The Effects of Brain Damage on Visual Functioning in Children

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Abstract: This article reviews the research literature on the direct effects of brain damage on the visual functioning of children and the electrodiagnostic tests used to diagnose them. Although the studies documented that brain damage affects visual functioning, they suggested that the prognosis for good functional vision after remedial intervention is more optimistic than was previously thought. They also found that electrodiagnostic testing is a valuable tool, but that the use of a combination of tests yields a more complete picture of the effects than does any single test.
retinal detachment, visual acuity and visual perception are affected. Regardless of the extent of the brain damage. The infrequent reporting of visual deficits and the failure to identify specific types of deficits, especially in children with multiple handicaps, may hamper rehabilitation. Many researchers (Bergman, 1957; Gardner, Morse. Isaacscon, & Trief, 1986; Isaacscon, 1976; Press. Cumnings, Siegfried, & Altman. 1982) have reported that it is difficult to assess the visual functioning of infants and young children and that in searching for information on visual deficits that are not physiologically based, examiners cannot determine function in patients who cannot respond spontaneously to them. In earlier studies of cerebral blindness, Bergman (1957) reported that, even with cooperative patients, testing often yielded conflicting information on visual functioning. For example, he frequently observed a pupillary response to light in patients who, after different and more intensive electrodiagnostic testing, showed lesions causing complete blindness. Diagnoses of and prognoses for children with brain damage and visual deficits are often conflicting and incomplete. Mellor and Fielder (1980) reported that children who were mentally retarded had a poorer prognosis for visual acuity, visual perceptual abilities, or both than did nonretarded children with the same deficits. With early intervention and therapy, however, the visual functioning of the children with mental retardation improved. In fact, in studying permanent cortical visual impairment, Whiting et al. (1985) discovered that most of the children they studied had some vision and that, with therapy, all the children, regardless of their mental ability, improved their use of vision. These findings contradicted the earlier poor prognosis for the recovery of vision, particularly in children who are mentally retarded. Because of the difficulty of testing and determining the effects of brain damage on visual functioning, physicians and educators do not have enough information K) enable them to work more effectively with these children (Mori & Olive. 1978). Studies are often limited because there are no suitable standardized tests of the visual functioning of mentally or physically handicapped infants and children (Robertson & Finer. 1985). Therefore, diagnoses may be imprecise, which directly affects the prognoses that will be given and, in turn, the interventions. Brain damage and vision loss

One of the most common problems during the perinatal period is cerebral hypoxic-ischemia, or the reduced flow of oxygen and blood to the brain, which causes extensive brain damage. As a result of this damage, there is considerable sensory impairment, particularly in visual functioning. Hof-Van Duin and Mohn (1984) found that cerebral hypoxic-ischemia causes cortical blindness, tunnel vision, and strabismus, among other forms of visual deficits. Robertson and Finer (1989) categorized the severity of hypoxic-ischemic damage to determine long-term developmental outcomes in sensory ability and found visual deficits of some kind to be frequent at all levels of severity. Brain damage from cerebral hypoxic-ischemia was also among the major causes of cerebral visual disturbance in the children studied by Van Nieuwenhuizen and Willems (1984). Although pseudotumor cerebri, or abnormal pressure on the brain, causes a substantial loss of vision in adults, it was previously believed to spare the visual system of children. However, in Baker, Carter, Hendrick, and Buncie's (1985) study, visual deficits in central visual acuity, as well as various degrees of field losses and improved residual field defects, were prominent in children with this condition. Ataxia telangiectasia, a hereditary progressive atrophy of the cerebellum that begins in infancy, is reported to affect visual functioning (Sridharan & Mehta. 1985). Sridharan and Mehta (1985) found that ataxia telangiectasia affected not only the visual pathway, but the oculomotor movements of some of the patients, who had multidi-directional nystagmus (rapid, involuntary eye movements) whether their eyes were open or closed and regardless of the position of the eyeball. Nass (1984) reported that unilateral brain damage directly affected visual functioning in children, primarily in the area of visual perception, regardless of which hemisphere of the brain was damaged. However she noted that the defects were more severe and less likely to improve after a lesion in the right hemisphere or if the damage occurred at a later age. This finding suggests that visual perception is laterialized in the right hemisphere and that damage to the right hemisphere will probably cause more damage to visual functioning.

