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Review Article

The 2024 Richardson Lecture: Prosopagnosia – A Classic Neurologic Deficit Meets the Modern Era

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ABSTRACT: Acquired prosopagnosia is a rare disorder, but it serves as a model for impairments in expert-level visual processing. This review discusses five key observations made over the past 30 years. First, there are variants, an apperceptive type linked to damage to the inferior occipitotemporal cortex and an amnestic type associated with anterior temporal lesions, both either right or bilateral. Second, these variants are clustered in syndromes with other perceptual deficits, the apperceptive type with field defects, dyschromatopsia and topographagnosia, and the amnestic type with topographagnosia and the auditory disorders of phonagnosia and acquired amusia. Third, extensive testing often shows additional problems with recognizing exemplars of other objects, especially when degrees of expertise are taken into account. Fourth, the prosopagnosic impairment does not affect all facial information. For example, the perception of expression and lip-reading likely depends on other neural substrates than those for processing facial identity. Last, face perception in prosopagnosia is not immutable but can improve with extensive training, though as yet this does not represent a cure for the condition. Continuing work with neural networks and animal models will enhance our understanding of this intriguing condition and what it tells us about how our brains process vision.

RÉSUMÉ : **La conférence Richardson de 2024 : la prosopagnosie, un déficit neurologique classique à l'aune de la science d'aujourd'hui.** La prosopagnosie acquise demeure un trouble rare, mais elle sert de modèle pour les troubles du traitement visuel. Cet article entend discuter de cinq observations clés faites au cours des trente dernières années. Premièrement, il existe des variantes à ce trouble, à savoir un type aperceptif lié à des lésions du cortex occipito-temporal inférieur et un type amnésique associé à des lésions temporales antérieures, toutes deux droites ou bilatérales. Deuxièmement, ces variantes sont regroupées dans des syndromes avec d'autres déficits perceptifs : le type aperceptif avec des défauts du champ visuel, la dyschromatopsie et l'agnosie topographique ; et le type amnésique, avec l'agnosie topographique de même que les difficultés auditives liées à la phonagnosie et l'amusie acquise. Troisièmement, des tests approfondis révèlent souvent des problèmes supplémentaires dans la reconnaissance d'exemplaires d'autres objets, en particulier lorsque les niveaux de capacité sont pris en compte. Quatrièmement, la déficience prosopagnosique n'affecte pas toutes les informations faciales. Par exemple, la perception de l'expression et la lecture labiale dépend probablement d'autres substrats neuronaux que ceux qui traitent l'identité faciale. Enfin, la perception des visages dans le cas de la prosopagnosie n'est pas immuable. Elle peut en effet s'améliorer grâce à un entraînement intensif même si cela ne constitue pas encore un remède à la maladie. La poursuite des travaux sur les réseaux neuronaux et les modèles animaux nous permettra de mieux comprendre cette pathologie intrigante ainsi que la façon dont notre cerveau traite la vision.

I accidentally walked into a full length mirror in an office hallway because I didn't realize it was my reflection and I expected that "guy" to move out of the way so we wouldn't collide! It was embarrassing and funny all at the same time. I also raised my son from the time he was 3 weeks and I wouldn't recognize him if I didn't know he was supposed to be in the room. He is now 28 years old. (Subject BIOT2)

It is hard not to be fascinated by the amusing and sometimes poignant stories of patients with acquired prosopagnosia. How is it that a person who can see and remember most things suddenly finds that the faces of their family and friends have become strange and that they ca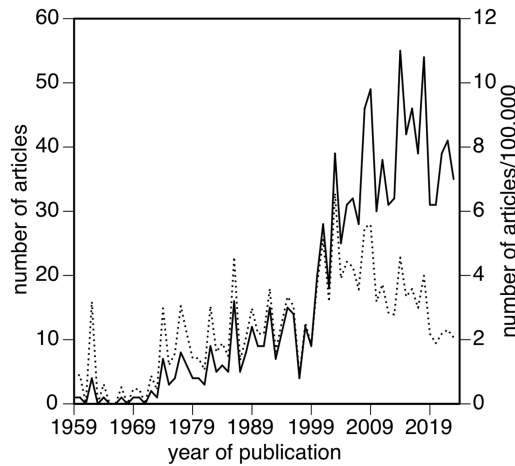n now no more tell faces apart than they can stones on the beach? And, more to the point for neurologists and neuroscientists, what does the existence of this condition tell us about the organization of vision in the human brain? Or about how we achieve the seemingly effortless task of recognizing faces, despite their complex three-dimensional structures, shifts in viewpoint, fleeting changes of expression, long-term warping from age and the high degree of similarity between all human faces?

