Preterm neonatal cardiovascular instability: understanding the fetus when evaluating the newborn

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Poor perfusion of the kidneys and gut, and associated functional impairment, are major problems in the first days of life in very premature infants. These complications can be associated with substantial mortality and further problems such as reduced kidney growth and chronic renal problems in later life. There is very little information, and consequently considerable debate, about how or even whether to improve perfusion of the vital organs of this most vulnerable group of babies. Indeed, significant variability exists in treatment options, and many therapies lack robust validation. Current treatments simply do not consistently improve babies' perfusion generally or kidney and gut perfusion and function in particular. In many infants, low blood flow is not mediated by low blood pressure. Rather hypoperfusion may be secondary to actively mediated vasoconstriction, which may have been initiated *in utero* as part of the fetal responses to hypoxia, and which is still active after birth as part of post-hypoxic recovery. Thus while the fetus has transitioned to newborn life, the responses must be considered as part of a continuum of responses from fetus to newborn. Most important are the post-hypoxic responses of the fetus, the mechanisms mediating hypoperfusion, and the role vasoconstriction plays in supporting blood pressure in the face of transient impairment of cardiac output. There are added complications faced by the newborn which further impair cardiac function such as a patent *ductus arteriosus* and the changes in metabolic demand of organs such as the kidney and newborn. There is value in taking a physiological approach to understanding the transition from fetal to newborn life, recognizing the need for evidence based science to develop and refine ways of improving perfusion of the kidneys and other vital organs in premature babies.