Timing the origins: acute vs chronic hypoxia

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A recent working group consensus publication has proposed a template to help define the timing of hypoxic cerebral palsy in the term infant. This was greeted with mixed views by many that felt its proposals were potentially restrictive. It suggested criteria that could be used to define an acute intrapartum hypoxic event. It also put forward a list of factors that might be used to suggest that a case of cerebral palsy was due to a cause other than acute intrapartum hypoxia.

Criteria considered essential to define acute intrapartum hypoxia:

1. A fetal or early neonatal blood measurement indicating metabolic acidaemia (pH<7.00 or base deficit > 12mmol/l).
2. Early onset of moderate or severe encephalopathy.
3. Spastic quadraplegic or dyskinetic cerebral palsy outcome.

Less specific criteria

4. A sentinel hypoxic event.
5. Subsequent rapid deterioration in fetal heart rate.
6. Apgar scores below 6 at 5 minutes.
7. Early evidence of multisystem failure
8. Early imaging for evident of cerebral abnomality'

The paper suggested that all three 'essential' criteria should be present in order to make a diagnosis of intrapartum hypoxia. The other signs were considered insufficiently specific to substitute for the confirmation of severe acidaemia.

The presence of the first three factors then allows the timing of intrapartum hypoxia to be considered. The presence of all remaining five factors would strongly suggest an acute event. Increasingly, absence of these factors would raise the possibility of doubt. Individually, other than criterion 4, the remaining factors are only weakly associated with an acute intrapartum damaging hypoxic event. Contrary evidence, such as a normal Apgar score at 5 minutes, would further raise a question about a serious acute event.