Review Article

The left ventricle in well newborns versus those with perinatal asphyxia, haemodynamically significant ductus arteriosus or fetal growth restriction

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Abstract: Hemodynamic changes accompanying the initial breaths at the time of birth are especially important for a smooth transition of fetal to neonatal circulation. Understanding the normal transitional physiology and the clinical impact of adverse adaptation is important for delineating pathology so as to guide physiologically relevant therapies. Disorders such as severe perinatal asphyxia, hemodynamically significant patent ductus arteriosus (and its surgical ligation) and utero-placental insufficiency underlying fetal growth restriction, can adversely affect left ventricular (LV) function. The left ventricle is the predominant chamber involved in systemic perfusion during postnatal life. Cardiac output is closely linked to afterload; the latter is determined by arterial properties such as stiffness and compliance. This article outlines normal transition in term and preterm infants. It also highlights the adverse impact of three not uncommon neonatal disorders on LV function. Perinatal asphyxia leads to a reduced LV output, superior vena cava and coronary artery blood flow and an increase in the troponin level. Multiple haemodynamic changes are observed in the premature infant with a large patent ductus arteriosus. They need careful analysis to determine when ligation should proceed. Ligation itself generally results in a dramatic increase in afterload which may lead to a reduction in LV contractility and the need for ionotropic support. Fetal growth restricted infants have a higher systolic pressure, a somewhat hypertrophied heart arising from an increased arterial wall thickness/stiffness and systemic peripheral resistance. Point of care ultrasound (POCUS) helps differentiate normal transition and that resulting from neonatal disorders. It may be increasingly utilized in guiding management.

Keywords: Neonatal circulation; left ventricular function (LV function); point of care ultrasound (POCUS)

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Introduction

The circulatory changes at the time of birth are perhaps the most sudden and dramatic adaptations in human life. During fetal life, the inferior vena caval blood streams via the eustachian valve, from the right to the left atrium through the foramen ovale, effectively ending into the left ventricle (LV) with the most highly oxygenated blood arising from the placenta. The circulatory changes must happen rapidly and in an orderly fashion, to enable successful transition from intrauterine life to "independent" extra-uterine existence (1). The in-utero high pulmonary vascular resistance (PVR) ensures that the majority of right ventricular (RV) blood bypasses the lungs and flows through the patent ductus arteriosus (PDA) into the aorta. There is transition from a high-resistance/low-compliance to a low-resistance/high-compliance pulmonary circulation. Fetal circulation functions in-parallel as both the right and left sides of the heart provide systemic blood flow. The in-parallel is converted to an in-series circulation with rapid reduction and subsequent closure initially functional and subsequently permanent, of the fetal shunts, namely the patent foramen ovale and PDA, generally over the first few minutes/hours or hours/days respectively. *In-utero*, the PVR is much greater than the systemic vascular resistance (SVR). The combined cardiac output at about 30 weeks gestational age (GA) increases as the fetus adapts toward extra-uterine existence (2). The RV is metabolically more active during fetal life contracting against a high PVR and supporting the systemic blood flow via the PDA as compared to the LV which contracts against a lower afterload represented by the placenta. The transition in the circulatory system, including cardiac authority from right to left heart dominance, is mediated by a reduction in vasoconstrictor thromboxanes, a greater production of nitric oxide which lowers the PVR, increased vasodilatory prostaglandins and a surge in catecholamines and cortisol secretion (3,4). Common neonatal interventions such as ventilation and surfactant replacement therapy (SRT) tend to influence this transition (5). Events before, at the time of birth or soon thereafter have the potential to hamper this orderly transition.

The objectives of this article aimed for clinicians involved in the care of newborns, include a discussion of the:

(I) Normal transition in term and preterm infants;
(II) The influence of acute neonatal disease on LV function for example perinatal asphyxia in term infants, PDA and its surgical ligation in preterms;
(III) The influence of chronic disease on LV function—*in-utero* maladaptation such as in fetal growth restriction (FGR);
(IV) Echocardiographic (Echo) parameters to assess neonatal LV function and systemic arterial dynamics in the normal and affected newborns.

