identification (Schyns et al., 2002), are fixated the most during identification (Barton et al., 2006), and generate more signal change in the fusiform face area than other face parts (Lai et al., 2014).

Third, occipitotemporal patients in the Vancouver cohort did worse when they had to monitor for changes in many parts of the face, than when they could focus on one change alone. This is reminiscent of an observation that patients 005 and 007 could perceive configural changes to the mouth better when allowed to focus on that one change (Barton et al., 2002), though this was not seen in patients 004 or 006. Likewise, LR could only detect changes in one feature only when he had to attend to changes that could occur anywhere in the face (Bukach et al., 2006). One might infer that a problem with attending to many changes across the entire face reflects loss of holistic processing, which is said to be another quality of expert face perception (Tanaka et al., 2003; Van Belle et al., 2010). However, one can also argue that it stems from limited capacity for processing facial shape, without necessarily invoking failure to process the face 'as a whole'.

In summary, our results indicate that occipitotemporal lesions cause a more severe apperceptive deficit than anterior temporal lesions, and that this deficit involves a failure to perceive various aspects of facial shape, particularly in the eye region, and particularly when multiple aspects of the face must be processed.

3.2. *Imagery*

The results for face imagery are more nuanced. For one, there are two outlying subjects with childhood epilepsy, LIOT2 and RAT1, in whom cerebral re-organization may confound correlations of structure with function. Even so, the group analysis that included them still showed that the anterior temporal group had worse imagery than the occipitotemporal group, as predicted.

Several points are apparent from the individual data. First, apart from LIOT2, patients with lesions sparing the anterior temporal lobes did not have severe impairments of imagery. Some of these patients even had normal imagery, including two with bilateral damage (BIOT1 and BIOT2). This contrasts with their often substantial problems with perceiving facial configuration. Thus their deficit is primarily an apperceptive rather than an amnestic one. This impressive preservation of face imagery despite severe perceptual problems for faces indicates that the occipitotemporal operations for perceiving faces contribute minimally to face imagery. This also adds to the debate about the relationship between perception and imagery in general (Bartolomeo, 2002, 2008), and shows that the neural substrates for these two phenomena are not identical.

Second, right anterior temporal damage alone abolished face imagery in only one patient, RAT3. The remainder had scores similar to patients with occipitotemporal lesions. Instead, imagery was near or below the limits of chance in the four patients with right anterior temporal damage *combined* with bilateral occipitotemporal damage, and the one patient with bilateral anterior temporal damage (008/BAT2). Thus the common feature of those with severe imagery deficits was right anterior temporal damage, but in all except RAT3, there was additional damage to either the left anterior temporal lobe or bilateral occipitotemporal cortex. This is the same conclusion made tentatively on the smaller data set of the Boston cohort (Barton, 2008).

The importance of the right anterior temporal lobe for facial memories is supported by the recent description in monkeys of cells in the right temporal pole that respond specifically to familiar faces (Landi et al., 2021). However the amnestic variant of prosopagnosia may require more complex changes in the face network than just loss of this one region. This is consistent with a model of visual imagery that propose interactions in a core network that includes the fusiform gyrus, medial temporal cortex and the anterior temporal lobes, with the potential for compensatory responses to damage (Spagna, 2022). Such a model may explain the moderate deficits in imagery we found from either fusiform or anterior temporal lobe damage alone, with severe deficits only emerging with more widespread damage to the network.

While the Boston cohort was not assessed for non-face visual imagery, this is not a significant limitation because this assessment is needed mainly to put in context severe deficits in face imagery, which were present in only two Boston subjects. One of these two (008/BAT2) was subsequently tested for visuospatial imagery in Vancouver, and the other (011) was shown to have intact visuospatial imagery in a prior study (Levine et al., 1985). In our Vancouver cohort, our baseline tests for visuospatial imagery with mental rotation were done well by most members. Thus the severe face imagery deficits seen in our five subjects were not likely due to a general failure of imagery generation.

Modern neuroimaging studies suggest that it is the left fusiform cortex that is activated broadly by imagery for various stimuli—for review, see (Spagna, 2022) and a meta-analysis (Spagna et al., 2021), which we note included three studies of mental rotation. This is supported by some unusual neuropsychological cases that point to left fusiform damage as the likely cause of general impairments in imagery, as assessed by symptoms, a vividness questionnaire (Thorudottir et al., 2020)

or imagery tests for objects, animals, colours and letters (Moro et al., 2008). The current model proposes that there is a 'fusiform imagery node' on the left that acts as an interface between the frontoparietal networks that initiate imagery and the stored semantic and episodic representations in the anterior and medial temporal lobes (Spagna, 2022).

What about face imagery? A neuroimaging review noted consistent left fusiform involvement in most forms of imagery *except* for faces and colours, which show more right fusiform activation (Spagna, 2022). While some studies claim left-sided activation for both face and non-face imagery (Ishai et al., 2000; Soddu et al., 2009), others showed only or predominantly right fusiform activation for imagery of familiar faces (Boly et al., 2007; Zeman et al., 2010). Artifacts make it difficult to examine the anterior temporal cortex with fMRI: our lesion results supplement the neuroimaging data and are consistent with a key role for the right anterior temporal lobe in visual semantic representations of faces that support both imagery and recognition.

One observation suggested in the Boston cohort (Barton, 2008) that was not borne out in this larger analysis was a selective loss of facial imagery for global shape. A specific imagery impairment for global face shape was reported for HJA, though this person had an integrative agnosia rather than prosopagnosia (Young et al., 1994). Looking at our entire group of 23 patients we did not find a selective defect, and a subset analyses also showed that this did not occur in those with anterior temporal lesions, or those with right-sided lesions. While the numbers involved in these secondary analyses are small, the feature/global differences do not approach significance. At the individual level there were a few patients with putative classical dissociations, but this could be for either global shape or feature imagery. Overall, there was a strong correlation between global shape and feature imagery. This has parallels in developmental prosopagnosia, where imagery for both global judgments and features were reduced together in a series of four subjects (Tree et al., 2010).

