Functional Echocardiography in Assessment of the Cardiovascular System in Asphyxiated Neonates

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Perinatal asphyxia commonly results in multi-organ damage, and cardiovascular dysfunction is a frequent association. Myocardial damage, right ventricular dysfunction, abnormal circulatory transition, and impaired autoregulation may all contribute to postnatal neurological damage. Adequate monitoring and appropriate targeted treatment therefore are essential after an asphyxial insult. Standard methods of cardiovascular monitoring in the neonate have limitations. Point of care ultrasound scanning or functional echocardiography offers extra information to assist the clinician in identifying when there is significant cardiovascular impairment, classifying the underlying abnormal physiology and potentially targeting appropriate therapy, thereby optimizing the post-insult cerebral blood flow and oxygen delivery. *(J Pediatr 2011;158:e13-8)*.

The neonate with symptoms of perinatal asphyxia usually has a multiorgan insult. The neurological manifestations are only a part of the underlying pathology. The common clinical appearance of a severely asphyxiated neonate—pale, poorly perfused, and tachycardic despite effective resuscitation—suggests cardiovascular compromise. Involvement of the cardiovascular system frequently includes primary cardiac dysfunction and circulatory inadequacy, which contributes to further postnatal neurological injury. After an asphyxial insult, maintenance of tissue oxygenation is a primary aim of management. Tissue oxygenation depends on oxygen content of the blood, blood flow to the organ, and the tissue's ability to extract and use oxygen. Consequently, cardiac output may be a more important determinant of cerebral blood flow than blood pressure.[1] Because cardiac dysfunction is a common outcome of perinatal asphyxia[2] and blood pressure is not necessarily reflective of cardiac output,[3] it is important to monitor cardiac output and blood pressure in asphyxiated neonates.

Echocardiography is used for diagnostic purposes in the asphyxiated neonate—to rule out primary congenital cardiac disease, assess neonates with suspected persistent pulmonary hypertension of the newborn (PPHN) and to allow assessment of the degree of myocardial dysfunction. Echocardiography can contribute to the assessment of myocardial ischemia and dysfunction because both the electrocardiographic changes and the biochemical changes seen in older children and adults are less common in neonates.[4,5] In adults with acute myocardial injury, two-dimensional echocardiographic measures of cardiac function have both high positive and negative predictive values of a poor prognosis.[6]

Traditionally, echocardiographic information is provided in a single consultation by an available cardiologist, with focus on exclusion of congenital heart disease and PPHN and a current assessment of function. The evolution of the use of point-of-care functional echocardiography, at the cot side by the clinician caring for the neonate, has resulted in extra hemodynamic data being available in a timely manner.[7,8]

Assessment of the Cardiovascular System

Transient myocardial ischemia is a recognized association of perinatal asphyxia, with an incidence from 30% to 82% of severely asphyxiated neonates.[2,9,10] The incidence of myocardial dysfunction may be higher in preterm neonates who already have risk factors for myocardial impairment because of immaturity of the myocardium.[11] Transient myocardial ischemia is often associated with evidence of myocardial damage, such as a rise in cardiac troponin levels.[12] Diagnosis of myocardial involvement after a hypoxic ischemic insult can be difficult and requires skills in echocardiography. There are two major patterns of myocardial dysfunction, depression of left ventricular (LV) function assessed with measures of contractility (fractional shortening, ejection fraction) and reduced cardiac output or aortic valve ejection velocity (Doppler velocity and flow measurements). Reduction in cardiac output of asphyxiated neonates is commonly observed.[4] The second pattern is moderate to severe pulmonary hypertension causing tricuspid regurgitation, reduced right ventricular (RV) output, and RV dysfunction.[13] In addition to the impairment of oxygenation that can occur with pulmonary hypertension, there is often an associated reduction in systemic

EF	Ejection fraction
FS	Fractional shortening
LV	Left ventricular
MPI	Myocardial performance index
PPHN	Persistent pulmonary hypertension of the newborn
RV	Right ventricular
SVC	Superior venal cava

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blood flow caused by impaired pulmonary venous return to the left atrium, which compounds the reduced systemic blood flow from LV dysfunction.

