Topographic restoration of visual spatial attention in the cortically blind cat

Claus C. Hilgetag\textsuperscript{a,b,c,*}, Stephen G. Lomber\textsuperscript{b,d}, Richard J. Rushmore\textsuperscript{b}, Bertram R. Payne\textsuperscript{b}

\textsuperscript{a}School of Engineering and Science, International University Bremen, Campus Ring 1, Bremen, D-28759 Germany
\textsuperscript{b}Laboratory for Visual Perception and Cognition, Department of Anatomy and Neurobiology, Boston University School of Medicine, 700 Albany Street, Boston, MA 02118, USA
\textsuperscript{c}Department of Health Sciences, Boston University, 635 Commonwealth Ave., Boston, MA 02218, USA
\textsuperscript{d}Cerebral Systems Laboratory, School of Human Development, University of Texas of Dallas, P.O. Box 830 688, GR41, Richardson, TX 75083, USA

Abstract

We explored the mechanisms of spatial attention in the cat with a combination of permanent and reversible lesion techniques. Animals with large unilateral visual cortical lesions displayed profound blindness of contralesional visual space. However, cooling deactivation of either posterior parietal cortex or the superior colliculus (SC) in the contralesional half of the brain restored visual orienting responses in the blind hemifield. Interestingly, gradual SC deactivation by decreasing cooling loop temperatures produced a topographic restoration of orienting responses, starting at the midline and progressing towards more peripheral eccentricities in the previously blind field. These effects can be explained within a computational model for spatial orienting, which represents the interhemispheric competition of bilateral brain structures that possess a topographic, center-magnified representation of the external visual space. © 2002 Published by Elsevier Science B.V.

Keywords: Orienting behavior; Spatial hemineglect; Posterior parietal cortex; Superior colliculus; Sprague effect; Reversible deactivation
1. Introduction

Previous work, e.g. [1], has shown that small unilateral lesions of posterior parietal cortex in the cat, at the posterior end of the middle suprasylvian sulcus (pMS), create behavioural deficits in visual orienting tasks. However, these deficits are transient, and the animals recover completely after only a few days [7]. On the other hand, large unilateral lesions of visual cortex produce permanent hemianopia that includes visual neglect. Earlier work has also shown that visual orienting deficits produced by focal pMS cortex deactivation can be reversed by additional, equivalent deactivation of pMS cortex in the contralateral hemisphere [4]. In the present study, we explored the opportunities for restoring spatial orienting capabilities after large unilateral visual cortical lesions, and related the experimental observations to an existing model for spatial attentional mechanisms in the cat [2,3]. For a detailed description of the experimental results see [5].

2. Methods

As in previous studies, an orienting paradigm was used to evaluate the impact of the experimental brain deactivations on attentional behavior. Cats were tested for their ability to redirect attention from a midline cynosure (such as a low-incentive food stimulus) to a new, moving stimulus presented between left and right 90° field eccentricity. Immediate and strong orienting responses to the correct stimulus eccentricity were rewarded with a morsel of high-incentive food. Orienting to both moving visual and white-noise auditory stimuli was tested.

Large permanent lesions were produced by aspiration of all contiguous visual cortical areas in either the left or right hemisphere. Additional focal deactivations of either parietal cortical sites or of the superior colliculus (SC) in the contralateral half of the brain were produced by cooling target structures to below 20°C with permanently implanted cryoloops brought into contact with the surface of the target sites [6]. The cryoloops were fitted with a microthermocouple for monitoring cooling temperatures, and cooling was effected by circulating chilled methanol through the cryoloop. Finally, the extent of the cooling deactivation was verified by 2-deoxyglucose (2DG) metabolic mapping.

We related experimental observations to the effects produced in a spatial orienting model based on known brain connectivity [2,3]. The computational model suggests that impaired orienting behavior is produced by imbalanced competition between bilateral brain structures possessing a topographic, center-magnified representation of the external space. The competitive bilateral model is based on anatomical and physiological features of the intermediate and deep layers of the SC in the cat. Important for the behavior of the model are its incorporated structural aspects: the two colliculi possess a topographically mapped representation of space; this representation strongly magnifies the midline region; mutually inhibitory connections mediate competitive interactions between the two colliculi, the interactions are also organized topographically such that mirror-symmetric field eccentricities inhibit each other; and finally, the bilateral system receives dominant visual input via cortical afferents, but also receives weaker extra-cortical visual input [3].
3. Results

Animals with intact cortex oriented without difficulties to visual stimuli at all field eccentricities. However, after unilateral removal of either the left or right contiguous visual cortices, the animals did no longer respond to visual stimuli presented in the contralesional hemifield. These deficits did not change during repeated testing over several weeks or months.

In addition to the permanent cortical lesion, we reversibly deactivated brain structures in the contralesional hemisphere, and verified, by 2DG-imaging, the depth and the focus of the cooling deactivation. Visual orienting responses were restored by deactivation of two of the tested contralesional brain structures, pMS cortex and the SC.