Neonatal transition in term and preterm infants

The transitional period involves rapidly evolving circulatory changes with impact on end-organ perfusion and function. Utilization of bedside clinician performed monitoring modality of point of care ultrasound (POCUS) is best suited to characterize the intricacies of the shift from RV to LV dominance. The transition from fetal circulation is facilitated by adequate alveolar aeration and recruitment, dilatation of pulmonary capillaries and constriction and eventual closure of shunts (foramen ovale and PDA), all aided by an adequate respiratory drive. The consequent changes in cardiac loading conditions and shunt-flow patterns may place the newborn, especially at extremes of prematurity, at risk of circulatory impairment. Pertinent to the LV, there is a significant increase in SVR and LV afterload once the umbilical cord is clamped, removing the placenta from the circulatory equation. LV contractility is influenced by variables of preload and afterload; with the inability of immature myocardium to increase contractility in response to increased preload more pronounced in the premature infant (*Frank Starling Law*); cardiac output increasing by a rise in heart rate. Myocardial performance is also impacted by ventricular interdependence (cross-talk between contractile fibres) and ventriculo-ventricular interactions (influence of changes in ventricular shape in response to pressure loading). A dilated-dysfunctional RV impinges on the LV relaxation capacity, restricting cardiac inflow and affecting contractility (6). Adequacy of LV preload is primarily influenced by pulmonary blood flow which in turn is affected by pulmonary distending pressures and affected by artificial ventilation strategies. We recently outlined the effects of spontaneous ventilation and various ventilator manoeuvres on LV pressures/function (5). SRT is a common neonatal intervention for preterm infants. The data on the influence of SRT on LV output have shown conflicting results. One study showed no change at two hours while the other noted a decrease in LV output following SRT administered in the delivery room (7,8). Both studies postulated that these changes were due to an increased left-to-right trans-atrial

flow. Evidently, more prospective data is needed to better characterize the impact of SRT on preterm circulation, and consequently cardiac function.

In summary, inherent neonatal issues such as prematurity and common neonatal disorders affect LV function which POCUS may allow for a better understanding and informed therapeutic choices.

Influence of acute neonatal disease on LV function

Perinatal asphyxia

Approximately 1–2 neonates/1,000 live births may be affected by severe perinatal asphyxia (9). It is characterized by significant disturbances in cardiovascular homeostasis and organ blood flow. Therapeutic hypothermia has been the standard of care for such infants (10). Recent data in neonates have highlighted the physiological interactions between 'disease' and therapeutic interventions. A recent study evaluated the relationship between LV function, LV perfusion [left anterior descending (LAD) coronary artery flow] and serum cardiac troponin (biochemical marker of myocardial injury) in infants with severe perinatal asphyxia (11). Echo data for fourteen infants with severe asphyxia were compared with 20 healthy term infants. Therapeutic hypothermia was initiated at a median of 1 hour, range or 1–5 hours. Echos in the asphyxiated subjects were done at a median of 7.7 hours, range of 3–10 hours. LV output and superior vena cava flow {median [range]} were significantly lower in the asphyxiated infants {108 [61–150] *vs.* 222 [156–321] mL/kg/min, P<0.001 and 34 [13–80] *vs.* 78 [60–99] mL/kg/min, P<0.001, respectively}. Serum cardiac troponin was high. Markedly low coronary flows were noted {median, 0.77 µg/L; range, 0.17–2.6). Coronary flow had a significantly positive correlation with LV output. Gebauer and colleagues also noted that in infants with severe asphyxia, the LV output was reduced to 67% of the post-hypothermic levels (9). Importantly, a dichotomy between blood pressure (BP) and flow parameters was noted, indicating the limitations of clinical assessments, and the importance of POCUS. The neonate's BP may only partially reflect the extent and profile of LV dysfunction due to the variation in SVR, necessitating the need for further haemodynamic evaluations (11). In essence, the LV bears the brunt in perinatal asphyxia; therapeutic choices for inotropes may be guided by the information provided by POCUS.

