

My Approach to Echocardiographic Assessment of the Premature Newborn

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Abstract

Echocardiographic assessment of preterm infants, why do it?

The incidence of premature births has been increasing worldwide and already affects about 10% of live births. Extremely preterm newborns (PTNB) are at increased risk of developing pulmonary immaturity, leading to pulmonary hypertension (PH), in addition to cardiovascular immaturity and patent ductus arteriosus (PDA).

Hemodynamic monitoring of this population is challenging and differs from critically ill pediatric patients due to hemodynamic lability. Thus, minimal manipulation is essential to avoid complications. In this context, non-invasive monitoring methods such as transthoracic echocardiography (TTE) gain importance for hemodynamic assessment. It is a low-cost, radiation-free test that offers good anatomical and functional visualization in pediatric patients, making it a useful tool in the clinical management of severe preterm newborns.

What you will find in this article:

We will take an objective, didactic, and at times humorous approach to the use of echocardiography in premature newborns in the neonatal intensive care unit (NICU), and we will discuss the echocardiographic particularities of premature infants, especially regarding equipment adjustments, PH, and persistence of the ductus arteriosus (DA).

Introduction

The incidence of premature births has increased significantly in recent decades and already affects about

10% of live births worldwide.¹ The advent of surfactants, corticosteroids and better understanding of respiratory and hemodynamic management resulted in a reduction in morbidity and mortality in these patients, although these advances are not yet widely accessible across the country.

Extremely preterm newborns (PTNB) are at greater risk of presenting pulmonary immaturity with consequent alterations in alveolar development, affecting the pulmonary vasculature, in addition to patent ductus arteriosus (PDA). It is also known that prematurity alone is an independent risk factor for cardiac remodeling, which, associated with increased pulmonary vascular resistance, results in overload of the cardiovascular system.²

It is also worth noting that the hemodynamic monitoring of this population differs from that of severe adult and pediatric patients, due to several factors such as low weight (many patients weighing less than 1 kilogram), hemodynamic lability, reduced blood volume and immaturity of all systems, especially the respiratory system. In this context, minimal manipulation of the PTNB is essential. Many of the invasive monitoring devices used in other groups of critically ill patients cannot be used here, increasing the importance of non-invasive monitoring methods. Transthoracic echocardiography (TTE) is a low-cost exam, without radiation, with good anatomical and dynamic functional visualization in pediatric patients, which therefore comes to add a lot to the clinical management of severe PTNB.

In this article, a didactic and objective approach was taken to the echocardiogram in premature infants in the neonatal intensive care unit (NICU); there was no pretension to discuss all the parameters contained in the neonatal echocardiogram, but rather the particularities inherent to premature babies.

Echocardiogram in patients weighing less than 1Kg, now what?

Given the scenario of minimal manipulation of the PTNB in the NICU, before starting TTE itself, one should observe an overview of the conditions found and try to obtain as much information as possible so that the examination is brief and brings as much information as possible to the handling of the case.

- Is the patient using invasive ventilation? If non-invasive, is there positive airway pressure in this ventilation mode?
- Is there use of vasoactive drugs?
- Are catheters used? What position?
- Age in days?

Keywords

Premature Infant; Echocardiography; Neonatology; Pulmonary Hypertension; Ductus Arteriosus

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Manuscript received April 4, 2023; revised manuscript April 3, 2023; accepted April 5, 2023

Editor responsible for the review: Daniela do Carmo Rassi Frota

DOI: <https://doi.org/10.36660/abcimg.20230035i>

It seems obvious, but it is worth noting that these patients have a tenuous hemodynamic balance, so an increase in pressure on the inferior vena cava (IVC) when evaluating the subcostal plane with the transducer, for example, may be enough to collapse it, preventing return venous system and reducing ventricular preload. Thus, attention should be paid to the amount of force applied to the transducer on the abdomen and chest. PTNB are also more susceptible to hypothermia; therefore, it is essential to open as few incubator doors as possible and not forget to close them at the end of TTE.³

How to adjust the equipment for preterm echocardiography?

