Hemodynamic Assessment and Monitoring of Premature Infants

Afif El-Khuffash, FRCPI, MD, DCh[a,b],
Patrick J. McNamara, MD, MSc[c,*]

KEYWORDS

• Hemodynamic assessment • Periviable • Preterm infant • Hypotension
• Echocardiography • NICOM • NIRS

KEY POINTS

- Management of the hemodynamic status of periviable premature infants is challenging owing to the multitude of etiologies and the unique characteristics of the circulatory system.
- There are difficulties in monitoring and identifying hemodynamic compromise and a lack of evidence supporting the current treatment approaches.
- A physiology-based approach to the diagnosis, monitoring and management of low blood flow sates in periviable infants is likely to produce the best outcomes.

INTRODUCTION

The cardiovascular care of critically ill preterm infants, particularly around the periviable period, remains a significant challenge in the neonatal intensive care unit for a multitude of reasons. First, the etiologic causes of hemodynamic compromise in this population are heterogeneous; second, the phenotypic presentation is oftentimes modified by the complex physiologic processes that occur during transition from fetal to neonatal life; third, the pharmacologic effects of therapeutic intervention are developmentally regulated; finally, thresholds to guide intervention, predominantly based on mean arterial pressure, lack scientific validation. Consequently, the approach to infants with low blood flow states needs to be individualized. The use of regimented protocols, which usually recommend the administration of fluids followed by stepwise incremental addition of specific cardiovascular agents, without consideration of their

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[a] Department of Neonatology, The Rotunda Hospital, Parnell Square, Dublin 1, DO1 P5W9, Ireland; [b] Department of Paediatrics, School of Medicine, Royal College of Surgeons in Ireland, 123 St Stephen's Green, Dublin 2, Ireland; [c] Division of Neonatology, The Hospital for Sick Children, University of Toronto, 555 University Avenue, Toronto, Ontario MG5 1X8, Canada
* Corresponding author.
E-mail address: patrick.mcnamara@sickkids.ca

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biological appropriateness for the active pathophysiologic state, have failed to produce tangible improvements in short- and long-term outcomes. In fact, recent evidence points toward causing harm.[1] Another increasingly recognized challenge is the lack of feasible and robust measurements of systemic blood flow that facilitate the identification of hemodynamic compromise. The overreliance on blood pressure, which is a poor surrogate for systemic blood flow, may result in both overtreatment and undertreatment of infants in certain physiologic situations. Although methods for intermittent and/or continuous monitoring of cardiac output and systemic blood flow are becoming increasingly used, the unique physiologic environment of preterm infants (with the persistence of fetal shunts) add further challenges to using those methods. There remains a lack of reliable data on normal blood pressure and cardiac output values in the neonatal population during the early transitional period and beyond. Identification of thresholds or clinical scenarios where hemodynamic intervention may modify patient outcomes represents the most important challenge for neonatal intensivists. Relevant to the clinical decision making process are the active disease state, phase of physiologic transition, and competing interventions, yet these are often not considered.

TRANSITIONAL PHYSIOLOGY: CARDIOVASCULAR AND PHYSIOLOGIC CONCEPTS

The transition from fetal to neonatal life is accompanied by important physiologic changes in the circulatory system: There is a significant increase in systemic vascular resistance (SVR) resulting in an increase in left ventricular (LV) afterload. This increase is a consequence of the loss of low resistance placental circulation, and a surge in vasoconstrictor substances including vasopressin (through vasopressin receptors, which increase intracellular calcium release and upregulate adrenaline receptors) and thromboxane A_2 (a potent vasoconstrictor).[2] In addition, there is a decrease in pulmonary vascular resistance (PVR) as a consequence of pulmonary arterial vasodilatation. This decrease is facilitated by the increase in the partial pressure of oxygen accompanying lung aeration, and the increased production of potent pulmonary arterial vasodilators including prostaglandins, bradykinins, and histamine.[3] The increase in SVR and decrease in PVR redirects right ventricular output from shunting across the ductus arteriosus (and supplying the brain) toward the pulmonary vascular bed (to supply the lungs). This is a crucial step during the early transition, which ensures that LV preload (which was derived from the placental circulation during fetal life) is maintained by adequate pulmonary venous return. The maintenance of adequate LV preload is essential for sustaining an adequate LV output (LVO) in the face of a rising LV afterload. Consequently, right ventricular preload becomes dependent on systemic venous return, and right ventricular afterload remains low owing to decreasing PVR (**Figs. 1** and **2**).

The additional effects of the timing of cord clamping after birth need to be considered as an important part of the transitional process. The placenta is thought to hold 30% to 50% of the fetal circulating volume at any one time; therefore, early clamping of the cord may result in a significant reduction of LV preload and effective LVO. This is a consequence of the reduction in blood flow to the left atrium from the placental circulation, which is not restored effectively until pulmonary flow blow is established.[4] Deferring cord clamping until the infant begins to breath and establish pulmonary blood flow may result in fewer fluctuations in LVO by ensuring the maintenance of LV preload (from the placental circulation) until pulmonary venous return takes over.[5] Knowledge of the approach taken in a particular infant (early or deferred clamping of the umbilical cord) will help with the individualized approach to managing that particular infant if a low blood flow state is identified.