Abstract

Hypoxic-Ischemic Encephalopathy (HIE) is a birth brain injury that precedes the development of Cerebral Palsy (CP). Visualized on MRI scanning, the lesions of HIE are infarcts resulting from deficient generalized perfusion of brain tissue. Birth asphyxia/hypoxia is the widely assumed cause of this brain injury, however, HIE may occur without significant hypoxia. Hypovolemia/hypovolemic shock, is evident in the great majority of HIE newborns. Cord compression prior to birth pools fetal blood in the placenta and immediate cord clamping finalizes the hypovolemia. Heart failure (hypovolemic shock) follows.

Retraction respiration—pulses of negative intra-thoracic pressure—pulls venous blood into the heart and, with hypotension, pulls arterial blood into the thoracic aorta from peripheral arteries. Circulation in the heart and lungs is maintained at the expense of perfusion of all peripheral organs, including the brain. Prolonged deficient perfusion of the actively metabolizing areas of the brain (basal ganglia and cerebral cortex) results in infarction.

Thus the primary pathology in HIE/CP cases is massive blood loss into the placenta, not hypoxia. HIE/CP can be avoided by not clamping the cord and resuscitating all babies with the placental circulation intact.

Keywords: birth brain injury, hypoxic-ischemic encephalopathy, cerebral palsy