Editorial

Appearances may not be deceiving: calculation deficits due to a brain structure abnormality in neurologically normal children

Calculation is an essential function learned in childhood that humans use in everyday life for many purposes. Calculation deficits typically occur in adults after left parietal lobe lesions (Grafman *et al*., 1982). Historical evidence suggested that, more specifically, the region of the left angular gyrus was crucial for the mediation of calculation, but more recent research using functional neuroimaging and patient studies indicates that the left intraparietal sulcus may be even more important for the representation of specific calculation processes (Levy *et al*., 1999; Rickard *et al*., 2000; Rueckert *et al*., 1996). Regardless of the exact location in the parietal lobe(s) of the key cortical structures concerned with supporting the computational mechanisms needed for aspects of calculation, significant advances have occurred regarding our knowledge about the cognitive architecture of calculation over the last two decades (Grafman and Rickard, 1997). These advances have allowed for more precise mapping of cognitive processes to brain sectors.

Dyscalculia is as prevalent as literacy problems in very low birthweight pre-term children. In this issue of *Brain*, Isaacs and colleagues used a new analytic technique, voxel-based morphometry, to identify a local region in the left parietal cortex where there was less grey matter in very low birthweight children who had dyscalculia than in very low birthweight children without dyscalculia (Isaacs *et al*., 2001). This difference in regional neural organization could not be seen with simple visual inspection of the MRI. While these dyscalculic children could typically perform simple calculations, they still performed significantly below expectation. Although developmental dyscalculia is a significant problem in children (Shalev *et al*., 2000), there has been some doubt as to the location of the pathology causing this problem. For example, as Isaacs and colleagues point out in their paper, there has been some consideration of a right hemisphere dysfunction causing developmental dyscalculia. Instead, Isaacs and colleagues have shown that morphometric changes in the grey matter in children with developmental dyscalculia occur in the same left parietal lobe region as the lesions that affect calculation ability in adults, and where functional neuroimaging in normal adults shows calculation-induced brain activation. It is worthwhile to note that these dyscalculic children did not have reading difficulty so their calculation problem cannot be attributed to a more general problem in language ability.

Voxel-based morphometry is a relatively new technique that enables brain sectors to be classified into various categories (grey matter, white matter, cerebrospinal fluid, scalp) (Ashburner and Friston, 2000). Although some smoothing of the image is necessary, regional analysis of proportion of voxels of these different categories within a region can be accomplished and then used, with appropriate correction for number of voxel comparisons across groups, to identify between-group differences in grey or white matter proportions within a region. This method does not require experts to draw regions of interest, but instead relies upon relatively automated comparisons. In this study, this comparison led to the identification of left intraparietal sulcus reduction in grey matter in children with dyscalculia. Clearly, this method could be important in determining differences in brain development in local brain regions in children who have, or who are at-risk for, other developmental disorders such as dyslexia so that we may identify the putative regional differences responsible for a range of cognitive deficits in individual children (Sowell *et al*., 2001). Could this technical advance bring us closer to a lesion-based developmental neuropsychology of children since the technique may be sensitive to neuronal migration defects or to damage to a local cortical neuronal network that minimally distorts the overall cortical structure, but results in slight changes to the local grey matter density and size (even if we do not know the exact course or timing of the neuronal abnormality)?

Isaacs and colleagues report that their results could not be accounted for by age or birthweight, although levels of plasma taurine and a measure of respiratory illness were predictors of dyscalculia (Isaacs *et al*., 2001). There were no differences in a 'freedom from distraction measure' between the groups they studied, so attentional deficits cannot account for the dyscalculia.

Several outstanding questions remain. Why didn't any neuroplasticity occur in these children so that they could have overcome the effects of their developmental lesion? Alternatively, could it be that the observed performance of these children is already the product of reorganization (i.e. plasticity) to compensate for the abnormality of these areas? What would an answer to either question teach us about the optimal circumstances to induce brain plasticity so that children with specific cognitive deficits may maximally

benefit from an intervention? Will these children ever be able to overcome their deficits through other compensatory mechanisms (i.e. deficit-specific training programmes that would induce the activation of brain areas distant from the structural abnormality, but associated with cognitive processes allied with the damaged function)? Would children with reading disorders have a different set of cortical areas involved in grey matter loss than the one seen here? Is a left intraparietal lesion sufficient to cause dyscalculia (these very low birthweight children only had partial deficits in calculation)? Perhaps this latter finding is evidence that other cortical regions do participate in calculation, or that some plasticity had occurred (Levin *et al*., 1996). Since most of the observed deficits were due to problems in carrying or borrowing numbers (as in multiplication), perhaps a selective memory deficit also plays a role in the children's impaired calculation skill? Of note is the fact that the investigators used only clinical, rather than more experimental, tests of number processing skill and calculation. Experimental arithmetic and skill learning tests would have helped better tease out more specific dyscalculia deficits (Stanescu-Cosson *et al*., 2000) and which learning stages might be affected.

In conclusion, Isaacs and colleagues used voxel-based morphometry to detect a grey matter abnormality in the left parietal cortex in children of very low birthweight who are dyscalculic. They have substantiated that dyscalculia in children and adults is due to cortical changes in a similar left parietal cortical region. This finding will not only be helpful to investigators studying normal and impaired number processing and calculation in children, but also should be of interest to researchers interested in neuroplasticity and in the use of novel analytic techniques for detecting abnormalities in the cortical architecture that might be hidden from simple visual inspection.

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