

2

Plasticity of the human visual brain after an early cortical lesion.

Mikellidou K, Arrighi R, Aghakhanyan G, Tinelli F, Frijia F, Crespi S, De Masi F, Montanaro D, Morrone MC

show author affiliations

Neuropsychologia. 2017 Oct 31

  Save/Follow  Get Article  ExLibris SFX

   Share

INTERESTING ARTICLE? GET MORE LIKE IT SMARTSEARCH 

RECOMMEND DISSENT

RECOMMENDATIONS 1 | ABSTRACT | COMMENTS

expand all 

Recommendations:

★★ Very Good

13 Dec 2017



FM Marlene Behrmann

F1000 Neuroscience

Carnegie Mellon University, Pittsburgh, PA, USA.

 FOLLOW

INTERESTING HYPOTHESIS | NEW FINDING

DOI: 10.3410/f.732073581.793538629

This fascinating paper documents near-normal central field vision in an individual with a massive unilateral right lesion to the optic radiations as a result of surgery to resect a subependymal giant cell astrocytoma at age 3 months. Very detailed psychophysics investigations (e.g. measuring visual acuity and contrast sensitivity) and neuroimaging (structural and functional) reveal surprisingly good residual vision, including conscious vision (in contrast with blindsight). An impairment in form and contrast vision was observed in the far periphery of the affected visual field. Intriguingly, the middle temporal complex (MT+) and the parieto-occipital sulcus (POS) in the intact hemisphere show responses to both contralateral and ipsilateral field stimulation and strong structural connections between MT+ and the lateral geniculate nucleus (LGN) were noted on diffusion measurement. This study reveals that strong thalamo-cortical projections may serve as the neurobiological basis of plasticity in such cases.

Disclosures

None declared

[Add a comment](#)

Abstract:

ABSTRACT

In adults, partial damage to V1 or optic radiations abolishes perception in the corresponding part of the visual field, causing a scotoma. However, it is widely accepted that the developing cortex has superior capacities to reorganize following an early lesion to endorse adaptive plasticity. Here we report a single patient case (G.S.) with near normal central field vision despite a massive unilateral lesion to the optic radiations acquired early in life. The patient underwent surgical removal of a right hemisphere parieto-temporal-occipital... [more »](#)

atypical choroid plexus papilloma of the right lateral ventricle at four months of age, which presumably altered the visual pathways during in utero development. Both the tumor and surgery severely compromised the optic radiations. Residual vision of G.S. was tested psychophysically when the patient was 7 years old. We found a close-to-normal visual acuity and contrast sensitivity within the central 25° and a great impairment in form and contrast vision in the far periphery (40-50°) of the left visual hemifield. BOLD response to full field luminance flicker was recorded from the primary visual cortex (V1) and in a region in the residual temporal-occipital region, presumably corresponding to the middle temporal complex (MT+), of the lesioned (right) hemisphere. A population receptive field analysis of the BOLD responses to contrast modulated stimuli revealed a retinotopic organization just for the MT+ region but not for the calcarine regions. Interestingly, consistent islands of ipsilateral activity were found in MT+ and in the parieto-occipital sulcus (POS) of the intact hemisphere. Probabilistic tractography revealed that optic radiations between LGN and V1 were very sparse in the lesioned hemisphere consistently with the post-surgery cerebral resection, while normal in the intact hemisphere. On the other hand, strong structural connections between MT+ and LGN were found in the lesioned hemisphere, while the equivalent tract in the spared hemisphere showed minimal structural connectivity. These results suggest that during development of the pathological brain, abnormal thalamic projections can lead to functional cortical changes, which may mediate functional recovery of vision.

Copyright © 2017 Elsevier Ltd. All rights reserved.

DOI: 10.1016/j.neuropsychologia.2017.10.033

PMID: 29100949



Abstract courtesy of PubMed: A service of the National Library of Medicine and the National Institutes of Health.

Comments:

COMMENTS

[add a comment](#)