New echocardiogram software have made great advances in recent years, favoring better image quality in very low weight patients. Many devices come with specific presets for the neonatal or even premature population, however, some specific tips can help to improve the exam:

- Use transducer with the higher frequency as possible, therefore, the higher the frequency, the higher the frame rate. There are, ideally, 12 Mhz transducers (Figure 1), however, if this type of transducer is not available, try to use one with a minimum frequency of 8 Mhz;
- Turn off the ultrasound harmonics (small structures very close to the transducer evaluated with high frequency transducers do not need this feature);
- You can use the device's "penetration/resolution" adjustment feature; in this case, reduce penetration and increase resolution (frequency); and
- PTNB smaller than 1Kg commonly have a limited parasternal window (especially when ventilated with positive pressure); therefore, the use of the subcostal window with alternative planes is very useful.

What is special about the echocardiogram of the preterm newborn?

Considering the importance of "looking at the whole", mentioned earlier, it is possible to make an analogy with a hunting eagle: it is important to fly free and look at the general panorama (patient conditions and acquisition of general parameters of the echocardiogram), however, when it spots its prey, it focuses on calculations of speed of descent and precision of attack. Like an eagle hunting, the TTE of PTNB should focus on the peculiarities inherent to prematurity. Therefore, this article will not detail parameters such as evaluation of left ventricular systolic function, evaluation of pericardial effusion repercussions, evaluation of intracardiac masses or complex congenital heart diseases. These themes are better addressed in other references, we will focus on the peculiarities of PTNB.

Lung immaturity plays a crucial role in this pathophysiology. The lung undergoes pre- and postnatal injuries, altering the growth of the pulmonary vasculature, which in turn leads to increased afterload imposed on the right ventricle (RV), leading to pulmonary hypertension (PH). In extreme cases, there is overload



Figure 1 – High frequency transducer used to assess nearby structures (4-6cm) to the sternum, useful for evaluating low weight patients.

of the cardiovascular system with right heart failure and consequent worsening of the left cardiac output.⁴ Thus, a cautious assessment of the entire right heart system, starting with the systemic venous return, RV dimensions, and systolic function, the estimation of pulmonary vascular resistance and the interrelationship with the left ventricle (LV) is necessary.

Another relevant point is the evaluation of the PDA. Its prevalence in premature newborns is inversely proportional to gestational age, occurring in 20% of patients with gestational age greater than 32 weeks and in 80-90% of premature infants with extremely low birth weight (less than 1,000g) and gestational age of less than 26 weeks.⁵

How to assess PH in preterm infants?

First, the presence of congenital heart diseases must be excluded, except for patent foramen ovale (PFO), atrial septal defect (ASD) and PDA. Once this is done, the assessment of PH in preterm infants can begin. Here, it is possible to think of it as a system of closed connections and to segment the reasoning as follows:

- The increase in pulmonary pressure is directly proportional to the increase in pulmonary vascular resistance (either secondary to the immaturity of the pulmonary vascular bed, or due to persistence of the fetal pattern or other injuries) while the pulmonary cardiac output is maintained;
- There is RV pressure overload, which evolves with dilation and systolic dysfunction;
- RV dilation (when important) added to suprasystemic pulmonary pressure physically makes the RV insinuate against the LV, disturbing the systemic cardiac output

by reducing the LV filling volume, in addition to the lower return preload of the pulmonary veins;

- This whole process leads to an increase in RV end-diastolic pressure (RV DP₂) which means enlargement of the right atrium (RA), leading to venous congestion; and
- Evaluation of flow pattern across communications.

1. Quantifying is good! Is it always possible?

1.1 Pulmonary artery systolic pressure (PASP)

PASP measurement can be performed using tricuspid regurgitation (TR) Doppler. PASP can be estimated using the modified Bernoulli formula⁶ (Equation 1):

$$PASP = 4 \times VTR^2 + RAP \quad (1)$$

Where:

PASP: pulmonary artery systolic pressure (mmHg);

VTR: maximum velocity of tricuspid regurgitation (m/s); and

RAP: RA pressure (mmHg).

RAP is often not measured and can be considered as 3-5 mmHg³, except in cases of invasive mechanical ventilation with high pressure parameters or IVC dilation and reduced collapsibility, considering 7-10 mmHg.

However, it is not always possible to estimate the PASP, recommending aligning the cursor with the TR orifice, usually in the apical 4-chamber plane (Figure 2). One should pay attention to the quality of the Doppler curve, since the PASP is proportional to the maximum velocity squared. Thus, if there is an error in the measurement, its dimension will be exponentially underestimated.

