

END-TIDAL CARBON MONOXIDE AND REGIONAL CEREBRAL OXYGENATION SATURATION IN PRETERM NEONATES WITH AND WITHOUT A PATENT DUCTUS ARTERIOSUS

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Background: Carbon monoxide (CO), an inflammatory marker, can be measured in exhaled air as end-tidal(et)CO. High levels of CO can induce vasodilatation and may alter cerebral perfusion/oxygenation. The inflammatory state can also influence the persistence of a patent ductus arteriosus (PDA). Cerebral oxygenation can be monitored by near-infrared spectrometry (NIRS).

Aim: To study the predictive value of etCO measured within 24 hours after birth for a PDA as well as the relationship between etCOc and NIRS on the first day of life in preterm infants.

Patients and methods: 91 preterm neonates (< 32 weeks GA) admitted to the neonatal intensive care unit of the Wilhelmina children's hospital were included. EtCOc was measured within 24 hours after birth. 78 of the included infants were NIRS-monitored for regional cerebral oxygen saturation (rScO₂) during the first day of life. PDA was diagnosed according to standard echocardiographic indices.

Results: EtCOc measured within 24 hours after birth was significantly higher in preterm neonates who developed PDA (mean 2.34 PPM \pm 0.71SD) compared to infants who did not develop PDA (mean 1.74 \pm 0.63SD) ($p < 0.001$). Measuring etCOc within 24 hours after birth has a sensitivity of 60% and a specificity of 86% in predicting PDA. High values of etCOc are determined by a cut-off value of 2,4 PPM. No statistically significant correlation could be found between etCOc and NIRS-measured rScO₂.

Conclusion: High values of etCOc are related with PDA. EtCOc seems not to be related to rScO₂ on the first day of life.