Cortical Visual Impairment in Childhood

Subjects: Neurosciences | Pediatrics

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Definition

Cortical visual impairment in childhood is a kind of visual damage congenitally sustained by children.

1. Introduction

A principal reason for visual dysfunction in childhood in developed countries is CVI [1][2][3]. This has occurred as technology development has led to better visual treatment for other conditions such as congenital glaucoma, retinopathy of prematurity, and congenital cataracts as well as the increased survival of infants with central nervous system damage or disease.

The incidence of CVI has increased, with it now being a highly significant public health concern. Approximately 30–40% of children with visual impairments have CVI. The National Institutes of Health website cites a CVI prevalence of 10.5% of all children with developmental disabilities [4]. Generally, the prevalence of visual impairment in children under 16 years ranges between 10–22 per 10,000 births in developed countries and 40 per 10,000 births in developing countries [5][6].

In children with cerebral palsy, approximately two-thirds also demonstrate impaired visual acuity and/or field defects indicative of CVI [6]. In an African study, 47.7 percent of cerebral palsied children also demonstrated CVI [6][7], and in India, reports of 28 percent have been described [6][8].

An infant or child is said to' have CVI if (a) the loss of functional vision cannot be explained completely by an eye examination; (b) has a history of neurological dysfunction even with brain imaging studies appear to be normal; (c) demonstrates an array of visual or behavioral features identified in medical, psychological, or educational research [9]. As CVI is a consequence of brain insult rather than ocular dysfunction, an understanding of the dynamic properties of neurological development of the infant and child can assist in planning and developing better treatment protocols that may influence the developing child's functional vision. The process of neuroplasticity related to the development and function of the visual system will be discussed.

Damage, insult, or dysfunction to the visual system during fetal, neonatal and infant development may well have long-term consequences that are, as we shall see, potentially more capable of alteration and restoration of function in the infant and child as compared to similar insult in adults [10].

2. Is Recovery of Normal Conscious Vision Possible?

Our visual perceptual abilities are dependent on the pathways represented in **Figure 1** [11][12][13]. Guzzetta and associates [14][15] propose that three criteria are necessary for the restoration of vision that includes: (a) pathology of involvement of the geniculostriatal pathway, (b) specific loss of vision that is independent of any other functional abnormality, and (c) regaining the formerly impaired function with concomitant empirical change in brain state or electrophysiological activity.

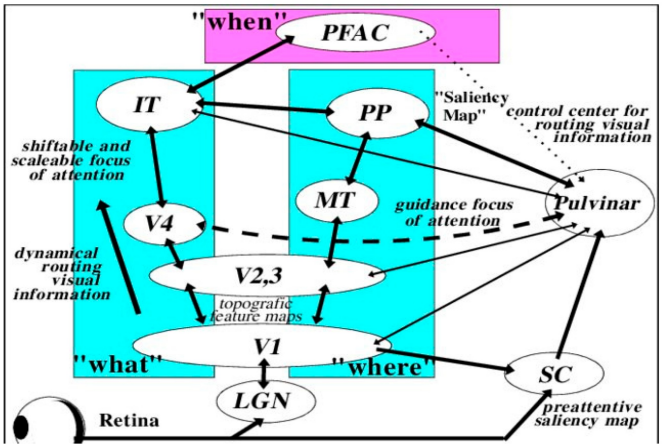

Figure 1. Major visual processing pathways of the primate brain considered in Gross et al.'s [13] model. Information from the retino-geniculostriatal pathway enters the visual cortex through area V1 and then proceeds through a hierarchy of visual areas that can be subdivided into two major functional pathways. The so-called "what"-pathway leads through V4 and the inferotemporal cortex (IT) and is mainly concerned with object-feature identification, regardless of position or size. V4 is the third area in the ventral stream obtaining strong feedforward signals from V2. Additionally, it receives projections directly from V1. The "where" pathway leads into the posterior parietal areas (PP) and is concerned with the locations and spatial relationships among objects, regardless of their identity. The "when" pathway involves the integration of signals from "What" and "Where" allowing for preplanning of movement and therefore response. (PFAC, prefrontal association cortex; IT, inferotemporal cortex; PP, posterior parietal areas; MT, middle temporal visual area; LGN, lateral geniculate nucleus; SC, superior colliculus) (after Gross et al. [13] with permission).

