Sir, Anderson et al. (2009) refuted the Kennard Principle, which states that the immature brain is more plastic and less vulnerable to insult than a mature brain (Montour-Proulx et al., 2004); however, there are important errors in Table 5 of their paper. The IQ data for their six age subgroups do not tally with the total group figures in the last column of Table 5. In each of these six subgroups, the IQ figure in the last row is intermediate between the first two rows, so clearly must be the full scale IQ, and not performance IQ as stated (V, P and FS refer to verbal, performance and full scale IQs on the Wechsler Intelligence Scales). One would then assume, as is usually reported, that the first subgroup row is verbal IQ, the second performance IQ and the third full scale IQ. However, there are two major problems with this reading:

(i) the mean figure for full scale IQ for the whole group (last column in Table 5), intermediate between verbal IQ and performance IQ, is now in row 1 and not row 3. The mean total group figures as printed do not correspond to subgroup row order from Table 5 (FS/V/P), to the more usual proposed order (V/P/FS), or to that expected from the previous literature (FS/P/V).

(ii) In all six subgroups, verbal IQs would be lower than performance IQs. For those aged 3–6 years at lesion, verbal IQ would be 94 and performance IQ 100; for age 7–9 years, verbal IQ would be 94 and performance IQ 102; for 10–16 years, verbal IQ would be 95 and performance IQ100. For the total group of 160 children, the figures in Table 5 claim a mean verbal IQ of 86 (see also p. 49) and performance IQ of 91. It was the implausibility of these roughly 6-point specific verbal IQ deficits that led to detailed scrutiny of Table 5.

To check the unlikelihood that brain lesions, as a whole, impair verbal IQ more than performance IQ, I have reviewed a sample of recent papers, most of which were not cited by Anderson et al. (2009):

(i) in a sample of 417 children (Montour-Proulx et al., 2004), those with small unilateral lesions had verbal IQ 94 and performance IQ 97, with verbal IQ 81 and performance IQ 75 for large lesions. Corresponding figures for adults were verbal IQ 100, performance IQ 92 and verbal IQ 88, performance IQ 80. Those with early lesions had lower verbal IQ. Traumatic and non-traumatic lesions resulted in similar IQs.

(ii) For lesions in each of four lobes for two sexes (sample size 635) (Braun et al., 2001, 2002), verbal IQs were 2–6 points higher than performance IQs. There were no generalized sex differences; ‘P IQ is simply more sensitive to brain damage in general’.

(iii) In a further analysis of this large Montreal database (now 725 cases) (Duval et al., 2008), ‘the so-called Kennard principle is refuted’ for full-scale IQ. Also, contrary to the statement ‘a further limitation of previous literature is a failure to account for age at testing’, (Anderson et al., 2009) lesions in childhood were associated with a full-scale IQ decline over time, whereas adults benefited from a slight test–retest IQ increase.

(iv) Those with attention deficit hyperactivity disorder (ADHD) after childhood stroke (Max et al., 2003) had verbal IQ 85, performance IQ 80; and those without current ADHD had verbal IQ 98, performance IQ 90. ‘V IQ is considered a more accurate measure of overall intelligence than P IQ or FS IQ’. A five-test short form of the Wechsler Intelligence Scale for Children (WISC) was used, so it does not look as if Anderson et al.’s use of the Wechsler Abbreviated Intelligence Scale accounted for apparent reversal of the usual V/P tilt in their sample.

(v) In 10 children after stroke at mean age 8 years, verbal IQ was 103, performance IQ 94 (Pavlovic et al., 2006). For ten
with neonatal strokes, Bayley Mental Quotients were below 50 for 3 with seizures, 84 for the 4th with seizures, and 90-110 for the 6 seizure-free. Anderson et al. briefly noted this study but did not comment on how their apparent findings for the corresponding age group (verbal IQ 94 and performance IQ 102) were the mirror opposite.

(vi) In 23 children with perinatal stroke (Ballantyne et al., 2008), those without seizures had verbal IQ 105 and performance IQ 101, and those with seizures had verbal IQ 79 and performance IQ 76. For small lesions, verbal IQ 95 and performance IQ 97; for multi-lobe lesions, verbal IQ 98 and performance IQ 91; for left hemisphere lesions, verbal IQ 96 and performance IQ 89; for right, verbal IQ 97 and performance IQ 97. Anderson et al. (2009) refer to this paper with respect to seizures only. In fact, there is a large highly relevant literature since 1924 (Hermann et al., 2002) on the cognitive outcome of seizures.