Acquired prosopagnosia has been part of the neurologic landscape for a long time. The term specifically refers to the loss of familiarity for the identity of the face, the lack of realization that it belongs to someone you have seen before. The first case was likely

Figure 1. Annual number of publications about prosopagnosia (solid line), also expressed as proportion of the scientific literature (dotted line).

LL, who was reported 157 years ago by the Italian ophthalmologist Quaglino.[1,2] It is a rare condition, with most neurologists unlikely to encounter more than one or two cases in their career. One or two also corresponded to the annual frequency of reports on prosopagnosia, until interest grew in the 1980s and then surged in the new century to about 40 papers a year, a 40-fold increase that exceeds the 10-fold growth in all scientific reports over this 60-year period (Figure 1).

Several factors have fueled the expansion of work in this unusual condition. In the 1980s, psychologists created cognitive models of face perception[3,4] and were interested in how these applied to prosopagnosia. At the same time, structural imaging with CT and MRI scans permitted the *in vivo* demonstration of the lesions that caused prosopagnosia,[5–7] an advance over indirect inferences from homonymous field defects and the wait for pathologic data.[8] In the 1990s, functional MRI emerged as a tool and revealed the network of regions involved in the face perception of healthy subjects,[9] including first and foremost the fusiform face area.[10] MRI techniques then expanded in several directions, allowing us to look at the structural and functional connectivity of this network[11] and to explore the functions of cerebral regions using fMRI adaptation.[12] For prosopagnosia, the development of face localizers robust enough to detect these networks in single subjects brought functional imaging to individual patients,[13] allowing the correlation of their functional defects with the structural impact of their lesions on the face network.

While these developments rapidly expanded our knowledge of the structural basis of face perception and prosopagnosia, advances in computerized image processing – some imported from the film industry – allowed more sophisticated means of measuring face perception. Instead of the simplistic Identikit drawings[14,15] used by police in the 1960s and 1970s, researchers could now morph faces between identities, ages and expressions,[16] separate the texture and reflectance of a face from its shape,[17,18] manipulate the size and position of different features[19] and so on. These were used in adaptation studies to probe the nature of the face representations our brains store[20,21] and to generate concepts of how these representations might be organized, as, for example, norm-based coding models.[22]

Finally, a major factor has been the discovery of developmental prosopagnosia. The first case was reported in 1976.[23] AB was a 12-year-old girl whose mother also had lifelong trouble recognizing faces. More cases were reported, leading to claims that as much as 2% of the population may have this condition,[24] though more recent estimates suggest the true incidence may be lower.[25] Regardless, this has led to larger samples of "face-blind" subjects available for research, though the relation of the developmental to the acquired version is not clear. In particular, it is not known whether the developmental variant is simply the low end of the normal distribution of face perception abilities in the population or a pathologically distinct group.[26] Recent cluster analyses of 1500 subjects suggest the former,[25] but more work with genetic and imaging biomarkers is needed to settle the issue.

This review focuses on acquired prosopagnosia. There are many intriguing aspects to this disorder, such as how patients with prosopagnosia scan faces,[8] whether they show covert recognition and what that means,[29] whether dissociations explain certain features[30] and the relation of face perception to other means of person recognition.[31] Here I will discuss five observations learned over 24 years of study.

Prosopagnosia is not a disorder but a family of disorders

People become prosopagnosic for different reasons, in both the structural and functional sense. The structural patterns of lesions in prosopagnosia are often complex but can be considered to differ mainly in two ways, whether they are (a) unilateral or bilateral or (b) anterior temporal or posterior occipitotemporal, though sometimes the latter occurs in combination.

