Neuropsychologia 105 (2017) 197–214

Contents lists available at ScienceDirect

Neuropsychologia

journal homepage: www.elsevier.com/locate/neuropsychologia

Review article

Functional outcomes following lesions in visual cortex: Implications for plasticity of high-level vision

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ARTICLE INFO

Keywords:
Plasticity
Vision
Hemispherectomy
Visual cortex
Face recognition
Object recognition
Word recognition

ABSTRACT

Understanding the nature and extent of neural plasticity in humans remains a key challenge for neuroscience. Importantly, however, a precise characterization of plasticity and its underlying mechanism has the potential to enable new approaches for enhancing reorganization of cortical function. Investigations of the impairment and subsequent recovery of cognitive and perceptual functions following early-onset cortical lesions in humans provide a unique opportunity to elucidate how the brain changes, adapts, and reorganizes. Specifically, here, we focus on restitution of visual function, and we review the findings on plasticity and re-organization of the ventral occipital temporal cortex (VOTC) in published reports of 46 patients with a lesion to or resection of the visual cortex early in life. Findings reveal that a lesion to the VOTC results in a deficit that affects the visual recognition of more than one category of stimuli (faces, objects and words). In addition, the majority of pediatric patients show limited recovery over time, especially those in whom deficits in low-level vision also persist. Last, given that neither the equipotentiality nor the modularity view on plasticity was clearly supported, we suggest some intermediate possibilities in which some plasticity may be evident but that this might depend on the area that was affected, its maturational trajectory as well as its structural and functional connectivity constraints. Finally, we offer suggestions for future research that can elucidate plasticity further.

1. Introduction

Plasticity is broadly defined as the brain's capacity to be shaped by experience, to adapt and learn, and to reorganize and recover after injury or lesion (Gleissner et al., 2005). This reorganization is a result of the nervous system's ability to respond to intrinsic or extrinsic stimuli by altering its structure and/or function and/or connections (Cramer et al., 2011). Understanding the nature and extent of neural plasticity in humans is of great interest from both a basic science and translational perspective, and recent findings have whet our scientific appetite. For example, although it is generally agreed that the capacity for learning and memory and the opportunities for neural malleability are disproportionately enhanced over the course of childhood compared with adulthood (Bourne, 2010), research in the last few decades has revealed extended capacity for neuroplasticity across the entire lifespan (for review, see Pascual-Leone et al., 2005). Also, recent efforts have focused on promoting plasticity and removing the “brakes” in order to increase circuit rewiring in the brain (Bavelier et al., 2010). Last, there has been increased emphasis on a clearer characterization of plasticity and its underlying mechanism as a means of developing new approaches for enhancing reorganization of cortical function both for clinical purposes (Cramer et al., 2011) and for improved function in the normal population (Lindenberger et al., 2017).

The extensive and growing body of research on plasticity and its underlying mechanisms covers a wide range of phenomena, including dynamic shifts in the strength of existing synaptic connections (cellular level), structural changes in cortico-cortical and cortico-subcortical pathways and connectivity (system level), and modifications of the mapping between behavior and neural activity (functional level) [for recent reviews, see Chandrasekaran et al. (2015) and Ganguly and Poo (2013)]. The focus of the current review is primarily on reactive plasticity, namely, the changes in the neural response following surgical resection or a lesion to the central nervous system (CNS) and the consequences for functional outcomes. Further, because we restrict our discussion to the findings from humans, and specifically those from studies of children, the approach that is most able to advance our knowledge is the detailed investigation of changes following perturbation of brain structure and function, either as a result of surgery or a lesion acquired early in life. A complementary approach is one in which changes over time are tracked in individuals with congenital neural abnormalities, and progress has been made in exploring plasticity in those with congenital sensory deficits such as blindness or deafness (for

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http://dx.doi.org/10.1016/j.neuropsychologia.2017.06.030
Received 27 December 2016; Received in revised form 27 June 2017; Accepted 28 June 2017
Available online 29 June 2017
0028-3932/

review, see Merabet and Pascual-Leone, 2009). Our focus here is primarily on the restitution of function in cases with frank damage to cortex but we also consider plasticity related to developmental atypicalities.

1.1. What can we learn from such investigations?

The impact of lesions and recovery from lesions in children is a potentially revealing means to better understand the organization of the visual system and its capacity to recover from damage. For example, functional neural reorganization has been reported after surgical resection for the treatment of pharmacologically intractable epilepsy (see Werth, 2008 for review). Of note, in many instances, the extent of the recovery is disproportionate relative to the magnitude of the damage – many compromised functions are regained partly or even completely, and so, understanding this dramatic recovery profile can shed light on the functional architecture of the brain.

To date, unsurprisingly given their importance, most investigations of post-resection plasticity have been concerned with the changes in intelligence (Vargha-Khadem and Polkey, 1992), memory (Meekes et al., 2013; Skirrow et al., 2015; Stretton et al., 2014), language (De Koning et al., 2009), or motor function (Bernasconi et al., 2000; Buckley et al., 2014; Hamad et al., 2013; Mullin et al., 2016). While such investigations offer rich descriptions of the neural and behavioral changes, many questions remain, including, what exact underlying mechanisms support the pattern of reorganization, whether reorganization follows a similar trajectory independent of etiology (e.g., stroke, resection) and domain of function (e.g., vision, language), and to what degree other factors (such as sex, education level, premorbid abilities, etc.) modulate the nature and extent of recovery. There are also a host of findings that, on the surface, appear difficult to explain: for example, whereas some children show good recovery of function in the language domain when the perinatal lesion to the left hemisphere occurred before age 1 (Woods and Carey, 1979) or after left hemispherectomy even beyond the critical age of acquisition (age 7–14 years at surgery) (Boatman et al., 1999), visual impairments appear to persist years after surgery and are less amenable to change (Haak et al., 2014). Last, because it is difficult to generalize findings from cases with different times of lesion onset, as well as varying times post-lesion, and to infer patterns of recovery from one cognitive domain to another (Dennis et al., 2014), there is a pressing need to characterize the multiplicity of patterns of recovery (or absence thereof) and discover general principles that characterize plasticity more generally.

1.2. Need for additional studies of plasticity of visual function

Aside from a host of studies on adults with blindsight who, following damage to primary visual cortex (V1), show some degree of conscious residual vision (Danckert and Culham, 2010; Georgy et al., 2016; Tamietto et al., 2010), there has been relatively little attention to the recovery of visual function post-cortical lesion. In particular, there is a paucity of studies examining post-injury changes to the cortical visual system in young patients and, in the few existing studies, the focus is primarily on the lower levels of the visual system (occipital lobe) (Muckli et al., 2009; Werth, 2006), leaving open many questions about recovery of higher levels of the visual system.

This relative dearth of studies of visual plasticity is remarkable given that vision is considered the dominant sense for perception in humans, and over half of the cerebral cortex is dedicated to visual processing (Felleman and Van Essen, 1991). Pressing questions specific to the plasticity of cortical vision include, for example: Does a lesion to different sites along the visual cortex affect functional recovery differently? Given the well-established hierarchical organization of the visual cortex (although see Rossion et al. (2011), Weiner et al. (2016)), does recovery of higher-order visual cortex depend strictly on the integrity of early visual cortex? What is the microgenesis i.e. the trajectory that is followed during reorganization of the visual system and what is the nature of the change in neural processes mediating such improvement?

2. Plasticity in higher-order visual cortex

Here, we review the successes and failures of restitution of function in patients with a lesion to or resection of VOTC early in life. For convenience, we subdivide the review into three domains of visual pattern recognition: face recognition (see Table 1), object recognition (see Table 2), and word recognition/reading (see Table 3). Many children manifest perceptual deficits in more than one of these domains simultaneously, but we assign an individual case to a subdivision based on the primary deficit. Note that we have also restricted our review to cases with relatively circumscribed lesions and so even though higher-order visual deficits may be apparent in neurodevelopmental disorders such as autism, Down syndrome, and Rett syndrome, we do not include these cases here. Following the analysis of the cases, we summarize and discuss factors that promote or constrain brain plasticity and describe other core principles gleaned from the cases. Last, we consider possible mechanisms that might underlie the plasticity and we relate these to the patterns of change noted in the human cases.

2.1. Theoretical considerations

Beyond V1, visual processing continues through well-characterized ventral and dorsal pathways, with functional specialization of “what” and “where/how”, respectively (Ungerleider and Mishkin, 1982; Kravitz et al., 2011, although see Freud et al., 2016 for a slightly different characterization). Within the ventral “what” pathway (projecting through the occipitotemporal cortex to the anterior temporal lobe), the topographic organization has been well characterized in adults, and a host of category-selective areas have been identified, including, for example, multiple face-selective regions such as the fusiform face area (FFA, Kanwisher et al., 1997), occipital face area (Rossion et al., 2003) and anterior temporal lobe (Avidan et al., 2014); multiple place- or scene-selective areas such as the retrosplenial cortex (RSC, Aminoff and Tarr, 2015) and parahippocampal place area (PPA, Epstein and Kanwisher, 1998), as well as object-selective regions such as the lateral occipital complex (LOC, Grill-Spector et al., 1999; Malach et al., 1995), and word-selective regions such as the visual word form area (VWFA, Cohen et al., 2000; McCandliss et al., 2003). Although most of these regions can be identified bilaterally, complementary patterns of hemispheric lateralization — greater face-selective activation in the right hemisphere (RH) than left hemisphere (LH) and the reverse pattern for word-selective activation — are consistently observed (Behrmann and Plaut, 2015). Category-selective topography is highly reproducible across individuals, but the mechanisms that give rise to this seemingly universal organization are not well understood. The mechanisms that determine the emergence of this topography, however, have direct implications for any reorganization that might occur (Anderson et al., 2011).

Theoretically, one might postulate a continuum of possible outcomes following brain damage that affects category-selective areas, ranging from complete reorganization and functional recovery to no recovery or reorganization. The extreme of no reorganization is supported by the hypothesis that the functional topographic map in ventral cortex is innately prespecified (e.g., McKone et al., 2012) and that individual regions have, to a large extent, a single assigned function (e.g., face processing or word processing, Kanwisher, 2010, 2017). To the extent that the organization is innately prewired and that a single region has a singular function, restitution of function following damage becomes less likely i.e. if the function is solely a property of a particular area, it is less likely that another area can assume this function. Farah et al. (2000) adopted just this view in the study of Adam who sustained bilateral lesions to VOTC (infarction of posterior cerebral arteries) at Day 1 of age. At age 16, Adam was profoundly impaired at recognizing

faces. That face recognition could not be supported by other parts of the brain (e.g., regions mediating object recognition) was taken as evidence that the brain areas for face recognition are predetermined. The unresolved face recognition impairment "implies that some distinction between face and object recognition, and the anatomical localization of face recognition, are explicitly specified in the genome." (Farah et al., 2010, p. 122).

At the opposite extreme is the view that all brain areas are equipotential (Basser, 1962; Dennis and Whitaker, 1977), in which case, following damage, any region might plausibly take over the function of another region, and recovery should be complete. This view is closest to the principle of 'mass action' (Lashley, 1929) which notes that the proportion of the brain that is injured is directly proportional to the decreased ability of a particular function, such as memory or visual recognition. In other words, a function cannot be localized to a single cortical area, but is instead distributed throughout the cortex. At least under conditions in which the lesion is not so extensive, the intact parts of cortex should be able to compensate for the damaged area.

As will be apparent when the evidence from the patient studies is presented below, neither of these extreme views seems to hold. There are numerous versions of views that fall intermediate along the plasticity continuum. In general, these views predict a flexible, dynamic and experience-dependent, but circumscribed, topographic outcome. One such view might predict that there are opportunities for plasticity but that these are constrained by existing cortical (and perhaps subcortical) connectivity (Bouhali et al., 2014; Plaut and Behrmann, 2011; Wandell and Yeatman, 2013). As a specific example, given that the word-preferring VWFA in the LH is tethered to both visual cortex as well as language regions (Stevens et al., 2017), damage to this area would require that a pre-requisite for the compensatory region is to have similar connectivity to visual and language regions. A related issue that is especially relevant for experience-dependent plasticity is that reorganization of higher-order visual cortex may be contingent on the quality of input from earlier regions (Fox et al., 2010). Even a brief period of deprivation of high quality visual input can lead to long-lasting deficits in face and holistic processing (Le Grand et al., 2004, 2003, 2001) and in object and form processing (Ostrovsky et al., 2009, 2006) as demonstrated in the studies of individuals with congenital blindness or cataracts.

There are a host of other factors that likely affect the outcome on the plasticity continuum. One such factor concerns the nature of the lesion: the more acute the onset, as in cases with sudden onset of lesion (e.g., stroke or traumatic brain injury) with minimal opportunity to slowly recruit other neural correlates (e.g., as in slow growing tumor), the less likely the recovery. Another factor concerns the timing of the lesion as well as the maturational chronology of the visual system i.e. not all regions necessarily obey the same developmental timetable. Research over the past two decades has indicated that cortical areal formation follows a unique, spatiotemporal time-lapse sequence during childhood and through early adulthood (Gogtay et al., 2004) with different maturational trajectories for different areas and some regions not fully mature until well into adolescence. For example, face-selective cortex follows a slower developmental trajectory, not fully mature even in early adulthood (Germine et al., 2011), compared to object-selective cortex, which may be adult-like as early as age 5 (Gogtay et al., 2004; Golarai et al., 2015, 2010, 2007; Haist et al., 2013; Scherf et al., 2011, 2007) or even younger (Emberson et al., 2017; Nishimura et al., 2015). The pattern of word selectivity in the VWFA also emerges slowly over development, as reading and writing experience accumulate (Maurer et al., 2006), and orthographic representations may develop at the expense of face representations in the LH (Cantlon et al., 2011; Dehaene et al., 2015; Dundas et al., 2013). One prediction then is that differential patterns of maturation in the VOTC during childhood may have potentially different trajectories for recovery of function with greater opportunities for plasticity in later (face- or word-selective) than in earlier (object-selective) maturing areas.

Below, we review the 46 cases with a cortical lesion/resection in childhood, and we report the observed effects of the lesion on face, object recognition and/or word recognition.

2.2. Face recognition in pediatric patients

2.2.1. Early-onset cases (before 1 year of age)

One strong case of early onset of prosopagnosia, as mentioned above, is Adam who contracted streptococcal meningitis on Day 1 of life with lesions affecting VOTC bilaterally (Farah et al., 2000; Farah and Rabinowitz, 2003) (see Table 1). When tested at 16 years of age, Adam showed a profound impairment in face recognition (unable to identify a single face from his favorite TV show) and a moderate deficit in object recognition (Naming: nonliving things 75% correct; living things 40% correct. Although the conclusion is that Adam's face recognition deficit is more severe than his object recognition deficit, the deficits may not be directly comparable given that the former was tested at the individual/exemplar level while the latter was tested at the basic level (and is seemingly less affected).

