Editorial



# Recovery from pediatric acquired brain injury: a guarded prognosis

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## Editorial

Traumatic brain injury (TBI) is the leading cause of death or disability in children in the United States (Centers for Disease Control and Prevention.[1] The incidence of long-term disability after severe TBI is high, with over 60% of children requiring educational or community based supportive services 12 months post-injury. There is increasing evidence that the young child's brain may be particularly vulnerable to early trauma owing to both physiological and developmental factors.[2,3] Research on pediatric brain injury suggests a more complex prognostic conceptualization process than occurs with adults. Specifically, while mild brain injuries are not usually associated with long-term neuropsychological deficits,[2] severe TBI's can result in a more complex recovery process than with adults and may affect subsequent cognitive development .[4] Research suggests that later developing brain structures and functions (such as executive functions) may be particularly vulnerable for anomalous development and the impairments may not be obvious until the years following the brain injury.[5,6] This potential outcome can complicate understanding the long-term implications of pediatric brain injury.

Injury to the immature brain in children may have a negative impact on cognitive skills that are either emerging or yet to reach maturity. The full development of later developing cognitive skills after an early life brain injury may be disrupted by various dynamic and persisting physiological processes.[2,3,5,6] Greater injury severity has been identified as a reliable predictor of longer term impairment in physical, cognitive and educational domains. Various other variables including a combination of social disadvantage and severe injury have also been found to be detrimental to recovery.[7] Other factors implicated in predicting outcome include younger age, or developmental level, at time of injury.[8,9] More research is greatly needed regarding long-term prognostic factors.

Systemic and cerebral hemodynamic factors such as hypotension, hypoxia, hyperglycemia, and fever are associated with poor outcome in pediatric TBI. Similarly, cerebral autoregulation of cerebral blood flow is often impaired after TBI and may adversely affect outcome, especially if systemic hemodynamics are altered. Furthermore, carbon dioxide vasoreactivity may be altered after pediatric TBI and lead to either cerebral ischemia or hyperemia.[10] Some of the relevant research shows greater early post injury autodysregulation in children under age 4 compared to older children and this was associated with worse 12 month outcomes. Developmental changes in excitatory neurotransmission may render the young brain more susceptible to excitotoxic injury[12] and altered development. Enlarged ventricles and brain atrophy have been reported after pediatric TBI.[13,14] Other findings include decreased growth of the corpus callosum in the years following severe pediatric TBI[14] and other areas of brain volume loss.

## Conclusions

In the context of moderate to severe TBI, there is evidence that subsequent cognitive development can be disrupted. Factors that may contribute to this situation include potential changes in cerebral hemodynamics, autodysregulation, excitatory neurotransmission, and potential volumetric brain changes. Given the complexities of an accurate long-term prognosis in the context of pediatric TBI, it may be important for clinicians to recommend appropriate comprehensive follow up examination to monitor recovery and subsequent cognitive development.

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## Conflicts of Interset

None.

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