If the neonate has PDA, PASP can be estimated by Doppler alignment of the DA through Equation 2:

$$PASP = SBP - 4 \times VAOPT^2 \quad (2)$$

Where

PASP: pulmonary artery systolic pressure (mmHg);

SBP: systolic blood pressure (mmHg); and

VAOPT: maximum flow velocity from the aorta (Ao) to the pulmonary trunk (PT) (m/s).

It is essential to compare the pulmonary pressure obtained with the patient's systemic systolic pressure and not just the absolute value. It is worth reporting if the pulmonary pressure obtained is less than 50% of the systemic pressure, greater than 50% of the systemic pressure, or greater than the systemic pressure (important PH).

1.2 Mean pulmonary artery pressure (MPAP)

The MPAP can be estimated using the pulmonary artery reflux (PR) curve, using Equation 3:^{6,7}

$$MPAP = 4 \times VPR^2 + PD2VD \quad (3)$$

Where

MPAP: mean pulmonary artery pressure (mmHg);

PRV: maximum pulmonary reflux velocity (m/s); and

RV DP₂: right ventricular end-diastolic pressure (mmHg).

1.3 Pulmonary vascular resistance: The pulmonary Doppler pattern

The Doppler pattern of the RV outflow tract can provide relevant information on the estimation of RV afterload. It is known that a shortening of the systolic acceleration time correlates with an increase in pulmonary pressure. Thus, a "symmetrical" flow pattern is expected during systole for normal pressures, a shortening of the systolic acceleration time as pulmonary pressure increases and, finally, a mid-systolic notch when there is suprasystemic PH⁸ (Figure 3). Analogously, the integral velocity over time (IVT) decreases proportionally with the increase in pulmonary vascular resistance.⁶

2. Brute force versus complacency: Dilation and dysfunction

2.1 The evaluation of the dimensions of the RV

The RV is a compliant cardiac chamber that evolves with hypertrophy and dilation in response to increased afterload. That said, one should actively look for signs of ventricular dilation in all planes. It is known that access to RV morphology is complex through the assessment of two-dimensional TTE,

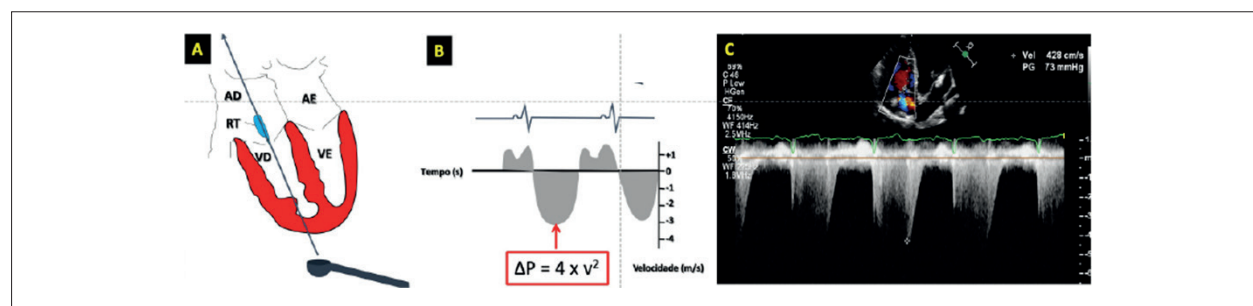


Figure 2 – A: Image showing how to align the cursor with the tricuspid regurgitation jet (TR) in the apical 4-chamber plane. B: TR Doppler. C: Tricuspid continuous Doppler flow pattern. RA: right atrium; LA: left atrium; RV: right ventricle; LV: left ventricle; TR: Tricuspid regurgitation; P: pressure (mmHg); v: flow velocity (m/s).