Neurological impairments and visual functioning

Other neurological impairments relate directly to visual loss. Many studies (Gaidner et al., 1986; Holman & Merritt, 1986; Ramani, 1985; Vu Nieuwenhuize & Willems, 1984; Whiting et al., 1989) have reported that prematurity, hydrocephalus, mental retardation and seizures are prominent etiologies of visual dysfunction in infants. Holman and Merritt found an increased incidence of neurologic impairment, especially from prematurity, in children with infantile esotropia (the inward turning of one or both eyes). Ramani (1985) reported that both icmal nystagmus (seizure-related, rapid, involuntary eye movements) and cortical blindness occurred periodically but prominently in patients with acute cerebral disorders from seizures.

Cerebral blindness requires a more detailed definition and description of its relationship to brain damage. Bergman (1957) defined cerebral blindness as the “bilateral absence of light perception... caused by disease of cerebral hemispheres.” Cerebral blindness, or “cortical blindness,” has been identified in adults as well as in children. It involves the “complete loss of all visual sensation, including appreciation of light and dark, loss of optokinetic nystagmus, preservation of pupillary response, normal motility of the eye and a normal retina on examination” (Whiting et al., 1985).

Cerebral blindness is often caused by bilateral cerebral softening in the calcanne cortex (the spur-shaped outer layer of the brain that is the visual pathway) from blockage of the cerebral arteries. Separate diagnoses for children have not yet been developed because the etiologies of the condition in adults and children have not been generally distinguished. Recently, many studies have reported that congenital cerebral blindness is different from the clinical entity found in adults. Mellor and Fielder (1980) presented another definition of cerebral blindness. They “dissociated visual development.” referring to the dysfunction as more of a “slow to see” phenomenon. Like Whiting et al. (1985), Mellor and Fielder reported that most children with cerebral blindness had some vision, many had functional vision, and all responded to photic stimulation after just a few sessions. The symptoms and effects of cerebral blindness are varied and often lead the
vision is present (Barnet et al., 1985; Bergman, 1997; Mellor & Fielder, 1980; Ramani, 1985; Whiting et al., 1989). The primary symptom of children with this disorder is visual inattentiveness. Other children have light perception only and still others can see objects at a distance, but minutes later appear to have no useful vision. Field tests from Whiting et al.'s study (1985) showed that many children bad tunnel vision or a right or left field loss bilaterally. Children with cerebral blindness could not maintain eye contact, while others looked away from visual stimulation as if it was physically bothersome or it hurt. Some children appeared to recognize familiar objects in one environment, but not in another. Many of these children supplemented their vision with (ouch by reaching out and exploring the object (actually, instead of simply looking at it).

Electrodiagnostic testing

Electrodiagnostic testing has proved to be a valuable form of assessment of visual functioning in children with brain damage (Baker et al., 1985; Barnet et al., 1970; Bergman, 1957; Fulton & Hansen, 1985; Hof-Van Duin & Mohn, 1984; Mellor & Fielder, 1980; Press et al., 1982; Ramani, 1985; Sridharan & Mehta, 1985; Van Nieuwenhuizen & Willemse, 1984; Whiting et al., 1985). These objective tests have made it possible to determine visual functioning in very young or severely mentally handicapped children. The following six major electrodiagnostic tests are useful in detecting visual loss in infants and children with brain damage.

