The Pathogenesis of Cerebral Injury in the Newborn

Phillip B James Wolfson Hyperbaric Medicine Unit
The University of Dundee, Scotland, UK.

Birth involves the transfer of the function of oxygenation, the most critical factor in life support from the placental circulation to breathing. This transfer of oxygenation to the lungs of the neonate requires major changes to the neonatal circulation. During labour, placental blood flow is inevitably compromised by uterine contractions, and if oxygenation falls below a critical level, then brain damage may occur at this time. Prystowsky has suggested that mothers should breathe 100% oxygen during labour as a preventative measure, because of the increased gradient for the delivery of oxygen to tissue. Premature placental separation may also severely compromise fetal oxygenation and delivery may also be associated with fetal brain tissue. In the last few weeks of intrauterine life the cerebral circulation of the fetus matures in preparation for birth. The changes are most prominent in the mid-brain with increased vascularisation and maturation of the blood-brain barrier. If delivery is premature the neonate is at special risk of damage because these critical changes have not yet taken place. Ultasonic imaging through the anterior fontanelle allowed damage to the mid0brain to be detected. Global oxygen deprivation may result in brain atrophy leading to microephaly, but lesser degrees of hypoxia cause cerebral edema, particularly in the midbrain where the nutrition of some the tissue is dependant on the venous drainage. The increased tissue water content reduces oxygen transport and if the edema is not resolved the basal ganlia and the critical areas of the midbrain can undergo cystic degeneration. The blood-brain barrier failure may also be associated with hemorrhage. Damage to the internal capsules in cerebral medulla leads often involves the internal capsules severing the pyramidal tracts. However, in many cases the tracts continue to function for some time but myelination is prevented leaving the axis cylinders exposed. This is associated with a delay in the development of spasticity which may be as long as two years. The additional gradient for the transport of oxygen that is provided by breathing oxygen under hyperbaric conditions may be able to prevent brain damage in many children as Magnetic Resonance Spectroscopy can detect hypoxia inferred from the presence of lactate it is possible to screen infants at birth to identify those at risk. Even after a delay, the use of hyperbaric oxygen therapy may be beneficial by resolving the edema. This may allow the completion of myelination and prevent the development of spasticity.