In light of the prominent and persistent prosopagnosia, Farah et al. (2000) stated that there is an early commitment of neural substrate to face recognition and that, because no other region of cortex was able to compensate for the VOTC lesions, recovery of function was not possible. We note, however, that Adam's bilateral lesion resulted in many elementary visual deficits such as esotropia, amblyopia, reduced acuity and visual field abnormalities, and thus the lack of reliable input, rather than a fundamental limit on plasticity per se, may have hindered Adam's development of face and object recognition.

KD also suffered from a bilateral occipital lesion due to meningococcal meningitis and was cortically blind for a few weeks in infancy (at 14 month) (Ellis and Young, 1988; Young and Ellis, 1989). Following this illness, KD had difficulties with face and object recognition and, between 8 and 11 years of age, KD had deficits in both low- and high-level vision. As with Adam, KD's low-level visual deficits included poor visual acuity, reduced contrast sensitivity, and impaired color vision. KD was also severely impaired in topographic orientation, copying and drawing, and object recognition, but was disproportionately impaired in face recognition (correct: unfamiliar faces 13/20; familiar faces 10/20). An 18-month training program to improve face processing in KD yielded no improvement, further cementing the hypothesis that there is an early commitment of neural substrate and that it is immutable (Ellis and Young, 1988).

Perhaps surprising given the low-level visual deficits, KD showed age-appropriate reading skills (although the assessment does not evaluate reaction time (RT) and this may be compromised for accuracy; Gerlach et al., 2005). There are a number of interpretations of this pattern of prosopagnosia without concurrent alexia. One anatomical explanation is that, even though the damage was bilateral, the lesion was more severe in the right hemisphere, as indicated by the persistent left-sided motor weakness. An alternative is of a dissociation between two- and three-dimensional representations (Ellis and Young, 1988): whereas the deficit in face and object processing might be accounted for by the inability to form 3D visual representations, the recognition of 2D shapes might have supported the preservation of letter and word processing (see Table 1 for more details).

These cases of persistent prosopagnosia can be contrasted with findings from Mancini et al. (1994) who report data from six patients with unilateral lesions acquired prenatally or before age 1.[1] Three

[1] Although each patient's performance was compared to only one control subject, we assume high representativeness of those controls reflecting normal and comparable performance across tasks from both Table II and the following sentence in Mancini et al. (1994). "In the control group, the scores in the various tasks therefore did not vary significantly from one child to another despite the differences in age and IQ, whereas in the patient group the scores in the various tasks did differ significantly from one patient to another".

(Patients 1–3) had damage to the RH and the other three had damage to the LH[2] (Patients 11–13) When tested in later childhood (from 7 to 11 years), all children were able to recognize famous faces except for Patient 11[3] although 3 patients (1, 3 and 11) showed impaired identity discrimination (sorting faces into pairs). This advantage for the recognition over face matching is a little surprising unless the famous faces had salient distinguishing characteristics. Variability in face and non-face task performance was high and two patients with the same etiology (Sturge-Weber angiomatosis) and similar extent of right occipital angioma performed differently on tasks like lip reading, emotional expression and identity judgment. Taken together, the results showed that some aspects of face processing (famous faces and sex discrimination) did not differ from controls, whereas other skills were severely impaired (identity and lip reading) although the pattern varied from one child to another. Given the variability and perhaps the difference in the complexity of different tasks, reaching conclusions from this study is difficult. Also, in the absence of premorbid data, we do not know whether the children's visual abilities were ever affected or whether there was a deficit early on that subsequently recovered.

2.2.2. Later-onset cases (between 1 and 6 years of age)

All cases reviewed above had perinatal or prenatal cortical perturbation but there are also cases with later lesions. Barton et al. (2003) reported findings from three patients (two with bilateral lesions) whose onset of lesion ranged between 1 and 6 years of age (see Table 1 for more details). When tested in adulthood, all three showed severe face recognition deficits, with varying degrees of impairment in object recognition and/or basic visual perception such as spatial contrast, luminance, and spatial resolution. In particular, the authors noted that all three patients were impaired in encoding the spatial configurations of face features, implicating configural processing in within-category discrimination as critical for face recognition. Note that a deficit in configural processing is also observed in the individuals with early cataracts, possibly implicating a similar early mechanism in the cortical lesion patients (Maurer et al., 2007).

Hadjikhani and De Gelder (2002) provided the first systematic study probing the neural basis of childhood prosopagnosia (see Table 1 for more details). They tested two adults, both of whom suffered from a closed head injury, one at 18 months of age (GA), and the other at age 7 years (RP). As is often true in such cases, neither patient had an obvious lesion on an MRI scan. However, both displayed severe impairment in recognizing faces while their object recognition abilities were reported to be preserved (but no RT is reported and the difficulty of the object recognition tests is not matched to that of the face tasks). During a localizer scan, GA showed no face-selective activation (i.e., a stronger response to faces than objects) anywhere in the VOTC whereas RP showed activation for both faces and objects in typical object-selective cortex, inferior occipital gyrus (IOG) and lateral occipital cortex (LOC). The absence of face-selective activation anywhere in GA is surprising and even the cataract patients show face-selective activation, albeit reduced relative to controls (Grady et al., 2014). The findings are also counterintuitive as the child with the earlier lesion does not show recovery of recognition skills whereas the child with the later lesion does.

Another comparable case, MJH, suffered a fall at age 5 and sustained extensive bilateral lesions to VOTC (including regions that would normally encompass FFA and OFA) (Michelon and Biederman, 2003; Xu and Biederman, 2014). Despite being completely blind for a period of time immediately following his accident, MJH regained close-to-normal visual acuity and normal contrast sensitivity except for the persistence of tunnel vision. Roughly 40 years later MJH was still impaired in face individuation (Michelon and Biederman, 2003) and detection (Xu and Biederman, 2014), suggesting insufficient plasticity to restore face perception (multiple tests conducted between ages 34 and 45 years). MJH is less impaired in face imagery than in perception (Michelon and Biederman, 2003) and his performance in discriminating facial expression and sex (Mangini and Biederman, 2004) as well as object naming was in the normal range (Michelon and Biederman, 2003). He did, however, have difficulty comprehending abstract drawings and showed a mild learning disability involving arithmetic, spelling, handwriting, and slowed reading.

The 14 childhood studies reviewed above all refer to individuals who have suffered an insult to cortex rather than a resection of cortical tissue. It appears, however, that etiology is not necessarily a determining factor of the presence/absence of recovery. Mixed results can also be found in cases of right hemispherectomy even when the patients' age and etiology (epilepsy) are closely matched as in the next two case studies described below. Damásio et al. (1975) reported a case of a 34-year-old woman who had undergone a right hemispherectomy at age 20. She sustained a severe closed head injury at age 5 which later triggered medically intractable seizures, eventually requiring a right hemispherectomy. Of course interpreting the nature of the neural changes in such a case is challenging given the long time line and the infrequent testing to map the trajectory of change: it is impossible to know whether the prosopagnosia occurred as a result of the initial injury, the ensuing epilepsy or the surgery itself. Surprisingly, this individual was not hemianopic after the surgery. She did, however, have bilateral optic atrophy prior to the surgery and, when tested 14 years post-surgery, there was a gradual suppression of vision in the left eye (eventually blind) and enlargement of the right eye's visual field. Post-surgical testing of face recognition did not show any impairment in the discrimination of familiar or unfamiliar faces, and reading of letters, words, digits, and numbers was normal. One possible interpretation is that considerable reorganization (shift of recognition skills to LH because of more severe insult to RH) might have occurred post injury and thus face and word/letter recognition were normal postsurgery. The absence of data pre-surgery and post injury also make it difficult to determine exactly when and what kind of reorganization occurred.

In contrast, patient BM was a 33-year-old woman who underwent right hemispherectomy to manage her medically intractable epilepsy at age 13 (Sergent and Villemure, 1989). Post-surgically BM had a dense left homonymous hemianopia, impaired contrast sensitivity in the low spatial frequency range and profound prosopagnosia across a range of tests, despite being unaware of her impairment. No obvious object agnosia was found for common objects but she did show a deficit in discriminating between highly similar objects. In addition, she had normal reading and good comprehension. Writing to copy and dictation, as well as spelling, were unimpaired, too.

2.2.3. Interim summary

Taken together, there appears to be no straightforward relationship between the etiology, time of onset, localization of the lesion and the severity or recovery of the impairment in face processing. Restitution or sparing of face recognition is clearly possible as shown in at least 5 out of 6 patients from Mancini et al. (1994) and Damásio et al. (1975), but persistent impairment is noted in the other 10 prosopagnosic cases, even when the nature of lesion, time of surgery and testing period post-surgery seem roughly similar. One definitive conclusion is that some aspects of face recognition can be spared or restored after both early and later lesion. These findings challenge the extreme hypothesis that there is a dedicated neural substrate for face recognition and no possibility for reorganization (Farah et al., 2000).

Of course this leaves open the question why recovery ensues in some but not all cases. One possibility, as alluded to previously concerns the

[2] Although a RH lesion is more commonly associated with prosopagnosia, there are reports of prosopagnosia associated with a left occipitotemporal lesion (Barton, 2008) and of a patient with pre-existing right-sided infarcts who only became prosopagnosic following a subsequent left occipitotemporal hemorrhage (Ettlin et al., 1992).

[3] Surprisingly, subject 11 is not considered prosopagnosic. "Despite his zero score recognition of famous faces, subject 11 was not prosopagnosic, since when presented with his mother's and his sister's photographs, he was able to recognize them." Mancini et al. (1994), p. 162.

quality of the input. If low-level vision is affected, with poor acuity, and poor contrast sensitivity, this may impede any experience-dependent maturation. The failure to acquire face recognition post-lesion (even with intervention (as in Ellis and Young, 1988; Young and Ellis, 1989)), then, may be a result of poor input to the system rather than the consequence of a dedicated and immutable cortical region. The absence of recovery may also result from the fact that the remaining hemisphere is 'crowded out' i.e. the LH is already committed to particular functions, making it incompatible with new skills although why this is true in some but not all cases is unclear.

A further possible determinant of recovery concerns whether the damage is unilateral or bilateral in nature. In the early onset cases, those with persistent prosopagnosia have bilateral deficits and those with recovery mostly have unilateral lesions. The same distinction may hold in those cases with later onset (Barton et al., 2003 with 2 of 3 cases having bilateral lesions, and Hadjikhani and De Gelder (2002) whose patients had closed head injuries). This unilateral/bilateral distinction, again, does not seem to hold in all the hemispherectomy cases, however, as two cases (Damásio et al., 1975; Sergent and Villemure, 1984), had unilateral right hemispherectomy and yet only one recovered face recognition abilities.

2.3. Object recognition in pediatric patients

Pediatric patients with only or predominantly object agnosia without major difficulties in other domains (e.g., faces, words) are relatively rare and perhaps even more rare than prosopagnosia without concomitant object agnosia.[4] One possible reason for this rarity is that objects typically elicit more extensive and distributed activation in the VOTC compared to face- or word-selective activation (Grill-Spector, 2003; Haxby et al., 2001) so that, even after damage, some neural tissue in this region may be preserved. In those cases who do show object agnosia, then, the chances of subsequent recovery might be low, as opportunities for functional reorganization may be precluded by the extensive lesion that likely caused the agnosia in the first place.

2.3.1. Cases with no or limited recovery

One of the most severe cases, AR, presented with object agnosia, prosopagnosia and color agnosia after contracting herpes encephalitis that resulted in extensive atrophy in the right temporal lobe and in a portion of the left inferotemporal region (Schiavetto et al., 1997) (see Table 2). Over the seven-year follow-up, AR showed limited recovery despite the proficient use of compensatory strategies such as feature-by-feature analysis for object identification and spatial localization for memorizing the position of objects (perhaps reflecting preserved parietal lobe function).

A similar but perhaps even more dramatic case is SB who contracted meningoencephalitis at age 3, resulting in extensive bilateral ventral lesions and a lesion in the right dorsal pathway (Lê et al., 2002) (see Table 2). Tested at age 30 (27 years post-onset), SB had a severe recognition deficit for objects, faces and words. He also had a range of low-level visual deficits such as impaired color vision, texture perception, and contrast sensitivity (in the high spatial frequency range) and no obvious recovery of his hemianopia. However, he showed surprising visuomotor competence, as reflected by his ability to orient and shape his hand for reaching and grasping objects. The authors concluded that the preservation of the visuomotor sensitivity is mediated by the residual dorsal pathway (Goodale and Milner, 1992), consistent with findings that the magnocellular-dorsal pathway supports residual visual perception functions for action (although see Rossit et al., 2017, for some recent counter-evidence).

[4] We have not included cases of childhood object agnosia with no obvious structural abnormalities such as patient LG (Ariel and Sadeh, 1996; Gilaie-Dotan et al., 2009) or MJ (Martinaud et al., 2015).

2.3.2. Cases with some recovery in low- or high-level vision

A different pattern, this time of some recovery of low-level visual skills, is reported in a case of object agnosia with relatively preserved face and word recognition (Bova et al., 2008). This patient suffered a bilateral V1 lesion at the age of 2 years 6 months (hemorrhagic infarcts and blood perfusion deficit were seen on imaging in both occipital lobes). Within 4 years of recovery, the patient progressively regained low-level visuoperceptual functions, starting with the recovery of motion perception, to an improvement in visual acuity and gradual reduction of the visual field defect. Comprehensive testing revealed average visuomotor integration skills (less than 50th percentile), but poor visual object recognition as measured on the Birmingham Object Recognition Battery (Riddoch and Humphreys, 1993), poor recognition of overlapping figures (Poppelreuter-Ghent Test, 3rd percentile), reduced gestalt perception (Street Completion Test, 10th percentile), and poor identification of objects viewed from unusual perspectives and under unusual lighting conditions (3rd and 1st percentiles respectively, see Table 2). Face and word recognition were reported to be relatively spared although performance was still poor (approximately 20th percentile on Test of Memory and Learning (TOMAL) facial memory subtest; no data on word recognition reported). Taken together, the pattern of visual recovery over a 4-year period in this child suggests differential patterns of functional recovery in low- vs. high-level vision: the acuity and the span of the visual field appear to have resolved, but the deficit in higher-order visual recognition persisted.

The hypothesis that intact low-level visual abilities early on are crucial for the development of high-level vision was directly examined in a recent fMRI study (see Table 2) (Hu et al., 2013). Patient CGN suffered a subdural hematoma in the left occipitoparietal cortex at the age of one year. At the age of 12.8 years, or about six years after the surgical removal of the subdural hematoma, CGN's low-level vision had improved significantly, including a largely alleviated hemianopia and this was reflected in the functional activation in the left occipital cortex. Importantly, although the left temporal cortex was activated by visual stimuli (faces, words, objects and scrambled objects minus the fixation baseline), the response was not object-selective (defined by the contrast of the object categories (faces, words and objects) versus scrambled objects). The right inferotemporal cortex revealed normal object-selective activation, indicating relatively uncompromised recognition when objects appeared in the good visual field. Taken together, the parallel findings between behavior and the neural basis of object recognition suggest that visual inputs from the early visual cortex at a young age may be necessary to trigger not just the activation but also the fine-tuned development of high-level visual functions in object-selective cortex (although note that the RH was spared at all stages and this may be the basis of the normal recognition). The differential trajectories of functional recovery in low- and high-level vision in both Bova et al. (2008) and Hu et al. (2013) echo the quality of the input argument mentioned previously. That is, the development of category-selective regions might depend on visual inputs at an early age, and early-onset disorders of low-level vision may impede experience-dependent maturation. Even the gradual recovery of low-level vision (within about 6 years) appears to be insufficient and category-selective behavior and neural responses remain abnormal (Bova et al., 2008; Hu et al., 2013).

