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Opinion Article

Recent Concepts about Hypothermia as a Treatment for Neonatal Hypoxic Ischemic Encephalopathy

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DESCRIPTION

Hypoxic-Ischemic Encephalopathy (HIE) in perinatal patients is still a leading cause of morbidity and mortality. Only moderate hypothermia (33.5 °C) has been approved as a regular treatment thus far. However, this therapy is ineffective for a sizable number of newborns. This sparked an international hunt for neuroprotectants to enhance the effects of mild hypothermia. Here, they look at Erythropoietin (EPO) as a leading contender. Neonatal animal studies demonstrate that both immediate and delayed EPO post-injury therapy can be neuroprotective and/or neurorestorative. However, the reported EPO therapy gains were typically not at the level of the control, unharmed animals. This implied that combining EPO therapy with an adjunctive therapeutic approach needed more study. In a non-human monkey model of prenatal hypoxia, treatment with EPO combined hypothermia reduced cerebral palsy, which prompted clinical trials. A recent Phase II clinical trial on neonatal children with HIE found that EPO with hypothermia resulted in superior 12-month motor results compared to hypothermia alone. As a result, the efficacy of combined treatment for neonatal HIE that includes moderate hypothermia and EPO now appears promising. It is now necessary to know the results of two ongoing clinical trials on neurological outcomes at ages 18 to 24 months and older. Additional study is needed to determine the ideal EPO dosage, timing of its administration, and length of treatment. Gender and the severity of the injury must also be carefully considered.

Because of the juvenile brain's greater resistance to damage from Hypoxia-Ischemia (HI) events, lower cerebral metabolic rate, immature Central Nervous System (CNS) plasticity, and immaturity in the formation of balance in the functional neurotransmitter, neonatal HIE clinical signs are sneaky. As a result, neonates with HIE won't be detected during the initial stages of HI, making them more vulnerable to secondary harm that happens 6 to 72 hours after the initial shocks. Similar to clinical examinations, these infants' disease condition and appearance can make them occasionally ambiguous or inconclusive. The American Academy of Pediatrics and the American College of Obstetrics and Gynecology have established diagnostic criteria for initial evaluation and suitable therapy approaches in neonates with HIE. The most frequently used diagnostic methods for identifying brain injury in newborns are routine serum biomarkers, Magnetic Resonance Imaging (MRI), and Electroencephalograms (EEG), which aid in prompt intervention, evaluation of treatment outcomes, and prognostication.

Several potential neuroprotective techniques have been studied in various animal models of neonatal HIE, including hypothermia, erythropoietin, magnesium, allopurinol, xenon, melatonin, growth factors, barbiturates, statins, and stem cells. These techniques target various pathways that result in neuronal cell death in response to hypoxic-ischemic insult. While others report ineffectiveness and/or negative effects, Therapeutic Hypothermia (TH) is the most frequently used standard treatment for neonatal HIE. It works by inhibiting inflammatory cascades, reducing production of reactive oxygen species, inhibiting apoptosis, having an endogenous neuroprotective effect, and reducing concentrations of free radicals and neurotransmitters like glutamate, glutamine, GABA, and aspartate. For instance, observed that unfavourable effects, such as cognitive impairment, occurred in around 50% of newborns treated with TH. According to a Randomised Control Experiment (RCT), cooling for 120 hours or to 32.0 °C, or both, may be harmful. When chilling was prolonged from 72 to 120 h, TH did not help EEG recovery and actually made neuronal survival worse. As a result, researchers are looking into additional neuroprotective substances that show potential either alone or in combination with therapeutic hypothermia.

Hypothermia is a safe technique that is successful in lowering death and moderate to severe neurodevelopmental dysfunction in neonates with postintrapartum asphyxial hypoxic-ischemic encephalopathy. Of the 4 million neonatal deaths that occur worldwide each year, 23% are caused by asphyxia. One to two cases of postintrapartum asphyxial HIE occur in per 1000 live

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births in affluent nations. The processes behind cellular harm during suffocation have been established by pathologic and imaging studies over the past 20 years. The focus of preventive measures is on the subsequent neuronal damage that results from the initial insult. Studies on animals, neonates, and humans have indicated that hypothermia, both systemic (whole body chilling) and selective (head cooling), has therapeutic potential. Reduced neuronal metabolic requirement, decreased cytotoxin buildup, and avoidance of apoptosis after secondary energy failure are some of the hypothermia's potential modes of action. There is insufficient evidence to justify the use of hypothermia, which is used to treat neonatal HIE, according to a systematic evaluation of the two studies described in four publications. Numerous RCTs have been carried out since then.

Higgins revealed gaps in our understanding of this intervention after reviewing just two sizable multicenter trials. Experimental investigations, pilot (feasibility and safety) studies, and multicenter studies were examined by Speer and Perlman. A number of researches, including RCTs, were not safety-evaluated, and their analysis included both randomised and nonrandomized studies but eliminated those that used non-English literature. Neither safety nor efficacy was described using meta-analytic methods. Our main goals were to compare the safety of hypothermia to normothermia in neonates with postintrapartum asphyxial HIE in order to discover which was more successful in preventing moderate to severe impairment in infancy and childhood. Secondary goals included subgroup analyses based on the degree of encephalopathy (mild, moderate, or severe), the method of head cooling (systemic or selective), and the degree of hypothermia (mild, 33.6°C, or moderate, 32.0-33.5°C).