

# Long-term Intellectual Outcome of Traumatic Brain Injury in Children: Limits to Neuroplasticity of the Young Brain?

Trauma is the most frequent cause of acquired brain injury in young children, but few studies of long-term intellectual outcome have been published. With a high incidence of inflicted injury in young children, long-term outcome data on accidental traumatic brain injury (TBI) in young children is especially sparse. In these 2 reports from the same children's hospital, there are noteworthy differences in the intellectual outcomes. Crowe et al<sup>1</sup> followed up 53 children who had sustained TBI (20 mild, 33 moderate to severe) before age 3 years and tested 27 healthy children with similar demographic features for comparison. Two-thirds of the children who had sustained moderate to severe TBI were injured in falls. Socioeconomic level of the family at the time of injury was the strongest predictor of intellectual level at follow-up when the children were 4 to 6 years old. The nonsignificant effect of acute TBI severity and the generally average range of intellectual function are unexpected findings. However, the classification of TBI severity based on the level of consciousness is less straightforward in very young children in comparison with older children whose verbal skills are further developed. Moreover, the high proportion of falls in the moderate to severe TBI group suggests that traumatic axonal injury due to acceleration-deceleration forces may have been mild in comparison with the effects of motor vehicle crashes and pedestrian-vehicular injuries that are thought to impart higher traumatic forces of acceleration and deceleration.<sup>2</sup>

In contrast to Crowe et al's findings, Anderson et al<sup>3</sup> found significant effects of acute TBI severity on the IQ measured at 12 months, 30 months, and 10 years after injury in children who ranged in age from 2 to 7 years when they were injured. The stronger relation of acute TBI severity to performance IQ than verbal IQ later may reflect the greater emphasis on speeded performance and solution of novel problems on the Performance Scale than the Verbal Scale of the Wechsler Intelligence Scale for Children. MRI at the 10-year follow-up also disclosed that white matter volume was related to intellectual level, a finding consistent with acute traumatic axonal injury and late effects on white matter development as my colleagues and I have reported previously.<sup>4</sup> Although socioeconomic background was also a predictor of intellectual level at 10 years postinjury, Anderson et al's finding of significant effects for the acute Glasgow Coma Scale<sup>5</sup> score and white matter volume at follow-up indicates that TBI severity had a stronger effect on IQ in children who ranged between 2 and 7 years at injury. In contrast to the younger sample of patients studied by Crowe et al, the moderate to severe injuries sustained by the children followed-up by Anderson and associates involved a high proportion of motor vehicle and pedestrian-automobile-related

**AUTHOR:** Harvey S. Levin, PhD

*Department of Physical Medicine & Rehabilitation, Baylor College of Medicine, Houston, Texas*

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Address correspondence to Harvey S. Levin, PhD, Baylor College of Medicine, 1709 Dryden Rd, Suite 1200, Houston, TX 77030. E-mail: [hlevin@bcm.edu](mailto:hlevin@bcm.edu)

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injuries that putatively imparted higher levels of acceleration and deceleration to the brain.

Psychosocial adjustment at follow-up in both studies was not significantly related to acute severity of TBI. However, this negative finding could also reflect reliance on parental report with the use of standard rating forms to evaluate psychosocial function. There is emerging evidence that child-based tasks that involve social problem solving of interpersonal dilemmas and generating solutions<sup>6,7</sup> may be more informative than parental reports or at least provide incremental information.

Taken together, these studies challenge views long held by clinicians and researchers, including that young children are more resilient to the effects of TBI on intellectual development than older children because of their greater capacity for neuroplasticity. The view that young children have greater capacity for cerebral reorganization of function may find support in early, focal vascular lesions, but not in severe diffuse white matter injury. The data reported by Anderson et al also challenge the contention that children who sustain early TBI “grow into their deficit,” an extrapolation from experimental lesions in regions

of motor cortex<sup>8</sup> and prefrontal cortex<sup>9</sup> in monkeys. Instead, the trajectory of intellectual development after early moderate to severe TBI appears to reflect a consistent lag in comparison with healthy children. Persistent intellectual impairment may be more likely to arise from TBI involving high levels of acceleration-deceleration that produce severe white matter injury that is diffuse or multifocal.

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