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CHAPTER

Functional Echocardiography in Neonates

Anup Thakur, Srikanth Kandi

ABSTRACT

Functional echocardiography (Fn echo) is a clinician driven echocardiography done at the bedside to assess cardiac function and hemodynamic condition of a neonate. It has grown to be an indispensable assessment tool in the newborn critical care unit. Traditionally, tissue perfusion is assessed by parameters such as blood pressure, heart rate monitoring, urine output, and capillary refill time. Although these parameters are useful but do not provide detailed information on rapidly changing hemodynamics in neonates. When combined with a comprehensive clinical examination, Fn echo can deliver quick, accurate, and real-time diagnostic information that can help in management of critically sick neonates. The most common scenarios in which Fn echo can help a neonatologist is to assess the cardiac functions, evaluate pulmonary pressures, and assess hemodynamic changes in a patent ductus arteriosus. This chapter deals with the various modes and views of echocardiography and assessment of neonatal cardiac function, and evaluation for hemodynamically significant patent ductus arteriosus (hsPDA) and pulmonary hypertension.

Keywords: Diastolic function, Functional echocardiography, Patent ductus arteriosus, Pulmonary hypertension, Systemic blood flow, Tissue Doppler, Volume assessment.

INTRODUCTION

Functional echocardiography (Fn echo) has grown to be an indispensable assessment tool in neonatal intensive care unit. When combined with a comprehensive clinical examination, it can deliver quick, accurate, and real-time diagnostic information that is crucial for patient care. Globally, Fn echo has different nomenclatures such as targeted neonatal echocardiography (TNE), clinician performed ultrasound (CPU), neonatologist-performed echocardiography (NPE), point-of-care ultrasound (POCUS), etc.[1] The basic principle remains the same, i.e., echocardiography is done by noncardiology and nonradiology professionals at the patient's bedside to conduct a timely assessment of a sick patient with prompt identification of pathologic processes that can guide life-saving and resuscitation measures. In a traditional clinical practice, the treating team orders an echocardiography, waits for the cardiologist to acquire and interpret the image and then the treating team tries to integrate the findings with the clinical context. On the

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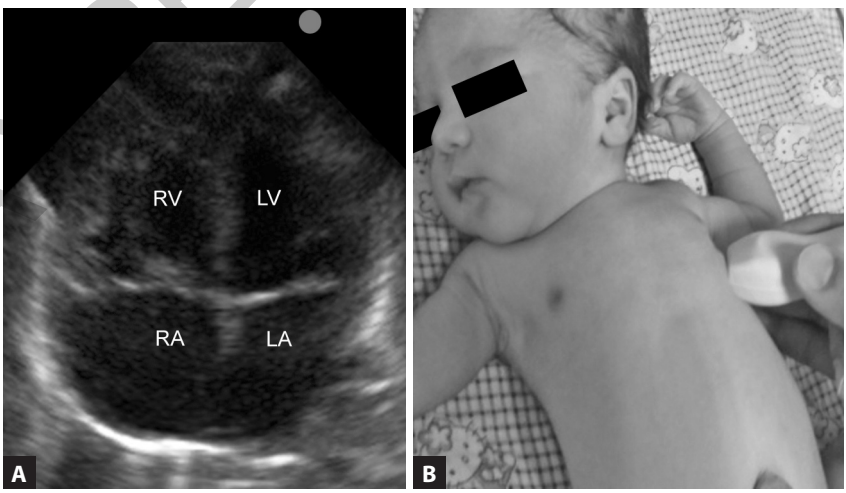
contrary, Fn echo is performed in real time; the treating team performs the study and integrates the information according to the clinical scenario and is also able to monitor changes associated with any intervention.[2]

Fn echo is clinician driven echocardiography done at the bedside to assess the cardiac functions, pulmonary pressures and hemodynamic changes in a patent ductus arteriosus. The Fn echo assessment done by neonatologist presumes that the heart is structurally normal. So, all such assessments should be preceded or followed by a comprehensive structural assessment by a paediatric cardiologist. This chapter deals with the various modes and views of echocardiography and assessment of neonatal cardiac function, and evaluation for hemodynamically significant patent ductus arteriosus (hsPDA) and pulmonary hypertension.

MODES OF ECHOCARDIOGRAPHY

The common modes of echocardiography are the B-mode, M-mode, color, and Doppler mode.[3]

- *Two-dimensional (2D)/B-mode:* This is the most common mode used where 2D image of the underlying structure is displayed. This mode is good for understanding the anatomy of heart structures **(Figs. 1A to D)**.
- *M-mode (motion):* This mode scans underlying moving tissue along a horizontal time line. Movement of the structures positioned in that line can be visualized across time line. Echoes from stationary structures trace out as flat lines parallel to horizontal line and echoes from moving structures are traced as curvy lines representing the range of motion. This mode is good for evaluating motion and variations in various heart chambers at different phases of cardiac cycle **(Figs. 2A to C)**.



Figs. 1A and B

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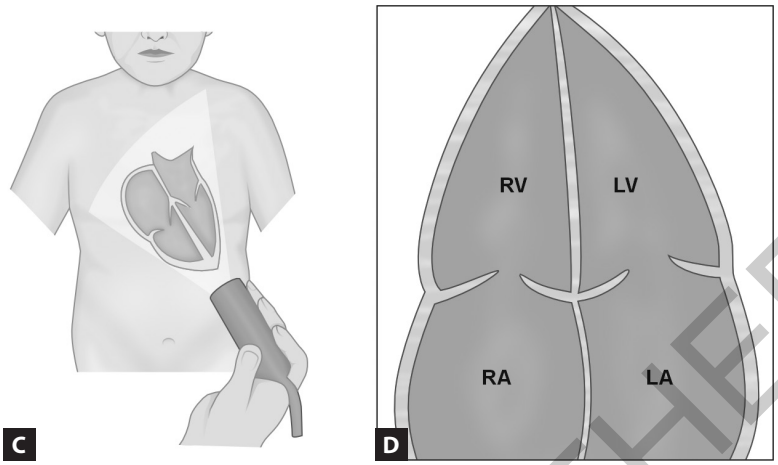
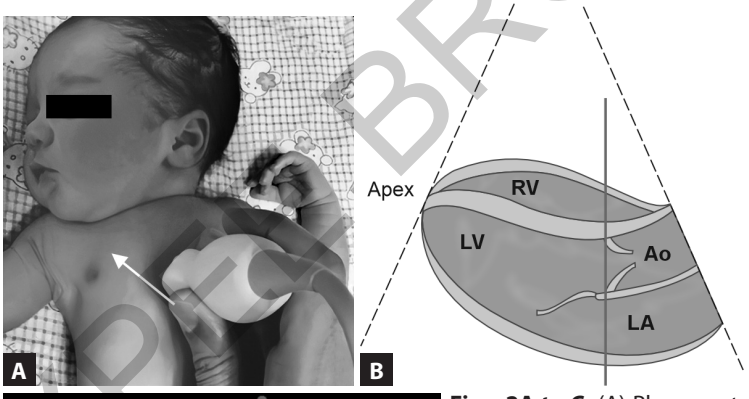
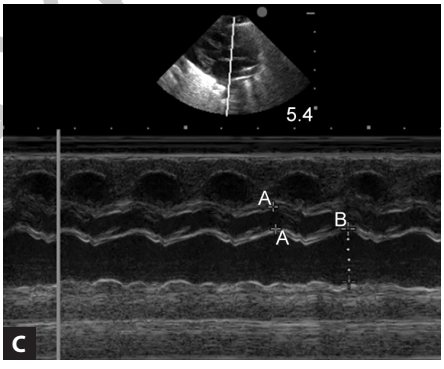


Figs. 1C and D

Figs. 1A to D: (A) B-mode showing apical or four-chamber view of the heart; (B and C) show placement of probe at the apex with pointer toward left and pointing toward 2–3 o'clock position; (D) Schematic representation of four-chamber/apical view. (LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle) *(For color version, see Plate 5)*





Figs. 2A to C: (A) Placement of probe for PLAX view—junction of upper two-thirds and lower one-third of sternum—3rd to 4th intercostal space with pointer toward right shoulder, at 10 to 11 o'clock position; (B) Schematic diagram showing structures in PLAX view and how to drop M-mode perpendicular to the area of interest; (C) M-mode image with M-mode being dropped at the hinge of aortic valve to measure LA/Ao ratio. A shows diameter of aortic valve and B show diameter of left atrium. (Ao: aorta; LA: left atrium; LV: left ventricle; PLAX: parasternal long axis view; RV: right ventricle) *(For color version, see Plate 5)*

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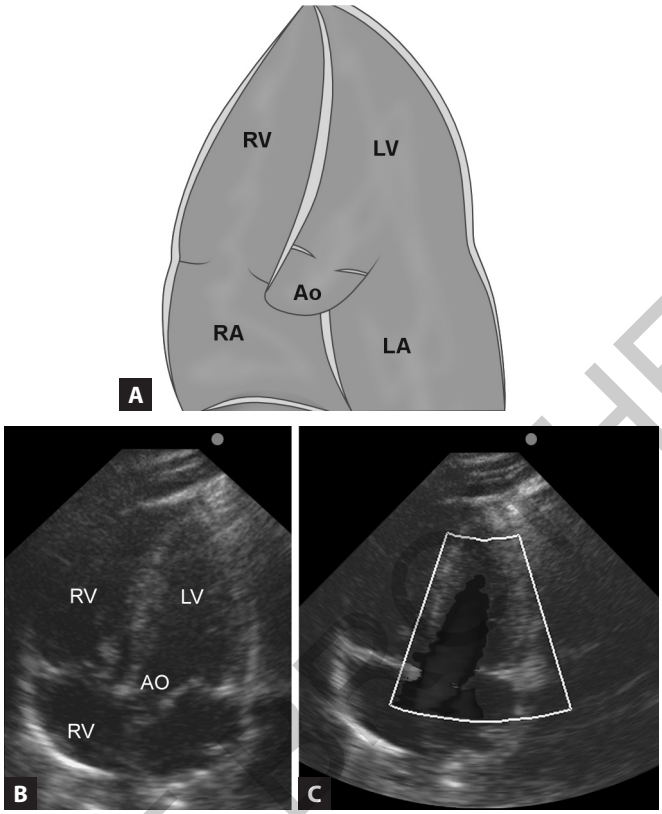