The electroencephalogram (EEG) amplifies, records, and analyzes the electrical activity of the brain, particularly the alpha rhythm (the rhythmic oscillations in electric potential occurring at a rate of 8 to 13 per second). The alpha rhythm does not respond to the opening or closing of the eyes, since those actions do not indicate that vision is present. In fact, normal alpha rhythm responses do not occur in those with congenital or acquired blindness. However, the alpha rhythm is sensitive to photic stimulation or intermittent flashing of light into the eye (Bergman, 1957) and is recorded over the occipital cortex, reflecting the integrity of the entire visual pathway.

The electroretinogram (ERG) measures the transient electrical response of parts of the outer layer of the retina to a change in luminance. This instrument is helpful in the detection and evaluation of hereditary disorders of the retina, such as retinoblastoma, a malignant tumor on the retina. The ERG does not distinguish between different diseases; rather, it indicates the presence of diffuse abnormalities. This test may also be used to rule out disorders of the retina as the cause of blindness in certain conditions, such as cortical blindness.

The electro-oculogram (EOG) records eye movements of both eyes. either separately or together. It is most useful in recording various forms of nystagmus and in situations in which the ERG is not sensitive enough to pick up macular degeneration. Thus, the EOG serves as a supplement to and complements the ERG.

The visually evoked response (VER), sometimes referred to as the visual evoked potential (VEP), enables the examiner to look at the macular functioning of the eyes. It is the EEG recorded at the occipital pole, using an intense flash stimulation or a pattern of stimulation. This test is a clinically objective technique for assessing the functional state of the visual system, like the retino-cortical function in infants. It does not show the pathological nature of the abnormalities that it finds.

The visual evoked potential mapping (VEPM) is a new electrodiagnostic test, first used by Whiting et al. (1985) to diagnose children with cortical visual impairment. Like the VER, the VEPM examines macular functioning, but displays it in map form to show the response of a much larger area of the macula. As Whiting et al. described it, the electrical activity is displayed as "a multicolored moving picture on the screen. Each state of color within a broad spectrum corresponds to the degree of electrical activity generated in response to flashes or pattern stimulation."

Computed tomography (CT) scanning takes sectional roentgenograms of the brain. This test uses 1-rrays of X-rays to obtain tissue-density values of highly detailed cross-sectional images of the head that are then formed into pictures by computers. CT simultaneously visualizes orbital and intracranial structures, including soft tissue and foreign bodies.

Some studies have found increased diagnostic accuracy when tests are used in conjunction with each other (Mellor & Fielder, 1980; Ramani, 1985; Whiting et al., 1985). Whiting et al. reported that they obtained their best results in diagnosing cerebral blindness by using VEPMs and CT-scanning. Although the VEPMs were more sensitive than was the CT-scan, the results of the two were correlated highly. The CT-scan was able to show structural defects better, while the VEPM reflected functioning. Therefore, the CT-scan revealed unilateral occipital lobe function, whereas the VEPM revealed bilateral but asymmetrical involvement of the occipital lobe.

Using both the EEC and the CT-scan, Ramani (1985) was able to identify the nystagmus exhibited by children with brain damage. The EEG showed the ictal (seizure-related) nature of the nystagmus, while the CT scan showed the occipital infarction. Mellor and Fielder (1980) reported that the combination of the VER and the VEPM was successful in determining dissociated visual development, which shows up as severe impairment in both the ERG and VER. These tests, then, can complement observations of an infant's visual behavior and an ophthalmological examination.

Some research has also found that these electrodiagnostic tests can be effective in some situations, but not in others. Barnet et al., (1985) reported that VERs sometimes indicated a normal response even when functional blindness existed. They also found that their recording conditions and the placement of electrodes could affect the detection of a peripheral field loss by the VER.

Bergman (1957) reported that the presence or absence of alpha waves in an EEG examination was directly related to the presence or absence of vision. Post-mortem examinations of some subjects showed physiological damage in the calcarine cortex with cerebral lesions that were consistent with the absence of alpha waves in the EEG examination.