What should we make of subjects RAT1, RAT2, RAT5, and 013, who had only modest or no imagery deficits, similar to those of some members of the occipitotemporal group? Unlike many of the latter, these three had either a mild (RAT1, RAT5) or no perceptual deficit (RAT2, 013), as shown in Table 2. Perhaps these patients come closest to meeting the definition of associative prosopagnosia: evidence of intact or at least relatively well preserved perception AND imagery, with an inference that the failure of face recognition represents a problem linking the two (Fox et al., 2008). Firmer diagnosis of an associative variant awaits a behavioural means of demonstrating dissociation. Neuroimaging markers such as abnormalities in the inferior longitudinal fasciculus (Morioka et al., 2024; Thomas et al., 2009) may provide a structural correlate but they do not prove dissociation as the functional mechanism, and the role of damage to white matter tracts may be hard to interpret in the presence of substantial cortical loss. Among our patients the most plausible structural case for an associative mechanism may be RAT1, whose surgical resection did not remove either the inferior cortex of the right anterior temporal lobe or the fusiform face area (Fig. 6).

3.3. *Variants in acquired prosopagnosia in the literature*

In the thirty years since the initial articles (Damasio et al., 1990; de Renzi et al., 1991; Young et al., 1994), other cases of acquired prosopagnosia have been labeled as apperceptive or associative, or described as having features consistent with those terms. The evidence used to make that distinction has varied, however (Table 4).

This is apparent in cases of reported apperceptive prosopagnosia. One case with right temporo-occipital atrophy was

Table 4 – Published cases of acquired prosopagnosia with potential classification.

Publication	Patient	Lesion	Etiology	Perceptual test	Other face perceptual tests	Imagery probe
Apperceptive						
Whiteley (1977)	LH729447	RIOT	Hemorrhage	**Unfamiliar face matching**	–	Verbal description
de Renzi (1991)	GD	RT+	Trauma/abscess	**BFRT 14/27**	**Age**	–
Bartolomeo (1998)	MmeD	BIOT	Hemorrhage	**Facial feature matching**	**Age, gender**	Questionnaire
Michelon (2003)	MJH	BIOT	Trauma	**Unfamiliar face matching**	Expression, age, gender	Questionnaire
Sugimoto (2012)	58M	BIOT	Atrophy	**Unfamiliar face matching**	**Expression, age, gender**	–
Jansari (2015)	DY	RIOT	Hemorrhage	**BFRT41/54 (slow)**	Expression, mooney	Questionnaire, recall
Apperceptive/amnestic						
Levine 1985, 1989	LH	BATOT	Trauma	**BFRT 33/54**	–	**Verbal description**
Young (1994)	PH	BIOT	Trauma	**BFRT 37/54**	–	**Questionnaire**
Non-apperceptive						
de Renzi (1991)	VA	RT?	Infection	BFRT 21/27	Age	–
Tábuas-Pereira et al., 2016	54F	BAT	Infection	BFRT 46/54	Expression	–
Papagno (2021)	56M	LIOT	Stroke	BFRT 44/54	Expression	–
Morioka (2024)	76M	R-ILF	Hemorrhage	–	Expression, gender	–

RIOT, BIOT = right, bilateral occipitotempora; BATOT = bilateral occipito- and anterior temporal, BAT = bilateral anterior temporal.
RT+ = right temporal lobe plus prior trauma, RT? = Right temporal lobe not further defined.
R-ILF = right inferior longitudinal fasciculus.
BFRT = Benton Face recognition test, with score.
Bold = impaired.

diagnosed as apperceptive on the basis of impaired unfamiliar face matching (Sugimoto et al., 2012), along with observations of impaired perception of facial expression, sex and age, which we consider less relevant. In another (Kesserwani et al., 2020) apperception was diagnosed anecdotally from poor face 'identification', by which they seem to mean knowing that a face is a face, which is also not relevant. Another may have indirectly inferred apperception from the fact that the lesion was in the right fusiform gyrus (Koh, 2022).

Some studies have provided more detailed data on both perception and imagery. Most have examined cases with occipitotemporal damage, and most of their findings support ours, that these lesions are associated with poor face perception but relatively intact face imagery. After a right occipitotemporal hemorrhage, LH 729447 had poor face matching but could still describe faces from memory (Whiteley et al., 1977). DY could not match faces after a right occipitotemporal hemorrhage but showed good face imagery on a questionnaire and free recall (Jansari et al., 2015). After bilateral occipitotemporal lesions Mme D could not match facial features or judge gender or age, but did well on a face-imagery questionnaire (Bartolomeo et al., 1998). At age 5 MJH had traumatic bilateral occipital lesions that included the right fusiform gyrus. He was impaired at face matching but his face imagery was in the low normal range (Michelon et al., 2003). These subjects with right or bilateral occipitotemporal lesions all fit the proposed profile for apperceptive prosopagnosia, of impaired face perception with good face imagery.