Figs. 3A to C: (A) Schematic diagram showing structures in five-chamber view; (B) Five-chamber view showing LA, LV, RA, RV, and aortic outflow; (C) Color Doppler being placed at left ventricular outflow tract shows nice laminar blue color. Blue color indicates the direction of blood flow is away from the probe. (Ao: aorta; LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle) *(For color version, see Plate 6)*

- *Color-mode:* In this mode, flow of blood is visualized across various heart chambers and vessels over 2D image. Blood flow toward the probe will result in red color stream, whereas blood flow away from the probe will yield a blue stream. A dark red color smooth stream denotes low velocity laminar flow toward probe and a dark blue color denotes a low velocity laminar flow away for probe. A bright yellow to orange stream with a lot of speckling and mosaics indicates a high velocity turbulent flow toward the probe and a bright sky-blue stream with a lot of speckling indicates a high velocity, turbulent flow away from the probe **(Figs. 3A to C)**.
- *Doppler mode:* It is used to measure velocity profile of flow across an area. Flow toward probe will give an upward stroke and flow away for the probe will give a downward stroke **(Figs. 4A to C)**. There are two types of Doppler modes: (1) pulse wave (PW) and (2) continuous wave (CW).

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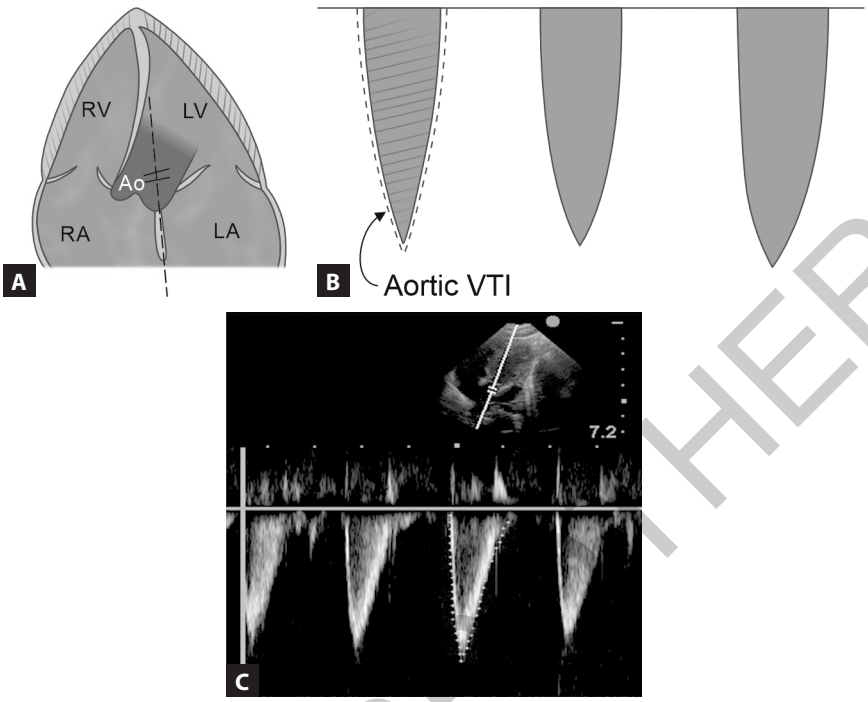

Figs. 4A to C: (A) Schematic diagram of pulse wave Doppler placement across aortic outflow; (B) Schematic diagram showing a downward stroke indicating flow away from probe; (C) Doppler trace across LV outflow tract. (LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle; VTI: velocity time integral)

When velocities are to be measured at a particular point, PW should be used (e.g., across all valves). Lesions with high velocities such as tricuspid regurgitation (TR) jet and valvular stenosis are best assessed by CW mode.

COMMON VIEWS OF ECHOCARDIOGRAPHY

Common views of echocardiography are discussed below:

- *Apical (four-chamber view):* The probe is held with the pointer toward the left shoulder (2–3 o'clock position) and placed at apex of heart (4th–5th left intercostal space between mid-clavicular line and anterior axillary line). The probe is aligned almost parallel to the bed. All the four chambers of the heart will be visible in its long axis **(Figs. 1A to D)**. This view helps in appreciating the relative size of all the four chambers of the heart. Tilting up anteriorly will bring left ventricular outflow tract (LVOT) and aortic valve in view (five-chamber view, see **Figure 3A to C**). Tilting up further anteriorly will open up the right ventricular outflow tract (RVOT).
- *Parasternal long axis (PLAX) view:* The probe is kept at the junction of upper two-thirds and lower one-third of sternum, just below nipple

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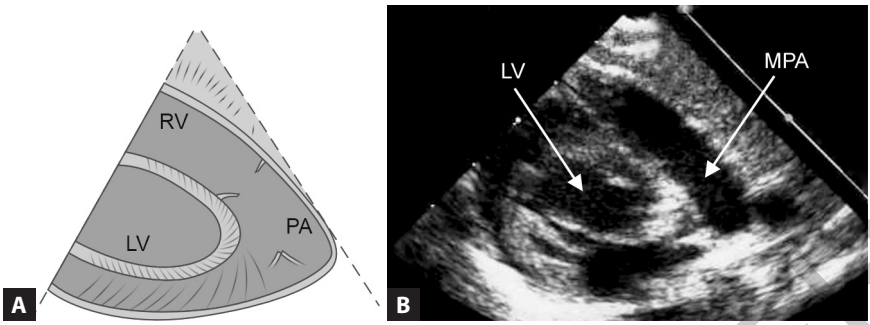

Figs. 5A and B: (A) Schematic diagram of modified PLAX view; (B). Modified PLAX view showing opening up of right ventricular outflow tract. (LV: left ventricle; PLAX: parasternal long axis; RV: right ventricle PA: pulmonary artery; MPA: main pulmonary artery)

line (3rd to 4th intercostal space) almost perpendicular to the surface with pointer toward right shoulder (10–11 o'clock position) **(Figs. 2A to C)**. The PLAX view cuts the heart in its long axis, from apex to aortic root. The standard long-axis view provides an image of the left heart structures—left atrium (LA), left ventricle (LV), aorta and interventricular septum (IVS), and a small part of right ventricle (RV) anteriorly. This view gives information about ventricular septal defect, aortic over-ride, myocardial contractility via fractional shortening (FS), and left atrium: aortic root diameter ratio in PDA evaluation. Slight tilt of the probe toward left shoulder results in modification of PLAX view and reveals main pulmonary artery (MPA) and the pulmonary valve (Modified PLAX view, **Figures 5A and B**).

- *Parasternal short-axis view:* The probe position is similar to parasternal long-axis view but with a 90° rotation. The probe is kept at the junction of upper two-thirds and lower one-third of sternum, just below nipple line (3rd to 4th intercostal space) almost perpendicular to surface with pointer toward left shoulder (1–2 o'clock position). The standard cut at aortic valve (AV) level shows cross-section of aortic root in the center with all three cusps of aortic valve along with left atrium and right heart structures—RA, RV, and RVOT with pulmonary artery bifurcation wrapping around the aortic root. Tilting the probe toward apex will show left ventricular cavity at mitral valve level (fish mouth view) and further tilting the probe toward the apex will cut the rounded left ventricular cavity at papillary muscles level with a surrounding crescentic RV surrounding **(Figs. 6A to C)**.
- *Ductal view:* Slight modification of short-axis view with slightly upward shift at left infra-clavicular region (2nd to 3rd intercostal space) will identify the ductus connecting the MPA and the descending aorta. Aortic root is seen in cross-section (Mercedes Benz sign). MPA will be seen anterior dividing into right pulmonary artery, which wraps around

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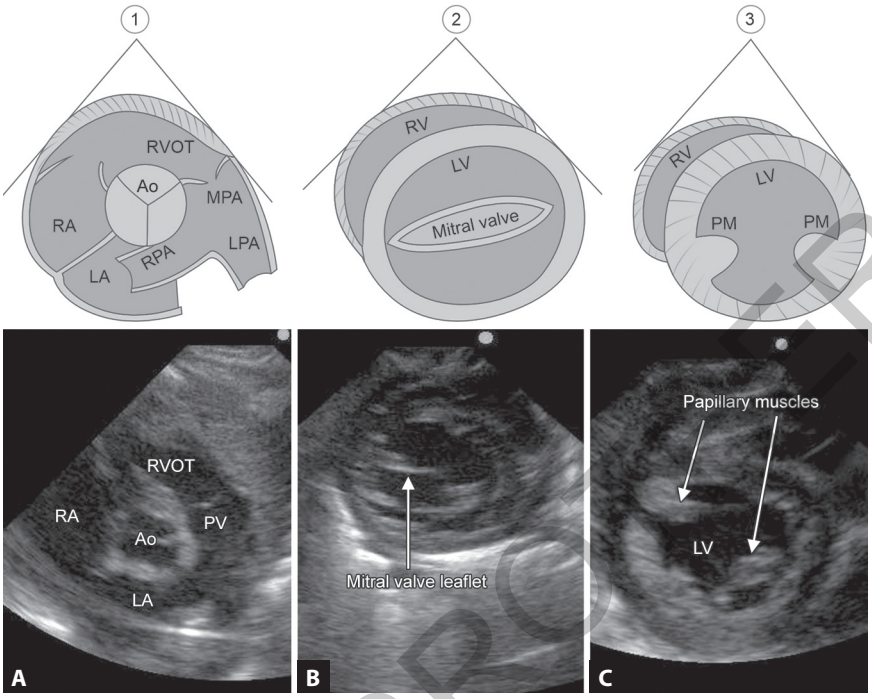
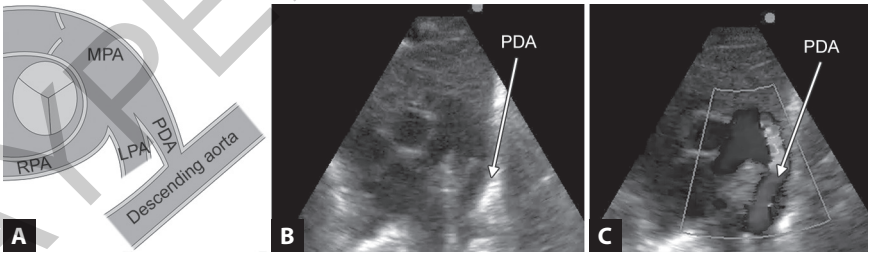


