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CEREBRAL AUTOREGULATION IN THE FIRST DAY AFTER PRETERM BIRTH: NO EFFECT OF SYSTEMIC INFLAMMATION

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Background and aims: Systemic inflammation as well as impaired cerebral autoregulation (CA) has been associated with brain injury in preterm infants. We hypothesized that impaired CA represents a hemodynamic link between inflammation and brain injury. Furthermore, we evaluated the relationship between hypotension and CA.

Methods: Sixty inborn infants (gestational age: 27+3; ± 9 days) with indwelling arterial catheter had continuous recording of mean arterial blood pressure (MAP) and cerebral oxygenation index (OI) by near-infrared spectroscopy for 2.3±0.5 hours, starting 18±9 hours after birth. Coherence and transfer function gain between MAP and OI was calculated and represented the presence and magnitude of impaired CA, respectively. We used fetal vasculitis (placenta histology) as an antenatal marker of inflammation, and level of blood interleukin-6 measured at 18±10 hours after birth as a postnatal marker of inflammation. Infants were considered hypotensive if MAP ≤ gestational age in weeks. The study was powered to detect a 1 SD difference in coherence between the vasculitis and the non-vasculitis group.

Results: Thirty-five infants (58%) had impaired CA. Twenty-one infants had fetal vasculitis. Neither fetal vasculitis nor interleukin-6 affected CA significantly. A high level of interleukin-6 was associated with hypotension (p=0.03) irrespectively of Dopamine therapy. Magnitude of impaired CA increased with increasing hypotension (p=0.02). No significant associations were found to intraventricular hemorrhage (n=10) or neonatal mortality (n=8).

Conclusions: Postnatal inflammation was weakly associated with arterial hypotension, and hypotension was weakly associated with impaired autoregulation. There was no direct association, however, between autoregulation and postnatal or antenatal signs of inflammation.