Patent ductus arteriosus

A hemodynamically significant PDA remains one of the most common cardiovascular problems in preterm infants, occurring in approximately 60% of infants <28 weeks GA (12). The assignment of hemodynamic significance (clinically or by echo) to a PDA remains a challenge for the neonatal clinician. The inability to accurately distinguish the PDA which needs treatment from the 'innocent' ductus arteriosus arises from the lack of scientific evidence of benefit or causality (13-15). The traditional definition of a PDA, which forms the basis of clinical trials conducted to date, is limited to the use of single parameters. Traditional echo markers such as ductal size and left atrial:aorta (LA:Ao) ratio do not predict neonatal outcomes (16,17). Ductal hemodynamics are influenced not only by ductal size but also by PVR and SVR and by the compensatory ability of the immature LV. We have proposed a ductal disease staging system to assess the cumulative effect of a hemodynamically significant PDA on LV performance as adjudicated by graded clinical and echo characteristics (15,18). A comprehensive appraisal of these echo markers to allow for a more meaningful and more systematic and structured approach for PDA evaluation. *Table 1* summarizes the range of cardiac parameters which may be used in such hemodynamic assessments though not all can or need be performed by the busy clinician (19). *Table 2* highlights those parameters that may be most helpful (19).

Left heart function characterization in PDA

The quantifications of left heart size and function (systolic and diastolic) are important indirect measures of the pressure and volume loading effects of increased pulmonary blood flow. The LA:Ao ratio is a well-recognized surrogate of ductal significance. It has been suggested that the rate of ductal misclassification is the lowest with LA:Ao ratio >1.4 (20). A ratio <1.4 may also represent a smaller shunt. Similarly, the ratio of left ventricular: atria diameter ratio, with the LV measured as an end diastolic dimension form a parasternal long axis view, has also been previously proposed as a surrogate marker; a value of >2.1 providing the lowest misclassification rate (20). However, patient related factors such as hydration, LV performance, or trans-atrial shunting may lead to over or underestimation of these unidimensional measures. The trans-mitral Doppler seems a useful marker of LA pressure/volume loading. A low E/A ratio amongst preterms reflects developmental immaturity of the myocardium, which is different from newly born

Table 1 Echocardiographic parameters available to evaluate systemic (cardiac and vascular) function