Regarding laterality, a frequent association with left hemifield defects led to early beliefs that prosopagnosia resulted predominantly from right hemispheric damage.[32] Bodamer, who coined the term "prosopagnosia," inferred the presence of bilateral occipital damage from clinical signs,[33] and the seminal studies of Meadows[8] and Damasio[5] described bilateral lesions on imaging and/or autopsy. These were countered by an increasing number of imaging reports in the 1980s of patients with unilateral right hemispheric lesions.[6,7,34–37] On the other hand, prosopagnosia from a unilateral left hemispheric lesion is distinctly rare.[38,39] Subjects with this condition are often left-handed, and sometimes, their imaging reveals subtle right hemispheric changes as well.[40]

These clinical observations on lateralization are consistent with functional imaging studies in healthy subjects that show bilateral activation with a right hemispheric dominance during face processing.[10,41] It is not clear if there is any qualitative difference in prosopagnosia between those with unilateral and those with bilateral lesions: the difference may be more quantitative, with slightly lower scores for face familiarity in those with bilateral lesions.[42] The degree of lateralization of the face network likely varies on a continuum, and this may determine whether a right-sided lesion is sufficient to create prosopagnosia or if bilateral lesions are needed.[43] A rightward bias of this continuum can explain why prosopagnosia is rare after left hemispheric lesions.

How about the second distinction, between posterior and anterior damage? The early cases also reported that damage was concentrated in the lingual and fusiform gyri.[5,8] It is probable that such lesions affected the fusiform and/or occipital face areas that were later discovered with functional imaging.[9] This has been confirmed in modern studies that used functional MRI with single-subject methods to show loss of one or both of these areas in patients with such lesions.[16,40,44] However, from the 1990s onward, reports of prosopagnosia with anterior temporal lesions emerged.[45–47] While some reported no difference in the perceptual deficits between those with fusiform and those with anterior temporal lesions,[48] we found
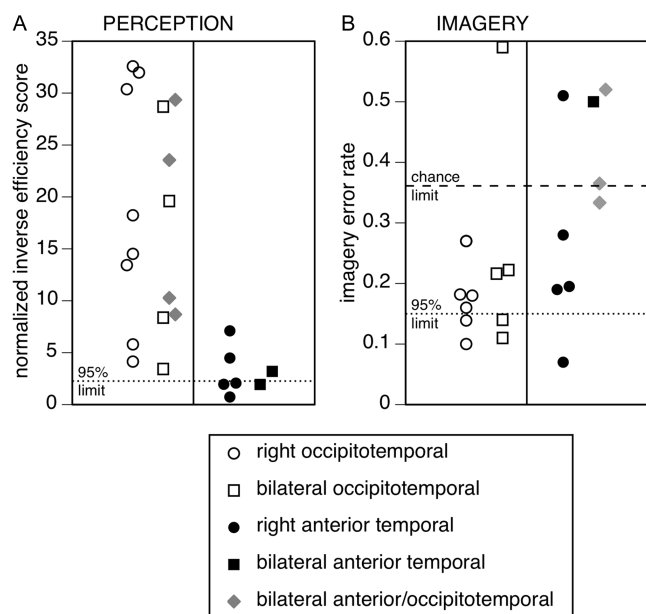
Figure 2. Performance on face tests by 23 patients with acquired prosopagnosia, classified by lesion location. (**A**) Inverse efficiency scores, normalized by the performance of the control group, for the perception of face configuration (interocular distance and nose–mouth distance combined). (**B**) Error rate on the famous face imagery test. Both graphs are arranged so that worse performance corresponds to higher scores. The dotted lines show 95% prediction limits from the control group, while the dashed line in B shows the 95% limit for chance performance of 0.5: scores above this are no better than random guessing. Data compiled from Barton, 2008[42] and Pancaroglu et al., 2016.[118]

regions (Figure 2B). Conversely, those with normal imagery scores were more likely to have lesions limited to the occipitotemporal cortex.