Two subjects showed a second, different pattern, with impairments in both face perception and face imagery. This is a profile that we found in patients with a combination of bilateral occipitotemporal and right anterior temporal damage. PH had both face-matching impairments and poor face imagery on a questionnaire (de Haan et al., 1991; Young et al., 1994). His traumatic lesions—which were not shown—were described as bilateral occipitotemporal (de Haan et al., 1991), but could have been more extensive, given the often diffuse nature of trauma and the limitation of MRI in the early 1990s. LH had a similar pattern of both impaired face matching and problems with face imagery (Levine et al., 1985, 1989), with loss of his face memories also inferred from the absence of covert face processing (Etcoff et al., 1991). We confirm LH's loss of face memories more directly here, as he is our subject 011. The prior findings are thus consistent with ours, but LH does not belong to the bilateral occipitotemporal group: rather, he is one of the four with bilateral occipitotemporal *and* right anterior temporal damage. Thus a conclusion that the right anterior temporal lobe plays a key role in the loss of facial memories can explain some well-known discrepancies in the literature, such as why of two prosopagnosic patients with bilateral occipitotemporal lesions, Mme D had intact imagery (Bartolomeo et al., 1998) while LH did not (Levine et al., 1985): the imaging shows that LH's lesion extended to the right anterior temporal lobe (Fig. 1A), while Mme D's did not.

Associative variants have often been inferred solely through exclusion of an apperceptive defect, usually by normal matching of unfamiliar faces. This was the case in one patient with a left occipitotemporal stroke (Papagno et al., 2021) and another with bilateral anterior temporal damage (Tábuas-Pereira et al., 2016). Less satisfactorily it was based on intact perception of gender and expression in a patient with damage to the inferior longitudinal fasciculus (Morioka et al., 2024). Without probing face memories, though, these do not distinguish between amnestic and associative variants. Others have rightly suggested that it is more accurate to call such cases 'non-apperceptive' (Biotti et al., 2016).

4. Conclusion

As previously remarked (Barton, 2008; Davies-Thompson et al., 2014), the distinction between apperceptive, associative and amnestic forms of prosopagnosia is likely relative rather than absolute—just as stated for visual agnosia in general (Damasio et al., 1990; Lissauer, 1890): the former commented that perceptual and mnemonic processes in face processing could not be 'rigidly compartmentalized' anatomically but existed on a continuum (p.92). Theories about imagery in general have proposed that perception and imagery share some of the same structures in their processing (Farah, 1989; Kosslyn, 1988). Nevertheless, dissociations between imagery and perception do occur (Bartolomeo, 2002). Our results show that some of our occipitotemporal group with significant perceptual difficulties had modest impairments of imagery, while some of the anterior temporal group with severe impairments of imagery had modest perceptual impairments. In line with the latter, some prior studies of prosopagnosic patients with anterior temporal damage have reported perceptual deficits also. LR with a right lesion had reduced whole-part advantages and composite face effects, suggesting a reduction in holistic processing similar to that seen in a patient with bilateral occipitotemporal damage (Busigny et al., 2014). As reported here, 008/BAT2 had mild problems on the configural components of the *AZ-test*, as well as a subtler problem with integrating combinations of changes in both the eye and mouth (Barton et al., 2003b).

Our work supports the proposal that acquired prosopagnosia is not a unitary disorder but has distinct variants. Occipitotemporal lesions caused an apperceptive impairment in processing facial shape, especially in the eye region, with either modest or no impairment in face imagery, and this was true regardless of whether the lesion was right or bilateral. An amnestic deficit, characterized by near-chance performance on face imagery, was seen with right anterior temporal lesions, but often required combination with left anterior temporal or bilateral occipitotemporal damage. Of these subjects with poor imagery, two, one with right and one with bilateral anterior temporal lesions, met the criteria of an amnestic variant, with severe imagery deficits and relatively spared perception of configuration. Another four with right anterior temporal and bilateral occipitotemporal lesions had combined amnestic and amnestic deficits, a possibility first envisioned by (de Renzi, 1986). Last, some patients with right anterior temporal lesions alone may approach the definition of an associative variant,

with intact or relatively preserved perception and imagery, but this remains an uncertain diagnosis of exclusion.

CRediT authorship contribution statement

Jason J.S. Barton: Writing – original draft, Supervision, Methodology, Funding acquisition, Data curation, Conceptualization. **Brad Duchaine:** Resources, Methodology, Data curation. **Andrea Albonico:** Writing – review & editing, Project administration, Formal analysis.

Co-author contributions

BD : Project administration, Resources, Writing – review and editing.

AA : Project administration, Formal analysis, Writing – review and editing.

Data for this study are available at: https://osf.io/yc5ea/. As the collection of these data began 20 years ago, no part of the study procedures or analyses were pre-registered prior to the research being conducted. The perceptual tests used were designed 20 years ago (Malcolm et al., 2005) and reported again 8 years ago (Pancaroglu et al., 2016). These involved photographs of people and permission for public storage had not been obtained. The imagery questionnaire was designed and reported in 2003 (Barton et al., 2003a) and is available at: https://osf.io/yc5ea/.

Acknowledgements

JB was supported by a Canada Research Chairs 950-202111, 950-228984, and 950-232752, and the Marianne Koerner Chair In Brain Diseases. This work was supported by NIMH grant 1R01 MH069898 and CIHR grants MOP-77615, MOP-85004, and MOP-102567. We thank all the trainees involved in evaluating these patients over the years, in particular M Cherkasova, R Hefter, G Iaria, C Fox, C Hills and R Pancaroglu.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cortex.2024.11.011.