Perhaps the most successful recovery of object recognition was evident in patient SR who suffered a diffuse lesion in the frontal, occipital, and parietal lobes as a result of encephalitis at 8 weeks of age (Funnell and Wilding, 2011). Although SR was not tested premorbidly (and testing of an 8 week old is not likely to be very informative), we assume that SR's poor recognition is a direct consequence of the encephalitis. SR then showed gradual, albeit slow, recovery of object recognition over the 8 years of the longitudinal investigation: visual assessment at age 10 years revealed normal basic visual abilities such as contrast sensitivity, convergence, color vision, and pursuit movement. Also, although her ability remained poorer than that of controls, SR was

Table 1
Studies on functional outcome/recovery of face recognition in patients with a lesion to higher-order visual cortex.

Study	Subject	Etiology	Lesion/resection	Onset	Surgery	Test	Vision test	Outcome
Barton et al. (2003)	GA	Cardiopulmonary arrest and coma	No lesions evident on the MRI scan	1 y	No surgery	21 y	**Low- and mid-level vision**: visual acuity, visual fields, contrast sensitivity, luminance discrimination, saturation discrimination, spatial resolution, dot displacement discrimination, curvature discrimination **High-level vision**: *Face*: BFRT,[a] WFMT,[b] Famous Faces test,[c] covert recognition, configurational perception *Object*: Benton line orientation, VOSP,[d] Ghent overlapping figures, within-category judgements	**Low- and mid-level vision**: 20/20 visual acuity, full visual fields, mildly impaired to normal low-level vision **High-level vision**: impaired face perception /recognition, mildly impaired to normal object perception/recognition
	KBN	Epilepsy	Focal developmental polymicrogyria in both occipital lobes	2 y	No surgery	31 y		**Low- and mid-level vision**: 20/50 visual acuity, mildly constricted visual fields, impaired low-level vision **High-level vision**: impaired in both face and object recognition/perception, particularly for unusual views and line drawings. Reading speed and comprehension were slow.
	KT	Respiratory arrest (idiopathic origin) and in vegetative state for 2 m	Diffuse gyral atrophy in bilateral posterior hemispheres	6 y	No surgery	36 y		**Low- and mid-level vision**: gradual recovery from near blind to 20/50 visual acuity, homonymous partial left inferior quadrantanopia, severely impaired low-level vision **High-level vision**: severely impaired in both face and object recognition/perception and reading (triad of alexia, prosopagnosia and visual agnosia)
Damasio et al. (1975)	N/A	Closed head injury followed by epilepsy	Presurgical bilateral optic atrophy, right frontal and parietal lesion, right hemispherectomy	5 y	20 y	34 y	**Low-level vision**: visual acuity, visual fields, detection of fixed or moving targets or flash of lights **High-level vision**: *Face*: BFRT[a]	**Low-level vision**: decreased visual acuity and blind left eye (pre-surgery), no hemianopia and enlarged visual field (post-surgery) **High-level vision**: no impairment in the discrimination of familiar faces nor of unfamiliar faces, normal reading for letters, words, digits, and numbers
Farah et al. (2000)	Adam	Group B streptococcal meningitis	Bilateral occipital and occipitotemporal lesions	Day 1 after birth	No surgery	16 y	**Low-level vision**: visual acuity, visual fields **High-level vision**: *Face*: recognition of face photographs, face matching (BFRT[a]) *Object*: recognition of object photographs, recognition of line drawings of objects[f]	**Low-level vision**: 20/80 visual acuity for distance and 20/40 for near vision, left central homonymous hemianopia, incomplete right homonymous hemianopia with macular sparing, esotropia, amblyopia (left eye) **High-level vision**: *Face*: profoundly impaired face recognition *Object*: no discernable object agnosia with real objects in everyday life, but have some difficulty with photographs of objects
Hadjikhani and de Gelder (2011)	GA	Closed head injury	No lesions evident on the MRI scan	18 m	No surgery	27 y	**Behavioral**: **Low-level vision**: Benton Visual Form Discrimination, Benton line orientation[g] **High-level vision**: *Face*: BFRT[a], Warrington recognition memory test[h] *Object*: Boston naming test, BORB,[i] picture naming[j] **fMRI**: localizer (faces, scrambled faces, objects, scrambled objects)	**Behavioral**: **Low-level vision**: within normal range **High-level vision**: impaired face recognition, preserved object recognition **fMRI**: *Face*: no face-selective (faces-objects) or face-sensitive (faces-houses) activation in the ventral visual pathway *Object*: object-related activation was found in inferior occipital gyrus (IOG) and lateral occipital cortex (LOC)
	RP	Closed head injury		7 y		49 y		**Behavioral**: **Low-level vision**: within normal range **High-level vision**: severely impaired face recognition, preserved object recognition

(continued on next page)

Table 1 *(continued)*

Study	Subject	Etiology	Lesion/resection	Onset	Surgery	Test	Vision test	Outcome
								fMRI: *Face*: no face-selective activation (faces-objects) in the ventral visual pathway, face-sensitive (faces-houses) activation was found in IOG *Object*: object-related activation was found in IOG and LOC
Mancini et al. (1994)	1	Sturge-Weber angiomatosis	Right occipital lesion	Prenatal	No surgery	7 y 9 m		**Low-level vision**: all had normal visual acuity and visual fields
	2	Sturge-Weber angiomatosis	Right occipital lesion	Prenatal		8 y 5 m	**Low-level vision**: visual acuity, visual fields	**High-level vision**: none was prosopagnosic, but with varying degrees of face recognition, reading and speech abilities
	3	Ischaemia	Right frontal porencephalic cyst and right hemispheric atrophy	10 m		11 y 9 m	**High-level vision**: *Face*: famous faces recognition, faces with speech/no-speech, lip reading, emotion expression, face identification, divided visual field of unfamiliar faces	
	11	Hypoxia-ischaemia	Left temporo-parietal porencephalic cyst	Prenatal		7 y 11 m		
	12	Ischaemia	Left anterior cerebral territory	Prenatal		8 y 1 m		
	13	Ischaemia	Sylvian territory	Prenatal		10 y		
Behavioral: Michelon and Biederman (2003) fMRI: Xu and Biederman (2014)	MJH	Head injury from fall off a high platform	Bilateral lesions (greater in the right hemisphere) to the ventral occipito-temporal cortices	5 y	No surgery	34 y, 45 y	**Low-level vision**: visual fields, visual acuity, contrast sensitivity **High-level vision**: *Face*: celebrity face naming, celebrity–non celebrity forced-choice task,[k] BFRT[a], CFMT,[l] match-to-sample test[m] *Object*: Boston naming test *Reading*: Wide Range Achievement Test, Gray Oral Reading test	**Low-level vision**: tunnel vision beyond approximately 10° in the RVF, re-gained close-to-normal vision, normal visual acuity and contrast sensitivity in foveal vision **High-level vision**: **Behavioral:** *Face*: severely impaired face perception, less impaired face imagery, a general reduced speed in making face judgments *Object*: average range in Boston naming test *Reading*: mild learning disability involving arithmetic, spelling, and handwriting, slowed reading speed **fMRI:** *Face*: no face-selective (faces-objects) activation in the ventral occipito-temporal lobe *Object*: normal activation to objects compared to their scrambles in the spared lateral occipital cortex (LO), but not in the lesioned posterior fusiform gyrus (pFs)
Sergent and Villemure (1989)	BM	Epilepsy	Right hemispherectomy	5 y	13 y	33 y	**Low-level vision**: visual acuity, visual fields, contrast sensitivity, color **High-level vision**: *Face*: age and sex classification, Mooney face, facial expressions, face matching at different resolution, BFRT,[a] face dissimilarity-judgement task, familiar persons	**Low-level vision**: 20/20 visual acuity, left homonymous hemianopia with macular splitting, impaired contrast sensitivity in the low spatial frequency range, no achromatopsia **High-level vision**: *Face*: severe prosopagnosia (unawareness of her impairment) *Object*: no object agnosia despite deficit at discriminating between closely similar objects

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Table 1 *(continued)*

Study	Subject	Etiology	Lesion/resection	Onset	Surgery	Test	Vision test	Outcome
							Object: VCVC,[o] object recognition in unconventional perspective[p]	*Reading*: normal reading
Young and Ellis (1989); Ellis and Young (1988)	KD	Meningococcal meningitis	Bilateral occipital damage	14 m	6 surgeries	Multiple tests between 8 y 9 m–11 y 7 m	**Low-level vision**: visual acuity, contrast sensitivity, color vision, spatial abilities **High-level vision**: *Face*: face classification, face descriptions, age and sex classification, facial expressions, identify matching, recognition of familiar faces, person recognition *Object*: object recognition using line drawings[f] and photographs, object matching, objects in unusual view,[r] etc	**Low-level vision**: gradual improvement of visual acuity to 6/18 at age 11 y, impaired contrast sensitivity at all spatial frequencies; achromatopsia, impaired spatial abilities **High-level vision**: *Face*: disproportionately severely impaired face recognition *Object*: impaired object recognition *Reading*: near normal for her age

[a] Benton face recognition test (Benton and Van Allen, 1972).
[b] Warrington recognition test for words and faces (Warrington, 1984).
[c] Famous Faces test (Albert et al., 1979).
[d] Warrington visual object and space perception battery (Warrington and James, 1991).
[f] Recognition of line drawings of objects, stimuli were selected from Snodgrass and Vanderwart (1980) corpus of line drawings.
[g] Subtests from the Birmingham Object Recognition Battery (BORB; Riddoch and Humphreys, 1993).
[h] Recognition memory test (Warrington, 1984).
[i] Birmingham Object Recognition Battery (Riddoch and Humphreys, 1993).
[j] Picture naming task (Snodgrass and Vanderwart, 1980).
[k] Celebrity–non celebrity forced-choice task (Mangini and Biederman, 2001).
[l] Cambridge Face Memory Test (Duchaine and Nakayama, 2006).
[m] Match-to-sample test (Yue et al., 2012).
[o] Visually Cued Verbal Cards (Foster et al., 1983).
[p] Object recognition in unconventional perspective (Warrington and Taylor, 1978).
[r] Objects in unusual view (Humphreys and Riddoch, 1984, 1985).

Table 2
Studies on functional outcome/recovery of object recognition in patients with a lesion to the higher-order visual cortex.

Study	Subject	Etiology	Lesion/resection	Onset	Surgery	Test	Vision test	Outcome
Bova et al. (2008)	N/A	Vascular lesions in both occipital lobes	Bilateral occipital lobe infarction	2 y 6 m	No surgery	Multiple tests between 2 y 6 m − 6 y 8 m	**Low-level vision**: visual acuity, visual fields **Mid-level vision**: DTVP,[a] gestalt perception[b] **High-level vision**: *Face*: TOMAL facial memory subtest[c] *Object*: BROB, overlapping figures,[d] unusual perspectives/lighting, internal representation	**Low-level vision**: progressive recovery of visual acuity and oculomotor abilities, progressive reduction of visual field defect **High-level vision**: *Face*: mildly impaired face recognition (< 20 percentile) *Object*: severely impaired object recognition (< 5 percentile) *Reading*: age-appropriate reading and writing skills
Fazzi et al. (2009)	22 children born preterm	Periventricular leukomalacia	Lesions along the primary optic pathway, in the occipitoparietal and occipitotemporal regions	Preterm	No surgery	Mean age: 8 y Age range: 6–15 y	**Low-level vision**: oculomotor abilities, visual acuity, contrast sensitivity, optokinetic nystagmus, visual field, stereopsis **Mid-level vision**: gestalt perception[b] **High-level vision**: *Face*: TOMAL facial memory subtest[c] *Object*: objects recognition using photographs, overlapping figures[d] *Letter*: recognition of the 21 letters of the Italian alphabet	**Low-level vision**: All had normal or near-normal visual acuity and full visual field, 17/22 had strabismus **Mid-level vision**: Impaired Gestalt perception: 5/22 **High-level vision**: Impaired object recognition: 15/22 Impaired face recognition: 4/22 Impaired letter recognition: 0/22
Funnell and Wilding (2011)	SR	Encephalitis	Extensive areas of attenuation in the frontal, occipital, and parietal lobes	8 w	No surgery	Multiple tests between 6–14 y	**Low-level vision**: visual acuity, visual fields, contrast sensitivity, color vision, oculomotor abilities **Mid-level vision**: form segmentation, illusory form/contour, proximity, collinearity, global and local processing, etc **High-level vision**: *Face*: face recognition *Object*: object recognition,[e] object naming[f]	**Low-level vision**: normal visual acuity, normal sensitivity for high spatial frequencies, impaired sensitivity in the middle to low range, normal color vision, convergence, and pursuit movement **Mid-level vision**: weak perceptual segmentation and limited use of perpetual grouping cues **High-level vision**: *Face*: profound prosopagnosia *Object*: gradual visual-perceptual development, slow acquisition of shape information *Reading*: learned to read and write all the letters of the alphabet and numerals
Hu et al. (2013)	CGN	Subdural hematoma and underdeveloped left occipitoparietal cortex	Left superior parietal lobule, significant atrophy in the left occipitoparietal cortex	1 y	6 y 7 m	12 y 8 m	**Low-level vision**: visual fields, clinical confrontation test, line bisection test[g] **High-level vision**: *Face*: not tested *Object*: rapid object naming test, spontaneous painting test (used to confirm hemianopia) **fMRI**: meridian mapping, category localizer (faces, objects, words, scrambled objects)	**Low-level vision**: gradual recovery from right hemianopia (6 y 7 m) to full visual fields (12 y 8 m) **High-level vision**: *Face*: N/A *Object*: named 3/6 objects in the LVF, 2/6 in the central field, and 0/6 in the RVF (due to hemianopia) *Reading*: age-appropriate reading **fMRI**: *Early visual cortex*: bilateral activation in early visual cortex, partially restored visual inputs

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Table 2 *(continued)*

Study	Subject	Etiology	Lesion/resection	Onset	Surgery	Test	Vision test	Outcome
								from early to higher-order visual cortex *Face*: normal face-selective activation in the right FFA and STS *Object*: normal object-selective activation in the right inferior temporal cortex, no object-selective response in the left temporal cortex *Reading*: word-selective activation in the right VWFA, despite left hemisphere dominant language processing
Lê et al. (2002)	SB	Meningoencepha-litis	Extensive bilateral lesion of the ventral pathway and lesions in the right dorsal pathway	3 y	No surgery	30 y	**Low-level vision**: visual acuity, contrast sensitivity, visual fields, orientation perception, brightness judgements, color perception, texture perception, depth perception **High-level vision**: *Face*: face discrimination, decision and detection task *Object*: object recognition (BORB[e], complex Rey's drawing), real object identification	**Low-level vision**: 6/10 acuity, left lateral homonymous hemianopia with macular sparing, impaired contrast sensitivity in the high spatial frequency range, achromatopsic, impaired texture perception, normal depth perception **High-level vision**: *Face*: severe prosopagnosia *Object*: severe object agnosia *Reading*: cannot read words/letters but can read and wrote Braille fluently
Schiavetto et al. (1997)	AR	Herpes encephalitis	Atrophy in the right temporal lobe and a portion of the left inferotemporal region	9 y	No surgery	Multiple tests between 9–16 y	**Low-level vision**: visual acuity, contrast sensitivity, convergence, stereoscopic vision, visual fields **High-level vision**: *Face*: recognition of primary emotion, face matching across orientation, famous faces test *Object*: naming drawings of common objects and geometric figures, identifying overlapping figures, etc	**Low-level vision**: normal visual acuity, contrast sensitivity, convergence, stereoscopic vision and visual fields **High-level vision**: *Object*: object agnosia *Face*: prosopagnosia *Reading*: acquired surface dyslexia and dysgraphia *Other aspects*: color agnosia

[a] Developmental Test of Visual Perception (DTVP; Voress et al., 2004).
[b] Street Completion Test (Street, 1931).
[c] TOMAL facial memory subtest (Reynolds and Bigler, 1994).
[d] Poppelreuter–Ghent Test (Della Sala et al., 1995).
[e] Birmingham Object Recognition Battery (BORB; Riddoch and Humphreys, 1993).
[f] Word Finding Vocabulary Test (Renfrew, 1995).
[g] Line bisection test (Barton and Black, 1998).