Adult functional vision is significantly different than that of the child with, among other things, evidence that the type of neuroplastic reorganization allowing for visual perception in the blind field seen in childhood cortical visual area insult has no adult analog [16]. A restricted broadening of the visual field can be normally seen shortly after the cerebral insult. This is a consequence of the resolution of temporary dysfunction of perilesional regions [17][18] or from modifications like the neural pathways adjacent to the lesion, including the size of the receptive field [17]. Beyond spontaneous recovery, partial or otherwise, support exists that interventions exist that can positively affect the size of the visual field through the recruitment of additional less efficient yet still intact visual pathways [17][19][20][21][22].

On the other hand, Bouwmeester and colleagues [23] found no support that limited broadening of visual field results in improved ocular motor scanning strategies or improvement in activities of daily living. This may be a consequence of the paucity of studies examining functions such as contrast sensitivity or line and edge detection in stroke in childhood or the lack of data on blindsight and certainly on the Sprague Effect in childhood CVI. The functional vision adaptation and visual pathway alterations noted after cerebral insult are likely not the result of direct recovery of lost vision, but rather the effect of learned compensatory visual-motor strategies [17][24].

3. Neuroplasticity and Developmental Damage to the Primary Visual Cortex (V1)

Our essential question is whether there is evidence to support the reestablishment of visual function when the insult occurs in infancy or early childhood? Related to developmental insult from animal studies, we have learned that lesions in early development generate greater recovery of functional visual capacities when compared with lesions acquired later in the life cycle.

The functions studied in cats and monkeys have included significantly enhanced performance in the discrimination of motion [25], shape [26], depth [27], and visual orientation [28], with early lesions in both animal and human revealing significantly greater neuronal rewiring and reorganization with lesions early in development [21][29]. Teuber, already in the 1970s [30] had examined individuals with lesions in the

occipital regions resulting from an injury that had occurred between adolescence through the twenties. Teuber found a relationship between the age at which the insult had occurred and the degree of shrinkage of the scotoma.

We know that children who have undergone hemispherectomy for conditions such as Rasmussen's syndrome [31] in early childhood fare better and, considering the drastic surgery, have relatively little functional impairment when compared with adults having undergone similar procedures [32][33]. There is much support since then for the notion that early developmental lesions, especially those that are congenital, are significantly more disposed to neuroplastic restructuring with the developing nervous system being more likely to employ compensatory pathways to counteract the effects of damaged brain areas thereby restoring functional vision.

Of great relevance to our argument of functional neuroplastic changes in early visual system insult, are the numerous reports of neonatal visual system damage highly associated with compensatory functions that are normally controlled by damaged regions of the brain [34][35]. A case was reported by Werth [36] of a hemispherectomized infant of 4 months who had later demonstrated intact visual fields. One reason that might support the neuroplastic changes in the pathways involved in hemianopsia in infancy maybe that area V1 of the integrated intact hemisphere also acts in response to the simulation of the ipsilateral blind hemifield. Cornwell and associates [37][38] proposed that a separate reorganizational pattern is evidenced in congenitally acquired lesioned individuals as opposed to lesions acquired later in life. Possible neuronal rearrangement patterns are represented in **Figure 2**.