Hence, the structural distinction between fusiform and anterior temporal lesions maps reasonably to a functional distinction between apperceptive and amnestic variants of prosopagnosia (Figure 3). There is not a perfect correspondence, as mild imagery deficits are commonly seen in the former and mild perceptual inefficiencies in the latter. This is consistent with claims that apperceptive and amnestic deficits lie along a continuum[49] and that, as Lissauer originally noted for general visual agnosia,[52] these distinctions are relative, not absolute.[42] Nevertheless, the proposal that right anterior temporal lesions are required – though not always sufficient – for an amnestic variant implies that the representations of familiar faces are stored primarily in that structure. This has received support from recent work in rhesus monkeys that found cells that respond specifically to familiar faces in face-activated patches of the right temporal pole.[53]

It has been argued that an associative form of prosopagnosia should have intact perception and intact memories yet fail to recognize faces because they cannot link the two.[30] As yet, we have not found a patient with acquired prosopagnosia who meets such strict criteria. However, a few patients with only right anterior temporal lesions come close, with only mild or no impairment in both perception and imagery.

Prosopagnosia is associated with other deficits in two syndromes

Human lesions are large. Vascular territories do not map onto the anatomic boundaries between one functional system and another, and tumors and infections do not respect sulcal or gyral divisions. It is no surprise that the damage that causes prosopagnosia will often affect adjacent neurologic circuits. The other functions that are impaired will be determined by this "neighbourhood principle."

The tetrad of prosopagnosia, homonymous field defects, dyschromatopsia and topographic disorientation is well recognized. This was described even in the earliest cases[2,33,54] and later

variations, as others had hypothesized.[49,50] Those with fusiform lesions struggled to see the precise shape of faces, but this was less of a problem for those with anterior temporal lesions[46] (Figure 2A). On the other hand, the latter had more trouble recalling the appearance of known faces on tests of imagery for famous faces.[51] In particular, those who performed no better than chance on imagery were more likely to have right anterior temporal lesions, often in combination with damage to bilateral occipitotemporal or left anterior temporal

Figure 3. Summary of functional and structural differences for apperceptive and associative variants of prosopagnosia, including associated visual field defects and other high-level sensory deficits.
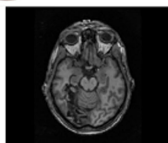
linked to bilateral fusiform damage.[5] It is a ventral occipitotemporal syndrome, in the same way that Bálint's syndrome is a dorsal occipitoparietal syndrome. Both are syndromes in that not every patient will have all components of the syndrome – that will depend on variations in both individual anatomy and the location and size of the lesions. The frequent co-occurrence of these deficits is due to the anatomic proximity of the networks involved, not because one problem causes the others. This may seem obvious, but in Borelli's commentary on Quaglino's case, he speculated that impaired color vision was the reason why LL could not recognize faces or places.[2] Even now, one encounters prosopagnosic patients who erroneously assume that their struggles with face recognition are due to their hemianopia.

More details about this long-established tetrad have emerged. First, it is specific to the apperceptive variant of prosopagnosia, not surprising given that fusiform damage is involved. Second, the topographic disorientation has two components: poor place recognition and impaired cognitive map formation.[55] Third, when the fusiform damage is bilateral, there may be a mild element of alexia,[40] as indexed by an elevated word-length effect, the time needed to read a word as a function of the number of letters it contains.[56] An occasional association with alexia is also consistent with earlier observations.[33]

