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# Childhood Stroke and Vision: A Review of the Literature

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**Abstract**

**Objective:** To review current literature regarding visual outcome after perinatal and childhood stroke.

**Background:** Visual deficits following stroke in adults are common and have been previously reviewed. Less is known about visual deficits following stroke in neonates and older children. Most of the literature regarding this subject has focused on preterm infants, or on other types of brain injury. This review summarizes the types of visual deficits seen in term infants following perinatal stroke and children following childhood stroke and predictors of outcome. This review suggests areas for future research.

**Methods:** The authors performed Ovid MEDLINE searches regarding visual testing in children, vision after childhood stroke, neuroplasticity of vision, treatment of visual impairment after stroke, and driving safety concerns after stroke.

**Results:** Visual field defects were the most commonly reported visual deficits after perinatal and childhood stroke. There is a significant lack of literature on this subject, and most is in the form of case reports and case series. Children can experience significant visual morbidity after stroke, and have the potential to show some recovery, but guidelines on assessment and treatment of this population are lacking.

**Conclusions:** There were limitations to this study given the small amount of literature available. Although stroke in children can result in severe visual deficits, most children regain at least a portion of their vision. However, more research is needed regarding

visual assessment of this population, long term visual outcomes, specific predictors of recovery, and treatment options.

## Introduction

The types and severity of visual loss following stroke have been well documented in adults. Visual recovery after childhood stroke, however, has not been well-studied. Most of the literature on visual outcomes in children with brain injury involves cases of hypoxia, periventricular leukomalacia (PVL), and traumatic brain injury (TBI). Much of the literature on visual recovery after perinatal brain injury focuses on visual outcomes in premature infants with ophthalmologic and neurological complications of prematurity, rather than on term infants. This review discusses techniques for assessing visual impairment in children and summarizes the limited literature on the presentation and outcomes of stroke in term infants and children. Treatment options for visual impairment after stroke are reviewed, and areas for further study are suggested.



## Methods

Five separate OVID searches from 1947-June 2017 were performed for this review: 1) childhood stroke and vision, 2) visual testing in children, 3) visual neuroplasticity, 4) treatment of visual impairment after stroke, 5) driving safety after stroke.

*Limitation and exclusion criteria for all studies:* All studies were limited to English language and date range of 1946 to June 2017. Human studies were excluded if stroke was not confirmed by computed tomography (CT) or magnetic resonance imaging (MRI) or if the stroke was not thought to be the direct cause of visual impairment. Subjects

were excluded if they had lesions not clearly identified as ischemic or hemorrhagic stroke.

***1.Childhood stroke and vision****:* Keywords: ("stroke" OR "cerebral infarction" OR "brain ischemia" OR "cerebrovascular accident" OR "Infarction, Posterior Cerebral Artery" OR "Infarction, Middle Cerebral Artery") AND ("vision or Vision, Ocular" OR "occipital lobe" OR "visual fields" OR "visual perception" OR "occipital cortex" OR "visual cortex") AND ("perinatal" OR "child" OR "infant" OR "prenatal" OR "neonatal").

*Articles meeting all criteria: 26*

***2. Early Childhood Vision Testing:*** Keywords: "Vision Tests" AND "Infant" AND "Child Development". Additional limits included "all child (0 to 18 years)"

*Additional exclusion criteria:* Articles were excluded if specific visual tests were not discussed.

*Articles meeting all criteria:* 7

***3. Vision and Neuroplasticity:*** Keywords: ("Stroke" OR "Brain Ischemia" OR "cerebrovascular accident" OR "Brain Injuries") AND ("vision" OR "Vision, Ocular" OR "Visual Cortex" OR "Visual Fields" OR "Visual Perception") AND ("neuroplasticity" OR "Neuronal Plasticity").

*Articles meeting all criteria:* 8

***4. Visual impairment treatment:*** Keywords "visual impairment" AND "treatment" AND "child" AND "stroke."

*Articles meeting all criteria:* 1. Another relevant article was identified in search 1.

