IN THE NAME OF GOD

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Asphyxia can occur before, during, or after birth

Antepartum events: maternal hypotension or trauma (4–20%)

Intrapartum events (placental abruption or umbilical cord prolapse (56-80%)

Evidence of intrapartum disturbance (eg, meconium-stained AF or severe FHR abnormalities) occurs in 10-35%, usually in association with an antenatal risk factor, such as diabetes mellitus, preeclampsia, or IUGR.

postnatal insult occurs (10% of cases), usually caused by severe cardiopulmonary abnormalities or associated with prematurity

RISK FACTORS

Maternal, obstetrics & neonatal

Antepartum conditions

- Abnormal maternal oxygenation (severe anemia, cardiopulmonary disease)
- Inadequate placental perfusion and/or gas exchange (maternal hypertension or severe hypotension, placental insufficiency caused by vascular disease)
- Congenital infection or anomalies

Intrapartum events

- Interruption of umbilical circulation (true knot, cord prolapse)

- Inadequate placental perfusion and/or gas exchange (placental abruption, uterine rupture, severe maternal hypotension, abnormal uterine contractions)

- Traumatic delivery (shoulder dystocia, difficult breech extraction)

- Abnormal maternal oxygenation (pulmonary edema)

Postnatal disorders

- Persistent pulmonary hypertension of the newborn (PPHN) (Persistent pulmonary hypertension of the newborn)
- Severe circulatory insufficiency (acute blood loss, septic shock)
- Congenital heart disease

The process of normal labor and vaginal delivery stresses the fetus such that there is the development of mild acidosis in nearly all labors

Human fetal arterial pH and pO2 are lower, and pCO2 is higher than maternal values.
Among non complicated pregnancies between 18 and 38 weeks, mean BE and pH were –2.3±0.6mmol/L and 7.39±0.05.
The normal fetus may be expected to enter labor with an umbilical artery BE of approximately –2mmol/L.

Assuming the normal fetus enters labor with a BE of –2mmol/L, an uncomplicated labor reduces BE by an additional –3mmol/L.

- BE does not change significantly during the latent phase of labor,
- whereas the stress of normal (active phase) labor may decrease fetal BE by –1mmol/L per 3 hours (assuming a 6h active phase).
- In the second stage, BE decreases by approximately –1mmol/L per hour in the normal fetus.

A protracted first stage of labor has been associated with increased risk of administration to the NICU & 5th min Apgar score but no increased risk of serious morbidity or mortality

A prolong 2th stage has been associated with increase in serious neonatal morbidity & mortality.

In one study the rate of birth asphyxia related complications progressively increase with duration of 2th stage.

- Among fetuses ‘‘exhibiting repetitive FHR decelerations for periods of hours,’’ BE decreased by approximately 4 mmol/L during the 2 hours before delivery (1mmol/L per 30min), whereas fetuses.
- With‘‘intrapartum hypoxemia resulting in asphyxia’’ showed a decrease in buffer base of 8 to 11mmol/L during the last 60 minutes of labor (1mmol/L per 6min).
- Among fetuses with terminal asphyxia, buffer base decreased an average of 7mmol/L in the 15 minutes before delivery (1mmol/L per 2min)

A single minute of umbilical cord occlusion results in a BE change of approximately 0.5mmol/L, regardless of the frequency of UCO.

Thus, a fetal bradycardia resulting from complete UCO results in a BE change of approximately 1mmol/L every 2 minutes.

BE normalizes at approximately 0.1mmol/L per min, between UCO or after a bradycardic event.

Thus, it would take approximately 2 hours for BE to normalize in a fetus with a BE of –12mmol/L, who remains undelivered and in which the insult was completely abated;

this recovery would be longer depending on the degree of continuing fetal hypoxic stress.

Importantly, these findings indicate that the fetus can potentially compensate if cord occlusion is sufficiently spaced (perhaps by maternal pushing on alternate contractions

With Thanks