Figs. 6A to C: *Top schematic diagram* of parasternal short-axis view at the level of aortic root (1), mitral valve (2), and papillary muscles (3).
Bottom echocardiographic image of parasternal short-axis view: At the level of aortic root and RVOT (A), at the level of mitral valve (B) and at the level of papillary muscle (C). (Ao: aorta; LA: left atrium; PV: pulmonary valve; RA: right atrium; RPA: right pulmonary artery; RVOT: right ventricular outflow tract)



Figs. 7A to C: (A) Schematic diagram of ductal view; (B) 2D image showing three-legged appearance of RPA, LPA and PDA; (C) Color Doppler of pulmonary arteries and PDA. (LPA: left pulmonary artery; MPA: main pulmonary artery; PDA: patent ductus arteriosus; RPA: right pulmonary artery). *(For color version, see Plate 6)*

the aortic root and left pulmonary artery which dips down. Ductus will be seen as third limb of MPA, which joins the descending aorta (Three-legged stool appearance) **(Figs. 7A to C)**. Ductal diameter is measured at the narrowest point usually at the pulmonary end.

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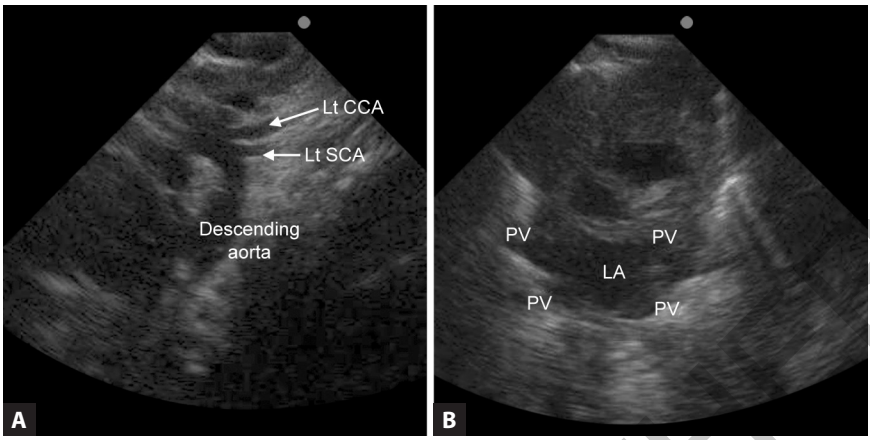

Figs. 8A and B: Suprasternal view. (A) Aortic arch and its branches; (B) Four pulmonary veins draining into the left atrium. (LA: left atrium; Lt CCA: left common carotid artery; Lt SCA: left subclavian artery; PV: pulmonary vein)

- *Subcostal views and suprasternal view:* For the subcostal view, the probe is positioned below subcoastal margin at junction of upper one-third and lower two-thirds of the line joining xiphisternum and umbilicus. The probe should be placed in coronal plane as flat as possible, parallel to bed with pointer of the probe kept at 2–3 o'clock position. In this view, the heart with all its four chambers will be lying horizontally. All the four chambers of the heart, interventricular septum and interatrial septum can be visualized in this view.

  Suprasternal window is good for visualizing aortic arch and pulmonary veins. To visualize aortic arch, the probe is kept almost in midline in suprasternal notch, and almost in sagittal plane with pointer at 1 o'clock position **(Fig. 8A)**. Suprasternal view is also good to visualize drainage of pulmonary veins into LA. To visualize pulmonary veins and LA, probe is rotated from current 1 o'clock plane to coronal plane and tilted posteriorly. All four pulmonary veins, draining into left atrium, can be seen in 2D and color mode (Crab view) **(Fig. 8B)**.

INDICATIONS OF FUNCTIONAL ECHOCARDIOGRAPHY

Traditional hemodynamic monitoring parameters, such as blood pressure, capillary refill time, heart rate, urine output, serum lactate, etc. are not completely reliable. Fn echo can help clinicians to understand the ongoing hemodynamic change in a more precise fashion by providing real-time estimation of volume status, cardiac output, systolic and diastolic function. Critical disorders such as acute pulmonary hypertension, PDA, and shock can reliably be diagnosed by Fn echo. In addition, Fn echo can help to guide therapeutic interventions in such situations. Other indications of Fn echo include detection and treatment of life-threatening complications such

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TABLE 1: Common indications of functional echocardiography in neonates.

Conditions	*Objective*
Patent ductus arteriosus in premature infants	• To confirm the diagnosis and assess for hemodynamic significance by evaluation of parameters such as ductal size, shunt direction, flow pattern, and shunt magnitude
Low systemic blood flow states during transition in premature neonates	• To evaluate volume status, ventricular systolic/diastolic performance and outputs
Acute pulmonary hypertension	• To confirm diagnosis of pulmonary hypertension • To assess and establish disease severity • To evaluate function of the ventricles and their outputs • To evaluate for presence of shunt and evaluate characteristics of flow • To assess sequentially in order to monitor the progression of disease
Shock	• To evaluate volume status, ventricular systolic/diastolic performance and outputs
Infants with perinatal asphyxia	• To evaluate biventricular systolic performance and outputs • To evaluate parameters for shock and acute pulmonary hypertension when relevant
Pericardial/pleural effusion	• To confirm or rule out diagnosis in neonates with sudden unexpected cardiorespiratory deterioration especially in neonates with a central venous catheter in situ
Chronic pulmonary hypertension	• To evaluate preterm infants and infants at risk of chronic lung disease for pulmonary artery hypertension, right ventricular function, and pulmonary venous stenosis

as cardiac tamponade, malpositioned central venous catheters and even identification of structural heart defects. The common indications of Fn Echo are listed in **Table 1**.

MEASUREMENTS IN FUNCTIONAL ECHOCARDIOGRAPHY

A comprehensive evaluation of heart includes assessment of volume status, systolic and diastolic functions, and evaluation of systemic blood flow. The following sections describe the measurements that should be performed in Fn Echo depending on the indications. For simplicity, the description is described under the following heads:

- Volume assessment
- Left ventricular systolic function

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- Left ventricular diastolic function
- Right ventricular systolic function
- Global myocardial function
- Systemic blood flow
- Pulmonary hemodynamics and assessment in pulmonary artery hypertension
- Assessment of patent ductus arteriosus
- Tissue Doppler

Volume Assessment

Cardiac output is the product of stroke volume and heart rate. Stroke volume is determined by preload, contractility, and afterload. In order to generate sufficient preload to the heart, adequate intravascular volume is needed. Volume expansion therapy is often the first line of treatment for hemodynamic compromise in neonates. Eyeballing of the heart in apical four-chamber view can give us a rough idea and a qualitative assessment about volume status. If the chambers appear small, it may reflect hypovolemia. On the contrary, if the chambers are overtly large and appear dilated, hypervolemia is a possibility. When hypovolemia is severe, there could be complete collapse of the left ventricular walls at end systole and as such the ventricular walls touch each other. This is called *left ventricular kissing sign.*[4] However, this assessment is subjective and neither precise nor reliable.

The inferior vena cava (IVC) delivers the major portion of preload to the heart. The fluid status determines the IVC diameter and collapsibility. In spontaneously breathing infants, during inspiration blood is drained from the IVC to right atrium; therefore, it will lead to collapse of IVC to a certain extent. In neonates, on mechanical ventilation, the relationship is reversed with IVC being distended with positive pressure ventilation. In order to visualize the IVC, one should form a subcostal view with pointer toward 12 o'clock position and slightly angulate the probe to right. In this frame, one can see the IVC coursing through the liver and entering the RA, visible on the right of the screen **(Fig. 9A)**. An IVC that is normally filled will have some pulsation and variation with respiratory cycle. An IVC that is hardly visible and tends to collapse entirely on inspiration is likely to be underfilled. On the other hand, an IVC that is overfilled appears large and has minimal collapsibility. As explained earlier, in ventilated patients, during inspiration IVC distends due to positive pressure.