***5. Driving safety:*** Keywords: ("driving after stroke") AND ("vision" OR "Vision, Ocular"). No articles were identified when "child" was added as a search term.

*Articles meeting all criteria and identified from references*: 3

## Results

The reports of visual outcomes after childhood stroke are summarized in Table 1 (Visual Outcomes after Perinatal Stroke) and Table 2 (Visual Outcomes After Childhood Stroke Past the Perinatal Period). In studies drawing subjects from the same longitudinal study [1-3], only the most recent data were reported. In studies using the same data [4-6], only the most recent and largest studies were reported.

*Reported Visual Outcomes after Perinatal Stroke (Table 1)*

There are a wide range of visual abnormalities seen in perinatal stroke survivors (Table 1); 136 children were included. Three of the identified studies were longitudinal cohort studies, and only the most recent data were reported [1, 4, 6]. Overall, visual field defects were the most common deficit [1, 4, 6-9]. Total number of patients displaying visual field defects could not be reported because visual field data for each patient were not described in one study [8]. The most common visual field abnormality was hemianopsia. In most patients, visual acuity was not specifically documented. Only one patient had a documented visual acuity deficit [6]. Other visual perceptual deficits noted

included “blindsight” (perception of visual stimuli without functional vision; 4 children); visual cancellation errors (38 children); orthoptic eye movement abnormalities (7 children); optokinetic nystagmus (3 children); hemispace bias (23 children); and crowding acuity (2 children) deficits [1, 6-10].

*Reported Visual Outcomes After Childhood Stroke Past the Perinatal Period (Table 2)*

Similar to perinatal stroke, there was also a wide variety of visual deficits reported after childhood stroke. Most studies in this area were case reports and small case series. Table 2 includes 43 children. As in perinatal stroke, visual field defects were the most common visual deficit reported [9, 11-19]. Of these, hemianopsia was the most common [9, 11-16]. Visual acuity was normal in three cases [17, 19, 20], and abnormal in one case [12]. Visual acuity in the other cases was not specifically addressed. There were two cases in which children presented with near complete or complete blindness [17, 21]. Various other visual abnormalities were documented in 33 children [11-13, 15-18, 20, 22-25]. These deficits included neglect, constructional apraxia, and optic ataxia.

## Discussion

### *Assessing Vision in Children*

Determining visual function in young stroke patients is challenging. Many aspects of vision are either not developed or difficult to assess in preverbal infants. Preverbal infant ophthalmological testing can include visual fields, visual acuity, refractive state, and ocular health [26]. As children get older, depth perception and use of vision in daily activities, or "functional vision," can also be assessed [27].

There are visual tests designed for young children. The behavioral visual field (BEFIE) screening test can be used in children older than 3 months [5]. For older children, toys or fingers can be used for confrontation testing. Commonly used tests of visual acuity include Teller Acuity Cards (TAC) and Cambridge Crowding Cards [28]. Infant random dot stereoacuity cards can be used in children 17-36 weeks old to measure depth perception; in older children, stereoscopy is used [29, 30]. The Atkinson Battery of Child Development for Examining Functional Vision (ABCDEFV) exam [31] can be used in infants from birth to 4 years of age [31]. However, these tests are not widely used in clinical practice because they are time-intensive and require specialized training to administer [26].

Functional magnetic resonance imaging (fMRI), magnetic resonance imaging (MRI), diffusion tensor imaging (DTI) and electroencephalography (EEG) have also been used to assess vision and predict visual outcome after childhood stroke in research studies [1, 6, 32, 33]. In the future they may become more common tests of vision after childhood stroke.

***Perinatal Stroke and Vision (Table 1)***

Up to 80% of infarctions in the perinatal period occur in the middle cerebral artery (MCA) territory [4]. Only 8-16% of perinatal strokes occur in the posterior cerebral artery (PCA) territory [4]. The MCA supplies portions of the frontal, parietal and temporal lobes, while the PCA supplies the occipital lobes, primary visual cortex, and brainstem. Ischemia in either the MCA or PCA may damage the optic radiations.