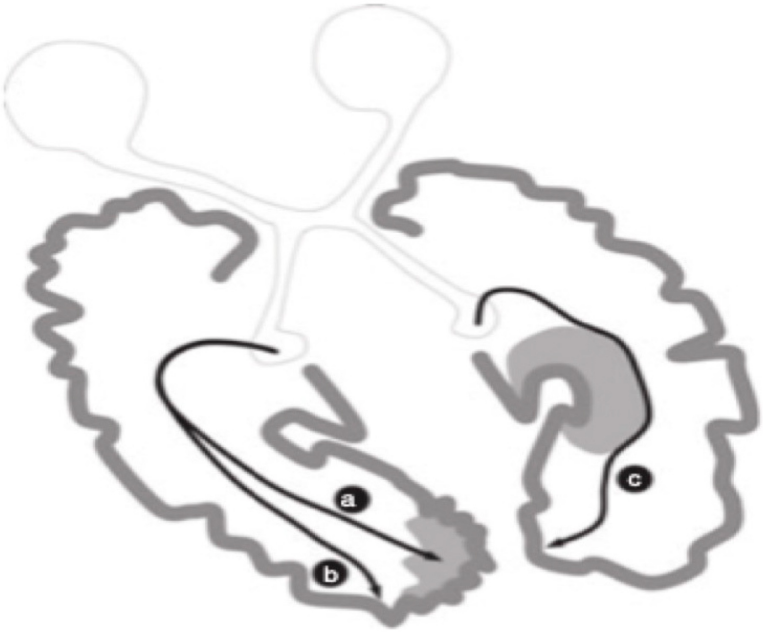

Figure 2. Potential mechanisms of neuroplasticity-based functional reorganization supporting normal visual function in congenitally brain-damaged individuals. (**a**) Represents damage to the PVC with functioning tissue existing within the lesion (**b**) aa reorganization occurring in regions external to the accepted boundaries of the PVC; (**c**) the geniculostriatal pathway bypassing the lesion and projecting to the calcarine cortex (after Guzzetta et al. [39] with permission).

Supporting the above arguments are animal studies in which cats lesioned in infancy demonstrate no significant difference with intact cats for complex pattern, shape, and hidden figure discrimination in contradistinction to adult-lesioned cats [37][38]. These findings have been confirmed by others [40][41]. Moore and colleagues [42] also noted that early lesioned monkeys demonstrated normal detection ability shortly after the lesion and recovered function relatively quickly thereafter. In subsequent studies, Moore and colleagues [43] also noted that monkeys with early striatal lesions demonstrated oculomotor movements appropriate to the direction of the stimulus presentation having seemingly had motion detection within the scotoma spared.

Also, in the 1990s, Mercuri, and associates [44] found significant visual field deficits with kinetic in infants who had suffered arterial stroke perinatally. When these investigators performed follow-up examinations of the same infants when they reached school age, no evidence of visual field defect was noted [17][45]. There are numerous possible explanations for the result, including geniculostriatal pathway changes, which will be discussed more fully later in the context of the Sprague Effect. However, as it is quite possible that infant visual field testing is performed by directing focus to the midline, the infant must uncouple attentiveness from the central field to a novel peripheral stimulus thus allowing the non-damaged visual cortical pathways to support a shift in attention. The findings reported in the follow-up studies of Mercuri and associates [46] may have been the result of the development or the capacity to modify the focus of attention as opposed to the expansion of the visual fields.

Reinforcing this notion is the fact that of the children studied, all of whom demonstrated deviant visual filed during their first year, also presented with parietal lesions evidenced by MRI with sparing of the optic radiations and the primary visual cortices [46]. We can add that there are multitudes of mechanisms of visual development occurring at different developmental stages confounding comparison between components of recovery.

A singularly important question is that when there exists physical damage to the visual pathways in infancy or early childhood, is it possible to know whether the maintenance of normal vision is a function of neuroplastic reorganization or rather a result of some other process? Developmentally we know that occipital lobe structural abnormalities do not necessarily universally produce defects of the visual fields [45][47][48][49] as it is known that with developmental cortical deformities, neuroplastic mechanisms are likely to correct such problems early in development.

