# Neonatal Encephalopathy

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Neonatal encephalopathy (NE) is a heterogeneous syndrome characterized by signs of central nervous system dysfunction in newborn infants. It can result from a wide variety of conditions but often remains unexplained. Approximately 70% of NE cases are associated with events arising before the onset of labor. On the other hand Hypoxic-ischemic encephalopathy (HIE) is one of the many possible contributors to NE. The term is appropriately used when NE due to hypoxic- ischemic brain injury. Guidelines from the American Academy of Pediatrics and the American College of Obstetrics and Gynecology for HIE indicate that all of the following must be present for the designation of perinatal asphyxia or HIE: 1- profound metabolic or mixed acidemia (pH <7) in an umbilical artery blood sample, 2- persistence of an Apgar score of 0 to 3 for more than 5 minutes, 3- neonatal neurologic abnormalities and 4- multiple organ involvement.

The asphyxia insult is due to impaired cerebral blood flow, as a consequence of interrupted maternal and/or fetal placental blood flow and gas exchange. The most important effects appear to include apoptosis and inflammation, which occur in the subacute phase after injury (hours to days after a hypoxic-ischemic event) ***Figure***.

There should be a comprehensive evaluation including assessment of neonatal clinical status, all potentially contributing factors and radiological studies. Therapeutic hypothermia is the only treatment currently shown to reduce death and/or disability after a hypoxic-ischemic insult in newborn infants with moderate to severe encephalopathy in the first 6 hours after birth. This intervention needs to be implemented according to the established published protocols and guidelines. Newborns with mild encephalopathy usually develop normally, while infants with moderate to severe encephalopathy are more likely to develop long-term neurologic sequela and morbidity.

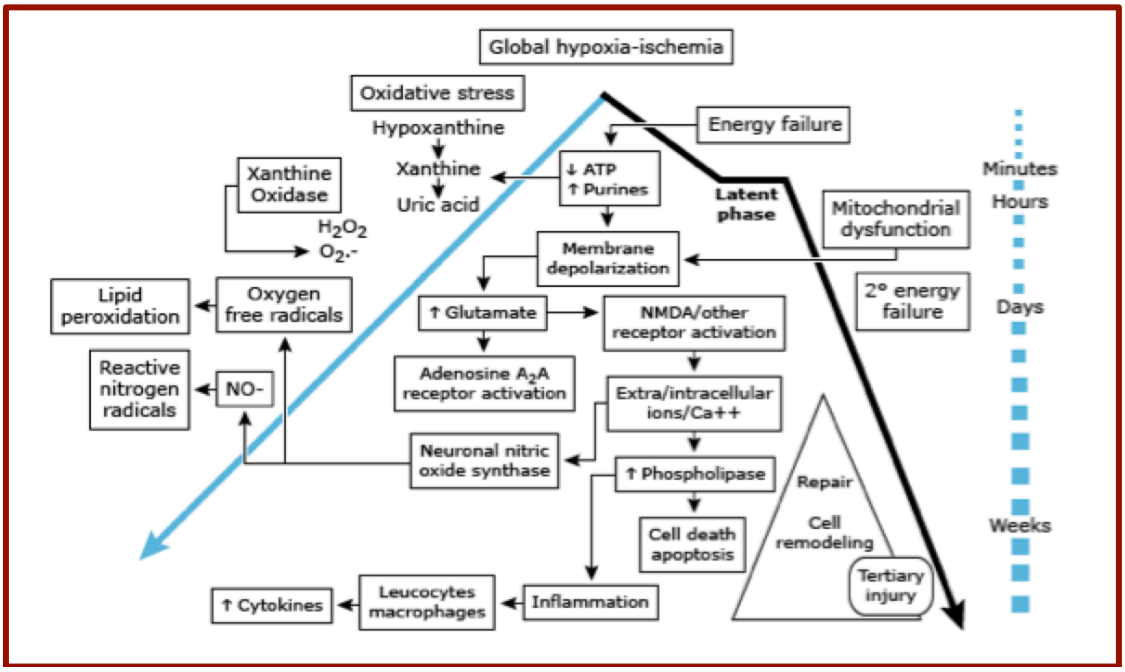



Figure: Pathogenesis of HIE. *The downward pointing blue line represents the cascade of events that occurs with oxidative stress, and the downward pointing black line depict events associated with energy failure.*

**Recent publication and researches:**


1. Sarnat HB, Sarnat MS. Neonatal encephalopathy following fetal distress. A clinical and electroencephalographic study. Arch Neurol 1976; 33:696.
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5. Higgins RD, Raju T, Edwards AD, et al. Hypothermia and other treatment options for neonatal encephalopathy: an executive summary of the Eunice Kennedy Shriver NICHD workshop. J Pediatr. 2011.;159(5):851–858
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**Biography:**



Amal Zubani is a consultant neonatologist at King Faisal Specialist Hospital and Research center- Jeddah, which is a tertiary care Hospital located in Jeddah on the West Coast of the Kingdom of Saudi Arabia. She is an advisor and active member in different committees in the ministry of heath in Saudi Arabia.

She graduated from King AbdulAziz University in Jeddah in 2000 then she joined university of Manitoba, Winnipeg, Canada as resident then fellow. She has her Canadian board in pediatric and Perinatal- Neonatal medicine in 2005 and 2007.

Her major interest and research subjects are nutrition in preterm infant and their neurodevelopmental outcome. She has several publications and presentations nationally and internationally.

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