There is tremendous variability in visual outcomes after perinatal stroke due to territory, stroke size, and possibly other factors. The abnormalities range from visual field deficits to optokinetic deficits. The visual impact of perinatal stroke is summarized in Table 1, which includes 136 patients. Visual field defects were the most common visual deficits reported [1, 4, 6-9].

A study of blindsight by Tinelli et al. [9] suggested there may be greater preservation of vision and capacity for visual recovery following perinatal stroke than after later childhood stroke. Patients with blindsight are able to detect visual stimuli, such as moving cars and lights, without functional vision [9]. Tinelli et al. [9] found that all children with perinatal stroke demonstrated blindsight, while those with later childhood stroke did not.

*Attempting to predict visual outcome after perinatal stroke*

Although multiple studies have suggested that most children experience at least some visual recovery after perinatal stroke few studies address predictors of outcome. The identified studies assessed the correlation of visual outcome with size, extent, and

hemisphere location on MRI lesions; with areas of activation on fMRI; with optic tract radiations on DTI; and with EEG.

A longitudinal study by Mercuri et al. [1, 2] examined the correlation of visual deficits with MRI images over time in 12 term perinatal stroke survivors Visual fields, visual acuity, orthoptic status, optokinetic nystagmus, fixation shift, visually evoked potentials, and global outcome were assessed within the first year of life. Seven out of the twelve (58%), had at least one visual abnormality. The size and extent of MRI lesions did not clearly correlate with the extent of visual impairment. The group was later re-evaluated at school age with repeat MRI [1]. Injury of the primary visual cortex was not always associated with visual field defects [1]. However, the vast majority of children showed huge visual improvement from their first evaluation [1]. Only 28% of the children retested at school age had a visual abnormality [1]. This longitudinal study highlighted how difficult it is to predict visual outcome in the perinatal period, given that extensive MRI lesions are not necessarily indicative of poor outcome.

There is some debate about whether involvement of optic radiations on MRI predicts visual field defects. While Mercuri et al. [1] found involvement of optic radiations on MRI to be a poor predictor of visual fields, a more recent study by Koenraads et al. [6] of 19 infants with perinatal stroke found a strong association between optic radiation asymmetry on MRI and DTI with visual field defects [6]. All infants with symmetrical optic radiations had normal visual fields. Seghier et al [33] described the association of optic radiation recovery with vision in a term boy with perinatal stroke who had brain DTI and functional MRI performed at 12 and 20 months of age. The increasing prominence of

optic radiations on serial DTI images and increased activation on functional MRI were associated with vision improvement.

Electroencephalography (EEG) was studied as a predictor of visual outcome after perinatal stroke by Biagioni et al. [32] in term infants with perinatal brain lesions. Unfortunately, this study included both children with perinatal stroke and children with hypoxic ischemic encephalopathy (HIE), and the authors did not differentiate between groups in the results. They reported that a normal EEG always correlated with normal visual fields, and nonreactivity to eye closure always correlated with visual field defects. Other abnormal features on EEG were less associated with visual field defects.

Infarct side (right vs left) may predict facial recognition difficulty and visuospatial neglect after perinatal strokes [7, 8, 10]. The right hemisphere processes more configural visual patterns than the left [7]. Face processing deficits after perinatal stroke occur more often in lesions of the left hemisphere than right [7].

Overall, visual outcome appears to be good for perinatal stroke survivors. The majority will recover some aspect of vision [1]. However, children with similar lesions may have different degrees of recovery. To date, only a few studies have looked specifically at visual outcome after perinatal stroke in term infants without hypoxic ischemic encephalopathy or some degree of perinatal asphyxia, and these studies are small. Larger studies are needed to further investigate predictors of visual outcome in term infants with perinatal strokes.

***Childhood Stroke and Vision***

As in perinatal stroke, the visual abnormalities seen after later childhood stroke vary greatly. The MCA territory is the most commonly affected in ischemic strokes, often resulting in hemianopsia [34]. Posterior cerebral artery strokes in the occipital lobe may present with visual field defects; strokes in the cerebellum may present with nystagmus; and strokes in the brainstem may present with abnormal eye movement. The types of visual abnormalities after childhood stroke are summarized in Table 2, which describes 43 patients. Visual field deficits were the most commonly reported visual abnormality post stroke [9, 11-19].