REFERENCES

Albert, M., Butters, N., & Levin, J. (1979). Temporal gradients in retrograde amnesia of patients with alcoholic korsakoff's disease. *Archives of Neurology, 36*, 211–216. https://doi.org/10.1001/archneur.1979.00500400065010

Avidan, G., Tanzer, M., & Behrmann, M. (2011). Impaired holistic processing in congenital prosopagnosia. *Neuropsychologia, 49*(9), 2541–2552. https://doi.org/10.1016/j.neuropsychologia.2011.05.002

Bartolomeo, P. (2002). The relationship between visual perception and visual mental imagery: A reappraisal of the neuropsychological evidence. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 38*(3), 357–378. https://doi.org/10.1016/s0010-9452(08)70665-8

Bartolomeo, P. (2008). The neural correlates of visual mental imagery: An ongoing debate. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 44*, 107–108. https://doi.org/10.1016/j.cortex.2006.07.001

Bartolomeo, P., Bachoud-Levi, A. C., De Gelder, B., Denes, G., Dalla Barba, G., Brugieres, P., & Degos, J. D. (1998). Multiple-domain dissociation between impaired visual perception and preserved mental imagery in a patient with bilateral extrastriate lesions. *Neuropsychologia, 36*(3), 239–249. https://doi.org/10.1016/s0028-3932(97)00103-6

Barton, J. J. S. (2008). Structure and function in acquired prosopagnosia: Lessons from a series of 10 patients with brain damage. *Journal of Neuropsychology, 2*(1), 197–225. https://doi.org/10.1348/174866407x214172

Barton, J. J. S., Albonico, A., Susilo, T., Duchaine, B., & Corrow, S. L. (2019). Object recognition in acquired and developmental prosopagnosia. *Cognitive Neuropsychology*, 1–31. https://doi.org/10.1080/02643294.2019.1593821

Barton, J., & Cherkasova, M. (2003a). Face imagery and its relation to perception and covert recognition in prosopagnosia. *Neurology, 61*, 220–225. https://doi.org/10.1212/01.wnl.0000071229.11658.f8

Barton, J., Cherkasova, M., & O'Connor, M. (2001). Covert recognition in acquired and developmental prosopagnosia. *Neurology, 57*, 1161–1167. https://doi.org/10.1212/wnl.57.7.1161

Barton, J. J., Keenan, J. P., & Bass, T. (2001). Discrimination of spatial relations and features in faces: Effects of inversion and viewing duration. *British Journal of Psychology, 92*(Pt 3), 527–549.

Barton, J. J., Press, D. Z., Keenan, J. P., & O'Connor, M. (2002). Lesions of the fusiform face area impair perception of facial configuration in prosopagnosia. *Neurology, 58*(1), 71–78. https://doi.org/10.1212/wnl.58.1.71

Barton, J. J., Radcliffe, N., Cherkasova, M. V., Edelman, J., & Intriligator, J. M. (2006). Information processing during face recognition: The effects of familiarity, inversion, and morphing on scanning fixations. *Perception, 35*(8), 1089–1105. https://doi.org/10.1068/p5547

Barton, J., Zhao, J., & Keenan, J. (2003b). Perception of global facial geometry in the inversion effect and prosopagnosia. *Neuropsychologia, 41*, 1703–1711. https://doi.org/10.1016/s0028-3932(03)00115-5

Benton, A., & van Allen, M. (1972). Prosopagnosia and facial discrimination. *Journal of the Neurological Sciences, 15*, 167–172.

Biotti, F., & Cook, R. (2016). Impaired perception of facial emotion in developmental prosopagnosia. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 81*, 126–136. https://doi.org/10.1016/j.cortex.2016.04.008

Biotti, F., Wu, E., Yang, H., Jiahui, G., Duchaine, B., & Cook, R. (2017). Normal composite face effects in developmental prosopagnosia. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 95*, 63–76. https://doi.org/10.1016/j.cortex.2017.07.018

Bodamer, J. (1947). Die prosop-agnosie. (die agnosie des physiognomieerkennens.). *Archives of Psychiatric Nervenkr Z Gesamtgebiete der Neurologie und Psychiatrie, 118*(1–2), 6–53. https://doi.org/10.1007/BF00352849

Bodamer, J. (1949). Über eine besondere form der optischen agnosie (physiognomieerkennungstörungen bei okzipitalhirnläslonen). *Deutsche Medizinische Wochenschrift, 74*(2), 48–50. https://doi.org/10.1055/s-0028-1118267

Boly, M., Coleman, M. R., Davis, M. H., Hampshire, A., Bor, D., Moonen, G., Maquet, P., Pickard, J. D., Laureys, S., & Owen, A. M. (2007). When thoughts become action: An fmri

paradigm to study volitional brain activity in non-communicative brain injured patients. *Neuroimage, 36*(3), 979–992. https://doi.org/10.1016/j.neuroimage.2007.02.047

Bukach, C. M., Bub, D. N., Gauthier, I., & Tarr, M. J. (2006). Perceptual expertise effects are not all or none: Spatially limited perceptual expertise for faces in a case of prosopagnosia. *Journal of Cognitive Neuroscience, 18*(1), 48–63. https://doi.org/10.1162/089892906775250094

Bukach, C. M., Le Grand, R., Kaiser, M. D., Bub, D. N., & Tanaka, J. W. (2008). Preservation of mouth region processing in two cases of prosopagnosia. *Journal of Neuropsychology, 2*(Pt 1), 227–244. https://doi.org/10.1348/174866407x231010

Busigny, T., Joubert, S., Felician, O., Ceccaldi, M., & Rossion, B. (2010). Holistic perception of the individual face is specific and necessary: Evidence from an extensive case study of acquired prosopagnosia. *Neuropsychologia, 48*, 4057–4092. https://doi.org/10.1016/j.neuropsychologia.2010.09.017

Busigny, T., & Rossion, B. (2011). Holistic processing impairment can be restricted to faces in acquired prosopagnosia: Evidence from the global/local navon effect. *Journal of Neuropsychology, 5*, 1–14. https://doi.org/10.1348/174866410X500116

