FETAL BRAIN INJURY

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Fetal brain injury may be of various forms although cerebral palsy is always high lighted with obstetric care. Cerebral palsy (CP) is a persistent disorder of movement and posture caused by non-progressive defects or lesions of the immature brain. Closer scrutiny enables us to identify several causes of brain injury in fetus and infants. They are: a) Malformation – genetic/chromosomal/anatomical; b) Toxic; c) Metabolic; d) Trauma; e) Infection; f) Hypoxia-ischaemia; g) Coagulation disorders; h) Maternal thyroid dysfunction; i) Inflammatory conditions /pyrexia in labour.

Birth defects in children with newborn encephalopathy are more common. In one study 276 term infants with moderate or severe encephalopathy and 564 controls were studied. Birth defects in the former group were 27.5% vs 4.3% in the controls (Felix JF et al Dev Med Child Neurol 2000; 42:803-8). In another series 29% had birth defects in those with moderate to severe CP compare with 4.9% in normal population (Palmer Antenatal antecedents Paed & Perinat Epidemiol 1993;16:6:298. However 90% of birth defects were not specific anomalies of the CNS. It is well established that birth defect is associated with increased risk of having severe encephalopathy, increased risk of death and of CP.

Maternal infection by TORCH organisms and neurological injury are well known especially with rubella and cytomegalovirus. The damage is more severe if infection is in 1'st and early 2'nd trimester. It is now clear that chorioamnionitis, and infection with mycoplasma/bacterial vaginosis associated with preterm labour, birth weight - <2500g and temp>38*C is associated with 4.2 - 12 fold risk of CP and 19 fold risk of Quadri Plegia. Raised cytokines, IL-1,IL-6, IL-8, TNF and coagulopathy in the cerebral circulation at micro vascular level is incriminated for such insults (Grether. JAMA 1997; 278:207; Nelson. Neurol 1998; 44:665; Murphy. Lancet 1995 346:1449; Wheater. Developmental medicine & Child Neurology 2000; 42:364). Infection not only damages the brain directly but also sensitises the brain to hypoxic insult. Experimental data from the rat combining ischemia from carotid artery occlusion and lipo-polysaccharide infusion (Bacterial endotoxin) is shown to sensitize the immature brain to hypoxic ischemic injury (Eklind S et al Eur J Neurosci. 2001; 13:1101-6)

Intrapartum maternal fever and even without infection is associated with neonatal encephalopathy and neurological damage. Maternal pyrexia (odds ratio 3.82),

A persistent occipitoposterior position (4.29), and an acute intrapartum event (4.44)

were all found to be risk factors for newborn encephalopathy (N. Badawi, et.al. Intrapartum risk factors for newborn encephalopathy: the Western Australian case-control study. BMJ1998; 317 (7172):1554-8). In another study of 4915 low risk term labours fever was associated with epidural use but controlling for epidural use, odds ratios associated with fever were: Encephalopathy 4.7 (1.3 - 17.4); metabolic acidosis 2.91 (1.14 - 7.39); Admission to SCBU 1.78 (1.1 - 2.89) (Impey L et.al.BJOG 2001;108;594-597). These studies suggest; that pyrexia in labour has a strong association with neonatal encephalopathy and cerebral palsy; Most pyrexia are not infective in origin; Neonatal brain cooling can reduce damage in hypoxic ischemic encephalopathy; Preventing temperature rise is known to limit hypoxic ischemic damage in animals and stroke victims; Trials of temperature control in labour in relation to both short term and long term outcome are urgently needed. There is also evidence that suggest that the rise in temperature associated with regional analgesia is labour is harmful and should be treated (Banerjee S & Steer PJ. International Journal of Obstetric Anaesthesia 2003; 12; 280-286).

Asphyxia both ante partum and intrapartum is a recognised cause. It may be a generalised hypoxia and asphyxial injury of the brain. This could be slow taking place over 30 minutes or more or may take place acutely within 30 minutes as in the case of abruption, cord prolapse, scar rupture or prolonged bradycardia. Localised asphyxial injury of the brain (fetal stroke) can take place due to blockage of a cerebral vessel or intracranial haemorrhage. Intrapartum cardiotocograph, neuroimaging of the infant and the extent of neurological damage provides some clue as to the type of injury.

Potential treatment for asphyxial damage is being explored. They are: prevention of energy depletion by strict pharmaceutical prevention of seizures; Hypothermia of the neonatal head to prevent energy utilisation; Stabilisation of the cell membrane there by minimising the need for sodium/potassium pump and depressing cerebral glutamate release through sodium channel blockage; inhibition of glutamate release through administration of adenosine or Calcium channel blockade.

Several mechanisms of neurological brain injury are identified and prevention and treatment strategies are being explored with the realisation that in many cases little could be done. It is important for the physicians from various disciplines to work as a team and to take all steps to prevent these incidents where possible and to offer parents the explanation and prognosis of their child.