Clinical Assessment

The mainstays of clinical assessment of asphyxiated neonates have been capillary refill time, acidosis, and blood pressure, which are all limited in both accuracy of assessment and the information obtained. Capillary refill time is used in adult and pediatric intensive care settings as an indicator of poor perfusion with some correlation;[14,15] however, accuracy in predicting low cardiac output is limited. Similarly, acidosis, as indicated by lactate level or base deficit, probably reflects poor perfusion and anaerobic metabolism some hours before. There is some usefulness in these measures, as shown by the correlation with important outcomes such as mortality rate.[16] It is generally assumed that normal blood pressure is indicative of normal cardiac output and thus adequate cerebral blood flow. Several clinical studies have demonstrated the disconnect between blood pressure and cardiac output caused by variability of peripheral vascular resistance, particularly in the newborn.[3,17] This is particularly important in the asphyxiated neonate with a normal systemic blood pressure but impaired myocardial function and resultant low cardiac output. With the exception of an impression of impaired perfusion and later development of acidosis, the only way to identify these neonates is to also measure cardiac output. Impaired cardiac output during the immediate post-asphyxia period may compound the original insult by impairing cerebral hemodynamics as measured with cerebral blood flow velocities and increased pulsatility/resistance indices. The more significant the cardiac dysfunction, the worse the outcome in hypoxic-ischemic encephalopathy.[18]

Qualitative Assessment of Cardiac Function

A simple visual assessment with echocardiography in either the parasternal long axis or a short-axis parasternal view can provide significant information about the function of the myocardium. Visual assessment of contractility is assessed with the movement of the septum and posterior myocardial wall and is reasonably accurate for clinicians experienced in echocardiography.[19] Similarly, the filling of the heart can be assessed by observing the residual volume in diastole—the LV end diastolic diameter. Generally, severely hypovolemic neonates will have very little residual cavity in diastole, and an infant with volume overload will have a significantly increased volume at end diastole. Both of these visual impressions are further quantified with measurement of fractional shortening, ejection fraction, or the more global function measures of stroke volume and cardiac output.

M Mode Measurements

M Mode imaging allows measurement of fractional shortening (FS) and ejection fraction (EF), which are more quantitative measures of the contractility of the ventricles. The reference range for FS in term neonates is 25% to 40%. EF is similar to FS, but each of the measures is cubed to allow assessment of volume changes; this also multiples any measurement error, particularly because in the neonate the RV dominance alters the shape of the ventricles. Other measures of EF, such as the modified Simpson's rule using biplane paired echocardiographic apical images in two views, although well validated, are labor intensive and not practical in sick infants. In a group of asphyxiated neonates, there was a gradual decrease in the FS with an increasing degree of asphyxia as determined with Apgar score and low cord pH level (<7.2).[20] Both EF and FS are of limited use in the neonate because of the presence of higher RV pressure and consequent reduced septal motion.[21] Newer techniques such as tissue Doppler ultrasound scanning imaging provide more specific information about regional cardiac function. Tissue Doppler ultrasound scanning imaging allows measurement of myocardial velocities and transmyocardial velocity gradient,[22] a potentially useful measure of function, but there is little data on the use of tissue Doppler ultrasound scanning imaging in the asphyxiated neonate.

Systolic/Diastolic Time Intervals

Other measures of cardiac function can be determined by measuring peak velocity, mean acceleration, acceleration time, and LV ejection time. The systolic time interval ratio (acceleration time/LV ejection time) was not predictive of asphyxia.[20] Use of more sophisticated measures of contractility that are independent of preload/afterload, such as the velocity of circumferential shortening or the ratio of velocity of circumferential shortening to end systolic wall stress, an afterload adjusted parameter,[23] may allow more accurate assessment of cardiac function in asphyxiated neonates, but there are few published clinical studies of these measurements in asphyxiated neonates.

Myocardial Performance Index

The myocardial performance index (MPI) combines a measure of both the systolic and diastolic intervals to characterize global myocardial performance.[24] The MPI is a Doppler ultrasound scanning-derived index of myocardial performance, combining the isovolumetric contraction and relaxation time intervals, used to assess ventricular function. The index is independent of heart rate and blood pressure and does not rely on geometric assumptions. Preterm neonates with evidence of mild perinatal asphyxia (Apgar score 5-7) have an increased MPI compared with a group of control neonates without evidence of asphyxia.[25] The index normalized by the third week of life.

Cardiac Output with Doppler Ultrasound Scanning

A more global assessment of cardiac function can be achieved by measuring cardiac output non-invasively by using

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