Busigny, T., Van Belle, G., Jemel, B., Hosein, A., Joubert, S., & Rossion, B. (2014). Face-specific impairment in holistic perception following focal lesion of the right anterior temporal lobe. *Neuropsychologia*. https://doi.org/10.1016/j.neuropsychologia.2014.01.018

Caldara, R., Schyns, P., Mayer, E., Smith, M., Gosselin, F., & Rossion, B. (2005). Does prosopagnosia take the eyes out of face representations? Evidence for a defect in representing diagnostic facial information following brain damage. *Journal of Cognitive Neuroscience, 17*(10), 1652–1666. https://doi.org/10.1162/089892905774597254

Corrow, J. C., Corrow, S. L., Lee, E., Pancaroglu, R., Burles, F., Duchaine, B., Iaria, G., & Barton, J. J. (2016). Getting lost: Topographic skills in acquired and developmental prosopagnosia. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 76*, 89–103. https://doi.org/10.1016/j.cortex.2016.01.003

Damasio, A. R., Tranel, D., & Damasio, H. (1990). Face agnosia and the neural substrates of memory. *Annual Review of Neuroscience, 13*, 89–109. https://doi.org/10.1146/annurev.ne.13.030190.000513

Davies-Thompson, J., Fletcher, K., Hills, C., Pancaroglu, R., Corrow, S. L., & Barton, J. J. (2017). Perceptual learning of faces: A rehabilitative study of acquired prosopagnosia. *Journal of Cognitive Neuroscience, 29*(3), 573–591. https://doi.org/10.1162/jocn_a_01063

Davies-Thompson, J., Pancaroglu, R., & Barton, J. (2014). Acquired prosopagnosia: Structural basis and processing impairments. *Frontiers in Bioscience (Elite Ed), 6*, 159–174. https://doi.org/10.2741/e699

de Haan, E., Young, A., & Newcombe, F. (1991). Covert and overt recognition in prosopagnosia. *Brain: a Journal of Neurology, 114*, 2575–2591. https://doi.org/10.1093/brain/114.6.2575

de Renzi, E. (1986). Current issues in prosopagnosia. In H. D. J. M. Ellis, F. Newcombe, & A. Young (Eds.), *Aspects of face processing* (pp. 243–252). Dordecht: Martinus Nijhoff.

de Renzi, E., Faglioni, P., Grossi, D., & Nichelli, P. (1991). Apperceptive and associative forms of prosopagnosia. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 27*, 213–221. https://doi.org/10.1016/s0010-9452(13)80125-6

Dietz, C. D., Malaspina, M., Albonico, A., & Barton, J. J. S. (2022). The persistence of remote visual semantic memory following ocular blindness. *Neuropsychologia, 165*, Article 108110. https://doi.org/10.1016/j.neuropsychologia.2021.108110

Duchaine, B., Germine, L., & Nakayama, K. (2007). Family resemblance: Ten family members with prosopagnosia and within-class object agnosia. *Cognitive Neuropsychology, 24*(4), 419–430. https://doi.org/10.1080/02643290701380491

Duchaine, B., & Nakayama, K. (2006). The cambridge face memory test: Results for neurologically intact individuals and an investigation of its validity using inverted face stimuli and prosopagnosic participants. *Neuropsychologia, 44*(4), 576–585. https://doi.org/10.1016/j.neuropsychologia.2005.07.001

Ellis, H. D., & Florence, M. (1990). Bodamer's (1947) paper on prosopagnosia. *Cognitive Neuropsychology, 7*(2), 81–105. https://doi.org/10.1080/02643299008253437

Etcoff, N. L., Freeman, R., & Cave, K. R. (1991). Can we lose memories for faces? Content specificity and awareness in a prosopagnosic. *Journal of Cognitive Neuroscience, 3*(1), 25–41. https://doi.org/10.1162/jocn.1991.3.1.25

Farah, M. J. (1989). The neural basis of mental imagery. *Trends in Neurosciences, 12*(10), 395–399. https://doi.org/10.1016/0166-2236(89)90079-9

Farah, M., Levinson, K., & Klein, K. (1995). Face perception and within-category discrimination in prosopagnosia. *Neuropsychologia, 33*, 661–674. https://doi.org/10.1016/0028-3932(95)00002-k

Farah, M., McMullen, P., & Meyer, M. (1991). Can recognition of living things be selectively impaired. *Neuropsychologia, 29*, 185–193. https://doi.org/10.1016/0028-3932(91)90020-9

Finzi, R. D., Susilo, T., Barton, J. J. S., & Duchaine, B. C. (2016). The role of holistic face processing in acquired prosopagnosia: Evidence from the composite face effect. *Visual Cognition, 24*(4), 304–320. https://doi.org/10.1080/13506285.2016.1261976

Fox, C. J., Hanif, H. M., Iaria, G., Duchaine, B. C., & Barton, J. J. (2011). Perceptual and anatomic patterns of selective deficits in facial identity and expression processing. *Neuropsychologia, 49*(12), 3188–3200. https://doi.org/10.1016/j.neuropsychologia.2011.07.018

Fox, C. J., Iaria, G., & Barton, J. J. (2008). Disconnection in prosopagnosia and face processing. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 44*(8), 996–1009. https://doi.org/10.1016/j.cortex.2008.04.003

Fox, C. J., Iaria, G., & Barton, J. J. S. (2009). Defining the face processing network: Optimization of the functional localizer in fmri. *Human Brain Mapping, 30*, 1637–1651. https://doi.org/10.1002/hbm.20630

Gainotti, G. (2010). Not all patients labeled as "prosopagnosia" have a real prosopagnosia. *Journal of Clinical and Experimental Neuropsychology, 32*(7), 763–766. https://doi.org/10.1080/13803390903512686