We can presume that the mechanism subserving the reorganization of pattern detection relates to the effectiveness of the tissue surrounding the visual system lesion as represented in **Figure 1**. Dumoulin and associates [50] had found involvement of dysplastic tissue active in visual information processing assumed to be the result of neuroplastic network reorganization. These results have been observed by numerous other authors in both adults [51][52][53][54][55][56][57] and especially in children [34][58][59]. The consistently observed degree of functional residual vision found in early lesioned animals is related to a substantial visual system rewiring and of neural system activity adjustment.

4. Conclusions

The elaborate network oftentimes referred to as the brain’s “wiring” or “circuitry” is known to expand exponentially during fetal, neonatal and infant development. If connectivities are not maintained to repeatedly employed they are oftentimes eliminated and conversely when employed and firing together we can create novel functional connectivities, a process we understand as neuroplasticity. This function, explained in detail elsewhere [60][61][62] supports the notion that early childhood is the time to build either a strong and supportive, or fragile and unreliable foundation, which continues into childhood, adolescence, and adulthood.

The capacity for vision develops early in the development of the fetus and therefore in life. Reid and colleagues [63] confirmed that a fetus of 34-weeks GA can rotate the head to track patterned visual stimuli projected into the uterine environment. They concluded that visual-motor development is facilitated by a “gestational clock” rather than by, as most think, interactive visual experience that facilitates neuroplasticity in the visual system. There inevitably must exist a relationship between the fetus’s environment and visual development. During the third trimester of pregnancy, the vulnerability of white matter damage can be significant in which the optic radiations may be sensitive to insult. This is largely evidenced by unilateral periventricular hemorrhagic infarcts as well as bilateral ischemic lesions. It is during the third trimester of pregnancy that we note the significant degree of plasticity in the development of thalamocortical afferents, as it is that during this period, with relevant axonal guidance, that there still exists migration of afferents from the sub- to the cortical plate.

A case reported by Seghier and colleagues [64][65] was discussed, about a three-month-old infant who had sustained a perinatal left arterial stroke with damage to the optic radiations but with sparing of the primary visual cortex PVC. Initial testing revealed cortical activation on the non-lesioned side with no evidence of the optic radiations on the ipsilesional side. When a follow-up study was performed at twenty months of age, fMRI demonstrated clear activation on the ipsilesional side representing neuroplastic changes in the thalamocortical pathway with attendant functional connectivities in the lateral geniculate. We can conclude from this and other cases that early insult to the PVC or optic pathways provides the infant with a critical period of developing neuroplastic changes that are better capable of allowing for the development of functional vision and with support for the conclusion that the child's brain has a better capacity for rewiring than that of the older brain. The hypothesized mechanism for the relatively low prevalence of visual abnormalities post-stroke in children as compared to adults is assumed to be due to neuroplasticity within the visual system.

Congenital or neonatal brain damage invariably leads to bilateral injury commencing in the periolandic areas and involving gray matter and cortical regions of the brain but with the capacity for restoration of aspects of vision. In particular, we have learned that there oftentimes exists involvement of the geniculostriatal pathway, a specific loss of vision that is independent of any other functional abnormality, and that the regaining of the formerly impaired function with concomitant empirical change in brain state or electrophysiological activity is evidenced.

Besides understanding developmental aspects of visual neuroplasticity, we also examined the Sprague Effect [66] in which small tectal lesions can reestablish visual orientation in the half field contralateral to the lesion in animals with significant unilateral geniculostriatal impairment. More specifically, the Sprague Effect can be better understood by an understanding of its relationship to hemispatial neglect, a failure to react to novel stimuli presented to aspects of visual space, and without being able to attribute the lack of responsivity to a specific lesion.

The consequence of our discussion leads to an understanding that in those with central visual field defects, extrastriatal visual connectivities are greater when a lesion occurs earlier in life as opposed to when it occurs in the neurologically mature adult. The result is a significantly more optimized system of visual and spatial exploration within the 'blind' field of view. This then can serve as a basis for developing interventional schemes in congenital visual system insult.

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