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Pathophysiology of foetal oxygenation and cell damage during labour

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A foetus exposed to oxygenation compromise is capable of several adaptive responses, which can be categorised into those affecting metabolism and those affecting oxygen transport. However, both the extent and duration of the impairment in oxygenation will have a bearing on these adaptive responses. Although intrapartum events may account for no more than one-third of cases with an adverse neurological outcome, they are important because they can be influenced successfully. This review describes the mechanisms underlying foetal hypoxia during labour, acid–base balance and gas exchange, and the current scientific understanding of the role of intrauterine asphyxia in the pathophysiology of neonatal encephalopathy and cerebral palsy. Although the mechanisms involved include similar initiating events, principally ischaemia and excitotoxicity, and similar final common pathways to cell death, there are certain unique maturational factors that influence the type and pattern of cellular injury.

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All human cells require oxygen and glucose to maintain aerobic metabolism and for energy production. The foetal oxygen requirement is determined by foetal size, activity and essential metabolic processes. During foetal life, the oxygen supply is dependent entirely on maternal respiration and circulation, placental perfusion, gas exchange across the placenta and the umbilical and foetal circulations. If the supply and requirement are in balance, the foetus has adequate oxygen to metabolise glucose aerobically to produce the energy required for organ function.

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During the human birth process, the foetus is squeezed through the birth canal over a period of up to several hours, during which the head sustains considerable pressure and the foetus is intermittently deprived of oxygen. A reduced oxygen supply is often categorised into three types: (1) hypoxaemia, involving a reduced oxygen in the arterial blood, but where cell and organ function are not usually affected; (2) hypoxia, with reduced oxygen and subsequent anaerobic metabolism, mainly in the peripheral tissues; and (3) asphyxia, where the hypoxia extends to the central organs – the heart, brain and adrenal glands – and potentially leads to metabolic acidosis.

The main direct causes of neonatal death worldwide are estimated to be preterm birth, severe infections and asphyxia [1,2]. Asphyxia is one of the main causes of neonatal and childhood mortality and morbidity, and it has been associated with neonatal death [3], hypoxic–ischaemic encephalopathy (HIE), seizures [4], intraventricular haemorrhage, cerebral palsy (CP) and delayed development [5].

The foetal blood supply

The placenta acts as the foetus's lungs, kidneys and gastrointestinal tract, allowing gas exchange, waste elimination and nutrient uptake via the mother's blood supply. In the intervillous space, oxygenated blood from the mother's spiral arteries flows around the foetal chorionic villi, which contain deoxygenated blood. The two umbilical arteries transport deoxygenated blood and waste products from the foetus to the placenta, whereas the umbilical vein provides the foetus with oxygenated blood and nutrients from the mother. The neonatal acid–base status is therefore best reflected by the umbilical arterial blood, whereas the venous umbilical blood contents depend on the maternal acid–base status and placental function.

The foetal cardiovascular system is designed such that the most highly oxygenated blood is delivered to the myocardium and brain. These circulatory adaptations are achieved in the foetus by both the preferential streaming of oxygenated blood and the presence of intracardiac and extracardiac shunts. Thus, the foetal circulation can be defined as a 'shunt-dependent' circulation.

Blood from the placenta passes via the umbilical vein almost unhindered through the ductus venosus across the right atrium, and through the foramen ovale to the left atrium and then to the left ventricle, which pumps into the aortic arch and neck and head vessels. The heart is equipped with baroreceptor and volume receptor, which sense changes in the pressure and volume of blood in the heart. The aortic arch and carotid bodies, which contain chemoreceptors, are well positioned to sense any alteration in the oxygen content of the blood coming from the placenta via the umbilical vein. This enables the foetus to produce a cardiovascular response to hypoxia when necessary [6].

Foetal oxygenation in labour

Labour increases the risk of compromised oxygenation in the foetus. Uterine contractions produce transient decreases in the blood flow to the placenta, which can lead to interruptions in gas exchange across the placental barrier. Certain degrees of hypoxaemia and acidaemia are normal in healthy foetuses during a normal labour, and so one of the challenges is to successfully identify foetuses experiencing levels of hypoxaemia and acidaemia that are sufficient to cause physiological damage. Table 1 lists acid–base values in the healthy, term foetus before and healthy neonates after birth, along with the adult values for comparison.

However, if the oxygen deficit is prolonged, foetal metabolism switches to anaerobic. This leads to a decrease in pH, accumulation of lactate and an increase in the base deficit (BD). The concentrations of free hydrogen ions (H^+; $[H^+]$) are expressed as pH values, and they are calculated as the negative logarithm of $[H^+]$; thus, pH decreases as $[H^+]$ increases.

According to the international consensus, metabolic acidosis in the umbilical cord arterial blood at birth is defined as umbilical cord pH <7.00 and BD $\geq$12.0 mmol/l [10]. The aim of calculating BD is to have a measure of the metabolic component of an acidosis. This measure should be expressed in such a way that it is independent of the respiratory component. An acute increase in the partial pressure of carbon dioxide (CO_2; pCO_2) in vivo causes an increase in the BD in the blood (BDblood) and a reduction in the BD in the plasma (BDplasma), whereas the BD in the extracellular fluid (BDecf) remains constant. The cause of these changes is a redistribution of H^+ within the extracellular volume and erythrocytes.

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