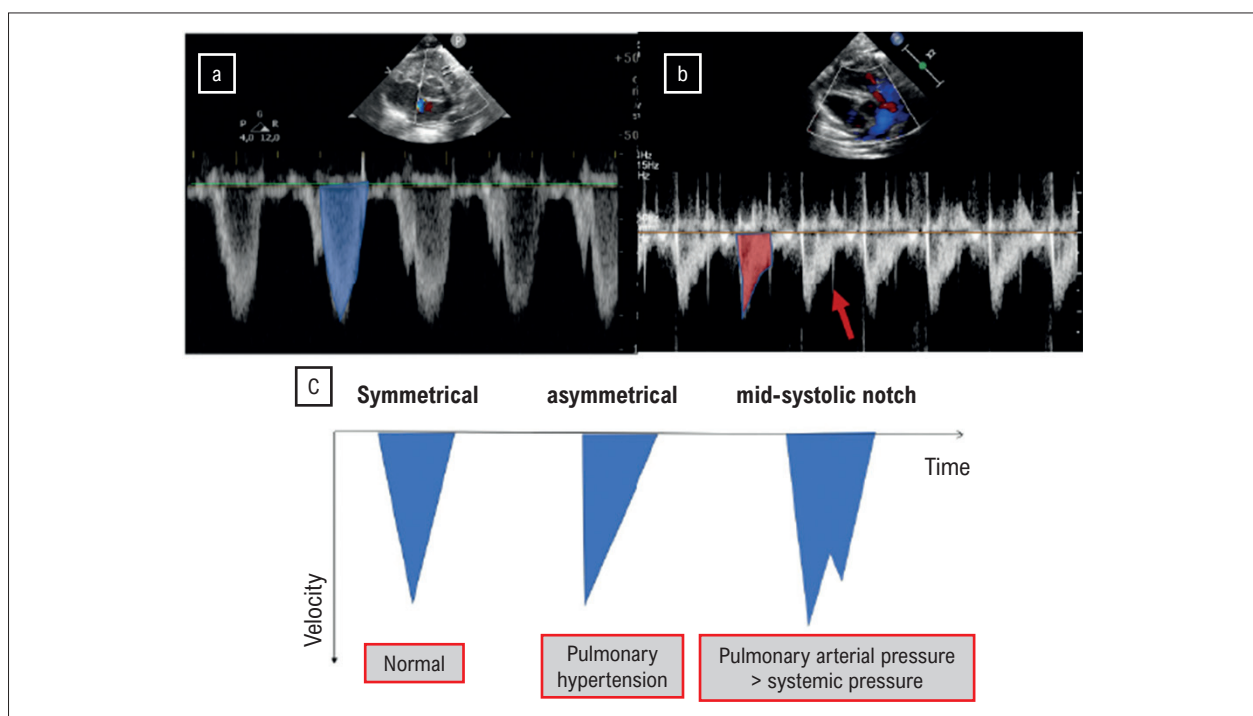


Figure 3 – Pulmonary artery Doppler pattern. In the first image (a) the symmetrical Doppler pattern of the pulmonary artery can be seen in the patient with normal pulmonary vascular resistance, in blue, the area under the curve proportional to the pulmonary VTI. In the second image (b), the mid-systolic notch (arrow) and the representation of the VTI (red area) of the patient with increased pulmonary vascular resistance can be seen. Image below shows representation of Doppler patterns.

as can be seen in Figure 4-A of the three-dimensional echocardiogram of the RV, reinforcing the importance of imaging assessment in different echocardiographic planes. The RV and LV commonly have similar dimensions in the apical 4-chamber view during the neonatal period, but the RV should not be larger than the LV (Figure 4-B and 4-C). The parasternal short axis plane complements the RV assessment and can be indexed by body surface area and age.⁹

2.2 Assessment of right ventricular systolic function:

2.2.1 Tricuspid Annular Plane Systolic Excursion (TAPSE)

TAPSE is the measure of the distance traveled by the tricuspid valve annulus toward the cardiac apex during systole. It is acquired in the apical four-chamber plane, usually positioning the M-mode cursor on the lateral portion of the valve annulus