Component of function	Technique	View	Cursor position	Comments
Cardiac systolic				
Stroke volume	PWD + diameter of LVO	Apical 5-chamber	Aligned with the flow, sample just beyond aortic valve	Angle and cursor position dependent LVO = heart rate × stroke volume
FS	M-mode	Parasternal long axis	Distal to mitral valve leaflet tips at end-diastole	(LVEDD – LVESD)/LVEDD
Myocardial performance index	PWD	Apical 4 & 5-chamber	Trans-mitral and apical 5-chamber	(IVCT + IVRT)/LVET
mVCFc	2D/M-mode	Combination of 1 and 2	Combination of above two	FS/LVET
Fractional area change*	2D	Apical 4-chamber	Include full view of the left ventricle (base to apex)	[(LV 4-chamber area at end-diastole – LV 4-chamber area at end-systole)/LV 4-chamber area at end-diastole] ×100%
Cardiac inflow	2D & PWD	Apical 4-chamber	Use mitral cross-sectional area and trans-mitral Doppler VTI	Include both E and A waves in VTI measurement
Mitral annular plane systolic excursion	M mode	Apical 4-chamber	Lateral aspect of mitral annulus	Maintain vertical alignment with the apex
Myocardial deformation imaging: strain-fraction or % change from the original dimension; strain rate-deformation per unit time	Endocardial tracing over a single frame, followed by automatic tracking of endocardial borders	Apical 4-chamber	Not dependent on angle of insonation; assessment in multiple dimensions (segments)	LV is divided into six segments: basal septal, middle septal, apical septal, apical lateral, middle lateral and basal lateral
Cardiac diastolic				
Tissue Doppler	TDI PWD	Apical 4-chamber	Sample just below the lateral mitral annulus	Peak systolic (S'), early diastolic (E'), late diastolic (A') and peak IVV
E/A ratio, EDT	PWD	Apical 4-chamber	Aligned with the flow, sample at tips of mitral leaflets	E wave: early passive filling; A wave: late active filling
IVRT	PWD/CWD	Apical 5-chamber	PWD: sample volume placed within LVOT (in proximity to the anterior mitral leaflet to record both inflow and outflow signals) CWD beam at an intermediate position (between inflow and outflow) to record both velocities	From closure of the aortic valve to the opening of mitral valve
Vasomotor function				
LAD coronary artery flow	PWD	Parasternal short-axis view, moving the transducer down one or two intercostal spaces, rotating it clockwise, and angling superiorly	Placed over the LAD distal to the bifurcation	Identify using colour Doppler, set to a low Nyquist limit (15–30 cm/s) Internal dimensions measured at end-diastole on 2D
Stiffness index	M-mode	Longitudinal abdominal	Straight, non-branched 1-cm segment of the abdominal aorta	ln (systolic BP/diastolic BP)/[(AAOs – AAOd)/AAOd]
Input impedance	PWD	Apical 5-chamber	Aligned with the flow, sample just beyond aortic valve	Pulse pressure/peak flow
Systemic vascular resistance	PWD	Apical 5-chamber	Aligned with the flow, sample just beyond aortic valve	(Mean BP – right atrial pressure)/LVO (estimated right atrial pressure 5 mmHg)
Distensibility coefficient	2D	Transverse subcostal view	Abdominal aorta in the supra-celiac region	[(Asyst – Adiast)/Adiast]/pulse pressure
Whole body arterial compliance	2D and PWD	Parasternal long axis and apical 5-chamber	Aortic valve cusps for aortic cross section and aligned with the flow, sample just beyond aortic valve for stroke volume	Stroke volume/pulse pressure
Morphology				
Sphericity index	2D	Apical 4-chamber	End-diastolic 2D view	(Base to apex length)/basal diameter
LVMI	M-mode	Parasternal long axis	Distal to mitral valve leaflet tips at end-diastole	0.8 [1.04 (diastolic LV internal diameter + diastolic LV septal thickness + diastolic LV posterior wall thickness)^3 – (diastolic LV internal diameter)^3] +0.6
Inter-ventricular septal hypertrophy	M-mode	Parasternal long axis	Distal to mitral valve leaflet tips at end-diastole	Septal thickness indexed to LV posterior wall thickness in diastole
Relative left ventricular dilatation	M-mode	Parasternal long axis	Distal to mitral valve leaflet tips at end-diastole	Wall thickness relative to end-diastolic LV cavity dimension
aIMT	M-mode	Longitudinal abdominal	Straight, non-branched 1-cm segment of the abdominal aorta	Measured at end-diastole

*, more commonly used for right ventricle evaluation. PWD, pulse wave Doppler; LVO, left ventricular output; FS, fractional shortening; LVEDD, left ventricular end diastolic dimension; LVESD, left ventricular end systolic dimension; IVCT, iso-volumetric contraction time; IVRT, Iso-volumetric relaxation time; LVET, left ventricular ejection time; mVCFc, mean velocity of circumferential fibre shortening; 2D, two-dimension; LV, left ventricle; VTI, velocity time integral; TDI, tissue Doppler imaging; EDT, E wave deceleration time; IVV, isovolumic contraction velocities; CWD, continuous wave Doppler; LVOT, left ventricular outflow tract; LAD, left anterior descending artery; BP, blood pressure; AAOs, abdominal aorta dimension at end systole; AAOd, abdominal aorta dimension at end diastole; Asyst, abdominal aorta cross-sectional area at end systole; Adiast, abdominal aorta cross-sectional area at end diastole; LVMI, left ventricular mass index; aIMT, aortic intima media thickness.