An interesting finding by Fulton and Hansen (1985) was that the ERG responses indicated that the infant retina was not yet fully developed. Infants' ERG responses were smaller and slower than those of adults, but sensitivity increased with age. As Fulton and Hansen noted, "sensitivity reaches adult values by about age six months while amplitudes are similar to adult values only near the end of the first post-natal year." Therefore, the study of the response of infants to ERGs may refine the diagnosis of retinal pathophysiology.

Mellor and Fielder (1980) reported a parallel delay in the development of the brain and of visual functioning, using the VER. That the development of the VER flash stimulation in premature infants showed marked individual variability suggests that the delay in the maturation of the VER response relates to visual development.
Development and its variability. It is important then, "that poor VER responses recorded in young infants with poor visual responsiveness should not in themselves be taken to imply a poor visual prognosis."

Whiting et al. (1985), however reported that EEGs and VERs elicited inconsistent responses from children with cortical blindness. The EEG changes reflected the underlying brain damage but did not diagnose cortical visual impairment. Children with some residual vision did not respond to photic stimulation. The VER was also inconsistent. Responses were abnormal for only a few children who had been diagnosed as having a visual loss.

**Diagnosis and prognosis**

The research on neurological impairments and their effects on visual functioning in infants and children has yielded much information on diagnosis and prognosis. The findings indicate that brain damage affects visual functioning in children, but that the prognosis for functional vision is more optimistic than was previously thought. For example, Barnett et al. (1970) reported that cerebral hypoxia was the proximate cause of blindness in the children they studied. Although some of these children were left with visual-perceptual defects, the vision of all the children improved over a period of weeks or months.

In studying infants with eye-muscle dysfunction from neurological impairment, Holman and Merritt (1986) reported that the prognosis for these children was worse than it was for children with esotropia that does not stem from neurological disorders. Despite the high incidence of over- and under correction, however, they found that children with neurological esotropia frequently made remarkable developmental gains following corrective surgery and that "the successes more than compensate for the failures." Baker et al. (1985) reported that children with pseudo-tumor cerebri may undergo a serious loss of visual acuity and visual fields that results in permanent damage, but these deficits can be reversed with rapid, appropriate therapy, and functional recovery can be good.

Mellor and Fielder's (1980) study of "slow-to-see" infants found that mentally retarded infants with normally functioning eyes often showed delayed visual awareness, implying that an abnormal VER response in such infants would suggest a poor prognosis for vision. The dissociated visual development could include delayed maturation of the macular photoreceptors, delayed myelination (the substance that sheaths parts of the brain) of the visual pathways, and the delayed formation of branches and development of synapses in the occipital cortex. Mellor and Fielder hypothesized that the difference between infants with and without mental handicaps could be that the delayed formation of branches is transient and strictly limited to the occipital cortex in mentally normal infants, but more generalized and permanent in mentally handicapped infants. Nevertheless, their study reported that vision improved in all the children, even if the improvement did not parallel the children's mental development.

**Brain damage and prognosis**

The many other transient effects of brain damage on vision may impede the correct diagnosis and prognosis of children, especially of those with cerebral blindness. Testing children with cerebral blindness, Barnett et al., (1970) reported that asymmetrical occipital responses, or "hemianinment," may be due to a defect in areas of visual association, rather than in the primary visual cortex, and that this defect would account for conflicting responses on electrodiagnostic tests. They also reported a distribution of brain lesions, with a relative sparing of cortical language areas, which they referred to as the "border zone." The "transient cerebral hypotensive episodes" within borderzone regions occurred between the distribution of the anterior posterior, and middle cerebral arteries. Lesions in this distribution would account for neurological findings, with the brunt of the lesions falling on the primary visual, sensory, and motor areas of the cortex and their respective association areas.

Almost all the studies reviewed expressed the need for the continued examination of brain damage in children and its effect on their visual functioning. Gardner et al. (1986) reported a lack of data on the visual functioning of visually impaired children from birth to age five. In their review of the literature, they found no recent complete estimates of the incidence or prevalence of multiple impairments in children who are visually impaired. Hot-Van Duin and Mohn (1984) discovered that in spite of the many children who sustain cerebral hypoxia in the perinatal period and the extensive brain damage incurred, few studies have explored sensory defects. They also found no detailed information about visual deficits in children with neurological abnormalities. Mellor and Fielder (1980) noted that electrodiagnostic studies had not been reported in any series of infants showing delayed visual maturation, either in the presence or absence of associated mental retardation, even though there has obviously been a direct relationship between the two conditions.