M-mode is used for calculating the minimum and maximum IVC diameter in both expiration and inspiration. *Collapsibility index* is obtained by subtracting maximum diameter in expiration and minimum diameter in inspiration and dividing it by maximum diameter in expiration. In mechanically ventilated neonates, *IVC distensibility index* is calculated by

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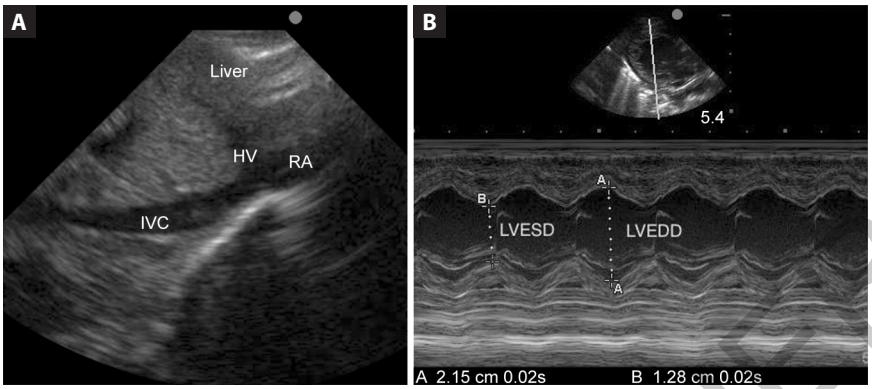

Figs. 9A and B: (A) IVC draining into the right atrium; (B) M-mode trace for calculation of shortening fraction. (HV: hepatic vein; IVC: inferior vena cava; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; RA: right atrium)

subtracting maximum diameter in inspiration and minimum diameter on expiration and dividing it by the minimum diameter on expiration.[5,6]

- Inferior vena cava collapsibility index = (maximum diameter on expiration – minimum diameter on inspiration)/maximum diameter on expiration (>50–55% may indicate a hypovolemic state)
- Inferior vena cava distensibility index = (maximum diameter on inspiration – minimum diameter on expiration)/minimum diameter on expiration (>18% may indicate a possibility that the neonate would respond to fluid bolus)

None of these measurements have been validated in the neonatal population and their utility is still being studied.

Left Ventricular Systolic Function

- *Shortening fraction:* Shortening fraction, also known as fractional shortening (FS), is the most commonly employed method of assessment of myocardial contractility. Using the parasternal long-axis view, M-mode is run with the line of M-mode cutting the tips of mitral valves. One should ensure that the line of M-mode should lie in the middle of LV cavity cutting the IVS perpendicularly. Maximum and minimum dimension of LV cavity will correspond to LV end-diastolic diameter (LVEDD) and LV end-systolic diameter (LVESD) **(Fig. 9B)**.

  There are certain caveats in assessment of systolic functions by FS. First, it is based on the assumption that the sampled region at the mitral valve tip would represent the global LV systolic function. In the presence of myocardial dysfunction, different parts of myocardium can have different degrees of fiber shortening, so estimation of FS would either overestimate or underestimate the overall LV systolic function. Secondly, it assumes that the LV cavity is circular and, therefore, this assumption may lead to

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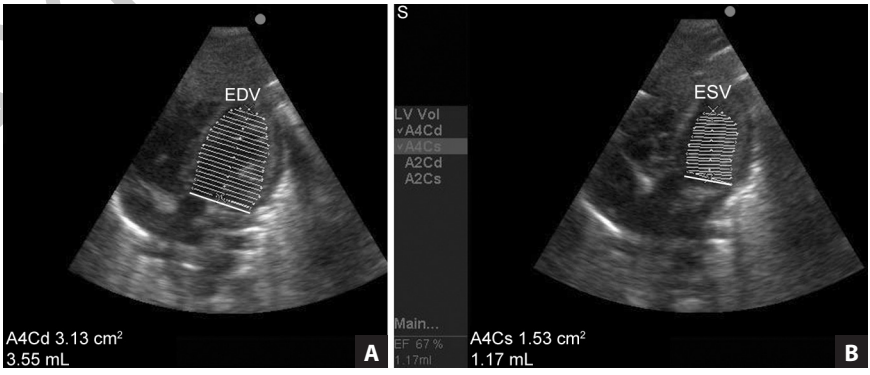
inaccuracy in the setting of septal flattening.[1] Another limitation is that extent of shortening is dependent on state of preload; fractional shortening will be more if preload is adequate and less if preload is inadequate.

$$FS = [(LVEDD - LVESD)/LVEDD] \times 100 \text{ (Normal range 30–45\% approximately)}$$ [7-9]

- *Ejection fraction:* LV ejection fraction (LVEF) is another measure of LV systolic function. It estimates the proportion of LV volume ejected during each cardiac cycle. There are three possible ways of measuring LVEF, based on different assumptions about the shape of LV.
  1. LVEF can be measured in M-mode method (same view as FS) but it assumes that the shape of LV is circular in cross-section. This is the least recommended method for calculation of LVEF and is prone to inaccuracies [$LVEF = \{(LVEDD^3 - LVESD^3)/LVEDD^3\} \times 100$].
  2. The area-length method or the bullet method is a hemicylindrical hemiellipsoid model assumes that the base of LV is cylindrical in shape and the apex is ellipsoid. The area is taken in cross-section in the parasternal short-axis view while the length is taken in the apical view. The LV volume is then calculated as (5/6 × LV area × LV length). The calculations are done in diastole and systole. The EF is then calculated as $LVEF = [(LVEDD - LVESD)/LVEDD] \times 100$, where LVEDV and LVESV are volume of LV in end of diastole and systole, respectively.
  3. The third method is the Simpson biplane method.[10] It assumes that the shape of cavity of LV is circular in cross-section. In both the methods, one should trace at the endocardial-blood pool interface in end diastole and end systole, with the mitral valve being closed **(Figs. 10A and B)**. Adjacent structures such as papillary muscle should be included as in the LV cavity. During measurements, ensure that the LV apex is not displaced much in end diastole or end systole. The normal LVEF is 55–70%.



Figs. 10A and B: Simpson biplane measurement of ejection fraction: (A) End-diastolic volume; (B) End-systolic volume. (EDV: end-diastolic volume; ESV: end-systolic volume)

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- *Velocity of circumferential fiber shortening (VcF):* Measurement of FS may be erroneous in early preterm life due to distortion of LV and abnormal septal wall motion, resulting from RV dominance. Velocity of circumferential fiber shortening describes the rate of shortening rather than the extent of shortening. To calculate this index, PLAX view along with M-mode is obtained and LVEDD and LVESD are calculated. Next one should obtain an apical five-chamber view and place the Doppler gate at the aortic root level, which will produce the spectral Doppler envelope of aortic flow velocity from where left ventricular ejection time (LVET) is calculated. The velocity of circumferential fiber shortening is calculated as [(LVEDD - LVESD)/LVEDD] × LVET. Normal value is 1.54 ± 0.04 circumference/second. Despite its merits over shortening fraction, its usefulness has not been established so far.

$$VcF = [(LVEDD - LVESD)/LVEDD] \times LVET$$

Diastolic Function

Immature myocardium of the preterm infants is known to have reduced diastolic function due to poor compliance. Diastole can be divided in four phases: (1) isovolumetric relaxation, where both the inlet and outlet valves are closed, (2) early rapid filling—which depends on ventricular relaxation and compliance, (3) diastasis, and (4) atrial contraction.

Diastolic function may be assessed by pulse Doppler assessment at mitral valves in standard apical four-chamber view. Normal forward flow will show two waves. The "E" wave corresponds to initial rapid passive filling followed by second "A" wave which corresponds to atrial contraction. In healthy term neonates "E" wave may be taller than "A" wave with ratio >1.1, while in preterm neonates transmitral passive flow (E wave) is less than active flow (A flow) resulting in E/A ratio <1 **(Table 2)**.[11,12] This is attributed to the developmental immaturity of the preterm myocardium with impaired diastolic function leading to limited passive flow. Increased velocity across inflow valves represents either stenosis or increased flow. The "E" wave increases with increasing preload. A large left to right (L–R) shunt in PDA may increase the E/A ratio across mitral valve **(Fig. 11)**. Isovolumic relaxation time (IVRT) is another frequently performed parameter to assess diastolic function. It is time between the closure of aortic valve and opening of mitral valve during isovolemic contraction. It, therefore, represents the time during

TABLE 2: E/A ratio in term and preterm neonates.

E/A ratio	*Term*	*Preterm*
Normal	1.1	0.8
Diastolic dysfunction	<0.7	<0.6

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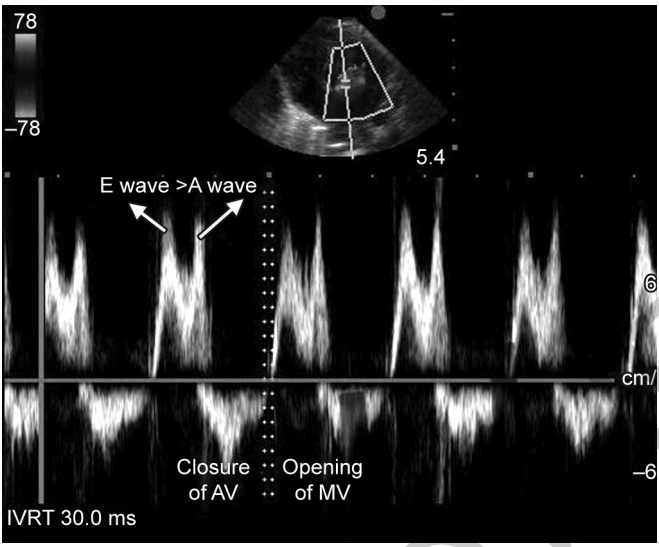

Fig. 11: E/A ratio >1 and short IVRT in presence of hsPDA. (AV: aortic valve; hsPDA: hemodynamically significant patent ductus arteriosus; IVRT: isovolumic relaxation time; MV: mitral valve) *(For color version, see Plate 7)*

which LV is closed and relaxing without any inflow or outflow across it. IVRT is prolonged in diastolic dysfunction or may be shortened in later state of ventricular stiffness due to increased LA pressure. There are other indices that have been proposed as markers of diastolic dysfunction such as acceleration and deceleration times of the "E" wave, velocities of E, A waves, etc.