Table 2 Summary of left ventricular function alterations in various neonatal disorders

Disorder	Echocardiographic parameters
Hemodynamically significant PDA	↑ LVO (normal 150–300 mL/kg/min)
	↑ Trans-mitral E/A ratio (0.5 at 24 weeks, 0.8 at term and 1.3 at 3 months of age)
	↑ LVO:SVC flow (no PDA =2.4±0.3 *vs.* significant PDA 4.5±0.6)
	↑ LV:Ao (no PDA =1.86±0.29 *vs.* significant PDA =2.27±0.37)
Surgical duct ligation	↓ LV contractility [↓FS (normal 25–45%)]
	↓ mVCFc (normal 2.7±0.5 circ/s)
	↓ LVO (normal 150–300 mL/kg/min)
Severe perinatal asphyxia	↓ LAD coronary artery flow (normal velocity time integral =1.6±0.8 cm)
	↓ LVO (normal 150–300 mL/kg/min)
	↑ Trans-mitral E/A ratio (0.5 at 24 weeks, 0.8 at term and 1.3 at 3 months of age)
	↓ LV longitudinal myocardial strain (21.45%±2.74%)
Fetal growth restriction	↓ LVO
	↓ MAPSE (0.36 cm at 26 weeks, 40 weeks 0.56 cm)
	↑ Myocardial performance index mitral [normal for term infant on day 2: median, 0.4 (interquartile range, 0.3–0.5)]
	↓ Tissue Doppler velocities mitral [normal for term infant on day 2: median, 6.1 cm/s (interquartile range, 5.5–6.3 cm/s)]
	↑ Trans-mitral E/A ratio (0.5 at 24 weeks, 0.8 at term and 1.3 at 3 months of age)

↑, increase; ↓, decrease; PDA, patent ductus arteriosus; LVO, left ventricular output; E/A, height of E wave/height of A wave of mitral inflow; LVO, left ventricular output; SVC, superior vena cava; LV, left ventricle; Ao, aorta; FS, fractional shortening; mVCFc, mean velocity of circumferential fibre shortening; LAD, left anterior descending coronary artery; MAPSE, mitral annular plane systolic excursion.

term infants where the E/A ratio is higher (>0.8). In infants with a hemodynamically significant PDA, an increase in passive trans-mitral flow due to a greater LA pressure leads to pseudo-normalization of the E/A ratio >1.0, resembling the normal term neonatal pattern (21). The isovolumic relaxation time reflects the time between closure of the mitral valve and opening of the aortic valve and decreases significantly in preterm infants with a hemodynamically significant PDA due to early pressure-related valve closure/opening (22). In keeping with increased pulmonary blood flow, the increased cardiac inflow leads to a higher LV output (accompanied by a higher LV output: superior vena cava flow ratio). Importantly, impairment of LV performance (lower LV output) in the presence of a hemodynamically significant PDA suggests a failing myocardium, which will lead to higher end-diastolic LA pressure and an increased the risk of pulmonary haemorrhage.

An international study performed serial evaluations at multiple time-points in the first week of life in preterm infants to identify the parameters with the best discriminatory ability (23). Among the various parameters, PDA diameter and flow velocity, LV output, and LV A' wave on the second day of life were independently associated with chronic lung disease (CLD)/death. In a similar study on preterm infants <32 weeks GA, an echo score (which included LV function parameters) was assigned on the day of therapy for hemodynamically significant PDA (24). Infants were then prospectively followed to ascertain the occurrence of CLD. Higher composite scores were associated with an increased subsequent risk of developing CLD. Together, these studies indicated merit in the use of early comprehensive echo examination which included LV parameters, to define the most suitable candidates where treatment is likely to improve neonatal outcomes. Our group has previously tabulated comprehensive echo assessments useful in the evaluation and assignment of hemodynamic significance to a PDA (13,15,18).

