Early plasticity versus early vulnerability: the problem of heterogeneous lesion types

Karen Lidzba, Marko Wilke, Martin Staudt and Inge Krägeloh-Mann

Department of Neuropediatrics, University Children’s Hospital, Tuebingen, Germany

Correspondence to: Karen Lidzba, Department of Neuropediatrics, University Children’s Hospital, Hoppe-Seyler-Str. 1, Tuebingen 72076, Germany
E-mail: karen.lidzba@med.uni-tuebingen.de

Sir, It was with great interest that we read the paper from Anderson et al. (2009) in which the authors addressed two competing hypotheses regarding developmental outcome after early versus late brain lesions. The first hypothesis assumes a greater compensatory potential during early brain development, while the second hypothesis proclaims that the young brain may be especially sensitive to insult. Both hypotheses are discussed controversially (Kennard, 1942; Taylor and Alden, 1997).

The authors have studied the consequences of the timing of lesion origin in a large group of children with brain lesions due to variable aetiologies. They interpret their data in favour of the second hypothesis. We would like to draw attention to two aspects which are well known to negatively interfere with cognitive function after brain lesions and which we believe the authors have not fully taken into account in their interpretation: (i) epilepsy (Vargha-Khadem et al., 1994; Muter et al., 1997); and (ii) bilaterality of lesions (Vargha-Khadem et al., 1985, 1997; Krägeloh-Mann et al., 1999).

First, the effects of seizures could not be studied in sufficient detail to exclude a confounding role, which was mentioned only briefly in the discussion. Due to the well-known interfering effects of epilepsy on cognitive functioning (for a review, see Nolan et al., 2003), we believe this to be a serious confound. Second, in comparing early (before second birthday) and late (after second birthday) lesions—the grouping used by the authors in discussing their results—bilateral lesions are significantly more frequent in the early group compared with the late group (Fisher’s exact test: 51 early unilateral; 57 late unilateral; 43 early bilateral; 23 late bilateral; P = 0.015). Therefore, the effect of lesion is confounded by the significantly higher presence of bilateral lesions in the early lesion group, which are known to have a worse prognosis than unilateral lesions (Vargha-Khadem et al., 1985, 1997; Krägeloh-Mann et al., 1999).

For these reasons, we find it difficult to share the authors’ conclusion that their results support the notion of higher vulnerability in early brain lesions. Instead, we believe that the study offers further evidence for the hypothesis that unilateral lesions offer more compensatory opportunities than bilateral lesions. Whether or not lesion timing is an additional or independent factor remains, for us, an open question.

References