PART 1: THE IMPACT OF PATENT DUCTUS ARTERIOSUS ON CEREBRAL OXYGEN SATURATION IN PREMATURE INFANTS

In the United States, patent ductus arteriosus (PDA) is present in approximately 20% of premature babies born at >32 weeks gestation and up to 60% of infants born at < 28 weeks gestation. Delayed or lack of appropriate closure of the ductus arteriosus after birth results in varying degrees of left-to-right shunt. Increased blood flow from the systemic to the pulmonary circulation puts stress on the pulmonary vasculature and can lead to heart failure and pulmonary hypotension if left untreated.

The presence of a PDA, the significance of which is in part determined by clinical symptoms and echocardiography to quantify ductal size and blood flow, may be associated with diminished perfusion of other organs in the body, including the brain.

In a 2008 study, Lemmers et al. discovered that the median cerebral oxygen saturation level in 20 premature infants upon diagnosis of PDA was significantly lower than that of a matched control cohort without PDAs (62±9% vs 72±10%, p<0.05). One 27-week-old infant within the PDA group experienced cerebral saturation levels less than 40% at the time of PDA diagnosis.

In a subsequent study, Chock et al. observed that a group of 12 infants requiring surgical ligation of their PDA experienced more hemodynamic instability and spent a significantly greater percentage of time with cerebral oxygen saturation levels below 20% of baseline (pre-surgical) levels than 12 unmatched control infants without PDA (Figure 2; p=0.0004).

Check out Part 2 of this two-part series to discover how cerebral oxygenation changes during treatment to close a PDA, and the impact of low cerebral saturation values on long-term neurodevelopmental outcomes.
REFERENCES