

Brain Injury in Perinatal Period: Neuroprotective Therapy

Melanie Badawi*

Department of Neurology and Neurosurgery, Montreal Children's Hospital-McGill University Health Center, Montreal, Canada

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Introduction

The most common type of brain injury in the perinatal period is hypoxic-ischemic disease-related brain injury. The focus of this article is on the most recent research developments in this field, particularly those that are expected to have the most profound impact on interventions in the first years of the new millennium. In the term infant, neuronal injury is the most common type of cellular injury. The main mechanisms leading to neuronal death after hypoxia-ischemia/reperfusion are energy depletion, extracellular glutamate accumulation, and glutamate receptor activation. The subsequent chain of events involves the accumulation of cytosolic calcium and the activation of a number of calcium-mediated deleterious events [1-3].

Notably, even if interventions are initiated after the insult has been terminated, this deleterious cascade, which evolves over many hours, may be interrupted; this is an important clinical point. Mild hypothermia, inhibitors of free radical production, and free radical scavengers are the leading candidates for application to the human infant in the relatively short term. There is promising clinical data for the use of mild hypothermia.

Perinatal brain injury in term infants is common in both developed and developing nations. Seizures and decreased reactivity are the most common clinical findings in neonatal encephalopathy, which results from almost all types of perinatal brain injury. Cortical blindness occurs in adults when the primary visual cortex and the geniculostriate pathway are injured, resulting in the loss of conscious visual perception [4].

Perinatal/neonatal stroke can also cause encephalopathy. Dehydration is added to the risk factors for neonatal stroke for cerebral venous sinus thrombosis, which include maternal risk factors such as infertility, primiparity, maternal fever, meconium-stained amniotic fluid, chorioamnionitis, pre-eclampsia, and intrauterine growth retardation. In infants with neonatal stroke, complicated deliveries, both instrumental and emergency caesarean sections, low Apgar scores, and hypoglycemia are more common [5]. Prothrombotic factors are becoming more common. Congenital heart disease also increases the risk of neonatal stroke.

Seizures are usual during neonatal encephalopathy, but they can also be caused by other conditions. They are frequently seen following neonatal stroke and may be the only clinical manifestation of this condition. The incidence of seizures in the newborn period can only be accurately estimated with EEG

Corresponding author:

Melanie Badawi

✉ melanie.badawi@mghc.ca

Department of Neurology and Neurosurgery, Montreal Children's Hospital-McGill University Health Center, Montreal, Canada

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monitoring, but the aetiology of seizures in term infants has recently been studied using extensive imaging, metabolic, and genetic examinations.

The long-term goal of neuroprotective therapy is to improve neurodevelopmental outcomes by preventing injury progression, salvaging and protecting cells that would otherwise be injured or die, repairing injured cells, and enhancing neurogenesis. Much has been learned from animal and adult studies on brain injury, including potential treatment strategies and the potential for translation into clinical research and trials. The success of moderate hypothermia in term hypoxic-ischaemic encephalopathy has shown that neural rescue by intervention after the hypoxic-ischaemic insult is possible.

Perinatal brain damage in the term infant is still a significant clinical issue. The adverse outcome rate after severe term perinatal asphyxia remains at 45 percent. Despite the fact that many aspects of the pathophysiology of hypoxic-ischaemic brain injury have been studied, therapeutic hypothermia remains the only neuroprotective strategy used in standard care [6]. Neonatal encephalopathy, arterial ischaemic stroke, prematurity, and systemic infections are all causes of perinatal brain injuries. Inflammation can either prime the brain for injury or protect it, depending on the timing and context.

Inflammation during preterm labour and intensive care for premature infants affects the very immature brain during critical stages of brain development, with serious implications for myelination and cortical development. Understanding the

role of inflammation in perinatal brain injury can help identify new prevention and treatment strategies that could reduce neurological and neuropsychiatric morbidities in maturing infants.

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