Only a few case studies report long-term follow up of visual function after childhood stroke; many experienced recovery [11, 12, 17, 20]. One case study reported visual recovery in a 2-year-old child following bilateral occipital lobe infarction [17]. At presentation, he was unable to fixate, and had no visual response. He had some residual visual perceptual ability (imitate hand movements, localize moving cars and light presented to lower visual fields). At 6 years of age, his visual exam was grossly normal, except residual upper field defect and deficits in visuospatial skills and object recognition. Another study by Ferro et al. [11] described left hemispatial neglect in three children following right hemispheric lesions. One child developed neglect following striatoinsular infarction, the other following intracerebral hemorrhage. Both patients had presenting symptoms similar to those seen in adult patients with similar lesions; however, these children had a complete recovery within one month of presentation.

*Attempting to predict visual outcome in childhood stroke past the perinatal period*

Predicting visual outcome after childhood stroke was difficult for many reasons. Many studies did not specify infarct location or size. For example, one study described the hemispheres in which stroke occurred, but not specific lobes or vascular territories [18]. Many articles only reported results on a few visual tests. For example, one study [16] described visual fields and visual hallucinations in a stroke patient, but no visual acuity data.

Schatz et al. [22] assessed the relationship of infarct volume and location with visual orienting deficits. Visual orientation is a complex cognitive activity involving the orientation of attention and visual scanning [22]. Fifteen children with sickle cell anemia-related infarction were given an orienting task, and results were compared with MRI scans. Lesions of the parietal lobe were more likely to cause problems with disengaging attention. Basal ganglia involvement was associated with decreased facilitation of attention in the hemifield contralateral to the injury. Volume of injury, however, did not correlate with any pattern of deficits.

Although some visual recovery after childhood stroke seems likely based on these studies, recovery varies dramatically among reported cases. Some children will not have significant visual recovery following stroke. The reasons some children recover vision better than others is still unknown; this is an area for further study.

### *Visual Outcomes and Neuroplasticity*

Long term prognosis for vision after childhood stroke, particularly perinatal stroke, can be good. Most children seem to regain at least a portion of their visual function over time. This is in contrast to adults, who appear to show less visual recovery post-stroke [35]. One theory behind this difference is changes in neuroplasticity with aging. In this discussion, we define neuroplasticity as the brain's ability to recruit undamaged neurons to perform the same function as the damaged neurons. Brain adaptability regarding visual loss has been discussed in numerous studies in humans [36-39], where visual function was recovered beyond what was expected given the extent of injury. However, much of the support for the theory of neuroplasticity and specific factors affecting it comes from animal studies. Several of these found that young age at stroke and environmental stimulation were associated with better outcomes [40-42].

### *Treatment of Visual Impairment after Childhood Stroke*

Two case reports were identified discussing children with stroke and their treatment with technology and coping strategies [12, 20], but no studies of treatment for childhood stroke were identified. This has been better addressed in the adult literature, where multiple treatment options have been reported. A recent review by Hanna et al. [43] addressed treatment of post-stroke visual impairment in adults. Adult patients with visual loss may be helped by various types of visual training. Visual field deficits and neglect have been addressed by eye movement training or scanning and search

training; use of a bright light at the edge of the visual field; or by the use of Peli prisms, which shift images from the area of visual field loss to the field that can be seen. Double vision and eye movement difficulties have been addressed with conservative measures such as patching, use of typoscopes, prisms and adaptive head positioning. Typoscopes, which are pieces of dark cardboard with a rectangle cut out in the middle, may help the patient focus on a small portion of reading text at a time. Medications, ocular muscle surgery, and counseling on coping strategies may also be helpful to patients [43]. However, the review concluded that further study was needed to assess which methods are the most effective. There is clearly a need for studies on treatment of visual deficits after childhood stroke.