Table 3
Studies on functional outcome/recovery of word recognition in patients with a lesion to the higher-order visual cortex.

Study	Subject	Etiology	Lesion/resection	Onset	Surgery	Test	Vision/language test	Outcome
O'Hare et al. (1998)	N/A	Haemophilus influenzae meningitis	Bilateral occipital lobe infarction	2.5 y	No surgery	Multiple tests between 2–10 y	**Low-level vision**: visual acuity, visual fields **High-level vision**: *Face*: face matching,[a] familiar face identification *Object*: picture and drawing identification *Reading*: letter and number identification	**Low-level vision**: gradual recovery of acuity left lower-quadrantanopia, impaired depth perception **High-level vision**: *Face*: prosopagnosia *Object*: object agnosia *Reading*: pure alexia with spared recognition of letters and numbers *Other aspects*: some degree of topographic agnosia
Cohen et al. (2004)	N/A	Sturge–Weber angioma, focal seizures	Left occipitotemporal resection	4 y	5 y	11 y	**Low-level vision**: N/A **High-level vision**: *Face*: N/A *Object*: N/A *Reading*: spoken and written language production and comprehension **fMRI**: speech processing (auditory), word reading (visual)	**Low-level vision**: N/A **High-level vision/cognition**: *Face*: N/A *Object*: N/A *Reading*: Normal reading abilities supported by right VWFA despite a strong left lateralization of all other language-related activations (Broca's area, superior temporal sulcus, inferior parietal lobule)
Danelli et al. (2013)	EB	A massive expanding process through the left frontal-insular area (diagnostic suspicion of a large cavernous angioma)	Left hemispherectomy	2 y 6 m	2 y 6 m	Multiple tests between 14–17 y	**Low-level vision**: visual acuity, visual fields, line orientation test[b] **High-level vision**: *Face*: Facial Recognition Task[c] *Object*: BORB[d] *Language*: phoneme discrimination test,[e] non-word repetition test,[f] Test for the Reception of Grammar,[g] the Token Test,[h] etc. *Reading*: Sartori et al. (1995) [i] battery, visual lexical decision task, visual homophone discrimination task, etc. **fMRI:** (1) automatic word generation (2) phonemic and semantic word fluency (3) word listening (4) plausibility decision task on sentences (5) word and non-word reading	**Low-level vision**: homonymous right hemianopia, absence of stereopsis, normal orientation perception **High-level vision/cognition**: *Face*: normal range *Object*: normal range *Language*: near-to-normal language production and comprehension Reading: below normal level in visual lexical decision task and visual homophone discrimination task, despite normal reading accuracy and speed **fMRI**: Activation in the right language network conforms to a left-like linguistic neural blueprint The right VWFA activation was not specific to reading

[a] Recognition memory test (Warrington, 1984).
[b] Line orientation test (Benton et al., 1978).
[c] Facial Recognition Task (Benton et al., 1978).
[d] Birmingham Object Recognition Battery (Riddoch and Humphreys, 1993).
[e] Phoneme discrimination test (Miceli and Caramazza, 1993).
[f] Non-word repetition test (Gathercole et al., 1994).
[g] Test for the Reception of Grammar (TROG Test; Bishop, 1982).
[h] The Token Test (De Renzi and Faglioni, 1978).
[i] Batteria per la valutazione della dislessia e della disortografia evolutiva (Sartori et al., 1995).

able to learn to perceive visual shape to some extent. Presumably the early timing of lesion, the fact that temporal cortex was spared and the etiology (encephalitis) might be key to the extent of plasticity and recovery.

Similar to these five in-depth case reports of childhood object agnosia (some of whom showed recovery and some did not), there are mixed reports of impaired or preserved object recognition associated with perinatal lesion. For example, 15 out of 22 children born preterm with periventricular leukomalacia affecting the retinogeniculate visual pathways (Fazzi et al., 2009, see Table 2) showed an object recognition deficit when tested at age 6–15 years. In contrast, letter recognition was intact in all cases and face recognition and Gestalt perception were relatively spared (4/22 were impaired in the former and 5/22 in the latter task).

2.3.3. Interim summary

A few key points can be derived from the studies of object recognition impairment. One speculative comment is that the number of object agnosic cases in the literature is rather small, perhaps indicative of high rates of recovery given the relatively large cortical region implicated in object recognition. A dissociation between recovery of early and later parts of cortex might be possible: pattern recognition may continue to be adversely affected even though some lower-level visual abilities may be functional or even recovered over time. This pattern of dissociation is found in some cases of object agnosia (gradually recovered low-level vision: Bova et al., 2008 and Hu et al., 2013; normal or near-normal low-level vision: Fazzi et al., 2009 and Schiavetto et al., 1997) and prosopagnosia (gradually recovered low-level vision: MJH in Michelon and Biederman, 2003; Xu and Biederman, 2014; functional low-level vision: GA in Barton et al., 2003; Hadjikhani and De Gelder, 2002). It is the case, however, that there are a number of cases that have moderate to severe impairment in basic visual abilities and persistent category-level impairments (e.g., object agnosia: Lê et al., 2002; prosopagnosia: KBN and KT in Barton et al., 2003; Farah et al., 2000; Sergent and Villemure, 1989; Young and Ellis, 1989). Of note, in all of the cases with category-level impairments, it is possible to see differential recovery e.g., recovery of reading skills but ongoing marked prosopagnosia.

2.4. Word recognition in patients with an early-onset lesion

In this last section, we review the word recognition deficits in the early lesion/resection cases. Given that letter and word recognition is not typically acquired until roughly 4–6 years of age and that the mastery of the mappings between visual print (orthography) and phonological representations takes considerable experience (Schlaggar et al., 2007), case reports of pure word recognition deficits in childhood are extremely rare. One prediction, however, is that, given that the left VWFA and other areas associated with more linguistics aspects of reading (such as supramarginal gyrus) may not mature until later childhood, the prolonged developmental trajectory of reading acquisition may potentially offer greater opportunities for plasticity and reorganization. Because our focus is primarily on the visual system, we focus more on the VWFA than on other areas that support reading (e.g., supramarginal gyrus) and so the question is, whether the right VWFA can be functionally recruited to subserve normal reading after lesion or resection encompassing the left VWFA?

2.4.1. Cases with no or limited recovery

Danelli et al. (2013) studied the spoken and written language recovery of a 14-year-old adolescent (EB) who underwent a left hemispherectomy at the age of 2 years 6 months (see Table 3). At test, EB's language production and comprehension was near-to-normal and activation in his right language network conformed to the linguistic neural blueprint of the LH (another case of dramatic reorganization of speech and language skills). Curiously, given that the left hemispherectomy occurred before the formal acquisition of reading, no reading-specific activation was found in EB's right ventral occipitotemporal cortex (spatially congruent with a right-sided VWFA). In fact, this region was more activated in response to elementary shape matching than to orthography, and his reading profile was one of surface dyslexia, typically due to damage of the left parietal or temporal lobe. It is unclear whether the persistent dyslexia is a direct result of the resection of the left VWFA and LH language regions, whether there is 'crowding' of the right hemisphere or whether some other mechanism is at work here.

Similar to EB above, O'Hare et al. (1998) reported an impairment in a 10-year-old child with normal intelligence who sustained bilateral occipital-lobe infarctions at the age of 2.5 years. This child showed a broad deficit in visual cognition including alexia (although recognition of letters and numbers was spared), object agnosia, prosopagnosia, and some degree of topographic agnosia in various investigations conducted between age 8.5 and 10 years (see Table 3).

2.4.2. Cases with functional reorganization of VWFA

A seemingly contradictory finding was reported in Cohen et al. (2004) in an 11-year-old left-handed girl with a history of Sturge–Weber disease, who had surgical resection of the left occipital lobe at age 5 years. Left lateralization of language-related activations was found, including Broca's area, superior temporal sulcus, and inferior parietal lobule, and clinical assessment showed normal spoken language production and comprehension. Her reading performance was within the lower range of normal adults (although it is not clear why adults were used as controls here), despite her right hemianopia. Interestingly, the right VWFA was functionally recruited for word reading such that it was activated more strongly by words than by checkerboards whereas a comparable level of activation is found in normal readers. According to the authors, this interhemispheric shift may have taken advantage of direct transcallosal projections from the right occipitotemporal cortex to the preserved language areas in the LH. It is also unclear whether the patient's left-handedness afforded any advantages in the reorganization of the VWFA. In fact, reorganization to right VWFA is not uncommon and has also been reported in some adult cases (Fischer-Baum et al., 2017, see more discussion in 3.5 Mechanism of plasticity). Furthermore, in the case of pediatric patient CGN in Hu et al. (2013)[5] with additional details presented in the previous Section 2.3.2, CGN showed age-appropriate reading and word-selective activation in the right VWFA at age 12 following lesion to the left occipitoparietal cortex at the age of one year.

2.4.3. Interim summary

Varying degrees of functional recovery of reading are evident in these three case studies with a lesion encompassing the left VWFA. Although causation cannot be easily established, the degree of recovery of reading ability (ranging from normal to none) is likely to be associated with the degree to which the right VWFA can be functionally recruited in word reading. A unilateral lesion offers potential for remapping to the other hemisphere (as in Cohen et al., 2004 and Hu et al., 2013 but not in Danelli et al., 2013) whereas a bilateral lesion to the occipital lobe precludes opportunities for functional reorganization (O'Hare et al., 1998). Aside from a difference in the extent of the lesion, other possible explanatory factors that differentiate the presence versus absence of recovery in the 3 cases is that the individual with recovered reading was left handed and the resection occurred later than in the other cases (5 years versus 2.5 years).

3. Discussion

The goal of this review paper is to examine the profile of recovery of

[5] This case was presented in 2.3 Object recognition in patients with early-onset lesion, based on the primary impairment.

visual recognition skills in pediatric patients who have undergone a cortical resection or who have suffered a lesion to the visual system (e.g., post-meningitis). Elucidating the nature and extent of recovery has the potential both to inform our theories of cortical organization and to provide possible opportunities for optimizing recovery in individuals with brain damage. To achieve this goal, we reviewed the findings from 46 children, all of who evinced a deficit in visual recognition.

Unsurprisingly perhaps, we have been unable to uncover any cut and dried answers. The profile of plasticity across the reported cases is highly variable, and this variability holds for the recovery of face, object and word recognition skills. Out of the 16 cases with a lesion affecting face recognition, 10 showed no recovery (see Table 1). There was also no recovery of high-level visual function in 18 out of 27 cases with a lesion affecting object recognition despite varying degrees of recovery of low-level vision (see Table 2), and no recovery of reading in 2 out of 3 cases with a lesion affecting word recognition (see Table 3). Together, out of 46 cases (which is admittedly a relatively small sample), 30 cases showed no recovery and the remaining 16 showed recovery to varying extents. Although no straightforward conclusions are possible, and this is disappointing, the finding of such variability is informative in and of itself and raises a host of questions for future study.

3.1. Plasticity in high-level vision

In addition to surveying the cases and laying out the presence/absence of plasticity, a further goal was to determine whether the nature and extent of recovery might permit conclusions about brain-behavior organization. Specifically, we hypothesized that the absence of plasticity might reflect a modular organization in which a specific cortical region subserves a specific function in which case, after damage, no other region can assume the function of the damaged area. The opposite end of the outcome continuum is one in which full recovery occurs irrespective of the size or site of the damage as the claim is that all cortical regions are equipotential (at least in early childhood).

That we do not see full recovery in all 46 cases essentially rules out the equipotential viewpoint. We do not even see recovery even in a subset of cases with the earliest possible lesions (perinatal or meningitis on Day 1 of age), which would afford maximal opportunity for rewiring. The absence of recovery might be taken as compatible with the strictly modular view. As articulated clearly by Farah et al. (2000), the persistence of a deficit supports the notion of prespecified function and structure in the genome and no ensuing malleability.

The problem with adopting this immutability viewpoint, however, is undermined by the fact that there are some cases who do show restitution of function: for example, this is so for the patient who underwent a right hemispherectomy (Damásio et al., 1975), and for some (or possibly all) of the 5 patients with prenatal or very early postnatal unilateral lesion in Mancini et al. (1994). In the domain of object recognition, the patient who suffered an early lesion at eight weeks of age showed a gradual recovery of object recognition over at least 8 years (Funnell and Wilding, 2011) and the functional reorganization to right VWFA in both Cohen et al. (2004) and Hu et al. (2013)[6] also reflect orthographic pattern recognition following lesion or surgical resection. These demonstrations of recovery challenge the strong conclusion that the neural substrate for visual recognition (even for face recognition) is committed early (Farah et al., 2000).

As clear from the above discussion, we have ruled out the extreme hypothesis of no recovery as well as the hypothesis of full recovery and, consequently, are left with an inconsistent picture of plasticity for face, object and word recognition. The remaining question then is what principles govern reorganization in some but not all individuals? There is no unambiguous conclusion. One limitation is that patients are almost never tested premorbidly (many are too young at the time of resection or lesion). Also, in some cases plasticity might have occurred prior to the surgery and the brain might already have reorganized so that good visual recognition post-surgery may already be in place. Other limitations are that many patients are tested only once post-brain damage so determining the nature, trajectory and mechanism of recovery when it exists, is difficult. Last, anatomical information about the lesion is often limited, and, across studies, patients are tested with different tests, making comparisons difficult.