Gainotti, G. (2018). The anterior temporal lobes: New frontiers opened to neuropsychological research by changes in health care and disease epidemiology. *The Journal of Neuropsychiatry and Clinical Neurosciences, 30*(1), 22–30. https://doi.org/10.1176/appi.neuropsych.17010002

Gerlach, C., Lissau, C. H., & Hildebrandt, N. K. (2018). On defining and interpreting dissociations. *Cognitive Neuropsychology, 35*(1–2), 66–69. https://doi.org/10.1080/02643294.2017.1414692

Geskin, J., & Behrmann, M. (2018). Congenital prosopagnosia without object agnosia? A literature review. *Cognitive Neuropsychology, 35*(1–2), 4–54. https://doi.org/10.1080/02643294.2017.1392295

Grüter, T., Grüter, M., Bell, V., & Carbon, C. C. (2009). Visual mental imagery in congenital prosopagnosia. *Neuroscience Letters, 453*(3), 135–140. https://doi.org/10.1016/j.neulet.2009.02.021

Grossi, D. (1991). *La riabilitazione dei disordini della cognizione spaziale. Secondary Titl*. Milano: Masson.

Hécaen, H., Angelergues, R., Bernhardt, C., & Chiarelli, J. (1957). Essai de distinction des modalités cliniques de l'agnosie des physionomies. *Revue Neurologique, 96*, 125–144.

Hamberger, M. J., & Cole, J. (2011). Language organization and reorganization in epilepsy. *Neuropsychology Review, 21*(3), 240–251. https://doi.org/10.1007/s11065-011-9180-z

Hills, C. S., Pancaroglu, R., Duchaine, B., & Barton, J. J. (2015). Word and text processing in acquired prosopagnosia. *Annals of Neurology, 78*(2), 258–271. https://doi.org/10.1002/ana.24437

Ishai, A., Ungerleider, L. G., & Haxby, J. V. (2000). Distributed neural systems for the generation of visual images. *Neuron, 28*(3), 979–990. https://doi.org/10.1016/s0896-6273(00)00168-9

Jansari, A., Miller, S., Pearce, L., Cobb, S., Sagiv, N., Williams, A. L., Tree, J. J., & Hanley, J. R. (2015). The man who mistook his neuropsychologist for a popstar: When configural processing fails in acquired prosopagnosia. *Frontiers in Human Neuroscience, 9*, 390. https://doi.org/10.3389/fnhum.2015.00390

Jenkins, R., Dowsett, A. J., & Burton, A. M. (2018). How many faces do people know? *Progress in Biological Sciences, 285*(1888). https://doi.org/10.1098/rspb.2018.1319

Kesserwani, H., & Kesserwani, A. (2020). Apperceptive prosopagnosia secondary to an ischemic infarct of the lingual gyrus: A case report and an update on the neuroanatomy, neurophysiology, and phenomenology of prosopagnosia. *Cureus, 12*(10), Article e11272. https://doi.org/10.7759/cureus.11272

Koh, Y.-H. (2022). Right fusiform gyrus infarct with acute prosopagnosia. *Acta Neurologica Taiwanica, 31*(4), 186–187.

Kosslyn, S. M. (1988). Aspects of a cognitive neuroscience of mental imagery. *Science, 240*(4859), 1621–1626. https://doi.org/10.1126/science.3289115

Lai, M., Oruc, I., & Barton, J. J. (2013). The role of skin texture and facial shape in representations of age and identity. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 49*(1), 252–265. https://doi.org/10.1016/j.cortex.2011.09.010

Lai, J., Pancaroglu, R., Oruc, I., Barton, J. J., & Davies-Thompson, J. (2014). Neuroanatomic correlates of the feature-salience hierarchy in face processing: An fmri -adaptation study. *Neuropsychologia, 53*, 274–283. https://doi.org/10.1016/j.neuropsychologia.2013.10.016

Landi, S. M., Viswanathan, P., Serene, S., & Freiwald, W. A. (2021). A fast link between face perception and memory in the temporal pole. *Science, 373*(6554), 581–585. https://doi.org/10.1126/science.abi6671

Levine, D., & Calvanio, R. (1989). Prosopagnosia: A defect in visual configural processing. *Brain and Cognition, 10*, 149–170. https://doi.org/10.1016/0278-2626(89)90051-1

Levine, D., Warach, J., & Farah, M. (1985). Two visual systems in mental imagery: Dissociation of "what" and "where" in imagery disorders due to bilateral posterior cerebral lesions. *Neurology, 35*, 1010–1018. https://doi.org/10.1212/wnl.35.7.1010

Lhermitte, F., & Pillon, B. (1975). La prosopagnosie. Role de l'hémisphère droit dans la perception visuelle. (apropos d'un cas consécutif à une lobectomie occipitale droite). *Revue Neurologique, 131*(11), 791–812.

Lissauer, H. (1890). Einfall von seelenblindheit nebst einem bintrag zur theorie derselben. *Archiv fuer Psychiatrie und Nervenkrankheiten, 2*(22).

Malcolm, G., Leung, C., & Barton, J. (2005). Regional variation in the inversion effect for faces: Different patterns for feature shape, spatial relations, and external contour. *Perception, 33*, 1221–1231. https://doi.org/10.1068/p5372

Michelon, P., & Biederman, I. (2003). Less impairment in face imagery than face perception in early prosopagnosia. *Neuropsychologia, 41*(4), 421–441. https://doi.org/10.1016/s0028-3932(02)00176-8

Money, J., Alexander, D., & Walker, H. T. (1965). *A standardized road map test of direction sense*. Baltimore: The Johns Hopkins press.