(vii) In 1141 patients with refractory seizures (mean verbal IQ 90 and performance IQ 90), age at seizure onset was the strongest single correlate of full-scale IQ, which increased linearly from onset before 1 year of age (IQ 84) to adult onset (IQ 93) (Strauss et al., 1995). This confirmed the literature review, particularly a previous study with corresponding IQs of 83 and 102. These figures are very similar to those of Anderson et al. (82/96), strongly contradicting the Kennard Principle. There was no effect due to duration of seizures. Reduced white matter volume was related to earlier onset of seizures but not to their duration or number, and correlated equally with verbal and performance IQ (Hermann et al., 2002). Those with onset of epilepsy over age 14 years were neuropsychologically similar to controls. In 242 with severe epilepsy, Glosser et al. (1997) found that those with early neurological risk factors and early onset seizures had verbal IQ 88 and performance IQ 84, full-scale IQ 85; those with early risks but late onset seizures had verbal IQ 92 and performance IQ 92, full-scale IQ 91; those with no early risks and late onset verbal IQ 96 and performance IQ 96, full-scale IQ 96. There was no effect of duration of epilepsy or extent of seizures.

(viii) In 10 children with perinatal brain damage (Sava et al., 2005), five had mean verbal IQ 129 and performance IQ 90, five with larger cortical lesions had verbal IQ 78 and performance IQ 73.

(ix) Thirty-six children with mostly severe traumatic brain injuries (Campbell et al., 2004) had baseline verbal IQ 84 and performance IQ 83. Longer hospital stay and head injury severity correlated with the performance IQ and not verbal IQ, consistent with prior literature. IQ was unrelated to age at injury onset (from 4 to 16 years).

Conclusions

In children with brain lesions, verbal IQs are higher than performance IQs, sometimes much higher. Mean verbal IQs are sometimes lower, but not by as much as five points. In fact, I am unaware of any clinical group with proven brain lesions and specific verbal IQ deficit. If any such group is produced, the first thing to check is that there is no excess of peripheral deafness, since low verbal IQ with normal performance IQ is found in the congenitally deaf. Deafness is likely if there is any problem with cerebrospinal fluid circulation or drainage into the cranial lymphatic system (Gordon, 1976). Anderson et al. (1997) found verbal IQ 93 and performance IQ 98 in children with meningitis in infancy, and verbal IQ 99 and performance IQ 103 in those diagnosed after the age of 12 months. Sensorineural deafness was found in 6%, and many more would probably have had otitis media in infancy.

It does not seem to matter so much what is done to the brain, rather that when it is done is crucial. The earlier the lesion, and the larger it is, the lower the eventual IQ. The largest effect is probably on ‘g’ (general intelligence).

The papers reviewed above were selected for their IQ data, but it is clear that they also contain consistent and compelling evidence against the widely believed Kennard Principle (Duval et al., 2008). Whilst their overall data are in close agreement, Anderson et al’s (2009) study was not ‘the first to systematically address these age-related hypotheses’ (plasticity versus early vulnerability). Anderson et al. (2009) said ‘A review of the literature to these issues provides little clarification’ but the papers reviewed here generate a simple rule—it is just a matter of time’. First, for onset, the earlier the brain insult, the worse the outcome; for duration, the longer the early timeout period, the worse it is. Factors contributing to timeout in infancy include environmental deprivation, seizures, effortful devising of compensatory strategies, brain reprogramming time, ‘spam’ elimination and anything that increases brain load, with eventual but delayed success at cognitive tasks. Meningitis in infancy can then be construed as a temporary period of brain chaos or shutdown at a critical period (reducing ‘g’), with added language delay from auditory reduction (cochlear deafness) or inconsistency (otitis media) (Gordon, 1976).

Anderson et al. (2009) need to correct the errors in Table 5 and explain how they arose. If the correct mean figures for their total group are in fact verbal IQ 91 and performance IQ 86, full-scale IQ 88, then their results are in line with the previous literature. If not, they need to discuss and explain their quite discrepant V:P tilts. It would also help if they supplied a 7 table of group.

References


Braun CMJ, Montour-Proulx I, Daigneault S, Rouleau I, Kuehn S, Piskopos M, et al. Prevalence, and intellectual outcome of unilateral...