We also raise a caveat in the interpretation of the sparing or recovery of visual cognition in pediatric patients. 'Recovery' implies that there was an impairment initially and that it was followed by gradual improvement over time. For example, we used "recovery" in our analysis of the Funnell and Wilding (2011) study: longitudinal investigations into S.R.'s visual object recognition and naming between ages 6 and 14 years showed an initial impairment followed by gradual improvement over time. However, in cases of perinatal lesion in Mancini et al. (1994) where no premorbid data are reported, the varied performance across different face-related tasks (e.g., identity, expression, sex) was used to infer that some aspects of face processing could be developed while other aspects could not be learned with experience. The assumption is that the children would have been impaired in particular domains and so the skills they manifest at testing must have been acquired but we do not have strong evidence for an initial impairment and subsequent recovery.

3.2. What factors affect plasticity?

A host of characteristic properties are known to predict post-surgery prognosis (Stiles et al., 2005). Also, the earlier the resection the better the recovery (Bourne, 2010), and the data we report are consistent with this as functional restitution of aspects of face recognition is observed in those with prenatal injury (Mancini et al., 1994). Although these data do not directly implicate a specific critical period for the acquisition of high-level visual skills, in theory, damage that occurs within the 'sensitive' period of development may afford an optimal outcome (Hensch, 2005) if the damage occurs earlier (Staudt, 2010).

Given that experience is well known to tune the visual system (Gauthier et al., 2000; Gauthier and Tarr, 1997; McGugin et al., 2017), and that a stimulating, rich environment delivering good quality and widely varying inputs has the potential to trigger experience-dependent changes in the patient (Kolb, 1995), one might expect greater plasticity in domains to which we are exposed through daily experience. Yet, the 10 childhood prosopagnosic individuals had many years of experience with faces post-lesion (including a training program targeting face processing in KD; Ellis and Young, 1988) and none recovered the ability to recognize faces. Although the lesions in most cases occurred prior to the emergence of a mature face-processing brain network (Golarai et al., 2007; Scherf et al., 2007), which has a prolonged developmental trajectory even extending to age 30 years (Germine et al., 2011), recovery may simply take longer. Additional follow-up of these cases in later years might further illuminate the nature and extent of recovery from childhood lesion or surgery.

The absence of plasticity in many cases is consistent with the surprising absence of plasticity in individuals with the developmental or congenital form of prosopagnosia (CP) (Avidan and Behrmann, 2009; Behrmann and Avidan, 2005; Geskin and Behrmann, in press). These CP individuals fail to master face recognition notwithstanding their normal vision and normal cognition and intelligence. The difficulties they experience appear to be lifelong and no substantial recovery has been documented but some training studies do report some improvement (DeGutis et al., 2011, 2014). Although the absence of recovery is puzzling in these cases given the lack of an observable lesion, there may be

[6] This case was presented in 2.3 Object recognition in patients with early-onset lesion, based on the primary impairment but the presence of right VWFA activation is important in this context.

alterations in VOTC such as compromised white matter integrity (Thomas et al., 2008; but see Song et al., 2015) that prevents incidental plasticity in a way that mimics a frank lesion. In such individuals, presumably, the dysfunctional mechanism is still in situ, precluding major changes in topography and selectivity.

A further predictive factor concerns the extent of the lesion: the more circumscribed the cortical resection/lesion, the better the outcome and, as noted above, there is minimal plasticity possible in either low- and high-level vision following a bilateral lesion (Farah et al., 2000; Young and Ellis, 1989). In addition, more chronic etiologies do better than those with acute onset (e.g., the relatively slow growth profile associated with tumor potentially affords greater opportunity for plasticity and reorganization than does stroke: a sudden lesion (Mancini et al., 1994) or meningitis (Farah et al., 2000; Young and Ellis, 1989) may preclude the opportunity for reorganization. In contrast, it remains possible, at least in principle, that a slow growing tumor or epilepsy, which is managed medically before becoming intractable, may offer time for slow reorganization of function.

3.3. Low-level deficits and experience-dependent plasticity?

To what extent does the deficit in low-level vision impede plasticity? A differential pattern of functional recovery may be associated with the status of low-level vision. For example, Bova et al. (2008) documented recovery of their patient's visual functions from the age of 2.6 years to 6.8 years. Motion perception was fully recovered first at 2.9 years, followed by gradual recovery of visual acuity and visual field from age 2.6–5 years, and then complete recovery of basic visual–perceptual abilities at age 6.8 years. Fox et al. (2010) also noted that high-level neural circuits are dependent on the quality of the information provided by lower level circuits. Consistent with this, in this review, cases with limited or no recovery of high-level vision tend to be those in whom deficits in low-level vision also persist (Farah et al., 2000; Mancini et al., 1994; Sergent and Villemure, 1989; Young and Ellis, 1989).

Of course, a persistent deficit in low-level vision deprives the observer of experience and this deprivation plays an important role (Daw, 2003; Lewis and Maurer, 2005). As alluded to above, in children with congenital blindness or cataracts, even if only for a short period, the brief, early visual deprivation can lead to long-lasting deficits in face and holistic/configural processing (congenital cataracts: Le Grand et al., 2004, 2003, 2001) and object and form processing (congenital blindness: Ostrovsky et al., 2006). Early visual deprivation can also affect aspects of mid-level vision including the perception of global form and global motion (Ellemberg et al., 2005; Lewis et al., 2002), especially after binocular deprivation (Ellemberg et al., 2002), and surgery does not immediately ameliorate all aspects of the impairment (Gandhi et al., 2015). Similarly, global processing deficits have been reported in amblyopic observers (see Hamm et al., 2014 for a review), concurrent with deficits in global motion perception (dorsal visual stream), global form perception and holistic face perception (ventral visual stream).

Unsurprisingly, deprivation of visual signals in those who are congenitally blind also impedes the emergence of some aspects of higher-order vision. However, functional neural reorganization such as cross-modal plasticity has often been reported after sensory deprivation: for example, in the blind, 'visual cortex' can be activated by tactile stimulation (Büchel, 1998; Sadato et al., 1996), or auditory stimulation (Gougoux et al., 2005; Striem-Amit et al., 2012) or can even be recruited during symbolic math calculation (Kanjlia et al., 2016) or syntactic functions (Lane et al., 2017). In particular, a functional dissociation between a ventral "What" stream for the processing of object shape and a dorsal "Where" stream for the processing of space exists in blind subjects in both the auditory and tactile domain (Amedi et al., 2007; Collignon et al., 2011). For recent reviews on category selectivity in the blind, see Bi et al. (2016).

Taken together, the timing and quality of visual experiences, particularly during an early developmental period, can have cascading effects on the development of mid- and high-level vision. Of note, even if these visual deficits recover, category-level recognition impairments may persist but other aspects of high-level visual function can be preserved.

3.4. Category-selective or broader deficits?

For convenience, we have divided the reviewed cases into three subgroups depending on the primary deficit (although which is primary is sometimes debatable). Almost all, if not all, cases do not show a deficit that is limited to one visual category (see Tables 1–3), and the degree to which deficits, other than the primary deficit, recover also varies in these cases. In particular, the extent to which recognition abilities are spared or recovered is not consistent, suggesting that the lack of plasticity in high-level vision in the immature brain may not be strictly category-specific (Farah et al., 1995; McNeil and Warrington, 1993). Ten out of 16 cases with a deficit in face recognition, as reviewed in Table 1, show an impairment in object recognition and/or reading abilities, although this varies in severity. In fact, the presence of a fairly widespread deficit is worth noting: because tests of object and word processing are generally not as demanding as face recognition tests (Behrmann et al., 1998; Gauthier et al., 1999), uncovering a more general deficit indicates that the deficits might be rather severe. In cases of childhood object agnosia, none of the cases demonstrated normal face recognition (Bova et al., 2008; Funnell and Wilding, 2011; Lê et al., 2002; Schiavetto et al., 1997) and, in studies of childhood pure alexia, if the face and object recognition were tested, the pure alexia co-occurred with prosopagnosia and object agnosia (O'Hare et al., 1998). In addition, considering the possibility that the development of high-level visual function is initially undifferentiated and becomes increasingly functionally specialized in the mature brain (Durston et al., 2006), it makes sense that the effects of a lesion sustained during early development may not be specific to one particular function. In sum, a common pattern evident from early brain lesions is that of a general impairment that affects recognition in multiple visual domains.

3.5. Mechanisms of plasticity

Although the cases reviewed in this paper do not speak directly to underlying mechanisms of plasticity, multiple mechanisms ranging from molecular and cellular changes through alterations to systems and networks are known to contribute to and govern plasticity, and hundreds of studies have been designed to elucidate these neurobiological processes. Among the major type of neurobiological changes are increased neural sensitivity, increased neural specificity, strengthened neural connectivity and/or improved neural efficiency. Morphology of dendritic spines also affects plasticity (Burke and Barnes, 2006) and alterations and regulation of excitation and inhibition (E/I) circuit balance support changes in cortex (Bavelier et al., 2010). A comprehensive review is beyond the scope of this review paper but the reader is referred to review papers such as those by Johnston and colleagues for extended discussion of these topics Johnston et al. (2009, 2001) and by Cramer et al. (2011).

It is challenging to infer direct links between the known neurobiological mechanisms and the changes noted in the pediatric cases following CNS damage. Perhaps more easily conceptualized are system-wide changes that involve spontaneous intra-hemispheric shifts, such as changes in representational maps, e.g., the hand area can shift dorsally to invade the shoulder region following injury to the motor network (Nudo et al., 1996), changes in inter-hemispheric balance (Cramer et al., 1997; Feydy et al., 2002), distribution of activation or alterations in weighting of connections of a network (Grefkes et al., 2008) or compensation by a homologous region in spared hemisphere (Fischer-Baum et al., 2017). Additionally, as reviewed by Anderson et al. (2011), reduction of diaschisis and anatomical reorganization may also be key

to some of the functional changes we have reviewed. One new approach that offers an opportunity to document and characterize change especially in reactive plasticity is one in which Multi-Voxel Pattern Analysis (MVPA) can be used to compare the functional profile of the recovered behavior such as reading in right VWFA with the MVPA pattern of controls' left VWFA to evaluate similarity in representational organization post recovery (Fischer-Baum et al., 2017).

We have not considered intervention or treatment in any detail in this review. While there are invasive and noninvasive interventions to restore visual acuity in adult rodents or cats (see Table 1 in Bavelier et al., 2010), this work has not been done in humans. There is great interest in promoting plasticity in humans, for example following amblyopia (Levi et al., 2015), and, in the last decade, non-invasive brain stimulation and neuropharmacological approaches have made great strides, as well (Cramer et al., 2011). Of high practical significance, understanding neuroplasticity and exploiting the potential for change would be a significant clinical advance, paving the way for new approaches to functional rehabilitation following cortical damage in childhood or adulthood. Some of the same ideas may be exploited in improving performance and promoting generalization of function in neurologically normal individuals (Green et al., 2010) and the recognition of the protracted developmental profile of normal individuals provides further clues for times and ways in which to facilitate optimization of neural circuits (Somerville, 2016).

4. Conclusion

This review examines the extent of plasticity or restitution of function in children with a lesion to higher-order visual cortex and explores the ramifications of the surgery for cortical vision. Perhaps unsurprisingly, no clear patterns emerge. Together, the findings from the various studies of prosopagnosia, object agnosia and pure alexia (arbitrarily divided as children have more than one disorder) suggest an inconsistent picture with many cases showing no plasticity and a few revealing instances of marked recovery. That the disorders persist in some (perhaps the majority) of the cases is rather surprising given the protracted development of the VOTC during childhood and the opportunities for experience-dependent change, which ought to potentially offer greater opportunities for alternative patterns of brain organization. Although plasticity was not evident in many cases (Bova et al., 2008; Farah et al., 2000; O'Hare et al., 1998), this negative evidence needs to be considered in the broader context in which childhood agnosia, prosopagnosia, or pure alexia are rare, and the possibility of many more cases with substantial recovery may be high. These cases would not necessarily have been seen by clinicians nor reported in the literature.

The relative paucity of cases of visual recovery seems strikingly different from the reports of substantial restitution of function in language function following left hemispherectomy (Boatman et al., 1999; Telfeian et al., 2002; Vining et al., 1997). Because we have no way of knowing the base rates, calculating the proportion of recovered versus non-recovered cases is not possible. Also, as noted previously, because the premorbid function is not always unequivocally established, the presence of good language function post-damage may reflect the sparing of language function or may be the result of shifts of organization that even occurred pre-surgery in response to the effects of the lesion.

The clearest conclusion we can reach is that attempts at researching functional outcome/recovery in children with a lesion to higher-order visual cortex are rather disjointed and that more research is required. As such, the review stands as a call to action laying out hypotheses that ought to be tested in future studies. Although most individuals receive standardized neuropsychological testing, there are few established tests for childhood prosopagnosia or object agnosia. Unsurprisingly then, researchers often end up having to create their own tests for these diagnoses, making cross-cases comparisons difficult. But perhaps more critical for our understanding of plasticity is the inability to derive causal inference. Pediatric patients, especially those with prenatal/perinatal lesions, are usually not tested experimentally premorbidly. Also, many patients have only been tested once post-lesion (or surgery). The absence of longitudinal data makes it hard to infer whether plasticity was a consequence of the surgery, pre-surgical adaptation or typical development. Moreover, in many cases, the anatomical information about the lesion or resection is quite limited (i.e. to what extent is a region spared vs. affected), which further limits the interpretation of plasticity. Future studies may benefit from rigorous and careful testing taking into consideration, for example, visual complexity of the input across categories, better tracking the trajectory of change before and after surgery, extending the length of the follow-up period with more frequent measurements and characterizing the nature and extent of the resection or lesion as precisely as possible.

Acknowledgements

The writing of this paper was supported by grants from the National Institutes of Health to MB (EY027018) and by a Presidential Fellowship from Carnegie Mellon University to TTL.