Morganti, F. (2018). Enacting space in virtual reality: A comparison between money's road map test and its virtual version. *Front Psychol, 9*, 2410. https://doi.org/10.3389/fpsyg.2018.02410

Morioka, H., Yanagihashi, M., Toda, M., Ikeda, T., Mizuno, M., Hori, M., Kamiya, K., & Kano, O. (2024). Isolated prosopagnosia caused by damage to the right inferior longitudinal fasciculus: A case report. *Journal of Nippon Medical School*. https://doi.org/10.1272/jnms.JNMS.2025_92-202. in press.

Moro, V., Berlucchi, G., Lerch, J., Tomaiuolo, F., & Aglioti, S. M. (2008). Selective deficit of mental visual imagery with intact primary visual cortex and visual perception. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 44*(2), 109–118. https://doi.org/10.1016/j.cortex.2006.06.004

Moroz, D., Corrow, S. L., Corrow, J. C., Barton, A. R., Duchaine, B., & Barton, J. J. (2016). Localization and patterns of cerebral dyschromatopsia: A study of subjects with prospagnosia. *Neuropsychologia*. https://doi.org/10.1016/j.neuropsychologia.2016.06.012

Ogden, J. A. (1993). Visual object agnosia, prosopagnosia, achromatopsia, loss of visual imagery, and autobiographical amnesia following recovery from cortical blindness: Case m.H. *Neuropsychologia, 31*(6), 571–589. https://doi.org/10.1016/0028-3932(93)90053-3

Palermo, R., Willis, M. L., Rivolta, D., McKone, E., Wilson, C. E., & Calder, A. J. (2011). Impaired holistic coding of facial expression and facial identity in congenital prosopagnosia. *Neuropsychologia, 49*, 1226-–1235. https://doi.org/10.1016/j.neuropsychologia.2011.02.021

Pallis, C. A. (1955). Impaired identification of faces and places with agnosia for colours: Report of a case due to cerebral embolism. *Journal of Neurology Neurosurgery and Psychiatry, 18*, 218–224. https://doi.org/10.1136/jnnp.18.3.218

Pancaroglu, R., Hills, C. S., Sekunova, A., Viswanathan, J., Duchaine, B., & Barton, J. J. (2016). Seeing the eyes in acquired prosopagnosia. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 81*, 251–265. https://doi.org/10.1016/j.cortex.2016.04.024

Papagno, C., Barvas, E., Tettamanti, M., & Gainotti, G. (2021). Selective defects of face familiarity associated to a left temporo-occipital lesion. *Neurological Sciences: Official Journal of the Italian Neurological Society and of the Italian Society of Clinical Neurophysiology, 42*(2), 613–623. https://doi.org/10.1007/s10072-020-04581-5

Pichler, P., Dosani, M., Oruc, I., & Barton, J. J. (2011). The nature of upright and inverted face representations: An adaptation-transfer study of configuration. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior*. https://doi.org/10.1016/j.cortex.2011.02.005

Ramon, M., Busigny, T., & Rossion, B. (2010a). Impaired holistic processing of unfamiliar individual faces in acquired prosopagnosia. *Neuropsychologia, 48*(4), 933–944. https://doi.org/10.1016/j.neuropsychologia.2009.11.014

Ramon, M., & Rossion, B. (2010b). Impaired processing of relative distances between features and of the eye region in acquired prosopagnosia–two sides of the same holistic coin? *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior, 46*(3), 374–389. https://doi.org/10.1016/j.cortex.2009.06.001

Rezlescu, C., Barton, J. J. S., Pitcher, D., & Duchaine, B. (2014). Normal acquisition of expertise with greebles in two cases of acquired prosopagnosia. *Proceedings of the National Academy of Sciences of the United States of America, 111*(14), 5123-–5128.

Riddoch, M., & Humphreys, G. (1987). A case of integrative visual agnosia. *Brain: a Journal of Neurology, 110*, 1431–1462. https://doi.org/10.1093/brain/110.6.1431

Rondot, P., Tzavaras, A., & Garcin, R. (1967). Sur un cas de prosopagnosie persistant depuis quinze ans. *Revue Neurologique, 117*(3), 424–428.

Rossion, B., Kaiser, M. D., Bub, D., & Tanaka, J. W. (2009). Is the loss of diagnosticity of the eye region of the face a common aspect of acquired prosopagnosia? *Journal of Neuropsychology, 3*(Pt 1), 69–78. https://doi.org/10.1348/174866408X289944

Russell, R., Chatterjee, G., & Nakayama, K. (2012). Developmental prosopagnosia and super-recognition: No special role for surface reflectance processing. *Neuropsychologia, 50*(2),