There are three basic types of abnormal filling patterns:

1. *Delayed (prolonged and impaired) relaxation pattern:* This will show prolonged IVRT and prolonged deceleration time of E wave, low E, and high A wave velocities with an E/A wave ratio typically <1.
2. *Restrictive pattern:* This is usually a late feature and is characterized by shortened IVRT, increased peak E wave velocity with very short deceleration time, and small (or even absent) A wave, leading to an E/A wave ratio 2.
3. *Pseudonormal pattern:* This is usually an intermediate stage between the two patterns described above and shows E/A ration of >1.

Pulmonary venous flow can be obtained by placing a Doppler in the pulmonary vein in the apical view. Three kinds of waves are seen: S wave that occurs during ventricular systole; D wave a diastolic wave that starts after the opening of mitral wave and lastly, AR wave reversal velocity that occurs during atrial contraction. Higher AR velocity values suggest increased LV end-diastolic pressure. In diastolic dysfunction, the compliance of LA decreases and there is rise in LA pressure. This leads to reduction in S velocity with an increase in D velocity, resulting in an S/D ratio <1.[10,13]

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Right Ventricular Systolic Function

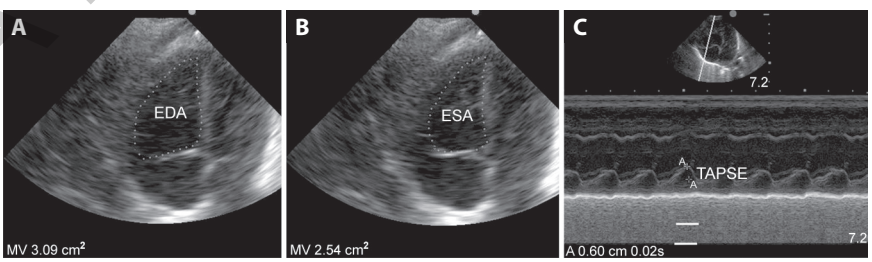
- *Fractional area change (FAC):* There is a geometric limitation of RV and, therefore, unlike LV volume estimation is not done with 2D echo. A surrogate marker of EF is used called *fractional area change (FAC)*. To calculate FAC, the endocardial border of RV is traced manually in systole and diastole in an apical four- or three-chamber view at the endocardial–blood pool interface **(Figs. 12A and B)**. FAC is affected not only by shortening of longitudinal fibers but also by apical, basal, and radial functions; however, it is prone to operator-dependent variations. Normal values range approximately 25–45%.[14-16]

$$\text{FAC} = [(\text{RV area diastole} - \text{RV area systole})/\text{RV area diastole}] \times 100$$

- *Tricuspid annular plane systolic excursion (TAPSE)* is another measure of RV systolic function. It measures longitudinal fiber shortening and is obtained from the four-chamber view using the M-mode with the cursor aligned along the direction of the lateral annulus **(Fig. 12C)**. In a term neonate, the mean value to TAPSE is 1.03 cm (Z-score ±2: 0.85–1.21 cm), while in a preterm neonate of gestation 26 weeks, the mean is 0.44 cm (Z-score ±2: 0.30–0.59 cm).[17] It has been found to increase linearly with advancing gestation. A value of <4 mm in a late preterm or a term neonate is predictive of severe PPHN and need for extracorporeal membrane oxygenation.[15]

Global Cardiac Function

Myocardial performance index (MPI) also known as Tei index is a measure of combined systolic and diastolic function. The higher the value of myocardial performance index, the worse is the cardiac function. As it is an indicator of global cardiac dysfunction, any abnormal value should be investigated further to determine whether systolic or diastolic components (or both) are abnormal. Postnatal changes correspond to changes in ventricular function reflecting the hemodynamic changes of transitional circulation **(Table 3)**.



Figs. 12A to C: (A) Right ventricular end-diastolic area (EDA); (B) Right ventricular end-systolic area (ESA); (C) Tricuspid annular plane systolic excursion (TAPSE) in a preterm baby of gestation 32 weeks.

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TABLE 3: Values of left ventricle myocardial performance index.[18]

	Day-1	*Day-28*
Preterm	0.44 ± 0.15	0.21 ± 0.08
Term	0.42 ± 0.14	0.22 ± 0.09

For LV MPI, in standard apical five-chamber view, pulse wave Doppler sample gate is placed at the LV outflow tract and the following parameters are calculated—isovolumic contraction time (IVCT) as time between closure of mitral valve and opening of aortic valve, IVRT as the time between the closure of aortic valve and opening of mitral valve and ejection time (ET) as time between opening and closure of aortic valve. MPI is calculated as MPI = IVRT + IVCT/ET. This is further explained under the section of tissue Doppler. For RV MPI, the ET is calculated in PLAX view by placing PW Doppler at the tip of pulmonary artery and the total cycle time is calculated from the apical view by placing Doppler gate at the tricuspid inflow. The sum of IVCT and IVRT are calculated by subtracting ET from total cycle time.

Measurement of Systemic Blood Flow

For measuring blood flow through any vessel, velocity of the blood flow and cross-sectional area of the vessel is required. Pulsed wave Doppler allows for measurement of velocity at one particular point of time. While measuring, the angle of insonation should be as parallel as possible to the direction of blood flow (close to zero). An angle <20° is acceptable. It assumes that the vessel is cylindrical in shape and so its cross-section is a circle. Therefore, the area of cross-section can be calculated using the formula $\pi D^2/4$ (D = diameter). Velocity time integral (VTI) is the area under the curve of Doppler envelope for each cardiac cycle. Using these parameters LV and RV outputs and superior vena cava flow can be estimated.

- *Left ventricular output (LVO):* In the absence of PDA, LVO equals systemic blood flow. An apical five-chamber view is obtained with pulse wave Doppler at the root of aortic valve, the area under one stroke of aortic pulse is VTI **(Figs. 4A to C)**. Next obtain parasternal long-axis view and diameter (D) of the aorta is calculated at the hinge of aortic valves **(Figs. 2A to C)**. The main reason for variability of output measurements is differences in calculating the aortic root diameter. It should be measured when the valve is open in systole. The value is divided by birth weight to get output in mL/kg/min.

$$\text{LVO} = \text{heart rate/min} \times \text{VTI} \times \pi D^2/4/\text{Weight (Kg)}$$

- *Right ventricular output (RVO):* Principal and formula are similar to that of LVO estimation. In presence of ductus, RVO may be a better indicator of perfusion status. In the absence of a significant flow through the patent

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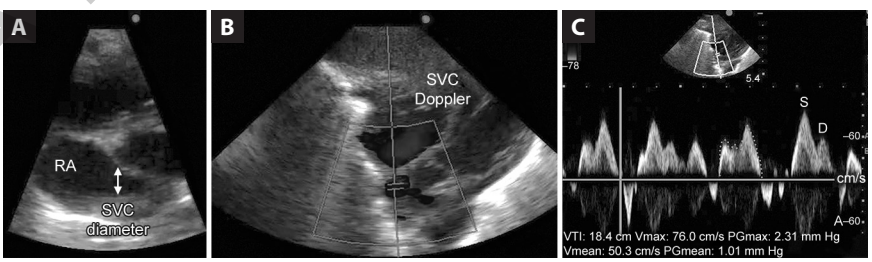
foramen ovale, systemic output is equal to the blood that returns to the right side of heart and exits through the pulmonary artery. As there is difficulty in visualizing the pulmonary hinge points, there is wide variability in the value of RVO, so serial values would be a more reliable indicator rather than a single value. One should obtain a parasternal long-axis view and tilt the probe toward left shoulder to view the pulmonary valve and artery. The diameter (D) of the pulmonary artery is obtained at the hinge points of the pulmonary valve. Next for calculating the VTI, the pulse wave Doppler sample gate is placed at the tips of pulmonary valve and pulmonary artery stroke is traced in the same way as for aortic flow **(Figs. 5A and B)**. Normal values range between 150 and 300 mL/kg/min.

$$\text{RVO} = \text{heart rate/min} \times \text{VTI} \times \pi D^2/4/\text{Weight (Kg)}$$

- *Superior vena cava (SVC) flow:* In presence of shunting at level of patent foramen ovale (PFO) and PDA, both LVO and RVO are not reliable. SVC flow is a measure of cerebral and systemic blood flow.[19] For calculating SVC diameter, place the probe in sagittal plane at left high parasternal position and tilt the probe as if trying to peep behind the sternum. SVC can be seen entering into right atrium, M-mode is placed at the point where SVC opens into right atrium. Cyclical variation with respiration is present, so an average of maximum and minimum diameters is taken. To measure the VTI of SVC flow, obtain subcostal four-chamber view and then angulate the beam anteriorly. SVC can be seen entering into the right atrium. Place the pulse wave Doppler sample at the junction of SVC and RA and record the triphasic SVC flow **(Figs. 13A to C)**. Normal range of SVC flow is between 40 and 160 mL/kg/min. Studies have reported high intra- and interobserver variability and poor correlation with MRI assessment. The main limitation of SVC flow and cardiac output measurements is that they are not a true measure of myocardial function.

$$\text{SVC flow} = \text{heart rate} \times \text{VTI} \times \pi D^2/4/\text{Weight (Kg)}$$

Some of the important cardiac measurements of systemic blood flow and LV function are shown in **Table 4**.



Figs. 13A to C: (A) Calculation of SVC diameter; (B) SVC Doppler; (C) Triphasic Doppler flow pattern of SVC. (A: atrial contraction; D: diastole; RA: right atrium; S: systole; SVC: superior vena cava) *(For color version, see Plate 7)*

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TABLE 4: Common cardiac measurements in neonates.