***Driving Safety after Stroke***

Visual impairment after pediatric stroke is highly variable and difficult to predict, but has the potential to significantly affect activities of daily living, particularly driving. This review did not identify any studies of driving safety or training in pediatric stroke survivors. This has been better addressed in the adult literature.

Evaluating a patient for driving after stroke requires a multidisciplinary approach, often involving visual, neuropsychological, and on-road testing [44]. Akinwuntan et al. [45] looked at the determinants of driving ability in adult stroke patients by assessing these three areas independently. Four facets of vision were examined: kinetic vision, monocular vision, binocular vision, and stereoscopy. These results were compared with team decisions regarding patient driving eligibility. They found that all four facets of the

visual exam were good indicators of the team's decisions, but kinetic vision was the best predictor of driving competency. Visual neglect can be seen in children following perinatal stroke [11, 18, 24], and has also been found to be a good predictor of driving ability in adults [45, 46]. In one adult stroke study, subjects displaying visual neglect after stroke consistently drifted to one side of the road and could not process multiple concurrent visual stimuli, making them unfit to drive [46].

Although a few adult studies have identified areas of visual assessment that are good indicators of driver ability, most of these tests are not required to obtain a license. Only visual acuity and visual fields are generally assessed before licensure, and specific requirements vary from state to state. Functional assessments of pediatric stroke survivors performed by driving rehabilitation specialists are strongly recommended for those preparing to drive. The Association for Driver Rehabilitation Specialists provides evaluations and recommendations for individuals with disabilities: http://www.aded.net/. Some branches of Easter Seals provide driver assessment and training for people with visual impairments: http://www.eastersealscrossroads.org/?page_id=171. .



**Limitations of this Review**

Although the field of childhood stroke has grown significantly over the past decades, it was difficult to gather data on visual outcomes. Some studies included children with multiple diagnoses, including periventricular leukomalacia, hypoxic ischemic encephalopathy, and traumatic brain injury and did not clarify which children had which outcomes. Many studies of childhood stroke patients did not include visual

outcomes. Treatment of visual impairment and issues of driving safety after stroke have not been well-studied in the pediatric stroke population, so this review extrapolated from the adult literature.

## Conclusions

Visual impairment after pediatric stroke is an important but significantly understudied area. Drawing conclusions and suggesting guidelines from published data was difficult. Most papers reported only portions of visual assessments or did not describe long-term outcomes. In some perinatal stroke studies, term infants were combined with premature infants in data analyses. Many studies of older children combined stroke-related visual impairments with impairments due to trauma and other forms of brain injury in their analyses. The few available publications suggest that both neonates and older children with stroke have the potential for some recovery. Based on the adult literature, we suggest pediatric stroke survivors have functional assessments by driving rehabilitation specialists before driving. The variability of visual outcome and lack of studies on visual outcome after childhood stroke makes it difficult to suggest additional guidelines. This paper identifies vision outcomes and treatment approaches after pediatric stroke as an important area for future study.

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**Table 1. Cases with Perinatal Stroke Meeting Inclusion Criteria**

