Critical periods for functional recovery after cortical injury during development

Bryan Kolb and Robbin Gibb

in Reprogramming the Cerebral Cortex: Plasticity following central and peripheral lesions

This chapter reviews studies in laboratory animals, and especially rats, in which precise developmental age has been manipulated and both functional and anatomical outcome has been examined. It also considers treatments that can modify the age-dependent effects of early cortical injuries.

Reprogramming the motor cortex for functional recovery after neonatal or adult unilateral lesion of the corticospinal system in the macaque monkey

E. M. Rouiller, T. Wannier, E. Schmidlin, and Y. Liu

in Reprogramming the Cerebral Cortex: Plasticity following central and peripheral lesions

This chapter describes the mechanisms underlying reprogramming of the motor cortex in order to rehabilitate some motor control after a lesion affecting the central nervous system, namely the cerebral cortex or the cervical spinal cord. The corticospinal projection was chosen as a model to address the issue of reprogramming the cerebral cortex following a lesion occurring either at early (neonatal) or late (adult) stages. Reprogramming the motor cortex is used to refer to the re-establishment of functional control on motoneurons deprived of cortical inputs as a result of cortical or cervical lesion.
Remodeling of cortical connections and enhanced long-term potentiation after lesions of the visual cortex
Ulf T. Eysel and Thomas Mittmann

in Reprogramming the Cerebral Cortex: Plasticity following central and peripheral lesions

Long-term potentiation (LTP) was first described in the mammalian hippocampus and was also elicited in the visual cortex of rats. LTP is most strongly expressed during early postnatal development when synaptic plasticity is high. To test the hypothesis that lesion-induced reorganization in the visual cortex is associated with increased LTP, this chapter examines synaptic plasticity in slices of the lesioned rat visual cortex in vitro. Characteristic changes are in plasticity are observed in the surround of lesions, supporting the hypothesis of enhanced LTP being involved in reprogramming of the visual cortex in response to local damage in the adult visual cortex.

Behavioral and neural alterations following V1 damage in immature primates
Hillary R. Rodman

in Reprogramming the Cerebral Cortex: Plasticity following central and peripheral lesions

This chapter describes the behavioral deficits observed following lesions of the primary visual cortex in both mature and infant macaque monkeys. Overall, the consequences of such lesions are far more severe in adulthood than during development. The chapter also presents anatomical evidence that alterations to the thalamocortical pathways may explain the greater behavioral abilities identified following visual cortex lesions incurred during development, but not maturity.
The longitudinal study of spatial cognitive development in children with pre- or perinatal focal brain injury
Joan Stiles, Pamela Moses, and Brianna M. Paul

in Reprogramming the Cerebral Cortex: Plasticity following central and peripheral lesions
Published in print: 2006 Published Online: September 2009
DOI: 10.1093/acprof:oso/9780198528999.003.0024
Item type: chapter
Publisher: Oxford University Press

This chapter discusses the perceptual effects of pre- or perinatal lesions. It shows that when subjects were asked to perform a task the outcome may appear normal, however, procedural affects may be present. Imaging techniques are used to study anatomical and functional changes related to recovered behavior.

Colour vision and its disturbances after cortical lesions
C.A. Heywood and A. Cowey

in The Neuropsychology of Vision
Published in print: 2003 Published Online: March 2012
DOI: 10.1093/acprof:oso/9780198505822.003.0008
Item type: chapter
Publisher: Oxford University Press

This chapter provides a discussion on colour vision and its disturbances after cortical lesions. It specifically describes that it is less clear whether colour constancy can be selectively impaired. It is shown that the orientation and direction selectivity of cells in the M-channel, along with their high luminance contrast gain, indicates that it plays a role in conveying motion and form information to visual areas in the parietal lobes. It is also noted that the loss of colour vision in achromatopsia can leave other wavelength-based processing intact. Moreover, the described studies demonstrate that wavelength variation in the visual scene can be used, not only to produce colour experience, but also to determine further attributes such as form and motion. It is stated that chromatic information plays a manifold role in vision.

The Processing of Motion-Defined Form
Deborah Giaschi

in Seeing Spatial Form
Published in print: 2005 Published Online: March 2012
Publisher: Oxford University Press
One type of motion-defined form is keeping a figure steady but moving the texture inside the figure in relation to that outside it. This chapter presents studies on this type of motion-defined form using patients and children. As seen in the results, in patients with multiple sclerosis, cortical lesions, amblyopia, or dyslexia, motion-defined form processing may not be associated with the processing of simple motion stimuli.

Disturbance of the 5-hydroxytryptamine metabolism in ageing and in Alzheimer's and vascular dementias

C. G. Gottfries

in 5-Hydroxytryptamine in Psychiatry: A Spectrum of Ideas

Syndromes of cognitive, emotional, and psychomotor disturbance in the elderly that are of disabling severity are called dementias and are classified as idiopathic, vascular (VD), and secondary dementias. The main groups of idiopathic dementias (primary degenerative or metabolic disturbances) are those of Alzheimer type. Originally Alzheimer's disease (AD) was the name of an early onset dementia with characteristic cortical lesions. However, similar neuropathological findings are made in the brains of patients with senile dementia; therefore, this group was named senile dementia of Alzheimer type (SDAT). The two forms are often brought into one group, Alzheimer-type dementia (AD/SDAT). Vascular dementia is diagnosed when there is an assumed causal relationship between vascular disorders and the appearance of dementia. Multiinfarct dementia (MID) is a subgroup of VD characterized by a temporary relationship between stroke attacks and the appearance of dementia. This chapter aims to review data indicating disturbance of the 5-HT system in patients with AD/SDAT and non-MID VD. Data about changes in the 5-HT metabolism in ageing are also reported.