Parameter	*View*	*Application*	*Normal range*
Left ventricular output = Heart rate/min × VTI × $\pi D^2/4$/ Weight (Kg)	Apical five-chamber view (VTI) and parasternal long-axis view for diameter	Estimates systemic blood flow	150–300 mL/kg/min
Right ventricular output = Heart rate/min × VTI × $\pi D^2/4$/ Weight (Kg)	Parasternal long-axis view (diameter and VTI)	Estimates pulmonary blood flow	150–300 mL/kg/min
Superior vena cava flow = Heart rate/min × VTI × $\pi D^2/4$/ Weight (Kg)	High parasternal position (diameter) and subcostal view (VTI)	Estimates systemic blood flow to upper part of body	40–160 mL/kg/min
Shortening fraction = [(LVEDD – LVESD)/LVEDD] × 100	Parasternal long-axis view (LVESD and LVEDD)	Myocardial contractility	27–42%
Ejection fraction = [(LVEDD - LVESD)/LVEDD] × 100	Simpson's biplanar method—apical four chamber or two-chamber view (LVESV and LVEDV)	Myocardial contractility and left ventricular systolic function	55–70%
Velocity of circumferential fiber shortening = [(LVEDD - LVESD)/LVEDD] × LVET	Parasternal long-axis view (LVESD and LVEDD) apical five-chamber view (LVET)	Myocardial contractility and ventricular systolic function	1.54 ± 0.04 circumference/ second

(LVEDV: left ventricle end-diastolic volume; LVESD: left ventricle end-systolic diameter; LVESV: left ventricle end-systolic volume; LVET: left ventricle ejection time; VTI: velocity time integral)

Pulmonary Hemodynamics and Assessment in Pulmonary Artery Hypertension

Persistent pulmonary hypertension (PPHN) occurs either due to maladaptation, maldevelopment or under development of pulmonary vasculature. The hallmark of PPHN is increased pulmonary vascular resistance (PVR) associated with shunting of deoxygenated blood from the pulmonary to the systemic circulation with resultant hypoxemia. PPHN is a clinical diagnosis and a comprehensive echocardiographic assessment is

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needed to evaluate for pulmonary artery hypertension (PAH) and to rule out any cyanotic congenital heart defect.

Severe PPHN with increased PVR results in right to left shunt at the ductal/atrial level. Increase in RV pressure may lead to tricuspid regurgitation (TR). Increase afterload leads to decreased pulmonary blood flow and right heart failure. There could be dilatation of RV and RA. Increased RV systolic pressure and RV dilatation lead to deviation of interventricular septum to left compressing the left ventricle and affecting both its contractility and preload. Cardiac functions may also be affected due to severe hypoxemia. Echocardiography is performed to assess pulmonary artery pressure, evaluate markers of pulmonary vascular resistance, ventricular function and to assess direction of shunt.

Fn echo markers commonly assessed in PAH are shown in **Table 5**.

- *Measurement of TR jet:* Pulmonary arterial pressure can be estimated by calculating the TR peak velocity and applying it in the modified Bernoulli's equation ($P = 4\ v^2$).

$$SPAP = RVSP = 4 \times (V_{max}\ TR^2) + RAP$$

[Where, SPAP = systolic pulmonary artery pressure, RVSP = right ventricular systolic pressure, V_{max} = peak velocity of tricuspid regurgitation, RAP = right atrial pressure (3–5 mm Hg)]

An apical four-chamber view is obtained with color flow across the tricuspid valve. Presence of TR will be identified by a blue color turbulent

TABLE 5: Common functional echocardiography markers in pulmonary hypertension.[20]

Pulmonary artery pressure	*Pulmonary vascular resistance*	*Right ventricular function*	*Assessment of shunts and their direction*
• Tricuspid regurgitation velocity • Peak velocity of right to left transductal shunt • Configuration of interventricular septum • Left ventricle systolic eccentricity index	• Right ventricular systolic time intervals • Time to peak velocity • Time to peak velocity/ Right ventricle ejection time	• Tricuspid annular plane systolic excursion • RV myocardial performance index • Fractional area change	• Transductal shunting • Interatrial shunting

(RV: right ventricle)

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jet across the valve during systole **(Fig. 14A)**. After identifying the TR jet, a continuous wave Doppler is placed with cursor aligned along the direction of the jet. Failure to align Doppler correctly will result in underestimation of pressure gradient. An optimal quality TR jet shows a well-demarcated envelope. TR jet velocity may be low in case of severe PAH when severe right ventricular dysfunction is present.

- *Transductal right-to-left blood flow peak velocity:* In the presence of ductus arteriosus, increased pulmonary pressure leads to pure right to left shunt or bidirectional shunt across the ductus. One can estimate the pulmonary artery pressure by measuring the transductal right-to-left blood flow if it lasts ≥30% of the cardiac cycle. Doppler gate is placed across the pulmonary end of the ductus and a trace of flow velocity is obtained. The same principle is applied and SPAP can be estimated by measuring the peak velocity of right to left shunt Doppler flow using the formula below:

$$SPAP = 4 \times (V_{max}\, DA^2) + SSAP$$

[Where, SPAP = systolic pulmonary artery pressure, V_{max} DA = peak velocity ductal right-to-left shunt, and SSAP = systolic systemic arterial pressure]

However, this is not reliable.

- *IVS configuration:* The alignment of the IVS serves as the basis for a different, albeit more subjective, estimate of PAP. The septum normally bends into the right ventricle (O-shaped LV). However, when RV pressure increases, the IVS flattens (D-shaped LV) **(Fig. 14B)** and, finally, curves into the LV (crescent-shaped LV).[21] The configuration and relation of LV and RV pressure is shown in **Table 6**.
- *Left ventricular systolic eccentric index (LV-sEI):* It is the ratio of the left ventricular dimension parallel and perpendicular to the septum. Parasternal short-axis view is obtained and left ventricular dimensions are measured. D1 is the left ventricle short-axis diameter parallel to the septum and D2 is the left ventricle short-axis diameter perpendicular to the septum. LV-sEI is defined as the ratio of D1/D2 **(Fig. 14C)**. The normal value is ≤1.3 and this increases in neonates with PPHN because with increase in right-sided pressures, the IVS will eventually curve into the left ventricle causing the diameter perpendicular to the septum (D2) to decrease and the value of LV-sEI to increase.
- *Right ventricular systolic time intervals:* The timing and duration of RV systolic event change with increasing pulmonary artery pressure. A parasternal short-axis view can be obtained and pulse wave Doppler is applied at the level of pulmonary valves in RV outflow tract. Time to peak velocity (TPV) also called pulmonary artery acceleration time and right ventricular ejection time (RVET) values are calculated from the Doppler

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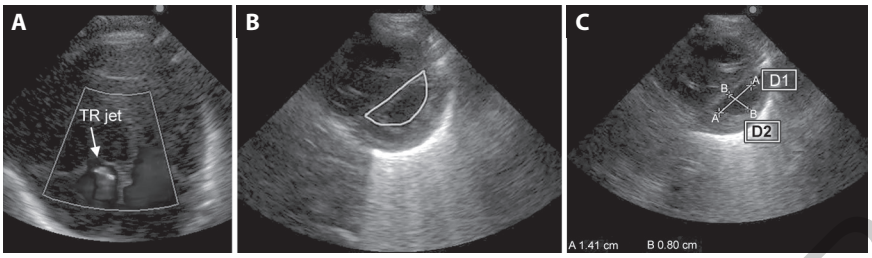

Figs. 14A to C: (A) Tricuspid regurgitation jet in four-chamber view (inverted); (B) D-shaped left ventricle cavity; (C) Left ventricular systolic eccentricity index calculation = D1/D2. (D1: left ventricular dimension parallel to IVS; D2: left ventricular dimension perpendicular to IVS; LV: left ventricle; IVS: interventricular septum) *(For color version, see Plate 7)*

TABLE 6: Estimation of right ventricular pressure (RVP) based on left ventricular configuration.

Left ventricular configuration	*Estimated RVP*
O-shaped	<50% of LVP
D-shaped	50–100% of LVP
Crescent shaped	>100% of LVP

(LVP: left ventricle pressure; RVP: right ventricle pressure)

sample **(Fig. 15A)**. In PPHN, TPV of pulmonary flow shortens and a value <90 ms indicates increased pulmonary artery pressure and a value <40 ms indicates severe PPHN. This measurement is expressed as a ratio to total RVET. Normal TPV/RVET ration is 0.38–0.40. In PPHN, the ratio is <0.23.[22] Sometimes, the Doppler wave form in PPHN may also show in mid-systolic notch or a flying W as seen in **Figure 15A**.[23]

- *Right ventricular function:* Parameters such as TAPSE, FAC, RVO, and RV MPI can be used to assess the RV function. These have been already described before.
- *Direction of shunts:* Direction and flow of blood through ductus depend upon the balance in pressure between aorta and pulmonary artery. A pure right to left (R–L) shunt or a R–L flow duration of >30% of cycle duration **(Fig. 15B)** suggests elevated pulmonary artery pressures. Closed or tiny ductus makes this method useless in estimation of PA pressure. Similarly, a bidirectional atrial shunt is found in PPHN. A pure R–L shunt at atrial level indicates TAPVC unless proved otherwise. In PPHN, a L–R shunt is possible at the atrial level, since the diastolic pulmonary pressure is usually subsystemic with suprasystemic systolic pulmonary pressure.