334–340. https://doi.org/10.1016/j.neuropsychologia.2011.12.004
Schiltz, C., Dricot, L., Goebel, R., & Rossion, B. (2010). Holistic perception of individual faces in the right middle fusiform gyrus as evidenced by the composite face illusion. *Journal of Vision, 10*(2), 25 1–2516. https://doi.org/10.1167/10.2.25
Schyns, P., Bonnar, L., & Gosselin, F. (2002). Show me the features! Understanding recognition from the use of visual information. *Psychological Science, 13*, 402–409. https://doi.org/10.1111/1467-9280.00472
Searcy, J., & Bartlett, J. (1996). Inversion and processing of component and spatial-relational information in faces. *Journal of Experimental Psychology, 22*, 904–915. https://doi.org/10.1037//0096-1523.22.4.904
Shuttleworth, E., Syring, V., & Allen, N. (1982). Further observations on the nature of prosopagnosia. *Brain and Cognition, 1*, 307–322. https://doi.org/10.1016/0278-2626(82)90031-8
Soddu, A., Boly, M., Nir, Y., Noirhomme, Q., Vanhaudenhuyse, A., Demertzi, A., Arzi, A., Ovadia, S., Stanziano, M., Papa, M., Laureys, S., & Malach, R. (2009). Reaching across the abyss: Recent advances in functional magnetic resonance imaging and their potential relevance to disorders of consciousness. *Progress in Brain Research, 177*, 261–274. https://doi.org/10.1016/S0079-6123(09)17718-X
Spagna, A. (2022). Visual mental imagery: Inside the mind's eyes. In G. Miceli, P. Bartolomeo, & V. Navarro (Eds.), *Handbook of clinical neurology* (pp. 145–160). Amsterdam: Elsevier.
Spagna, A., Hajhajate, D., Liu, J., & Bartolomeo, P. (2021). Visual mental imagery engages the left fusiform gyrus, but not the early visual cortex: A meta-analysis of neuroimaging evidence. *Neuroscience and Biobehavioral Reviews, 122*, 201–217. https://doi.org/10.1016/j.neubiorev.2020.12.029
Sugimoto, A., Koyama, S., Midorikawa, A., Futamura, A., Ishiwata, K., Ishii, K., Miller, M. W., & Kawamura, M. (2012). Is this a new type of primary prosopagnosia, both progressive and apperceptive? *Neuropsychiatric Disease and Treatment, 8*, 169–173. https://doi.org/10.2147/NDT.S30541
Tábuas-Pereira, M., Vicente, M., Coelho, F., & Santana, I. (2016). Prosopagnosia as the presenting symptom of whipple disease. *Cognitive and Behavioral Neurology: Official Journal of the Society for Behavioral and Cognitive Neurology, 29*(2), 100–106. https://doi.org/10.1097/WNN.0000000000000091
Tanaka, J., & Farah, M. (2003). The holistic representation of faces. In M. Peterson, & G. Rhodes (Eds.), *Perception of faces, objects and scenes* (pp. 53–74). Oxford: Oxford University Press.
Thomas, C., Avidan, G., Humphreys, K., Jung, K. J., Gao, F., & Behrmann, M. (2009). Reduced structural connectivity in ventral visual cortex in congenital prosopagnosia. *Nature Neuroscience, 12*(1), 29–31. https://doi.org/10.1038/nn.2224
Thorudottir, S., Sigurdardottir, H. M., Rice, G. E., Kerry, S. J., Robotham, R. J., Leff, A. P., & Starrfelt, R. (2020). The architect who lost the ability to imagine: The cerebral basis of visual imagery. *Brain Science, 10*, 59. https://doi.org/10.3390/brainsci10020059
Townsend, J. T., & Ashby, F. G. (1983). *Stochastic modeling of elementary psychological processes*. Cambridge: Cambridge University Press.
Tree, J. J., & Wilkie, J. (2010). Face and object imagery in congenital prosopagnosia: A case series. *Cortex; a Journal Devoted To the Study of the Nervous System and Behavior*. https://doi.org/10.1016/j.cortex.2010.03.005
Trojano, L., Siciliano, M., Pedone, R., Cristinzio, C., & Grossi, D. (2015). Italian normative data for the battery for visuospatial abilities (teradic). *Neurological Sciences, 36*(8), 1353–1361. https://doi.org/10.1007/s10072-015-2114-4
Ulrich, P. I. N., Wilkinson, D. T., Ferguson, H. J., Smith, L. J., Bindemann, M., Johnston, R. A., & Schmalzl, L. (2017). Perceptual and memorial contributions to developmental prosopagnosia. *The Quarterly Journal of Experimental Psychology: QJEP, 70*(2), 298–315. https://doi.org/10.1080/17470218.2016.1177101
Van Belle, G., De Graef, P., Verfaillie, K., Busigny, T., & Rossion, B. (2010). Whole not hole: Expert face recognition requires holistic perception. *Neuropsychologia, 48*(9), 2620–2629. https://doi.org/10.1016/j.neuropsychologia.2010.04.034
Warrington, E. (1984). *Warrington recognition memory test*. Los Angeles: Western Psychological Services.
Whiteley, A., & Prosopagnosia, W. E. (1977). A clinical, psychological, and anatomical study of three patients. *Journal of Neurology Neurosurgery and Psychiatry, 40*, 395–403. https://doi.org/10.1136/jnnp.40.4.395
Young, A. W., & Bruce, V. (2011). Understanding person perception. *British Journal of Psychology, 102*(4), 959–974. https://doi.org/10.1111/j.2044-8295.2011.02045.x
Young, A. W., Hellawell, D., & Hay, D. C. (1987). Configurational information in face perception. *Perception, 16*(6), 747–759. https://doi.org/10.1068/p160747
Young, A. W., Humphreys, G. W., Riddoch, M. J., Hellawell, D. J., & de Haan, E. H. (1994). Recognition impairments and face imagery. *Neuropsychologia, 32*(6), 693–702. https://doi.org/10.1016/0028-3932(94)90029-9
Yovel, G., & Duchaine, B. (2006). Specialized face perception mechanisms extract both part and spacing information: Evidence from developmental prosopagnosia. *Journal of Cognitive Neuroscience, 18*(4), 580–593. https://doi.org/10.1162/jocn.2006.18.4.580
Zeman, A. Z., Della Sala, S., Torrens, L. A., Gountouna, V. E., McGonigle, D. J., & Logie, R. H. (2010). Loss of imagery phenomenology with intact visuo-spatial task performance: A case of 'blind imagination'. *Neuropsychologia, 48*(1), 145–155. https://doi.org/10.1016/j.neuropsychologia.2009.08.024