Whereas cooling of the pMS cryoloop down to about 10°C did not produce any change in behavior, a deactivation temperature of about 8°C led to a reversal of the visual orienting deficit. This restoration result was specific for deactivation of pMS cortex, as cooling deactivation of posterior extrasylvian cortex, on the gyrus immediately lateral to pMS cortex, or of area 7, on the crown of the gyrus adjacent and medial to pMS cortex, did not restore orienting, at any deactivation temperature.

In a different animal, the effects of gradual contralesional collicular deactivation were explored. Whereas no changes in behavior were observed for cryoloop temperatures of 20°C and above, a gradual decrease of deactivation temperatures below this threshold induced a graded and ordered restoration of orienting capabilities, expanding from the center to the periphery of the previously ignored visual hemifield. Restoration was complete when the superficial layers of the SC were completely deactivated. This effect is surprising, since the cooling deactivation was not specifically matched to collicular topography. We therefore explored the graded collicular restoration effect with the help of a simple theoretical model proposed previously to simulate intact, impaired and restored spatial orienting in the cat.

The model simulations considered the gradual deactivation of visual afferents into a SC that is contralateral to a colliculus which has been depleted of all its visual cortical inputs (cf. Fig. 1). We derived the activity distribution in the collicular model for decreasing inputs to the intact side both in the presence and absence of external stimuli. The modeled stimulus-related activity indicated that for the strongly imbalanced system (lesioned cortical inputs opposite to completely intact inputs) none of the stimuli presented anywhere in the blind hemifield produced activity higher than the baseline of spontaneous activity. No orienting responses into the blind hemifield would be expected on the basis of such an activity distribution. For moderate deactivation of the contralesional collicular inputs, however, activity related to stimuli presented near the midline in the blind hemifield rose above the activity baseline, and so orienting could be expected to stimuli at more central eccentricities. Finally, a strong deactivation of the contralesional collicular input restored the competitive balance between the bilateral structures, and produced stimulus-related activity that would exceed the baseline threshold for all stimulus eccentricities in the blind hemifield. In summary, the outcome in the model followed the experimental results of an orderly, graded restoration of orienting from the midline to the periphery for a gradual deactivation of contralesional collicular inputs (Fig. 1). This graded reversal can be linked to the overrepresentation
of central visual space in the modeled brain structures which follows the known magnification factors for the SC.

4. Conclusions

Our results show that deactivation of a small, specific region of posterior parietal cortex can reverse visual orienting deficits resulting from a large visual cortical lesion in the opposite hemisphere. Such a reversal is also possible through contralesional collicular deactivation, with the reversal proceeding in a topographically ordered fashion. The deactivation effects observed in our study are in agreement with an orienting model that is based on topographically organized connectivity and inter-hemispheric competition.

The results confirm that pMS cortex and the SC represent important cortical and midbrain nodes in a bilaterally-balanced brain network for visual orienting. This is in line with earlier studies showing that collicular lesions can reverse attentional deficits produced by large contralateral visual cortical lesions [8], and that visual hemineglect induced by unilateral deactivation of pMS cortex can be reversed also by deactivating contralateral pMS cortex [4]. The latter study also found an intriguing hysteresis-like effect, in which the induction of hemineglect required deactivation of all pMS cortex...
layers, whereas restoration of attention only required deactivation of the superficial layers of contralateral pMS cortex. In the present study as well, restoration of attention to the blind hemifield was produced by just deactivating the upper layers of contralesional pMS cortex, hinting on the significance of intrinsic and transcortical pathways that originate in these layers and that may provide the connectional basis for signal amplification in, as yet unknown, cortical and subcortical regions. It remains an important challenge for the future to identify all members of this distributed attentional network, characterize their specific functions, and explore their functional interactions.

Acknowledgements

Supported by NINDS, NSF and the Wellcome Trust.

References


Claus C. Hilgetag studied Biophysics in Berlin and Neuroscience in Edinburgh, Oxford, Newcastle and Boston. He is now an Assistant Professor of Neuroscience at the newly founded International University Bremen. His current research focuses on computational analyses of neural architecture and connectivity, and on understanding the mechanisms of spatial attention and inattention in mammalian brains.

Stephen G. Lomber is an Assistant Professor of Neuroscience at the School of Human Development of the University Texas at Dallas and a Research Assistant Professor of Anatomy and Neurobiology at the Boston University School of Medicine. His principle interests concern functional interactions and behavioral cartography of extrastriate visual cortex.

R. Jarrett Rushmore is a graduate student in the Department of Anatomy and Neurobiology at Boston University School of Medicine. He is interested in the dynamic and adaptive properties of visual system circuitry.

Bertram R. Payne is a Professor of Anatomy and Neurobiology at Boston University School of Medicine. His interests lie in the functional organization and plasticity of cerebral cortex.