In contrast, anterior temporal lesions cause a different cluster of deficits (Figure 3). Dyschromatopsia is not a feature.[57] Field defects are less common despite the fact that Meyer's loop is in the vicinity.[58] We found that only one of seven prosopagnosic patients with lesions limited to the anterior temporal lobes had an upper quadrantanopia – in contrast, only 1 of 12 patients with fusiform lesions alone had full visual fields. Patients with anterior temporal lesions get lost in familiar places too, but their topographic problems are due to place agnosia only, not impaired use of cognitive maps.[55] A more distinctive difference is the presence of auditory deficits, reflecting the fact that the anterior temporal cortex is a multimodal sensory convergent zone. Those with bilateral lesions may be impaired in recognizing voices as well as faces, a deficit called "phonagnosia."[59] These patients still retain a store of biographic information about people, which distinguishes them from patients with a multimodal person recognition disorder, or "people-specific amnesia,"[60–62] Some also have acquired amusia, or tone deafness, with altered esthetic musical experiences, either musicophilia or its opposite, anhedonia.[63] This may account for occasional reports from the pre-imaging era of patients who also lost the ability to sing or recognize music when they developed prosopagnosia.[64] With our patients these auditory deficits were not mentioned spontaneously, and more sensory deficits may await characterization in this group – consider, for example, the description of an impairment in yet another modality, an agnosia for odors that accompanied prosopagnosia in a man with progressive bitemporal atrophy.[65]

Not all agnosia in prosopagnosia is face-related

Unlike patients with general visual agnosia, prosopagnosic subjects do not mistake wives for hats. They know that a face is a face, a hat is a hat and a car is a car. What they cannot tell is whose face this is, and the natural question then is whether they can tell which hat and which car. For decades, people have argued about whether prosopagnosia is specific to faces – for a review, see.[66] For every report of a prosopagnosic patient who could recognize birds, dogs, butterflies, fruits, vegetables, cars or tools, there is a countering

article about another whose recognition troubles included one or more of these other objects. This speaks to a larger issue, about whether visual processing in the human brain is modular or distributed. The modular view proposes that at least some components of the network involved in face recognition process faces alone,[67] whereas the distributed view argues that no region is dedicated to a single object.[68]

The contribution of prosopagnosia to this debate is time-honored but complicated. The large scale of human pathology means that, if non-face object modules are close to face modules, there is a high chance that they will be damaged too. If so, problems with recognizing other objects will often accompany prosopagnosia, even if face processing is accomplished by a dedicated face module. One could argue that the discovery of a single patient with completely intact recognition of non-face objects would settle the argument, but exactly how does one show "completely intact recognition"?

A review of the studies of object recognition in developmental prosopagnosia[69] and its accompanying commentaries grappled with the difficulties involved. These were summarized later as 11 points that studies should consider,[70] such as firm diagnostic criteria,[71] assessing reaction time as well as accuracy, showing putative classical dissociations,[72] testing equivalent processing stages, matching for test difficulty,[73] matching the number of exemplars in the decision space[74] and testing of at least three categories of objects.

One especially challenging task is accounting for variations in premorbid expertise for different types of objects. Recognition abilities depend not just on prior exposure but also on interest. While we assume that most humans have a similar interest in recognizing faces, this cannot be assumed for most other objects. Consider two hypothetical prosopagnosic patients: subject A, who has no interest in automobiles, and subject B, who is a car buff. Cars are a good category because all people in modern society are exposed to them on a daily basis. If we ask these subjects to name the car in Figure 4A, answering "Porsche" may be a sign of intact car recognition in subject A. However, we would expect subject B to do better, to say that it is a 911, perhaps more specifically the 964 variant, and judging by the wheels, a 1994 model. If subject B can only say "Porsche," we would be concerned that something is amiss.

To take into account premorbid expertise, we devised a comparison between verbal semantic knowledge about cars – reasoning that semantic knowledge should be intact in prosopagnosia – and visual car recognition.[75] The two are highly correlated in healthy subjects, with an r of 0.91. This allowed us to predict how many of the 150 car images a given prosopagnosic patient should recognize on the basis of their verbal knowledge, knowing that the Pinto was made by Ford, for example. We found poorer car recognition than expected in almost all prosopagnosic patients, and this was particularly clear in those who were car experts.[66,70,75]