Surgical ligation of a hemodynamically significant PDA and the effects on LV performance

Failure of medical intervention or contraindication to medical therapy necessitates surgical intervention.

This scenario is often times complicated by systemic hypotension and oxygenation failure (21,25). The ability of the immature LV myocardium to compensate for the dramatic elevation in afterload has been the focus of recent research. McNamara *et al.* prospectively studied 46 preterm infants undergoing surgical duct ligation (25). Echos were done pre-operatively, and then 1, 8 and 24 hours after the procedure. Duct ligation was followed by an increased SVR, temporally coinciding with reduced LV contractility and output. Infants with pre-operatively impaired LV systolic performance were more likely to require inotropic support for a low systolic BP and abnormal base-deficit in the post-operative period, which are accompanied by cardiorespiratory deterioration, greater respiratory support requirements, pulmonary edema on chest radiographs, possibly related to an increased SVR and end-diastolic LA pressure. This scenario is termed 'Post-Ligation Cardiac Syndrome'. Multiple authors have documented that it may happen approximately 6–12 hours after surgery (26,27). Clinical/demographic parameters like age at surgery <28 days of age, weight at surgery <1,000 g, previous use of inotropes and low Apgar scores at birth, identified this high-risk sub-cohort, allowing for focussed physiologically-appropriate peri-operative care. Echocardiographically ductal size and pre-operative lower coronary perfusion identify this sub-cohort (21).

One of the features of a hemodynamically significant PDA is a wide pulse pressure (low diastolic BP). Since coronary artery perfusion depends on aortic diastolic BP, a low diastolic BP may compromise coronary perfusion in premature infants with a PDA, causing chronic myocardial ischemia (28). Previous data demonstrating ST-segment depression on the electrocardiogram and elevated plasma troponin that normalized after ductal closure in premature infants with hemodynamically significant PDA (29,30) support this hypothesis. A prospective study performed serial echos in 20 preterm infants requiring duct ligation to document systolic and diastolic LV function. Simultaneous coronary flow was evaluated by LAD diastolic Doppler flow, before and after (1, 8, and 24 hours) of surgical ligation (21). A low baseline LAD velocity time integral was associated with a lower systolic BP, low LV output and the post-operative need for inotropes. Lastly, the role of POCUS has been investigated in 'predicting' cardiorespiratory instability after PDA ligation. In a study of 62 preterm infants, a predictive model was developed based on the echo criteria (in *Table 1*). LV output <200 mL/kg/min at 1 hour after PDA ligation was noted to be a sensitive predictor of systemic hypotension and the need for inotropes (31). In essence, duct ligation is associated with major adaptive changes in systemic hemodynamics. The greater risk of clinical deterioration in infants with impaired pre-operative LV systolic performance further emphasizes the need to better characterize this critical peri-operative period to neonatal outcomes.

