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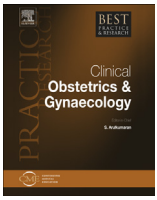

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Neurological damage arising from intrapartum hypoxia/acidosis

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Complications occurring at any level of foetal oxygen supply will result in hypoxaemia, and this may ultimately lead to hypoxia/acidosis and neurological damage. Hypoxic–ischaemic encephalopathy (HIE) is the short-term neurological dysfunction caused by intrapartum hypoxia/acidosis, and this diagnosis requires the presence of a number of findings, including the confirmation of newborn metabolic acidosis, low Apgar scores, early imaging evidence of cerebral oedema and the appearance of clinical signs of neurological dysfunction in the first 48 h of life. Cerebral palsy (CP) consists of a heterogeneous group of nonprogressive movement and posture disorders, frequently accompanied by cognitive and sensory impairments, epilepsy, nutritional deficiencies and secondary musculoskeletal lesions. Although CP is the most common long-term neurological complication associated with intrapartum hypoxia/acidosis, >80% of cases are caused by other phenomena. Data on minor long-term neurological deficits are scarce, but they suggest that less serious intellectual and motor impairments may result from intrapartum hypoxia/acidosis. This chapter focuses on the existing evidence of neurological damage associated with poor foetal oxygenation during labour.

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Intrapartum events leading to hypoxia/acidosis

The foetus is totally dependent on the oxygen supply provided by the mother, and any interruption in the flow will result in transient or permanent foetal hypoxaemia. Rhythmic uterine contractions during labour put a particular stress on foetal oxygenation, as they cause reduced blood flow in vessels running inside the myometrium, and they may also compress the umbilical cord. Other intrapartum complications compromising foetal oxygenation include those of maternal origin (sudden maternal hypotension, cardiorespiratory arrest and respiratory or circulatory disorders), mechanical complications of delivery (umbilical cord prolapse, shoulder dystocia and retention of the aftercoming head), major placental abruption, uterine rupture and foetal haemorrhage (ruptured vasa previa or foetal–maternal haemorrhage). The intensity and repetitive nature of foetal hypoxaemia will determine the severity and duration of the resulting hypoxia/acidosis.

Metabolic acidosis is defined as the measurement in umbilical blood, or in the newborn circulation during the first minutes of life, of a pH value below 7.00 and a base deficit (BD) exceeding 12 mmol/l, but there is already an association with adverse short-term outcome when pH values are below 7.05 and BD in the extracellular fluid (BD_{ecf}) is above 10 mmol/l [1]. Depending on the intensity, duration and repetitive nature of the hypoxic insult, as well as on the individual capacity to cope with the situation, the newborn may recover promptly or may suffer irreparable cell damage. Four distinct patterns of foetal hypoxia have been described: slowly evolving hypoxia, subacute hypoxia, acute hypoxia and pre-existing hypoxia [2]. In a few cases, the hypoxic insult will result in death or in long-term neurological morbidity. Coexisting foetal inflammation may have a synergistic interaction with hypoxia, lowering the threshold for neuronal apoptosis and brain damage.

Hypoxic–ischaemic encephalopathy

Neonatal encephalopathy is reported to occur in 3.0 per 1000 live term births [3], and it is a major predictor of later neurodevelopmental disability, leading to death in the newborn period in 15–20% of affected infants and permanent neurologic deficits in 25%. Although there is still no universally accepted definition for this entity, it is a clinical syndrome of near-term neonates manifested by a subnormal level of consciousness, seizures, respiratory difficulties and/or a depression of tone and reflexes [4].

An estimated 70% of cases of neonatal encephalopathy are thought to be consequent to events arising before the onset of labour [5], and <10% to postnatal complications such as severe respiratory distress, sepsis and shock [6]. Most of the remaining cases are due to intrapartum hypoxia/acidosis, but their incidence varies according to health-care settings. Hypoxia/acidosis can also occur before labour and in the postnatal period, and they cause neonatal encephalopathy, but these cases will not display metabolic acidosis at the time of birth.

Hypoxic–ischaemic encephalopathy (HIE) is the short-term neurological dysfunction caused by intrapartum hypoxia/acidosis, and it has an estimated incidence of 1.5 per 1000 live births [3]. As there are multiple non-hypoxic causes of neonatal encephalopathy, this diagnosis requires the confirmation of metabolic acidosis in the umbilical cord blood or in the newborn circulation during the first minutes of life [7], together with low Apgar scores at 5 and 10 min, and early imaging evidence of cerebral oedema [4]. Multisystem organ failure may coexist, involving the renal, hepatic, haematologic, cardiac, metabolic and gastrointestinal systems, but the severity of neurological injury does not necessarily correlate with this.

HIE occurs in the first 48 h of life, and it can be classified as mild, moderate or severe, according to the criteria described by Sarnat and Sarnat (grades 1, 2 and 3, respectively). Grade 1 is characterised by hyperalertness, irritability, jitteriness and a normal electroencephalogram. Grade 2 is characterised by obtundation, hypotonia, strong distal flexion and occasional multifocal seizures, and it has a 20–30% risk of death or major neurological sequelae such as cerebral palsy (CP). Grade 3 is associated with coma, and in the majority of cases neonatal death or long-term neurological sequelae such as CP occur [8]. Permanent neurological damage is rare in cases with mild HIE, whereas moderate

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