However, the car is only one type of object. We supplemented this expertise-adjusted car test with two other probes, the Old/New Test,[76] which includes subtests for cars, guns, horses and glasses, and the Cambridge Bicycle Memory Test,[77] which uses the same format as the Cambridge Face Memory Test. We found that none of the 15 patients with acquired prosopagnosia (and only 1 of the 12 with developmental prosopagnosia) had conclusive evidence for intact non-face object recognition across all three tests.[70]
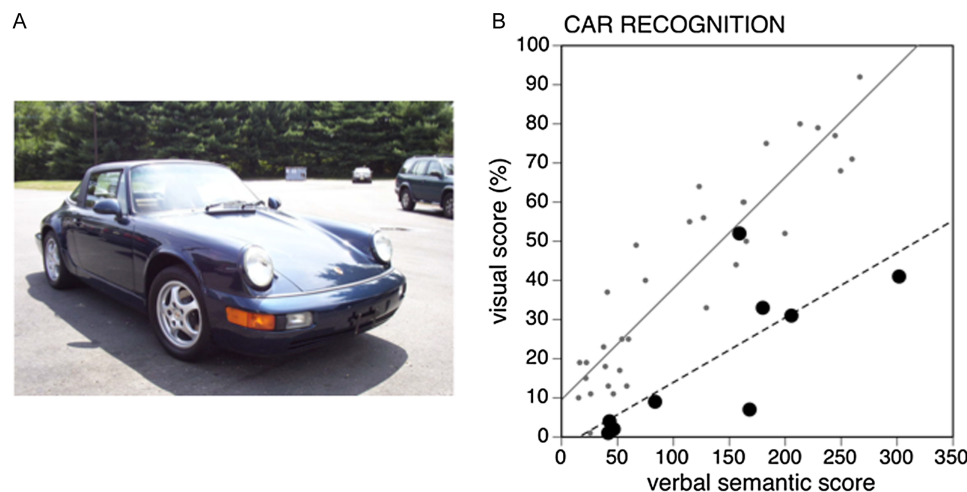
Figure 4. (A) What car is this? How precise an answer you can give depends on your perceptual expertise. (Answer: Porsche 911, 964 Targa variant, 1994 model year) (B) Expertise-indexed test of car recognition. The proportion of a set of 150 pictures of cars that were correctly named by a subject is plotted as a function of their score on a verbal semantic test, which asked them to match the manufacturer to a named model (e.g., Pinto, answer: "Ford"). In control subjects, these abilities are highly correlated (small gray dots, $r = 0.91$). With one exception, the nine prosopagnosic patients tested (large black discs) recognized fewer cars than predicted from their verbal semantic score. Data compiled from Barton et al., 2009[75] and Davies-Thompson et al., 2014.[119]

One special class of objects is visual text. People in literate societies also have universal exposure to and interest in written words. Like face recognition, reading is considered an expert visual process. In fact, the bilateral networks for text and face perception overlap each other,[41] with the key difference being that reading generates stronger activation in the left hemisphere while face recognition emphasizes the right. The left visual word form area occupies a region that is almost the mirror location of the right fusiform face area, underscoring the concept of the fusiform gyrus as a key structure in expert visual perception.[78] Not surprisingly, prosopagnosic subjects with bilateral fusiform lesions have an elevated word-length effect, indicating a mild degree of alexia. However, prosopagnosic patients with right unilateral lesions – either anterior temporal or fusiform – do not show any difficulty in reading.[40,79] Rather, all but 1 of the 11 prosopagnosic patients in our study struggled to identify the handwriting or the font of the text.[40,66] Hence, the right hemisphere appears to contain a network that is important for decoding identity information in text, just as it has a network for identifying faces, but is less involved in reading text.

Not all face deficits are confined to prosopagnosia

If the recognition difficulties of many prosopagnosic patients extend to other objects besides faces, it is also true that these difficulties sometimes spare other types of facial information besides the identity of the person. Like many complex objects, faces convey a wide range of information, including gender, emotional state, age and attractiveness. Cognitive models of face perception proposed that dynamic information such as expression is processed separately from the static structural data upon which most judgments of face identity are based.[3] However, a few early studies disagreed on (a) the type of anatomic damage that impaired the perception of facial expression[80–82] and (b) whether prosopagnosic patients were impaired in perceiving expression as well as identity – for review, see.[16] New data from functional imaging then emerged, suggesting that the superior temporal sulcus played a key role in processing facial expression.[9,83,84] A study using morphed faces as stimuli found intact perception of facial expression in four prosopagnosic patients, all of whom had preserved face-related activation of the right posterior superior

temporal sulcus, while one patient with a lesion that eliminated this area – but spared the right occipital and fusiform face areas – was impaired in expression but not identity perception.[16]