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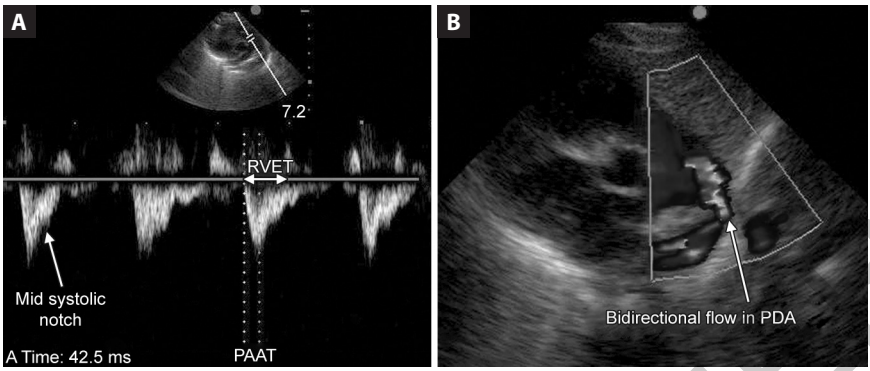

Figs. 15A and B: (A) Shortened PAAT and presence of mid systolic notch; (B) Bidirectional shunt in PDA. (PAAT: pulmonary artery acceleration time; PDA: patent ductus arteriosus; RVET: right ventricular ejection time) *(For color version, see Plate 8)*

Assessment of Hemodynamic Significance of Patent Ductus Arteriosus

The effect of PDA depends on the volume and direction of flow across the duct. During neonatal transition as the pulmonary pressures come down, there is a reversal in direction of shunt from R–L or bi-directional to L–R. A large PDA shunting L–R can result in pulmonary overcirculation and systemic hypoperfusion. In such a scenario, Fn echo can help to assess hemodynamic significance. In the presence of a hsPDA diagnosed on Fn echo, it is essential to confirm that structurally heart is normal and avoid treatment of ductus in presence of any duct dependent lesion.

The ductus can be evaluated under the parameters as mentioned in **Table 7**. These are further discussed below.

Ductal Parameters

- *Size of ductus:* To visualize the duct, a high parasternal short-axis view is obtained, with slight anticlockwise rotation and caudal tilt (beam toward sternal notch). In 2D mode, we can see the cross-section of the aorta, right pulmonary artery wrapping around it, left pulmonary artery and ductus (three leg stool appearance) **(Figs. 16A to C)**. Duct diameter should be measured in 2D mode at the pulmonary end by a frame-by-frame analysis at the smallest dimension that is at the point of maximum constriction. Transductal diameter is an important parameter in evaluating whether ductus is hemodynamically significant or not. Placement of calliper is important to decrease error. The diameter of the duct can be expressed as an absolute value in mm (smallest cut-off 1.5 mm) or indexed either to the diameter of the LPA (smallest cutoff 0.5) or to the patient's weight (cut-off 1.4 mm/kg).

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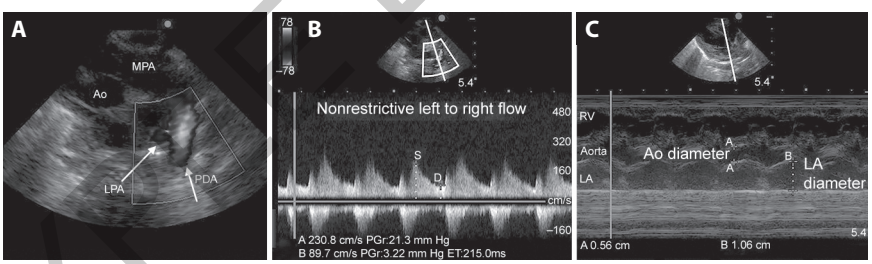
TABLE 7: Assessment of hemodynamic significance of ductus arteriosus.[24]

Characteristics of ductal dimension and flow	*Parameters of pulmonary overcirculation*	*Parameters of systemic steal*
• *Size:* Diameter (mm)/ comparison to left pulmonary artery • *Direction of shunt:* (Left to right, bidirectional or right to left) • Doppler of the ductus to assess velocity in systole and diastole (m/s) and gradient	• *Volume overload of left heart:* – Diastolic velocity in main pulmonary artery and left pulmonary artery (m/s) – Pulmonary vein D wave velocity (m/s) – Left atrium/aortic diameter ratio – Left ventricle end-diastolic diameter (mm) – Increased left ventricle output • Pressure overload of left heart – Mitral E/A ratio – Isovolumic relaxation time	• Diastolic flow in systemic arteries (postductal) – Descending aorta – Celiac artery – Middle cerebral artery



Figs. 16A to C: (A) Ductal view showing left to right shunt with laminar flow; (B) hsPDA with pulsatile flow pattern (ratio of V_{max}:V_{min} is >2); (C) LA/Ao ratio >1.5 suggestive of hsPDA. (Ao: aorta; LA: left atrium; LPA: left pulmonary artery; MPA: main pulmonary artery; PDA: patent ductus arteriosus; RV: right ventricle) *(For color version, see Plate 8)*

- *Direction of shunt:* Color Doppler can be used to know about the direction of ductal flow. A L–R shunt is indicated by red jet and R–L shunt is indicated by blue jet. Obtain a Doppler trace through the ductus to see the flow pattern which can be pure R–L, pure L–R, or bidirectional flow. Presence of pure R–L shunt or bidirectional shunt with R–L >30% of blood flow can indicate PPHN. Such a scenario may indicate pulmonary hypertension or a duct dependent cardiac lesion and in these situations ductal closure

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should not be attempted. A duct that is purely L–R with smooth red color signifying laminar flow is more likely to benefit from treatment.

- *Ductal velocity:* Duct can either be restrictive/nonrestrictive based on the velocity of flow through it. In a closing duct (restrictive type), the velocity is high throughout the cardiac cycle (V_{max} >1.5-2 m/s) and the ratio of V_{max}:V_{min} is <2. A pulsatile pattern of duct is in which the ratio of V_{max}:V_{min} is >2. Such ducts are nonrestrictive and may need treatment depending on the clinical scenario and presence of other markers of hsPDA.

Parameters of Pulmonary Overcirculation

- *Volume overload of left heart:*
  - *Diastolic velocity in main pulmonary artery and left pulmonary artery (m/s):* The main pulmonary artery is visualized in modified PLAX view. In absence of a PDA, on placing a pulsed wave Doppler beyond the pulmonary valve, normal pulmonary artery flow is seen as normal forward systolic flow with no or minimal flow or turbulence in diastole. In the presence of PDA, blood rushes back from descending aorta to MPA during diastole creating a turbulence. Broadly speaking a diastolic flow velocity of >0.5 m/s in MPA may indicate the hsPDA. Similarly, the LPA can be evaluated for turbulence. It has been found that both a mean and end-diastolic velocity of 0.42 m/s and 0.2 m/s correlate with a high transductal flow.
  - *Pulmonary vein D wave velocity (m/s):* In the presence of a hsPDA increased blood flow though the MPA is guided to the left atrium by the pulmonary veins. This increased blood flow can be demonstrated by interrogating a pulmonary venous Doppler. The pulmonary veins are demonstrated in a high parasternal view with pointer toward 2–3 o'clock position and dorsal tilt (crab view). A pulse waved Doppler can be put in the lower pulmonary veins and a triphasic flow pattern with a systolic (S-wave), diastolic (D-wave), and atrial contraction (A-wave) can be obtained. Broadly speaking, a D-wave peak velocity of >0.5 m/s may indicate a moderate shunt **(Fig. 17A)**.
  - *Left atrium to aortic ratio (LA/Ao), Left ventricle end-diastolic diameter (mm) and increased LVO:* The hsPDA causes volume overload of left heart, leading to enlargement of LA and LV. This enlargement is quantified using the LA/Ao ratio. One can obtain a standard parasternal long-axis view with M-mode cursor aligned along the root of aortic valve. Aortic root diameter (Ao) is measured at the hinge of aortic valves and LA dimension is measured at the end of diastole **(Fig. 16C)**. A value >1.5 is taken as a cut-off of hsPDA and a ratio of >2 is suggestive of moderate to large shunt. Using z-scores for LVEDD, the effect of volume loading on the LV can be evaluated over time. In

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presence of hsPDA with dehydration/large L–R shunt at the foramen ovale may offload the left side of heart and can give rise to low or normal LA/Ao ratio or LVEDD.

A large L–R shunt across the PDA can lead to increase in LVO to >300 mL/kg/min. A ratio of LVO and superior vena cava (SVC) flow (representing upper body blood flow) (LVO/SVC ratio) has been suggested as one of the markers for severity of ductal flow. Broadly speaking the LVO/SVC ratio ≥ 4 would suggest the hsPDA.

- *Pressure overload of left heart*
  - *Transmitral Doppler flow (E/A ratio):* One can interrogate the mitral inflow by obtaining a pulse wave Doppler across the mitral valve. As discussed in the section of diastolic function, in the presence of a significant PDA, there will be increase in the passive transmitral flow due to increased LA pressure leading to change of E/A ratio to >1 similar to term neonates. This is called pseudo-normalization of E/A ratio **(Fig. 11)**.
  - *Isovolumic relaxation time:* It is the phase of the cardiac cycle from closure of aortic valve after LV ejection is over till opening of mitral valve, which allows blood to flow from atria to ventricle in diastole. It can be measured in an apical five-chamber view with Doppler sample kept between mitral inflow and aortic outflow streams. Doppler sample will include both transmitral flow (E- and A-wave) and aortic flow (negative deflection). The time between the end of aortic stroke (negative deflection) and the subsequent mitral inflow wave (positive deflection) is called IVRT. It decreases in presence of hsPDA **(Fig. 11)** due to earlier opening of mitral valve due to high left atrial pressure.

