EXPERT CONSENSUS STATEMENT

Targeted Neonatal Echocardiography in the Neonatal Intensive Care Unit: Practice Guidelines and Recommendations for Training

Writing group of the American Society of Echocardiography (ASE) in collaboration with the European Association of Echocardiography (EAE) and the Association for European Pediatric Cardiologists (AEPC)

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Abbreviations

AAP = American Academy of Pediatrics

AEPC = Association for European Paediatric Cardiology

ASE = American Society of Echocardiography

CDH = Congenital diaphragmatic hernia

CHD = Congenital heart disease

EAE = European Association of Echocardiography

ECMO = Extracorporeal membrane oxygenation

EF = Ejection fraction

LV = Left ventricular

MPI = Myocardial performance index

mVCFc = Mean velocity of circumferential fiber shortening

NICU = Neonatal intensive care unit

PA = Pulmonary artery

PDA = Patent ductus arteriosus

RA = Right atrial

RV = Right ventricular

RVSp = Right ventricular systolic pressure

SF = Shortening fraction

SVC = Superior vena cava

TEE = Transesophageal echocardiography

TNE = Targeted neonatal echocardiography

TVI = Time-velocity integral

2D = Two-dimensional

VLBW = Very low birth weight

1. TARGETED NEONATAL ECHOCARDIOGRAPHY: BACKGROUND AND INDICATIONS

The role of echocardiography in the neonatal intensive care unit (NICU) has changed over the past few years. Previously, nearly all echocardiographic studies in the NICU were performed by pediatric cardiologists to diagnose or monitor congenital heart disease (CHD) and to screen for patent ductus arteriosus (PDA). More recently, neonatologists have become interested in the echocardiographic assessment of hemodynamic instability in infants. The terms *functional echocardiography* and *point-of-care echocardiography* have been introduced to describe the use of echocardiography as an adjunct in the clinical assessment of the hemodynamic status in neonates.[1-4] The increasing availability of echocardiography, with miniaturization of the technology, has resulted in more widespread use of echocardiography in NICUs around the world.[5] Perhaps the most significant challenge for the application of so-called functional studies is that newborns in the NICU with hemodynamic instability are at a much higher risk for having underlying CHD. In addition, newborns in the NICU are unique in that they are in the process of transition from fetal to postnatal circulation.

In this document, we make clear distinctions between initial echocardiographic studies in neonates with the suspicion of CHD and studies performed on infants without any clinical suspicion of CHD. If CHD has been excluded, subsequent studies in children with structurally normal hearts can focus on hemodynamic or functional assessment. The initial echocardiographic examination should always be a comprehensive study of both anatomy and function that is to be interpreted by a pediatric cardiologist within a reasonable time frame. Some structural defects, such as anomalous pulmonary venous return or coarctation of the aorta, can be difficult to detect using echocardiography and require extensive training and continued practice. Once significant congenital defects have been ruled out, more focused studies can be performed and interpreted by a trained echocardiographer for specific indications, as defined later in this document. We propose to use the term *targeted neonatal echocardiography* (TNE) for the more focused studies. The aims of the current document are: (1) to review the current indications of TNE; (2) to define recommendations for the performance of TNE; and (3) to propose training requirements for the operators performing and interpreting TNE.

1.1. Cardiovascular Adaptations in the Neonatal Period

The neonatal cardiovascular system differs from those of fetal, pediatric, and adult patients. At term, a neonate must successfully transition through abrupt changes in the cardiorespiratory system, including changes in lung volume and compliance and changes in left and right heart preload and afterload. Intracardiac and extracardiac shunts via the foramen ovale and ductus arteriosus, physiologic in the fetus, have varying effects on immediate postnatal hemodynamics. The neonatal heart may also have to cope with structural heart disease and/or extracardiac congenital and acquired conditions, such as congenital diaphragmatic hernia (CDH), sepsis, or pulmonary hypertension, that are tolerated differently compared with an older child. In the setting of a preterm delivery, the immaturity of the cardiovascular system and other organ systems makes it more difficult for the neonate to appropriately respond to the challenges of postnatal transition and extrauterine existence. Detailed understanding of fetal, transitional, and neonatal cardiovascular physiology is necessary to enable understanding of echocardiographic information obtained during the perinatal period.

The Transition From Fetus to Neonate. It is important for those performing TNE to understand the normal development of the myocardium and cardiopulmonary circulation and how preterm delivery may disrupt this process. In the fetus, myocytes are smaller and typically have a single nucleus, compared with the multinucleated myocytes that are prevalent postnatally.[6] Although the fetus has a higher indexed myocardial mass,[7] the fetal myocardium is less organized at the cellular level, with fewer sarcomeres per unit mass, different isoforms of contractile proteins,[8] a developing sarcoplasmic reticulum,[9] an overall higher water content,[10] and a decreased number of mitochondria.[11] In addition, the heart is enclosed within a poorly compliant thorax.[12] As a result of these factors, the fetal heart is less compliant and less contractile than a term newborn or adult heart. These differences are manifested in the developmentally regulated limited fetal cardiac response to changes in preload or afterload, with heart rate changes being the major mechanism to alter fetal cardiac output, although some response to preload occurs if afterload remains constant.[13-18] The fetal right and left ventricles also differ in myofiber architecture throughout gestation, with the right ventricle less tolerant to increases in afterload, despite its contributing slightly more to the combined ventricular output than the left ventricle during fetal life.[15] The fetal pulmonary vasculature also exhibits significant differences from the adult pulmonary circulation.[10] In the