Clearly, more work on expression deficits remains to be done. Studies of other social signals have yielded mixed results in prosopagnosic patients, with some finding variably preserved judgments of trustworthiness, attractiveness and aggression[85] and others a reduced sense of facial attractiveness.[86]

Lip-reading is another task with faces that raises interesting questions about the lateralization of function. On the one hand, it involves faces, and faces have a network that is more active in the right hemisphere. On the other hand, it contributes to linguistic operations, which in most people are located in the left hemisphere. Lip-reading has been studied in a patient with prosopagnosia after a right posterior cerebral artery stroke and another with pure alexia after a left occipitotemporal stroke[87,88] and, more recently, in four patients with pure alexia and one prosopagnosic patient, all with unilateral lesions.[89] Though small in number, the results are consistent. Neither of the two prosopagnosic patients had difficulty in lip-reading, while all five of the patients with pure alexia were impaired.

The poor lip-reading of alexic patients caused interesting anomalies in the McGurk effect.[90] This effect occurs when subjects see a face pronouncing one syllable while at the same time hearing a voice pronouncing a different one: subjects typically report perceiving a syllable that is a blend of the two. The pure alexic subjects failed to report either the blend or the visually presented syllable, but the subjects with acquired prosopagnosia performed normally.

From these observations, lip-reading appears to lateralize to the left. This is consistent with tachistoscopic reports of a right hemifield advantage for lip-reading[91] and functional imaging studies showing that activity in the left superior temporal sulcus correlates with lip-reading proficiency[92] and the McGurk effect.[93]

Both the results for lip-reading and those for handwriting and font identification make the same point: it is not the stimulus that is lateralized but the nature of the operation being performed. Prosopagnosic patients with right hemisphere lesions have trouble identifying not only faces but also handwriting and font, though they can read text and can lip-read with faces. Alexic patients with left fusiform lesions have almost the converse: they cannot do the
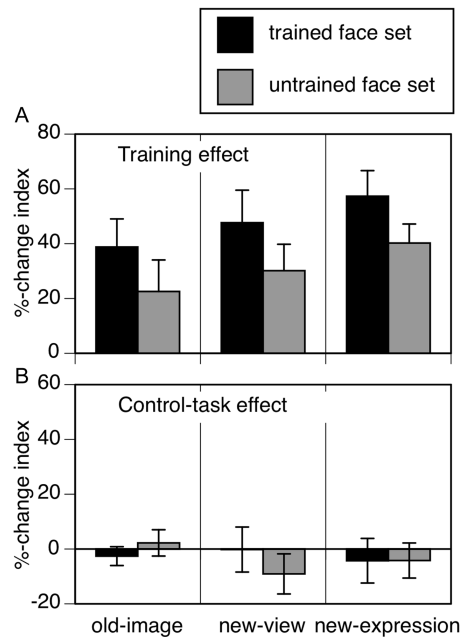
Figure 5. Training the face perception of prosopagnosic patients. Subjects performed 11 weeks of daily work with a perceptual learning method. Graphs show the percent change from their test performance before training. After training (A), there is a nearly 40% improvement with the same types of images used in training (black bar in "old image"). The improvement does "generalize" to new views and new expressions of the same faces that had not been seen during training. Also, when tested on a set of faces from different people, there is "transfer" of benefit (gray bars, untrained face set). No benefit is seen after the control task (B), which provided exposure to faces but not feedback or a formal training structure. Data from Davies-Thompson et al., 2017.[101]

linguistically directed tasks of reading text or lips, but they can identify faces. What we do not know yet is how they are with identifying handwriting or font.