Many of the authors considered that the previous research was incomplete. Holman and Merritt (1986) stated that despite the finding by many researchers of the "increased incidence of infantile esotropia among neurologically impaired patients...few clinicians have [performed] the same surgical procedures for" neurologic and 'normal' esotropes," perhaps because they thought that the neurological involvement in these children would respond differently to treatment. Nass's study (1984) of unilateral brain damage concluded that "further studies comparing children of different ages at lesion and testing with comparable unilateral lesions will be required to map fully the effects of early brain injury." In addition, few studies have looked longitudinally at the developmental levels of term infants with hypoxic-ischemic encephalopathy. Robertson & Finel (1985) or have provided information on the visual potential of children with congenital or acquired visual disorder (Zeit & Biglan, 1985).

Finally, Gardner et al. (1986) cited the need for the standardized detection, evaluation, and recording of visual impairment, particularly in children with multiple handicaps, whose visual impairments are usually underreported. As was mentioned earlier, the increase in the population of children with brain damage who have visual deficits should encourage the accurate documentation of the type and frequency of these residual deficits.

**Conclusion**

This review of the literature has focused on the reported effects of brain damage on the visual functioning of infants and children. Regardless of the severity of brain damage, the vision of all the children was affected. Not only was there a high incidence of visual dysfunction, but visual loss was the most troublesome residual effect. The most common form of brain damage was hypoxic-ischemia, which caused many visual disorders, but primarily cortical visual impairment, tunnel vision, field losses, and strabismus. Lesions in the right hemisphere caused the most severe damage to visual perception, with
a lower probability of recovery. The levels of severity of visual dysfunction ranged from severe functional blindness to the loss of visual perceptual abilities.

The specific diagnostic tests reviewed in this article indicate that more information is gained using a combination of tests and that the prognosis is more optimistic than was previously thought, especially for children with multiple handicaps or mental impairments. The studies suggested that no one electrodiagnostic test was capable of giving a complete view of both brain damage and related visual dysfunction. The use of complementary tests not only yielded a more complete picture, but indicated that the visual functioning of the children could improve. Many of these studies demonstrated that testing alone frequently caused improvement in some of the children, but some of these studies found that electrodiagnostic testing may yield inconsistent results. Since cortical visual impairment shows no physiological involvement, it is often underreported and frequently overlooked. These tests, however, suggest that cortical visual impairment—a frequent, direct effect of brain damage—had far more potential for improvement and even recovery than was previously thought, particularly in infants and children who are mentally impaired. Holman and Mertz (1986) found that surgery to correct esotropia caused by neurological impairment, even though not as accurate as with esotropia that is not related to neurological impairment, caused developmental gains almost immediately. Isaacsong (1976) reported that growth occurs in the brain whatever the severity of damage. As long as growth occurs, rehabilitation can occur; this is an important finding.

This review of current research on brain damage and its effect on visual functioning in children has brought to light an important point: Brain damage often results in visual impairment. All the studies found relationships between brain damage and deficits in visual acuity or in visual perception. Since the prognoses was frequently determined to be more optimistic than was previously thought, perhaps treatment could be more successful. However, research in this area has been limited, and some of it is not current. Although electrodiagnostic testing could be used to confirm information that has already been obtained on the treatment of children with visual impairments caused by certain types of brain damage, more research is clearly needed. This research should focus on more detailed information regarding the relationship between specific types of brain damage and certain types of visual dysfunctioning. Information from current and future studies, could be used to develop standardized identification, evaluation, and recording procedures for children who are considered to be at a high risk, especially those with multiple handicaps.

References