Figure 1. Normal circulation

Part 1 of this two-part series discusses the relationship between the presence of a patent ductus arteriosus (PDA) and cerebral oxygen saturation in the premature or very low birth weight infant. In Part 2, we discuss how cerebral oxygenation changes during treatment to close a PDA, and the impact of low cerebral saturation values on long-term neurodevelopmental outcomes.

In 20 premature infants with hemodynamically significant PDA treated with indomethacin, Lemmers et al. found that at 24 hours post-treatment there was no longer a significant difference in median cerebral oxygen saturation value between this group and a matched control group without PDAs.1

In a separate study, the same group also showed that the mean cerebral oxygen saturation was significantly higher 24 hours after performing surgery to clip the PDA compared to pre-clip values (Figure 2; p<0.05).2 Chock et al. published similar findings, demonstrating a statistically significant increase in cerebral oxygen saturation values from baseline in a group who underwent ligation compared to a group managed with conservative measures (e.g., fluid restriction, diuretics).3

Timely treatment of a PDA, and subsequent improvement in cerebral oxygen saturation, may have important implications on long-term neurodevelopmental health. In a pivotal 2014 longitudinal study of 67 premature infants, Verhagen et al. found that those who experienced prolonged desaturation at values less than 50% on day 1 post-birth demonstrated worse gross motor skills at two and three years of age.4

Figure 2.
REFERENCES


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