References

Albert, M.S., Butters, N., Levin, J., 1979. Temporal gradients in the retrograde amnesia of patients with alcoholic Korsakoff's disease. Arch. Neurol. 36, 211–216.
Amedi, A., Stern, W.M., Camprodon, J.A., Bermpohl, F., Merabet, L., Rotman, S., Hemond, C., Meijer, P., Pascual-Leone, A., 2007. Shape conveyed by visual-to-auditory sensory substitution activates the lateral occipital complex. Nat. Neurosci. 10, 687–689. http://dx.doi.org/10.1038/nn1912.
Aminoff, E.M., Tarr, M.J., 2015. Associative processing is inherent in scene perception. PLoS One 10, e0128840. http://dx.doi.org/10.1371/journal.pone.0128840.
Anderson, V., Spencer-Smith, M., Wood, A., 2011. Do children really recover better? Neurobehavioural plasticity after early brain insult. Brain 134, 2197–2221. http://dx.doi.org/10.1093/brain/awr103.
Ariel, R., Sadeh, M., 1996. Congenital visual agnosia and prosopagnosia in a child: a case report. Cortex 32 (2), 221–240.
Avidan, G., Behrmann, M., 2009. Functional MRI reveals compromised neural integrity of the face processing network in congenital prosopagnosia. Curr. Biol. 19, 1146–1150. http://dx.doi.org/10.1016/j.cub.2009.04.060.
Avidan, G., Tanzer, M., Hadj-Bouziane, F., Liu, N., Ungerleider, L.G., Behrmann, M., 2014. Selective dissociation between core and extended regions of the face processing network in congenital prosopagnosia. Cereb. Cortex 24, 1565–1578. http://dx.doi.org/10.1093/cercor/bht007.
Barton, J.J., Black, S.E., 1998. Line bisection in hemianopia. J. Neurol. Neurosurg. Psychiatry 64, 660–662.
Barton, J.J.S., 2008. Prosopagnosia associated with a left occipitotemporal lesion. Neuropsychologia 46, 2214–2224. http://dx.doi.org/10.1016/j.neuropsychologia.2008.02.014.
Barton, J.J.S., Cherkasova, M.V., Press, D.Z., Intriligator, J.M., O'Connor, M., 2003. Developmental prosopagnosia: a study of three patients. Brain Cogn. 51, 12–30. http://dx.doi.org/10.1016/S0278-2626(02)00516-X.
Basser, L.S., 1962. Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. Brain 85, 427–460. http://dx.doi.org/10.1093/brain/85.3.427.
Bavelier, D., Levi, D.M., Li, R.W., Dan, Y., Hensch, T.K., 2010. Removing brakes on adult brain plasticity: from molecular to behavioral interventions. J. Neurosci. 30, 14964–14971. http://dx.doi.org/10.1523/JNEUROSCI.4812-10.2010.
Behrmann, M., Avidan, G., 2005. Congenital prosopagnosia: face-blind from birth. Trends Cogn. Sci. 9, 180–187. http://dx.doi.org/10.1016/j.tics.2005.02.011.
Behrmann, M., Nelson, J., Sekuler, E.B., 1998. Visual complexity in letter-by-letter reading: "pure" alexia is not pure. Neuropsychologia 36, 1115–1132. http://dx.doi.org/10.1016/S0028-3932(98)00005-0.
Behrmann, M., Plaut, D.C., 2015. A vision of graded hemispheric specialization. Ann. N. Y. Acad. Sci. 1359, 30–46. http://dx.doi.org/10.1111/nyas.12833.
Benton, A.L., Van Allen, M.W., 1972. Prosopagnosia and facial discrimination. J. Neurol. Sci. 15, 167–172.
Benton, A.L., Varney, N.R., Hamsher, K.D., 1978. Visuospatial judgment. A Clin. Test. Arch. Neurol. 35, 364–367.
Bernasconi, A., Bernasconi, N., Lassonde, M., Toussaint, P.J., Meyer, E., Reutens, D.C., Gotman, J., Andermann, F., Villemure, J.G., 2000. Sensorimotor organization in patients who have undergone hemispherectomy: a study with (15)O-water PET and somatosensory evoked potentials. Neuroreport 11, 3085–3090.
Bi, Y., Wang, X., Caramazza, A., 2016. Object Domain and Modality in the Ventral Visual Pathway. Trends Cogn. Sci. 20, 282–290. http://dx.doi.org/10.1016/j.tics.2016.02.002.
Bishop, D.V.M., 1982. Test for reception of grammar: trog-2. Pearson.
Boatman, D., Freeman, J., Vining, E., Pulsifer, M., Miglioretti, D., Minahan, R., Carson, B.,

Brandt, J., McKhann, G., 1999. Language recovery after left hemispherectomy in children with late-onset seizures. Ann. Neurol. 46, 579–586. http://dx.doi.org/10.1002/1531-8249(199910)46:4<579::AID-ANA5>3.0.CO;2-K.
Bouhali, F., de Schotten, Thiebaut, Pinel, M., Poupon, P., Mangin, C., Dehaene, J.-F., Cohen, L, S., 2014. Anatomical connections of the visual word form area. J. Neurosci. 34, 15402–15414. http://dx.doi.org/10.1523/JNEUROSCI.4918-13.2014.
Bourne, J.A., 2010. Unravelling the development of the visual cortex: implications for plasticity and repair. J. Anat. 217, 449–468. http://dx.doi.org/10.1111/j.1469-7580.2010.01275.x.
Bova, S.M., Giovenzana, A., Signorini, S., La Piana, R., Uggetti, C., Bianchi, P.E., Fazzi, E., 2008. Recovery of visual functions after early acquired occipital damage. Dev. Med. Child Neurol. 50, 311–315. http://dx.doi.org/10.1111/j.1469-8749.2008.02044.x.
Büchel, C., 1998. Functional neuroimaging studies of Braille reading: cross-modal reorganization and its implications. Brain 1193–1194. http://dx.doi.org/10.1093/brain/121.7.1193.
Buckley, R.T., Morgan, T., Saneto, R.P., Barber, J., Ellenbogen, R.G., Ojemann, J.G., 2014. Dysphagia after pediatric functional hemispherectomy. J. Neurosurg. Pediatr. 13, 95–100. http://dx.doi.org/10.3171/2013.10.PEDS13182.
Burke, S.N., Barnes, C.A., 2006. Neural plasticity in the ageing brain. Nat. Rev. Neurosci. 7, 30–40. http://dx.doi.org/10.1038/nrn1809.
Cantlon, J.F., Pinel, P., Dehaene, S., Pelphrey, K.A., 2011. Cortical representations of symbols, objects, and faces are pruned back during early childhood. Cereb. Cortex 21, 191–199. http://dx.doi.org/10.1093/cercor/bhq078.
Chandrasekaran, S., Navlakha, S., Audette, N.J., McCreary, D.D., Suhan, J., Bar-Joseph, Z., Barth, A.L., 2015. Unbiased, high-throughput electron microscopy analysis of experience-dependent synaptic changes in the neocortex. J. Neurosci. 35.
Cohen, L., Dehaene, S., Naccache, L., Lehericy, S., Dehaene-Lambertz, G., Henaff, M.-A., Michel, F., 2000. The visual word form area: spatial and temporal characterization of an initial stage of reading in normal subjects and posterior split-brain patients. Brain 123, 291–307. http://dx.doi.org/10.1093/brain/123.2.291.
Cohen, L., Lehéricy, S., Henry, C., Bourgeois, M., Larroque, C., Sainte-Rose, C., Dehaene, S., Hertz-Pannier, L., 2004. Learning to read without a left occipital lobe: right-hemispheric shift of visual word form area. Ann. Neurol. 56, 890–894. http://dx.doi.org/10.1002/ana.20326.
Collignon, O., Vandewalle, G., Voss, P., Albouy, G., Charbonneau, G., Lassonde, M., Lepore, F., 2011. Functional specialization for auditory-spatial processing in the occipital cortex of congenitally blind humans. Proc. Natl. Acad. Sci. USA. 108, 4435–4440. http://dx.doi.org/10.1073/pnas.1013928108.
Cramer, S.C., Nelles, G., Benson, R.R., Kaplan, J.D., Parker, R.A., Kwong, K.K., Kennedy, D.N., Finklestein, S.P., Rosen, B.R., 1997. A functional MRI study of subjects recovered from hemiparetic stroke. Stroke 28, 2518–2527. http://dx.doi.org/10.1161/01.STR.28.12.2518.
Cramer, S.C., Sur, M., Dobkin, B.H., O'Brien, C., Sanger, T.D., Trojanowski, J.Q., Rumsey, J.M., Hicks, R., Cameron, J., Chen, D., Chen, W.G., Cohen, L.G., deCharms, C., Duffy, C.J., Eden, G.F., Fetz, E.E., Filart, R., Freund, M., Grant, S.J., Haber, S., Kalivas, P.W., Kolb, B., Kramer, A.F., Lynch, M., Mayberg, H.S., McQuillen, P.S., Nitkin, R., Pascual-Leone, A., Reuter-Lorenz, P., Schiff, N., Sharma, A., Shekim, L., Stryker, M., Sullivan, E.V., Vinogradov, S., 2011. Harnessing neuroplasticity for clinical applications. Brain 134, 1591–1609. http://dx.doi.org/10.1093/brain/awr039.
Damasio, A., Lima, A., Damásio, H., 1975. Nervous function after right hemispherectomy. Neurology 25 (1), 89.
Danckert, J., Culham, J.C., 2010. Reflections on blindsight: neuroimaging and behavioural explorations clarify a case of reversed localisation in the blind field of a patient with hemianopia. Can. J. Exp. Psychol. 64, 86–101. http://dx.doi.org/10.1037/a0017426.
Danelli, L., Cossu, G., Berlingeri, M., Bottini, G., Sberna, M., Paulesu, E., 2013. Is a lone right hemisphere enough? Neurolinguistic architecture in a case with a very early left hemispherectomy. Neurocase 19, 209–231. http://dx.doi.org/10.1080/13554794.2011.654226.
Daw, N.W., 2003. Critical periods in the visual system. In: In: Hopkins, B., Johnson, S.P. (Eds.), Neurobiology of infant vision Praeger, Westport, CT, pp. 43–103.
De Koning, T., Versnel, H., Jennekens-Schinkel, A., Van Schooneveld, M.M.J., Dejonckere, P.H., Van Rijen, P.C., Van Nieuwenhuizen, O., 2009. Language development before and after temporal surgery in children with intractable epilepsy. Epilepsia 50, 2408–2419. http://dx.doi.org/10.1111/j.1528-1167.2009.02264.x.
De Renzi, E., Faglioni, P., 1978. Normative Data and Screening Power of a Shortened Version of the Token Test. Cortex 14, 41–49. http://dx.doi.org/10.1016/S0010-9452(78)80006-9.
DeGutis, J., DeNicola, C., Zink, T., McGlinchey, R., Milberg, W., 2011. Training with own-race faces can improve processing of other-race faces: evidence from developmental prosopagnosia. Neuropsychologia 49, 2505–2513. http://dx.doi.org/10.1016/j.neuropsychologia.2011.04.031.
DeGutis, J.M., Chiu, C., Grosso, M.E., Cohan, S., 2014. Face processing improvements in prosopagnosia: successes and failures over the last 50 years. Front. Hum. Neurosci. 8, 561. http://dx.doi.org/10.3389/fnhum.2014.00561.
Dehaene, S., Cohen, L., Morais, J., Kolinsky, R., 2015. Illiterate to literate: behavioural and cerebral changes induced by reading acquisition. Nat. Rev. Neurosci. 16, 234–244. http://dx.doi.org/10.1038/nrn3924.
Della Sala, S., Laiacona, M., Spinnler, H., Trivelli, C., 1995. Poppelreuter-Ghent's Overlapping Figures Test: its sensitivity to age, and its clinical use. Arch. Clin. Neuropsychol. 10, 511–534.
Dennis, M., Spiegler, B.J., Simic, N., Sinopoli, K.J., Wilkinson, A., Yeates, K.O., Taylor, H.G., Bigler, E.D., Fletcher, J.M., 2014. Functional plasticity in childhood brain disorders: when, what, how, and whom to assess. Neuropsychol. Rev. 24, 389–408. http://dx.doi.org/10.1007/s11065-014-9261-x.
Dennis, M., Whitaker, H.A., 1977. Hemispheric Equipotentiality and Language Acquisition. In: Language Development and Neurological Theory. pp. 93–106. doi: http://dx.doi.org/10.1016/B978-0-12-635650-2.50014-5.
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, 576–585. http://dx.doi.org/10.1016/j.neuropsychologia.2005.07.001.
Dundas, E.M., Plaut, D.C., Behrmann, M., 2013. The joint development of hemispheric lateralization for words and faces. J. Exp. Psychol. Gen. 142, 348–358. http://dx.doi.org/10.1037/a0029503.
Durston, S., Davidson, M.C., Tottenham, N., Galvan, A., Spicer, J., Fossella, J.A., Casey, B.J., 2006. A shift from diffuse to focal cortical activity with development: commentary. Dev. Sci. http://dx.doi.org/10.1111/j.1467-7687.2005.00454.x.
Ellemberg, D., Lewis, T.L., Defina, N., Maurer, D., Brent, H.P., Guillemot, J.P., Lepore, F., 2005. Greater losses in sensitivity to second-order local motion than to first-order local motion after early visual deprivation in humans. Vision. Res. 45, 2877–2884. http://dx.doi.org/10.1016/j.visres.2004.11.019.
Ellemberg, D., Lewis, T.L., Maurer, D., Brar, S., Brent, H.P., 2002. Better perception of global motion after monocular than after binocular deprivation. Vision. Res. 42, 169–179. http://dx.doi.org/10.1016/S0042-6989(01)00278-4.
Ellis, H.D., Young, A.W., 1988. Training in face-processing skills for a child with acquired prosopagnosia. Dev. Neuropsychol. 4, 283–294. http://dx.doi.org/10.1080/87565648809540412.
Emberson, L.L., Crosswhite, S.L., Richards, J.E., Aslin, R.N., 2017. The lateral occipital cortex is selective for object shape, not texture/color, at Six months. J. Neurosci. 37, 3698–3703. http://dx.doi.org/10.1523/JNEUROSCI.3300-16.2017.
Epstein, R., Kanwisher, N., 1998. A cortical representation of the local visual environment. Nature 392, 598–601. http://dx.doi.org/10.1038/33402.
Ettlin, T.M., Beckson, M., Benson, D.F., Langfitt, J.T., Amos, E.C., Pineda, G.S., 1992. Prosopagnosia: a bihemispheric disorder. Cortex 28, 129–134.
Farah, M.J., Levinson, K.L., Klein, K.L., 1995. Face perception and within-category discrimination in prosopagnosia. Neuropsychologia 33, 661–674. http://dx.doi.org/10.1016/0028-3932(95)00002-K.
Farah, M.J., Rabinowitz, C., 2003. the brain: is "living things" an innate the organisation of semantic memory in the brain: I S " living things" an innate Category? Cogn. Neuropsychol. 3294. http://dx.doi.org/10.1080/02643290244000293.
Farah, M.J., Rabinowitz, C., Quinn, G.E., Liu, G.T., 2010. Early Commitment of Neural Substrates for Face Recognition early commitment of neural substrates for face recognition, 3294, pp. 37–41. doi: http://dx.doi.org10./1080/026432900380526.
Farah, M.J., Rabinowitz, C., Quinn, G.E., Liu, G.T., 2000. Early commitment of neural substrates for face recognition. Cogn. Neuropsychol. 17, 117–123. http://dx.doi.org/10.1080/026432900380526.
Fazzi, E., Bova, S., Giovenzana, A., Signorini, S., Uggetti, C., Bianchi, P., 2009. Cognitive visual dysfunctions in preterm children with periventricular leukomalacia. Dev. Med. Child Neurol. 51, 974–981. http://dx.doi.org/10.1111/j.1469-8749.2009.03272.x.
Felleman, D.J., Van Essen, D.C., 1991. Distributed hierachical processing in the primate cerebral cortex. Cereb. Cortex 1, 1–47.
Feydy, A., Carlier, R., Roby-Brami, A., Bussel, B., Cazalis, F., Pierot, L., Burnod, Y., Maier, M.A., 2002. Longitudinal study of motor recovery after stroke: recruitment and. Stroke 33, 1610–1617.
Fischer-Baum, S., Jang, A., Kajander, D., 2017. The cognitive neuroplasticity of reading recovery following chronic stroke: a representational similarity analysis approach. Neural Plast. 2017, 1–16. http://dx.doi.org/10.1155/2017/2761913.
Foster, R., Giddan, J., Stark, J., 1983. Assessment of children's language comprehension. Palo Alto, CA. Consulting Psychologists Press.
Fox, S.E., Levitt, P., Nelson, C.A., 2010. How the timing and quality of early experiences influence the development of brain architecture. Child Dev. http://dx.doi.org/10.1111/j.1467-8624.2009.01380.x.
Freud, E., Plaut, D.C., Behrmann, M., 2016. "What" Is Happening in the Dorsal Visual Pathway. Trends Cogn. Sci. xx, 1–12. http://dx.doi.org/10.1016/j.tics.2016.08.003.
Funnell, E., Wilding, J., 2011. Development of a vocabulary of object shapes in a child with a very-early-acquired visual agnosia: a unique case. Q. J. Exp. Psychol. (Hove). 64, 261–282. http://dx.doi.org/10.1080/17470218.2010.498922.
Gandhi, T., Kalia, A., Ganesh, S., Sinha, P., 2015. Immediate susceptibility to visual illusions after sight onset. Curr. Biol. http://dx.doi.org/10.1016/j.cub.2015.03.005.
Ganguly, K., Poo, M., 2013. Activity-dependent neural plasticity from bench to bedside. Neuron 80, 729–741. http://dx.doi.org/10.1016/j.neuron.2013.10.028.
Gathercole, S.E., Willis, C.S., Baddeley, A.D., Emslie, H., 1994. The children's test of nonword repetition: a test of phonological working memory. Memory 2, 103–127. http://dx.doi.org/10.1080/09658219408258940.
Gauthier, I., Behrmann, M., Tarr, M.J., 1999. Can face recognition really be dissociated from object recognition? J. Cogn. Neurosci. 11, 349–370.
Gauthier, I., Skudlarski, P., Gore, J.C., Anderson, A.W., 2000. Expertise for cars and birds recruits brain areas involved in face recognition. Nat. Neurosci. 3, 191–197. http://dx.doi.org/10.1038/72140.
Gauthier, I., Tarr, M.J., 1997. Becoming a "Greeble" expert: exploring mechanisms for face recognition. Vision. Res. 37, 1673–1682. http://dx.doi.org/10.1016/S0042-6989(96)00286-6.
Georgy, L., Celeghin, A., Marzi, C.A., Tamietto, M., Ptito, A., 2016. The superior colliculus is sensitive to gestalt-like stimulus configuration in hemispherectomy patients. Cortex 81, 151–161. http://dx.doi.org/10.1016/j.cortex.2016.04.018.
Gerlach, C., Marstrand, L., Habekost, T., Gade, A., 2005. A case of impaired shape integration: implications for models of visual object processing. Vis. Cogn. 12, 1409–1443. http://dx.doi.org/10.1080/13506280444000751.
Germine, L.T., Duchaine, B., Nakayama, K., 2011. Where cognitive development and aging meet: face learning ability peaks after age 30. Cognition 118, 201–210. http://dx.doi.org/10.1016/j.cognition.2010.11.002.