| Reference | No. of Patients | Ages at Visual Assessment | Etiology | Territories/Lobes Affected | Other Areas Affected | Visual Fields | Visual Acuity | Other Aspects of Vision |
|---|---|---|---|---|---|---|---|---|
| Cioni et al. 1998 [7] | 5 | 9-12 months | unspecified | Left FTP (1)<br>Left FTPO (1)<br>Right FTP (2)<br>Right P (1) | Optic radiations (3). | Normal (2)<br>Partial field loss (3) | Normal (5) | *Orthoptic status:* alternating strabismus (4)<br>*Optokinetic nystagmus*: normal (2), asymmetric (3) |
| Biagioni et al. 2002 [32] | 1 | infant | unspecified | Bilateral borderzone | Optic radiations | normal | Normal | Not assessed |
| Mercuri et al. 2003 [1] | 16 | *Initial:* 3-14 months<br>*School age:* 4-6 years | unspecified | MCA cortical branch (9)<br>MCA main branch (4)<br>Borderzone (3) | Optic radiations (7).<br>Basal ganglia (6). | Initial: normal (4), left narrow (1), right narrow (5), right total neglect (1), not assessed (1)<br>*School Age:* normal (14), right abnormal (2) | Normal (16) | *Initial:* orthoptic status normal (16), fixation shift normal (6), abnormal (10)<br>*School Age:* orthoptic status normal (14)/strabismus (2), stereopsis normal (14)/ abnormal (2), crowding acuity normal (14)/abnormal (2) |
| Seghier et al. 2005 [33] | 1 | Serial fMRI-DTI imaging at 3, 12 & 20 months | unspecified | Left TPO | Left optic radiations | Not assessed | Not assessed | + increased activity in visual cortex over time |
| Bava et al. 2005 [10] | 46 | 4-18 years | unspecified | Left hemisphere (23)<br>Right hemisphere (23) | unspecified | Normal | Unspecified | Left hemispace bias (23)<br>No perceptual asymmetry (23) |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Thareja et al. 2012 [8] | 38 | 4-16 years | unspecified | All focal unilateral left hemisphere (19) and right hemisphere (19) | Unspecified | Author noted some defects in subjects but not specific about type or number of children | Unspecified | Visual cancellation task: more errors in right and left hemispace in left hemisphere stroke, more errors on left hemispace in right hemisphere stroke |
| Tinelli et al. 2013 [9] | 4 | 13-17 years | unspecified | Left MCA main branch (2)<br>Right MCA main branch (2) | left optic radiations (2)<br>Right optic radiations (2) | Left homonymous hemaniopia (1)<br>Left lower quadrant (1)<br>Right lower and part of upper quadrant (2) | unspecified | Blindsight (4) |
| Van der Aa et al. 2013 [4] | 6 | 12 months-10 years | Unspecified (2)<br>Factor V Leiden and heterozygous MTHFR mutation (1)<br>Heterozygous MTHFR (2)<br>Homozygous MTHFR (1) | PCA (6) | Optic radiations (4).<br>Corpus callosum (4).<br>Basal ganglia (3). | Hemianopia (3)<br>Quadrantinopia (2)<br>Normal (1) | unspecified | Not assessed |
| Koenraads et al. 2016 [6] | 19 | 4 months-6 years | Unspecified | Complete MCA (10)<br>Cortical MCA branch (3)<br>PCA (6) | Unspecified | Hemianopia (6)<br>Quadrantanopia (2)<br>Inconclusive (2) | Normal (12)<br>Abnormal (1)<br>Not assessed | *Fixation*: all normal<br>*Orthoptic status*: normal (18), V pattern (1) |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | (6) | |