Influence of chronic disease on LV function: FGR

Cardiac maladaptation may occur in infants with FGR, secondary to utero-placental insufficiency. FGR is defined as a birthweight <10th centile for GA and sex with abnormal fetal Doppler recordings. It affects 21–30% of pregnancies delivered prematurely at <32 weeks GA (32-34). Amongst infants <32 weeks born in Australia and New Zealand in 2020, 668 (21%) were born <10th centile for GA and sex; approximately half (45%) amongst them were <3rd centile (severe FGR) (32). A decade of circulatory research done by our group has shed light on LV adaptation amongst infants with FGR. In a prospective echo study, we compared preterm infants 28–32 weeks GA with FGR with infants who were appropriate for GA. Preterm FGR infants had significantly greater inter-ventricular septal thickness and LV free wall thickening and a lower sphericity index (1.53±0.15 *vs.* 1.88±0.2, P<0.001), signifying globular and hypertrophied hearts. The diastolic and systolic dysfunction were characterized by a significantly raised trans-mitral E/A ratio and isovolumic relaxation time in the FGR cohort (0.84±0.05 *vs.* 0.78±0.03, P<0.001 and 61.4±4.1 *vs.* 53.2±3.2 ms, P<0.001, respectively). The rate corrected mean velocity of circumferential fibre shortening was reduced (1.93±0.4 *vs.* 2.77±0.5 circ/s, P<0.001) in the FGR cohort (35). Essentially, on assessments done in the initial postnatal weeks of life, preterm infants with FGR had altered cardiac function. FGR infants also had significantly higher systolic BP, aorta intima-media thickness, wall stiffness and peripheral resistance (36). Similar findings were noted in term FGR infants, in comparison with those whose weights were appropriate for GA infants. These included lower LV output, impaired LV relaxation, and thicker and stiffer systemic arteries (37,38).

Table 2 summarizes salient alterations in LV function in the aforementioned neonatal disorders. They may further complement the echo parameters used in the retrospective study of Vijayashankar *et al.* (39).

Echo parameters to assess LV function and systemic arterial dynamics

Although perinatal care has seen major advances, the diagnostic and therapeutic approach to cardiovascular dysfunction remains suboptimal owing to an overreliance on poorly predictive clinical parameters such as urine output, heart rate or capillary refill time. In the hands of appropriately trained frontline neonatal care providers, POCUS enables real-time evaluation of LV performance to characterize accurate acute physiology and aetiology of cardiovascular compromise, to guide physiologically-appropriate therapeutic decisions (18). The ability to monitor response to therapy is an added benefit. It is also a useful tool to determine the 'need to treat', differentiating normal transitional physiological changes from pathological ones. Its utility in improving clinical outcomes in newborns has been demonstrated (40,41). Echo assessments may provide greater insight into a rapidly evolving circulatory milieu in premature and term infants. *Table 1* summarizes the range of cardiac and vascular echo parameters which may be used in hemodynamic assessments in acute and chronic neonatal conditions (19,35-38). Few however have had the training, skill or time to carry out such a wide range of investigations suggesting the need to select those that may be most helpful as summarised in *Table 2* (19).

Afterload forces play a major role in determining cardiac performance, supporting the view that systemic arterial dynamics is a salient variable. Meta-analyses found arterial stiffness as an independent risk marker for future cardiovascular disease risk (42,43). Longitudinal appraisal between arterial stiffness and hypertension indicates a precursor role for arterial properties (44). Recent data in neonates have noted a similar value in evaluating arterial stiffness and distensibility which may provide indices of early vascular changes that predispose to the development of further vascular disease (36,37). These include arterial intima-media thickness, stiffness index and compliance, which have emerged as important predictors of coronary artery disease and cerebrovascular accident in adults (45-48). FGR is the archetypal disease process which involves primary vascular remodelling which may explain the increased predisposition to cardiovascular disease in adult life (49).

Conclusions

The transition from a dominant RV to LV can be hampered by a range of medical conditions in preterm and term infants. The skillset of POCUS has a seminal role in delineating physiologic detail to facilitate rational therapy. Timely and physiologically appropriate inotropic support, as guided by clinical decision making and aided by POCUS has the potential to improve outcomes. The assessment of the LV as the major contractile force in the postnatal period is facilitated by coalescing clinical and echo information. Assuming that there are adequate training facilities to develop the skills to carry out the range of echo investigations, further study is needed to clarify which parameters may be the most helpful in each clinical scenario as time remains a limited luxury in a busy nursery.

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Footnote

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