Abnormal Diastolic Flow in Systemic Arteries

Normal diastolic flow in the postductal descending aorta is antegrade. Although the PDA shunts blood away from the systemic circulation throughout the cardiac cycle, ductal steal is more apparent during diastole. To visualize this, one should obtain an arch view (high parasternal view) and pulse wave Doppler gate is placed on the descending aorta beyond the opening of the duct. In case of a hemodynamically significant duct, these diastolic flows progressively become absent and then retrograde. These patterns can be also seen if a sagittal abdominal view is obtained with pointer toward the head and if a pulse wave Doppler is placed in the celiac trunk and the superior mesenteric artery. Other vessels such as cerebral arteries and renal arteries can also be interrogated to assess for ductal steal during diastole **(Figs. 17B and C)**.

Table 8 depicts the cut-off values of various parameters for an hsPDA.

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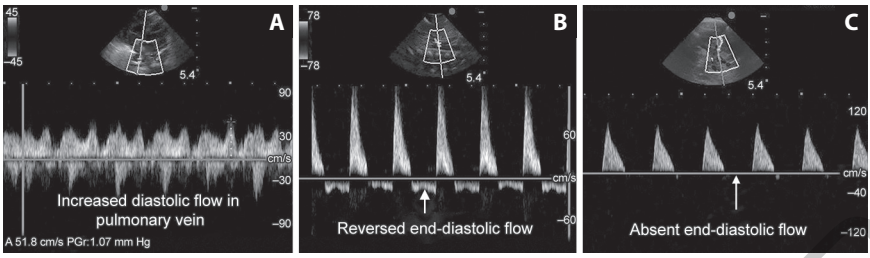

Figs. 17A to C: (A) Diastolic velocity >0.5 m/s in pulmonary vein; (B) Reverse end-diastolic flow in coeliac trunk; (C) Absent end diastolic flow in middle cerebral artery. *(For color version, see Plate 8)*

TABLE 8: Assessment of patent ductus arteriosus.[25]

Echocardiography indices	*Small*	*Moderate*	*Large*
Diameter (mm)	<1.5	1.5–2	≥2
PDA:LPA ratio	<0.5	0.5–1	≥1
Transductal peak velocity (m/s)	>2	1.5–2	<1.5
Transductal systolic to diastolic velocity gradient	<2	2–4	>4
LA:Ao ratio	<1.5	1.5–2	>2
Mitral valve E/A ratio	<1	1	>1
IVRT (ms)	>40	30–40	<30
Diastolic flow pattern	Antegrade	Absent	Retrograde

(IVRT: isovolumic relaxation time; LA:Ao; left atrium/aortic; LPA: left pulmonary artery; PDA: patent ductus arteriosus)

Tissue Doppler Imaging

Tissue Doppler imaging (TDI) is a modality that helps to assess myocardial function. It measures velocity of movement of tissues during various phases of cardiac cycle. The measurements of movement of myocardium are captured as low velocity Doppler signals, while high-velocity signals of blood flow are filtered out. During systole, the myocardial fibers move from base toward the cardiac apex, while the reverse occurs in diastole. This longitudinal myocardial motion velocity can be measured at the annulus of mitral and tricuspid valve as well as the base of interventricular septum. The ventricular systolic function is assessed on TDI by measuring the maximum systolic velocity (S′). Assessment of diastolic function is done by measuring early diastolic velocity (E′) and late diastolic velocity (A′). Another parameter that can be measured using TDI trace is myocardial performance index (MPI) that assesses global cardiac function.

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Following are the common parameters assessed in pwTDI **(Fig. 18)**:

- *Peak systolic velocity (S′):* When atrioventricular (AV) valve contracts in the ventricular systolic phase and moves toward the echocardiography probe, a positive deflection of spectral curve is obtained. This is called systolic velocity curve and denotes the ventricle ejection phase. The maximum amplitude of systolic velocity curve is called peak systolic velocity (S′) and is measured in cm/s.
- *Early diastolic velocity (E′) and late diastolic velocity/atrial systolic velocity (A′):* When AV valve relaxes in the ventricular diastolic phase and the valve moves away from the echocardiography probe, two negative deflections of pwTDI curve is obtained. The first curve is called early diastolic velocity curve and denotes early filling phase of the ventricles due to mitral valve opening. The maximum amplitude of early diastolic velocity curve is called E′ and is measured in cm/s. The second curve formed in the ventricular diastolic phase is called as late diastolic velocity curve and denotes the late diastolic filling phase which coincides with atrial systolic phase. The maximum amplitude of the late diastolic velocity curve is called late diastolic velocity (A′) and is also measured in cm/s.
- *Peak diastolic velocity (D′):* Occasionally, a single negative/downstream velocity curve is formed in the ventricular diastolic phase due to increased heart rate leading to fusion of E′ and A′. In such cases, assessment of two separate waves is not possible and the velocity of the single negative diastolic downstream curve will be taken. The maximum amplitude of the diastolic velocity curve is called as D′ and is measured in cm/s.

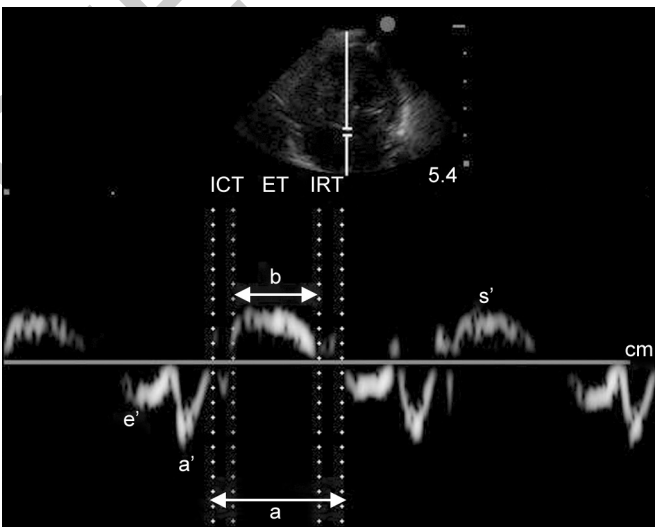

Fig. 18: Various parameters of tissue Doppler. (a′: atrial systolic velocity; e′: early diastolic velocity; ET: ejection time; ICT: isovolumetric contraction time; IRT: isovolumetric relaxation time; MPI: myocardial performance index (a-b/b); S′: peak systolic velocity)

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- *Myocardial performance index (MPI):* MPI incorporates both systolic and diastolic time intervals in expressing global systolic and diastolic ventricular function. MPI is the sum of isovolumetric phases as a fraction of the ejection phase [time interval of isovolumetric contraction (ICT) + isovolumetric relaxation time (IRT) divided by time interval in ejection phase (ET), usually assessed at the left lateral and right lateral hinge of AV-plane. ICT is taken as time period on the pwTDI velocity curve from end of systolic wave to beginning of diastolic wave, i.e., end of S′ till beginning of E′. ICT is time period from end of diastolic wave till beginning of systolic wave. ET is the time period in which the LV or RV ejects the blood to aorta or pulmonary artery during ventricular systole respectively. Both systolic and diastolic dysfunctions result in an abnormality in the myocardial relaxation, which prolongs IRT. Increase in MPI denotes poor cardiac function.

Normal values of commonly used TDI indicators of ventricular performance are not well-defined or sufficiently evaluated in prematurely born newborns. Studies have suggested TDI values at day 1 of life in term neonates LV S′: 5.7 ± 1.2 cm/sec, LV E′: 7.46 ± 1.4 cm/sec, LV A′ 7.45 ± 2.3 cm/sec, RV S′ 6.5 ± 1.4 cm/sec, RV E′ 6.4 ± 1.2 cm/sec, RV A′ 8.7 ± 1.3 cm/sec, LV MPI: 0.53 ± 0.1, and RV MPI: 0.46 ± 0.02 cm/sec. Similarly, TDI values of preterm neonates gestational age: 30–36 weeks at day 1 of life, LV S′: 4.3 ± 0.7 cm/sec, E′: 5.7 ± 1.4 cm/sec, A′: 6.0 ± 1.9 cm/sec, RV S′: 5.9 ± 0.9 cm/sec, E′: 6.2 ± 1 cm/sec, and A′: 7.7 ± 1.8 cm/sec.

CONCLUSION

Functional echocardiography, a clinician driven echocardiography done at the bedside, has grown to be an indispensable assessment tool in the NICU. It is crucial for obtaining detailed information on rapidly changing hemodynamics in neonates. In conjunction with a comprehensive clinical examination, Fn Echo can deliver quick, accurate, and real-time diagnostic information that can help in management of critically sick neonates. The most common indications are assessment of the cardiac functions, evaluation of the pulmonary pressures and determination of the hemodynamic changes in a patent ductus arteriosus.

KEY LEARNING POINTS

- Fn echo is clinician driven echocardiography done at the bedside to assess the cardiac functions, pulmonary pressures and assess hemodynamic changes in a patent ductus arteriosus.
- Fn echo can help clinicians to understand the ongoing hemodynamic change in a more precise fashion by providing real-time estimation of volume status, cardiac output, systolic and diastolic function.
- The IVC delivers the major portion of preload to the heart. The fluid status determines the IVC diameter and collapsibility.

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- Shortening fraction is the most commonly employed method of assessment of myocardial contractility.
- Left ventricular ejection fraction is another measure of LV systolic function. It estimates the proportion of LV volume ejected during each cardiac cycle.
- Fn echo is important for diagnosis of pulmonary hypertension to assess its severity and to exclude congenital heart defects.
- Fn echo helps to identify a significant PDA by assessing features of pulmonary over circulation and systemic steal.
- Tissue Doppler imaging is a modality that helps to assess myocardial function by measuring velocity of movement of tissues during various phases of cardiac cycle.
- Clinicians should be aware of caveats of Fn echo. All Fn echo studies should be preceded or followed at the earliest possible window by a comprehensive echocardiography done by a pediatric cardiologist.

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