T=temporal, F=frontal, P=parietal

**Table 2. Cases With Childhood Stroke Past the Perinatal Period Meeting Inclusion Criteria**

| Reference | No. of Patients | Age at Stroke (years) | Etiology | Territory/Lobes Affected by Stroke | Additional Structures Affected | Visual Fields | Visual Acuity | Other Aspects of Vision |
|---|---|---|---|---|---|---|---|---|
| Ferro et al. 1984 [11] | 2 | 6 and 9 | Fibromuscular dysplasia (1)<br>Idiopathic thrombocytopenic purpura (1) | Periventricular (1)<br>Right PO (1) | Unspecified (2) | Left hemianopia (1)<br>Unspecified (1) | Unspecified (2) | Visuoconstructional ability impaired at onset and resolved two weeks later(1)<br>Left hemispatial inattention (1) |
| Lewis et al. 1994 [18] | 2 | 7 and 12 | unspecified | R hemispheric | unspecified | Left visual field disturbance (1) | unspecified | Ipsilateral neglect/ constructional apraxia |
| O'Hare et al. 1998 [12] | 1 | 2.5 | unspecified | Bilateral O | Unspecified | Left hemianopia at age 5, resolved by age 9 | 3/60 on Vernier Catford drum at age 3, 6/5 in right and 6/9 in left with Preferential Acuity Card procedure at age 9 | *Visual processing deficits*: alexia, prosopagnosia, topographic agnosia, peripheral neglect<br>*Stereopsis*: decreased<br>*Color vision*: normal |
| Schatz et al. 2000 [22] | 15 | 10.8 +/- 3.2 | Sickle cell anemia | Bilateral FP, Left T (1)<br>Left F (1)<br>Left FP (3) | Bilateral basal ganglia (3)<br>Left basal ganglia only (1) | Unspecified | Unspecified | *Orienting task*: difficulties disengaging with parietal and mid |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | Bilateral F, R P (1)<br>White matter only (4)<br>Bilateral F (2)<br>Bilateral F, Left P (1)<br>Left FPT (1) | | | | frontal gyrus, less facilitation of attention with basal ganglia injury |
| Gillen et al. 2003 [20] | 1 | 10 | Infective endocarditis | Right and Left P | unspecified | Normal | Normal | *Orthoptic status:* + optic ataxia<br>*Stereoacuity*: normal<br>*Color vision*: normal<br>*Strabismus:* none, jerky smooth pursuit<br>*Refraction*: normal<br>*Simultagnosia*: present |
| Delorio et al. 2004 [21] | 1 | 8 | phenylpropanol amine ingestion | Bilateral PO | Unspecified | No light perception bilaterally, resolved after 7 wks | No light perception bilaterally, resolved after 7 wks | Unspecified |
| Ortiz et al. 2007 [23] | 1 | 8 | Unspecified | Left MCA | unspecified | unspecified | unspecified | "Left gaze preference" |
| Radoeva et al. 2008 [13] | 1 | 15 | Arterial compression after traumatic occipital subdural collection | Left PCA | unspecified | Right homonymous hemianopia | Unspecified | + perception of motion in damaged visual field |
| Bova et al. 2008 [17] | 1 | 2 | Post colon resection for | Bilateral occipital hemorrhagic | unspecified | Presentation: Not tested | Presentation: Not tested | Presentation: Flash VEPs delayed, |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | Hirschsprung disease | infarcts | | due to inability to fixate<br>Four years later: upper visual field defect | due to inability to fixate<br>Four years later: recognition acuity 10/10 both eyes | fixation absent, refraction normal<br>Four years later: impairment of higher visual functions |
| Fernandez et al. 2010 [14] | 1 | 11 months | post liver transplant | Intraventricular hemorrhage | unspecified | hemianopia | unspecified | unspecified |
| Everts et al. 2010 [15] | 10 | 3 months to 15 years | unspecified | Left sided (5)<br>Right sided (5) | unspecified | Hemianopsia (1),<br>unspecified (9) | Unspecified | Lateralization of visual search tasks (8) |
| Kleinman et al. 2010 [24] | 1 | 9 | Antiphospholipid syndrome, decreased protein C activity, decreased antithrombin III | Right MCA, Right ACA, left PCA | unspecified | normal | normal | Severe unilateral spatial neglect on presentation, resolution after 1 month |
| Sampaio et al. 2011 [25] | 1 | 14 | none | Left MCA | unspecified | unspecified | unspecified | Left gaze preference |
| Tinelli et al. 2013 [9] | 3 | 6, 13, 15 | Moya Moya (1)<br>AVM rupture (2) | MCA infarct (1)<br>Right subcortical (1)<br>Left subcortical (1) | Optic radiations (3) | Left homonymous hemianopia (2)<br>Right homonymous hemianopia (2) | unspecified | Unspecified |
| Sorrentino | 1 | 8 | Dissected right | Bilateral O | Unspecified | Left | Normal | *Orthoptic status:* |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| et al. 2015 [19] | | | vertebral artery embolus | | | homonymous superior quarantanopia | | normal<br>*Color vision:* intact<br>*Stereopsis*: normal |
| Balakrishnan et al. 2016 [16] | 1 | 11 | *B. henselae* vasculitis | Right P | Unspecified | homonymous hemianopia | unspecified | Visual hallucinations |

O=occipital, F=Frontal, T=temporal, P=parietal