Face recognition in prosopagnosia is malleable and can be trained

The preceding observations contribute to our understanding of neurologic structure and function, but on a practical level, can we fix prosopagnosia? With the exception of rare cases with resolution after small strokes or migraine[94–96] – which admittedly may be underreported – most patients are likely to have prosopagnosia permanently. Most of the prosopagnosic patients studied have had the condition for months to years. While many are resourceful in finding strategies to work around their face recognition problems, they may still experience social difficulties. This has been studied best in subjects with developmental prosopagnosia, who report anxiety in social situations that can lead to avoidance behavior.[97]

Early studies of single cases or small series tried a variety of strategies, as recently summarized.[98–100] These included creative approaches such as trying to convert covert into overt recognition,[101] learning of face-name associations,[102,103] employing the tricks of professional mnemonists[104] or emphasizing the recognition of facial features.[102,105,106] The results have been mixed.[100] The studies are also difficult to compare given the variations in training techniques and the means used to assess efficacy and because these studied single subjects for the most part.

To advance upon this situation, we performed a randomized online training study in a cohort of 10 patients with acquired prosopagnosia.[100] The 10 were divided into 2 groups matched

approximately for lesion location. One started with the training protocol and the other with a control condition, namely, watching British murder mysteries. We employed a perceptual learning technique.[107] This used a large volume of repetitive sensory training with feedback over 11 weeks. On each trial, a subject saw a target face, below which were two images, each made by morphing a variable degree between this target face and another face. The task was to choose which of the two was most similar to the target face. Initially, the difference between the two choice faces was set quite large, and the answer was obvious. With every correct response, the physical differences between the two choice faces were lessened until the subject was training at a level where they were getting about 85% correct. As training progressed, subjects began to perceive subtler differences. Once they achieved a learning criterion, they moved on to blocks with variable viewpoint, then variable expression, then variations in both viewpoint and expression, with the aim of learning the three-dimensional aspects of facial structure that would apply to real-life situations.

The result was a 39% improvement in subjects' ability to see facial structure (Figure 5). Critically this improvement generalized to viewpoints and expressions that hadn't been used in training. It also "transferred," in that there was a 30% improvement for faces of new people not seen in training. Thus, subjects were not just doing better with a set of trained faces to which they had massive exposure, but they had learned a skill that could be applied to new faces. This effect was still apparent 3 months later. It is unclear how best to assess whether benefits from training impact face recognition in daily life, but some subjects related positive anecdotes about their experience with family and friends, though others did not.

The same protocol improved face perception in a cohort of 10 subjects with developmental prosopagnosia,[108] and other training studies have yielded positive results with the developmental variant.[109–111] Such findings indicate that face perception is malleable and can be improved in prosopagnosia. However, the current methods lead only to partial benefits and clearly do not "cure" the condition. In the future, refinements of these protocols and combination with techniques that promote cerebral network plasticity may yield even better results.

Summary

Reports of acquired prosopagnosia have been an invaluable complement to the studies of face perception in healthy subjects, investigations of the functional anatomy of the face network and electrophysiological work in nonhuman primates. As with any complex cognitive process, face recognition has component operations, and this fact is reflected in variants of prosopagnosia that correspond to lesions of different cerebral regions. These variants are marked clinically by associations with other perceptual deficits that form syndromes, one with occipitotemporal damage and one with anterior temporal lesions. Whether faces have a special modular status in visual processing continues to be debated. Acquired prosopagnosia is frequently accompanied by problems with recognition of other objects, particularly once the variability of human expertise with the latter is taken into account. On the other hand, the prosopagnosic deficit does not extend to all aspects of face perception. It is also not a fixed deficit but can be improved by training, which opens the possibility of rehabilitation.

Future directions promise interesting new developments. Most people are aware of the use of artificial intelligence to perform face recognition in forensic and commercial settings. More recent work

in this area is starting to ask how well artificial neural networks can replicate the various properties of human face processing and provide insights into how our brains achieve this feat.[112–114] While functional MRI was initially applied to human face networks, it is now being combined with electrophysiological techniques in nonhuman primates, where this has revealed a network of interconnected face patches extending from the posterior occipital to anterior temporal regions.[115,116] A major goal will be to understand the roles of these different patches and their interactions. Observations from these approaches will refine our understanding of prosopagnosia and clarify several theoretically important questions about its basis and the nature of visual processing in the human brain.

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