Geskin, J., Behrmann, M., Congenital prosopagnosia without object agnosia: A literature review. Cogn. Neuropsychol. (in press).
Gilaie-Dotan, S., Perry, A., Bonneh, Y., Malach, R., Bentin, S., 2009. Seeing with profoundly deactivated mid-level visual areas: non-hierarchical functioning in the human visual cortex. Cereb. Cortex 19, 1687–1703. http://dx.doi.org/10.1093/cercor/bhn205.
Gleissner, U., Sassen, R., Schramm, J., Elger, C.E., Helmstaedter, C., 2005. Greater functional recovery after temporal lobe epilepsy surgery in children. Brain 128, 2822–2829. http://dx.doi.org/10.1093/brain/awh597.
Gogtay, N., Giedd, J.N., Lusk, L., Hayashi, K.M., Greenstein, D., Vaituzis, A.C., Nugent, T.F., Herman, D.H., Clasen, L.S., Toga, A.W., Rapoport, J.L., Thompson, P.M., Thompson, P.M., 2004. Dynamic mapping of human cortical development during childhood through early adulthood. Proc. Natl. Acad. Sci. USA 101, 8174–8179. http://dx.doi.org/10.1073/pnas.0402680101.
Golarai, G., Ghahremani, D.G., Whitfield-Gabrieli, S., Reiss, A., Eberhardt, J.L., Gabrieli, J.D.E., Grill-Spector, K., 2007. Differential development of high-level visual cortex correlates with category-specific recognition memory. Nat. Neurosci. http://dx.doi.org/10.1038/nn1865.
Golarai, G., Liberman, A., Grill-Spector, K., 2015. Experience shapes the development of neural substrates of face processing in human ventral temporal cortex. Cereb. Cortex bhv 314. http://dx.doi.org/10.1093/cercor/bhv314.
Golarai, G., Liberman, A., Yoon, J.M.D., Grill-Spector, K., 2010. Differential development of the ventral visual cortex extends through adolescence. Front. Hum. Neurosci. 3, 80. http://dx.doi.org/10.3389/neuro.09.080.2009.
Goodale, M.A., Milner, A.D., 1992. Separate visual pathways for perception and action. Trends Neurosci.
Gougoux, F., Zatorre, R.J., Lassonde, M., Voss, P., Lepore, F., 2005. A functional neuroimaging study of sound localization: visual cortex activity predicts performance in early-blind individuals. PLoS Biol. 3, 0324–0333. http://dx.doi.org/10.1371/journal.pbio.0030027.
Grady, C.L., Mondloch, C.J., Lewis, T.L., Maurer, D., 2014. Early visual deprivation from congenital cataracts disrupts activity and functional connectivity in the face network. Neuropsychologia 57, 122–139. http://dx.doi.org/10.1016/j.neuropsychologia.2014.03.005.
Green, C.S., Li, R., Bavelier, D., 2010. Perceptual Learning During Action Video Game Playing. Top. Cogn. Sci. 2 (2), 202–216. http://dx.doi.org/10.1111/j.1756-8765.2009.01054.x. (Epub 2009 Oct 30). https://www.ncbi.nlm.nih.gov/pubmed/25163784.
Grefkes, C., Nowak, D.A., Eickhoff, S.B., Dafotakis, M., Küst, J., Karbe, H., Fink, G.R., 2008. Cortical connectivity after subcortical stroke assessed with functional magnetic resonance imaging. Ann. Neurol. 63, 236–246. http://dx.doi.org/10.1002/ana.21228.
Grill-Spector, K., 2003. The neural basis of object perception. Curr. Opin. Neurobiol. 13, 159–166. http://dx.doi.org/10.1016/S0959-4388(03)00040-0.
Grill-Spector, K., Kushnir, T., Edelman, S., Avidan, G., Itzchak, Y., Malach, R., 1999. Differential processing of objects under various viewing conditions in the human lateral occipital complex. Neuron 24, 187–203. http://dx.doi.org/10.1016/S0896-6273(00)80832-6.
Haak, K.V., Langers, D.R.M., Renken, R., van Dijk, P., Borgstein, J., Cornelissen, F.W., 2014. Abnormal visual field maps in human cortex: a mini-review and a case report. Cortex 56, 14–25. http://dx.doi.org/10.1016/j.cortex.2012.12.005.
Hadjikhani, N., De Gelder, B., 2002. Neural basis of prosopagnosia: an fMRI study. Hum. Brain Mapp. 16, 176–182. http://dx.doi.org/10.1002/hbm.10043.
Haist, F., Adamo, M., Han Wazny, J., Lee, K., Stiles, J., 2013. The functional architecture for face-processing expertise: FMRI evidence of the developmental trajectory of the core and the extended face systems. Neuropsychologia 51, 2893–2908. http://dx.doi.org/10.1016/j.neuropsychologia.2013.08.005.
Hamad, A.P., Caboclo, L.O., Centeno, R., Costa, L.V., Ladeia-Frota, C., Carrete, H., Gomez, N.G., Marinho, M., Yacubian, E.M.T., Sakamoto, A.C., 2013. Hemispheric surgery for refractory epilepsy in children and adolescents: outcome regarding seizures, motor skills and adaptive function. Seizure 22, 752–756. http://dx.doi.org/10.1016/j.seizure.2013.06.001.
Hamm, L.M., Black, J., Dai, S., Thompson, B., 2014. Global processing in amblyopia: a review. Front. Psychol. 5, 1–21. http://dx.doi.org/10.3389/fpsyg.2014.00583.
Haxby, J.V., Gobbini, M.I., Furey, M.L., Ishai, A., Schouten, J.L., Pietrini, P., 2001. Distributed and overlapping representations of faces and objects in ventral temporal cortex. Science 293, 2425–2430. http://dx.doi.org/10.1126/science.1063736.
Hensch, T.K., 2005. Critical period mechanisms in developing visual cortex. Curr. Top. Dev. Biol. 69, 215–237. http://dx.doi.org/10.1016/S0070-2153(05)69008-4.
Hu, S., Jin, H., Chen, Z., Mo, L., Liu, J., 2013. Failure in developing high-level visual functions after occipitoparietal lesions at an early age: a case study. Cortex 49, 2689–2699. http://dx.doi.org/10.1016/j.cortex.2013.07.009.
Humphreys, G.W., Riddoch, M.J., 1985. Authors' correction to “Routes to object constancy. QJEP 37A, 493–495. http://dx.doi.org/10.1080/14640748508400946.
Humphreys, G.W., Riddoch, M.J., 1984. Routes of object constancy: implications from neurological impairments from neurological impairments of object constancy. Q. J. Exp. Psychol. Sect. A 36, 385–415. http://dx.doi.org/10.1080/14640748408402169.
Johnston, M.V., Ishida, A., Ishida, W.N., Matsushita, H.B., Nishimura, A., Tsuji, M., 2009. Plasticity and injury in the developing brain. Brain Dev. 31, 1–10. http://dx.doi.org/10.1016/j.braindev.2008.03.014.
Johnston, M.V., Nishimura, A., Harum, K., Pekar, J., Blue, M.E., 2001. Sculpting the developing brain. Adv. Pediatr. 48, 1–38.
Kanjlia, S., Lane, C., Feigenson, L., Bedny, M., 2016. Absence of visual experience modifies the neural basis of numerical thinking. Proc. Natl. Acad. Sci. 113, 201524982. http://dx.doi.org/10.1073/pnas.1524982113.
Kanwisher, N., 2017. The Quest for the FFA and where it led. J. Neurosci. 37, 1056–1061. http://dx.doi.org/10.1523/JNEUROSCI.1706-16.2016.
Kanwisher, N., 2010. Functional specificity in the human brain: a window into the functional architecture of the mind. Proc. Natl. Acad. Sci. Usa. 107, 11163–11170. http://dx.doi.org/10.1073/pnas.1005062107.
Kanwisher, N., McDermott, J., Chun, M.M., 1997. The fusiform face area: a module in human extrastriate cortex specialized for face perception. J. Neurosci. 17, 4302–4311.
Kolb, B., 1995. Brain Plasticity and Behavior. Erlbaum, Mahwah, NJ.
Kravitz, D.J., Saleem, K.S., Baker, C.I., Mishkin, M., 2011. A new neural framework for visuospatial processing. Nat. Publ. Gr. 12. http://dx.doi.org/10.1038/nrn3008.
Lane, C., Kanjlia, S., Richardson, H., Fulton, A., Omaki, A., Bedny, M., 2017. Reduced Left Lateralization of Language in Congenitally Blind Individuals. J. Cogn. Neurosci. 29, 65–78. http://dx.doi.org/10.1162/jocn_a_01045.
Lashley, K., 1929. Brain Mechanisms and Intelligence: A quantitative Study of Inhuries to the Barin. Chicago Univ. Chigago Presshttp://dx.doi.org/10.1037/10017-000.
Lê, S., Cardebat, D., Boulanouar, K., Hénaff, M.-A., Michel, F., Milner, D., Dijkerman, C., Puel, M., Démonet, J.-F., 2002. Seeing, since childhood, without ventral stream: a behavioural study. Brain 125, 58–74. http://dx.doi.org/10.1093/brain/awf004.
Le Grand, R., Mondloch, C.J., Maurer, D., Brent, H.P., 2004. Impairment in holistic face processing following early visual deprivation. Psychol. Sci. a J. Am. Psychol. Soc. / APS 15, 762–768. http://dx.doi.org/10.1111/j.0956-7976.2004.00753.x.
Le Grand, R., Mondloch, C.J., Maurer, D., Brent, H.P., 2003. Expert face processing requires visual input to the right hemisphere during infancy. Nat. Neurosci. 6, 1108–1112. http://dx.doi.org/10.1038/nn1121.
Le Grand, R., Mondloch, C.J., Maurer, D., Brent, H.P., 2001. Neuroperception: early visual experience and face processing. Nature 410. http://dx.doi.org/10.1038/35073749.
Levi, D.M., Knill, D.C., Bavelier, D., 2015. Stereopsis and amblyopia: a mini-review. Vision. Res. 114, 17–30. http://dx.doi.org/10.1016/j.visres.2015.01.002.
Lewis, T.L., Ellemberg, D., Maurer, D., Wilkinson, F., Wilson, H.R., Dirks, M., Brent, H.P., 2002. Sensitivity to global form in glass patterns after early visual deprivation in humans. Vision. Res. 42, 939–948.
Lewis, T.L., Maurer, D., 2005. Multiple sensitive periods in human visual development: evidence from visually deprived children. Developmental psychobiology 46 (3), 163–183.
Lindenberger, U., Wenger, E., Lövdén, M., 2017. Towards a stronger science of human plasticity. Nat. Rev. Neurosci. 18, 261–262. http://dx.doi.org/10.1038/nrn.2017.44.
Malach, R., Reppas, J.B., Benson, R.R., Kwong, K.K., Jiang, H., Kennedy, W.A., Ledden, P.J., Brady, T.J., Rosen, B.R., Tootell, R.B., 1995. Object-related activity revealed by functional magnetic resonance imaging in human occipital cortex. Proc. Natl. Acad. Sci. USA. 92, 8135–8139.
Mancini, J., de Schonen, S., Deruelle, C., Massoulier, A., 1994. Face recognition in children with early right or left brain damage. Dev. Med. Child Neurol. 36, 156–166.
Mangini, M.C., Biederman, I. may 2001. Differentiating expression, gender and identity in faces: comparing normals, the ideal observer, and a prosopagnosic. In: Proceedings of the Paper Presentation at the Meeting of the Vision Sciences Society, Sarasota, FL.
Mangini, M.C., Biederman, I., 2004. Making the ineffable explicit: esti- mating the information employed for face classification. Cogn. Sci. 28, 209–226.
Martinaud, O., Pouliquen, D., Parain, D., Goldenberg, A., Gérardin, E., Hannequin, D., Altarelli, I., Ramus, F., Hertz-Pannier, L., Dehaene-Lambertz, G., Cohen, L., 2015. Impaired functional differentiation for categories of objects in the ventral visual stream: a case of developmental visual impairment. Neuropsychologia 77, 52–61. http://dx.doi.org/10.1016/j.neuropsychologia.2015.08.009.
Maurer, D., Mondloch, C.J., Lewis, T.L., 2007. Sleeper effects. Dev. Sci. 10, 40–47. http://dx.doi.org/10.1111/j.1467-7687.2007.00562.x.
Maurer, U., Brem, S., Kranz, F., Bucher, K., Benz, R., Halder, P., Steinhausen, H.-C., Brandeis, D., 2006. Coarse neural tuning for print peaks when children learn to read. Neuroimage 33, 749–758. http://dx.doi.org/10.1016/j.neuroimage.2006.06.025.
McCandliss, B.D., Cohen, L., Dehaene, S., 2003. The visual word form area: expertise for reading in the fusiform gyrus. Trends Cogn. Sci. 7, 293–299.
McGugin, R.W., Ryan, K.F., Tamber-Rosenau, B.J., Gauthier, I., 2017. The role of experience in the face-selective response in right FFA. Cereb. Cortex 1–14. http://dx.doi.org/10.1093/cercor/bhx113.
McKone, E., Crookes, K., Jeffery, L., Dilks, D.D., 2012. A critical review of the development of face recognition: experience is less important than previously believed. Cogn. Neuropsychol. 29, 174–212. http://dx.doi.org/10.1080/02643294.2012.660138.
McNeil, J.E., Warrington, E.K., 1993. Prosopagnosia: a face-specific disorder. Q. J. Exp. Psychol. A. 46, 1–10.
Meekes, J., Braams, O., Braun, K.P.J., Jennekens-Schinkel, A., van Nieuwenhuizen, O., 2013. Verbal memory after epilepsy surgery in childhood. Epilepsy Res. 107, 146–155. http://dx.doi.org/10.1016/j.eplepsyres.2013.08.017.
Merabet, L.B., Pascual-Leone, A., 2009. Neural reorganization following sensory loss: the opportunity of change. Nat. Rev. Neurosci. 11. http://dx.doi.org/10.1038/nrn2758.
Miceli, G., Caramazza, A., 1993. The assignment of word stress in oral reading: evidence from a case of acquired dyslexia. Cogn. Neuropsychol. 10, 273–295. http://dx.doi.org/10.1080/02643299308253465.
Michelon, P., Biederman, I., 2003. Less impairment in face imagery than face perception in early prosopagnosia. Neuropsychologia 41, 421–441. http://dx.doi.org/10.1016/S0028-3932(02)00176-8.
Muckli, L., Naumer, M.J., Singer, W., 2009. Bilateral visual field maps in a patient with only one hemisphere. Proc. Natl. Acad. Sci. USA. 106, 13034–13039. http://dx.doi.org/10.1073/pnas.0809688106.
Mullin, J.P., Soni, P., Lee, S., Jehi, L., Valappi, A.M.N., Bingaman, W., Gonzalez-Martinez, J., 2016. Volumetric Analysis of Cerebral Peduncles and Cerebellar Hemispheres for Predicting Hemiparesis after Hemispherectomy. Neurosurgery 79, 499–507. http://dx.doi.org/10.1227/NEU.0000000000001307.
Nishimura, M., Scherf, K.S., Zachariou, V., Tarr, M.J., Behrmann, M., 2015. Size precedes

