

Electrocortical activity during repetitive umbilical cord occlusions with worsening acidemia in the ovine fetus near term

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Objective: In the ovine fetus electrocortical (ECoG) activity as a measure of the brain's functional activity shows behavioural state alterations and a flattening in amplitude with induced hypoxia of a moderate to severe degree reflecting an 'adaptive brain shutdown' as a protective mechanism occurring in advance of asphyxia-mediated brain injury. However, there is limited study of ECoG activity in the ovine fetus with repetitive umbilical cord occlusions (UCOs) leading to concerning acidemia as might be seen during human labour. The aim of this study was to induce repetitive UCOs in fetal sheep leading to worsening acidemia to determine the related change in ECoG behavioural state activity and amplitude/frequency characteristics and thereby their predictive value for fetal compromise with asphyxial brain injury.

Methods: Near-term fetal sheep (n=10) underwent chronic preparation with arterial catheters, electrocardiogram (ECG) and ECoG electrodes, and placement of an inflatable umbilical cord occluder. Following a baseline recording period, fetuses underwent a series of mild UCOs (1 min every 5 min for 1 h), moderate UCOs (1 min every 3 min for 1 h), and severe UCOs (1 min every 2 min continuing), until arterial pH decreased to <7.0 when UCOs were stopped and animals allowed to recover. Fetal arterial blood samples for blood gases and pH were taken at selected time points while mean arterial blood pressure (MABP), fetal heart rate (FHR), and ECoG activity were continuously monitored through the baseline, UCOs, and recovery period. ECoG activity was analyzed for amplitude and frequency, and thereby incidence of low-voltage/high frequency (LV/HF), high-voltage/ low frequency (HV/LF), and indeterminate voltage/frequency (IV/F) behavioural state activity, using computerized spectral edge analysis techniques. ECoG parameters were then related to measures of fetal acidosis and cardiovascular change using adjusted baseline comparisons or Spearman correlations with significance assumed for $p < 0.05$.

Results: Fetal arterial pH was little changed during the mild UCOs, but showed a progressive worsening thereafter to 7.25 ± 0.02 (SEM) during the moderate UCOs ($p < 0.05$), and to 6.91 ± 0.04 during the severe UCOs ($p < 0.01$), from the baseline value of 7.36 ± 0.01 . Behavioural state activity was increasingly disrupted with a stepwise decrease in the incidence of LV/HF through the mild, moderate and severe UCO series and conversely a stepwise increase in the incidence of IV/F (all $p < 0.05$) such that LV/HF-HV/LF cycling was largely abolished during the moderate and severe UCOs. At this time ECoG amplitude during the one minute of cord occlusions was found to be decreased the lower the pH ($r = 0.65$, $p = 0.001$) and the higher the base deficit ($r = -0.63$, $p = 0.002$). Additionally, 52 ± 13 min prior to the fetal pH drop < 7.0 , the spectral edge frequency of the ECoG began to increase abruptly during each FHR deceleration, from 3 ± 1 Hz up to 23 ± 2 Hz ($p < 0.001$) and was correlated to both FHR change and a pathological decrease in MABP during each FHR deceleration at this time ($p < 0.001$).

Conclusion: Repetitive UCOs with worsening acidemia in the ovine fetus alters ECoG activity in a predictable manner with an early decrease in the high energy LV/HF state, and thereafter amplitude and frequency changes in relation to cord occlusions and thereby FHR change which are well correlated with the degree of acidemia and in advance of that associated with asphyxial brain injury.