view: developmental emergence of invariant object representations in lateral occipital complex. J. Cogn. Neurosci. 27, 474–491. http://dx.doi.org/10.1162/jocn_a_00720.
Nudo, R.J., Wise, B.M., SiFuentes, F., Milliken, G.W., 1996. Neural substrates for the effects of rehabilitative training on motor recovery after ischemic infarct. Science 272, 1791–1794.
O'Hare, A.E., Dutton, G.N., Green, D., Coull, R., 1998. Evolution of a form of pure alexia without agraphia in a child sustaining occipital lobe infarction at 2 1/2 years. Dev. Med. Child Neurol. 40, 417–420.
Ostrovsky, Y., Andalman, A., Sinha, P., 2006. Vision following extended congenital blindness. Psychol. Sci. 17, 1009–1014. http://dx.doi.org/10.1111/j.1467-9280.2006.01827.x.
Ostrovsky, Y., Meyers, E., Ganesh, S., Mathur, U., Sinha, P., 2009. Visual parsing after recovery from blindness. Psychol. Sci. 20, 1484–1491. http://dx.doi.org/10.1111/j.1467-9280.2009.02471.x.
Pascual-Leone, A., Amedi, A., Fregni, F., Merabet, L.B., 2005. The plastic human brain cortex. Annu. Rev. Neurosci. 28, 377–401. http://dx.doi.org/10.1146/annurev.neuro.27.070203.144216.
Plaut, D.C., Behrmann, M., 2011. Complementary neural representations for faces and words: a computational exploration. Cogn. Neuropsychol. 28, 251–275. http://dx.doi.org/10.1080/02643294.2011.609812.
Renfrew, C., 1995. Word Finding Vocabulary Test. UK: Winslow Press, Bicester.
Reynolds, C.R., Bigler, E.D., 1994. Test of memory and learning. Pro-Ed., Austin, Tex.
Riddoch, J.M., Humphreys, G.W., 1993. BORB: Birmingham object recognition battery. BORB: Birm. Object Recognit. Battery.
Rossion, B., Caldara, R., Seghier, M., Schuller, A.M., Lazeyras, F., Mayer, E., 2003. A network of occipito-temporal face-sensitive areas besides the right middle fusiform gyrus is necessary for normal face processing. Brain 126, 2381–2395. http://dx.doi.org/10.1093/brain/awg241.
Rossion, B., Dricot, L., Goebel, R., Busigny, T., 2011. Holistic face categorization in higher order visual areas of the normal and prosopagnosic brain: toward a non-hierarchical view of face perception. Front. Hum. Neurosci. 4, 225. http://dx.doi.org/10.3389/fnhum.2010.00225.
Rossit, S., Benwell, C.S.Y., Szymanek, L., Learmonth, G., McKernan-Ward, L., Corrigan, E., Muir, K., Reeves, I., Duncan, G., Birschel, P., Roberts, M., Livingstone, K., Jackson, H., Castle, P., Harvey, M., 2017. Efficacy of home-based visuomotor feedback training in stroke patients with chronic hemispatial neglect. Neuropsychol. Rehabil. 1–22. http://dx.doi.org/10.1080/09602011.2016.1273119.
Sadato, N., Pascual-Leone, A., Grafman, J., Ibanez, V., Deiber, M.P., Dold, G., Hallett, M., 1996. Activation of the primary visual cortex by Braille reading in blind subjects.[see comment]. Nature 380, 526–528. http://dx.doi.org/10.1038/380526a0.
Sartori, G., Job, R., Tressoldi, P.E., 1995. Batteria per la valutazione della dislessia e della disortografia evolutiva. O.S. Organizzazioni Speciali, Firenze, Italy.
Scherf, K.S., Behrmann, M., Humphreys, K., Luna, B., 2007. Visual category-selectivity for faces, places and objects emerges along different developmental trajectories. Dev. Sci. 10, F15–F30. http://dx.doi.org/10.1111/j.1467-7687.2007.00595.x.
Scherf, K.S., Luna, B., Avidan, G., Behrmann, M., 2011. "What" precedes "which": developmental neural tuning in face- and place-related cortex. Cereb. Cortex 21, 1963–1980. http://dx.doi.org/10.1093/cercor/bhq269.
Schiavetto, A., Décarie, J.C., Flessas, J., Geoffroy, G., Lassonde, M., 1997. Childhood visual agnosia: a seven-year follow-up. Neurocase 3, 1–17. http://dx.doi.org/10.1093/neucas/3.1.1.
Schlaggar, Mc.Candliss, Schlaggar, B.L., McCandliss, B.D., 2007. Development of neural systems for reading. Annu. Rev. Neurosci. 30, 475–503. http://dx.doi.org/10.1146/annurev.neuro.28.061604.135645.
Sergent, J., Villemure, J.G., 1989. Prosopagnosia in a right hemispherectomized patient. Brain 112, 975–995. http://dx.doi.org/10.1093/brain/112.4.975.
Skirrow, C., Cross, J.H., Harrison, S., Cormack, F., Harkness, W., Coleman, R., Meierotto, E., Gaiottino, J., Vargha-Khadem, F., Baldeweg, T., 2015. Temporal lobe surgery in childhood and neuroanatomical predictors of long-term declarative memory outcome. Brain 138, 80–93. http://dx.doi.org/10.1093/brain/awu313.
Snodgrass, J.G., Vanderwart, M., 1980. A standardized set of 260 Pictures: norms for name agreement, image agreement, familiarity, and visual complexity. J. Exp. Psychol. Hum. Learn. Mem. 6, 174–215.
Somerville, L.H., 2016. Searching for signatures of brain maturity: what are we searching for? Neuron 92, 1164–1167. http://dx.doi.org/10.1016/j.neuron.2016.10.059.
Song, S., Garrido, L., Nagy, Z., Mohammadi, S., Steel, A., Driver, J., Dolan, R.J., Duchaine, B., Furl, N., 2015. Local but not long-range microstructural differences of the ventral temporal cortex in developmental prosopagnosia. Neuropsychologia 78, 195–206. http://dx.doi.org/10.1016/j.neuropsychologia.2015.10.010.
Staudt, M., 2010. Reorganization after pre- and perinatal brain lesions. J. Anat. 217, 469–474. http://dx.doi.org/10.1111/j.1469-7580.2010.01262.x.
Stevens, W.D., Kravitz, D.J., Peng, C.S., Henry Tessler, M., Martin, A., 2017. Privileged functional connectivity between the visual word form area and the language system. J. Neurosci. http://dx.doi.org/10.1523/JNEUROSCI.0138-17.2017.
Stiles, J., Reilly, J., Paul, B., Moses, P., 2005. Cognitive development following early brain injury: Evidence for neural adaptation. Trends Cogn. Sci. 136–143. http://dx.doi.org/10.1016/j.tics.2005.01.002.
Street, R.F., 1931. A gestalt completion contribution to education. New York: Bureau of Publication, Teachers College. Columbia University.
Stretton, J., Sidhu, M.K., Winston, G.P., Bartlett, P., McEvoy, A.W., Symms, M.R., Koepp, M.J., Thompson, P.J., Duncan, J.S., 2014. Working memory network plasticity after anterior temporal lobe resection: a longitudinal functional magnetic resonance imaging study. Brain 137, 1439–1453. http://dx.doi.org/10.1093/brain/awu061.
Striem-Amit, E., Cohen, L., Dehaene, S., Amedi, A., 2012. Reading with sounds: sensory substitution selectively activates the visual word form area in the blind. Neuron 76, 640–652. http://dx.doi.org/10.1016/j.neuron.2012.08.026.
Tamietto, M., Cauda, F., Corazzini, L.L., Savazzi, S., Marzi, C.A., Goebel, R., Weiskrantz, L., de Gelder, B., 2010. Collicular vision guides nonconscious behavior. J. Cogn. Neurosci. 22, 888–902. http://dx.doi.org/10.1162/jocn.2009.21225.
Telfeian, A.E., Berqvist, C., Danielak, C., Simon, S.L., Duhaime, A.-C., 2002. Recovery of language after left hemispherectomy in a sixteen-year-old girl with late-onset seizures. Pediatr. Neurosurg. 37, 19–21.
Thomas, C., Moya, L., Avidan, G., Humphreys, K., Jung, K.J., Peterson, M.A., Behrmann, M., 2008. Reduction in white matter connectivity, revealed by diffusion tensor imaging, may account for age-related changes in face perception. J. Cogn. Neurosci. 20, 268–284. http://dx.doi.org/10.1162/jocn.2008.20025.
Ungerleider, L.G., Mishkin, M., 1982. Two cortical visual systems. In: Ingle, D.J., Goodale, M.A., Mansfield, R.J.W. (Eds.), Analysis of Visual Behavior. MIT Press, Cambridge, MA, pp. 549–586.
Vargha-Khadem, F., Polkey, C.E., 1992. A Review of Cognitive Outcome after Hemidecortication in Humans. Springer, US, pp. 137–151. http://dx.doi.org/10.1007/978-1-4615-3420-4_8.
Vining, E.P., Freeman, J.M., Pillas, D.J., Uematsu, S., Carson, B.S., Brandt, J., Boatman, D., Pulsifer, M.B., Zuckerberg, a., 1997. Why would you remove half a brain? The outcome of 58 children after hemispherectomy-the Johns Hopkins experience: 1968 to 1996. Pediatrics 100, 163–171. http://dx.doi.org/10.1542/peds.100.2.163.
Voress, J.K., Pearson, N.A., H.D.D, 2004. Developmental Test of VisualPerception (DTVP). Erickson Ed, Trento, Italy.
Wandell, B.A., Yeatman, J.D., 2013. Biological development of reading circuits. Curr. Opin. Neurobiol. 23, 261–268. http://dx.doi.org/10.1016/j.conb.2012.12.005.
Warrington, E.K., 1984. Recognition Memory Test: Manual. NFER-Nelson, Berkshire, UK.
Warrington, E.K., James, M., 1991. The Visual Object and Space Perception Battery: VOSP. Pearson.
Warrington, E.K., Taylor, A.M., 1978. Two categorical stages of object recognition. Perception 7, 695–705.
Weiner, K.S., Jonas, J., Gomez, J., Maillard, L., Brissart, H., Hossu, G., Jacques, C., Loftus, D., Colnat-Coulbois, S., Stigliani, A., Barnett, M.A., Grill-Spector, K., Rossion, B., 2016. The face-processing network is resilient to focal resection of human visual cortex. J. Neurosci. 36, 8425–8440. http://dx.doi.org/10.1523/JNEUROSCI.4509-15.2016.
Werth, R., 2008. Cerebral blindness and plasticity of the visual system in children. A review of visual capacities in patients with occipital lesions, hemispherectomy or hydranencephaly. Restor. Neurol. Neurosci. 26, 377–389.
Werth, R., 2006. Visual functions without the occipital lobe or after cerebral hemispherectomy in infancy. Eur. J. Neurosci. 24, 2932–2944. http://dx.doi.org/10.1111/j.1460-9568.2006.05171.x.
Woods, B.T., Carey, S., 1979. Language deficits after apparent clinical recovery from childhood aphasia. Ann. Neurol. 6, 405–409. http://dx.doi.org/10.1002/ana.410060505.
Xu, X., Biederman, I., 2014. Neural correlates of face detection. Cereb. Cortex 24, 1555–1564. http://dx.doi.org/10.1093/cercor/bht005.
Young, A.W., Ellis, H.D., 1989. Childhood prosopagnosia. Brain Cogn. 9, 16–47. http://dx.doi.org/10.1016/0278-2626(89)90042-0.
Yue, X., Biederman, I., Mangini, M.C., von der Malsburg, C., Amir, O., 2012. Predicting the psychophysical similarity of faces and non-face complex shapes by image-based measures. Vision. Res. 55, 41–46. http://dx.doi.org/10.1016/j.visres.2011.12.012. https://www.ncbi.nlm.nih.gov/pubmed/22248730.