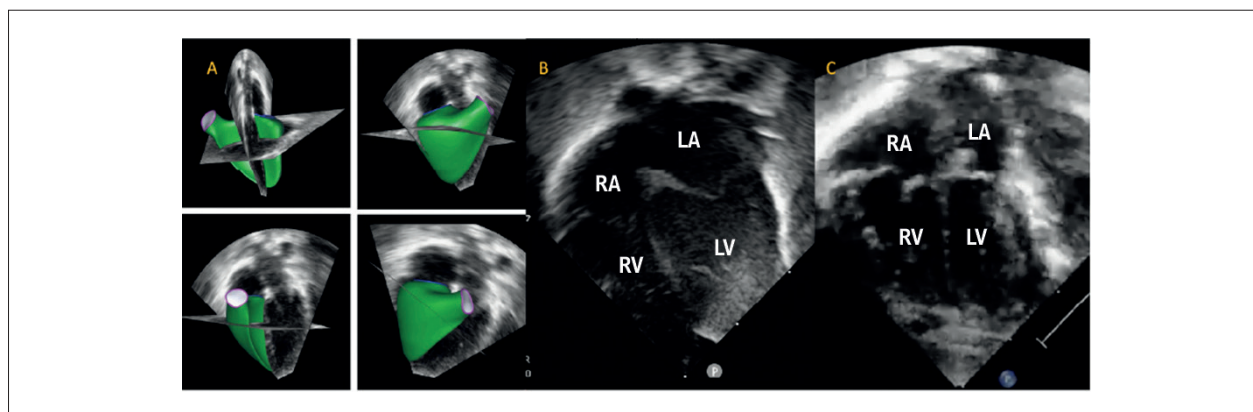


Figure 4 – Three-dimensional echocardiogram of the right ventricle, seen from different angles, showing the complexity of its morphology (A). Two-dimensional echocardiogram showing the apical 4-chamber view in a normal PTNB without pulmonary hypertension with normal proportions of LV and RV dimensions (B) and PTNB with pulmonary hypertension showing dilation of the right chambers (C). RA: Right atrium; LA: Left atrium; RV: Right ventricle; LV: left ventricle; PTNB: Preterm newborn

(Figure 5). The TAPSE value is negatively correlated with pulmonary vascular resistance and pulmonary pressure values.¹⁰ Normality values for TAPSE in neonates are indexed by body surface area and vary over the days of life. Jain et al. found a mean TAPSE of 0.92 cm on the first day of life.⁹

2.2.2 Fractional area change (FAC)

The FAC is the variation of the RV area in systole compared to diastole. It incorporates the global RV systolic function and can be obtained with a two-dimensional image of the apical four-chamber view modified for the RV, in which the endocardial walls should be traced in diastole (end diastolic area) and in systole (end systolic area), as shown in Figure 5. The normal value of FAC in adults is greater than 35%; in neonates it can also vary with the days of life and with the body surface.⁹ It can be obtained by the following equation:

$$\text{FAC} = \frac{\text{end diastolic area} - \text{end systolic area} \times 100}{\text{end diastolic area}}$$

There are other ways of assessing RV systolic function that will not be approached in this article.^{6,7} The ones mentioned above are the most used in practice.

3. High pressure: Two bodies do not occupy the same space. Straightening of the interventricular septum (IVS)

RV pressure overload leads to rectification of the IVS at the end of ventricular systole, resulting in a change in LV configuration that goes from an "O" shape (normal) to a "crescent" shape (in severe PH) passing through the "D" shape, when visualized in the short axis parasternal view. The assessment of IVS offers indirect evidence of increased pressure in the right chambers and is especially important when it is impossible to obtain an objective estimate of pulmonary pressure (Figure 6). It is even possible to infer the relationship between pulmonary and systemic pressure according to the curvature of the IVS,⁶ as seen in Table 1.

3.1. Eccentricity Index (EI)

The EI is derived from the ratio between the anteroposterior and septolateral diameters of the LV obtained in the parasternal short axis view at the level of the papillary muscle at the end of ventricular systole (Figure 7). $\text{EI} > 1.3$ is related to pulmonary pressure greater than half the systemic pressure, with good specificity.¹¹

4. Pipes and connections: Impaired systemic venous return

It has been observed so far that PTNB with PH show an increase in RV DP_2 , with a consequent increase in RA pressure and often show IVC dilation and reduced collapsibility. In this population, the evaluation of the dimensions of the IVC is done subjectively and collapsibility can be calculated using Equation 4:

Observou-se até aqui que o RNPT com HP apresenta aumento da PD_2 do VD, com consequente aumento da pressão do AD e frequentemente apresenta dilatação e redução de colapsabilidade da VCI. Nessa população, a avaliação das dimensões da VCI é feita de forma subjetiva e a colapsabilidade pode ser calculada pela Equação 4:

$$\frac{D_{\max} - D_{\min}}{D_{\max}} \quad (4)$$

Where $D_{\max}$ is the maximum diameter of the IVC and $D_{\min}$ is the minimum diameter measured, as in Figure 1. This measurement is expressed as a percentage.⁷ Another subjective indicator of increased pulmonary pressure that can be observed in the systemic venous return is the evident reverse flow in the hepatic veins (Figure 8). It is worth emphasizing that the increase in RA pressure can be due to several causes in addition to PH (hypervolemia, important tricuspid insufficiency, RV systolic or diastolic dysfunction, etc.), which highlights the importance of correlation with clinical data.

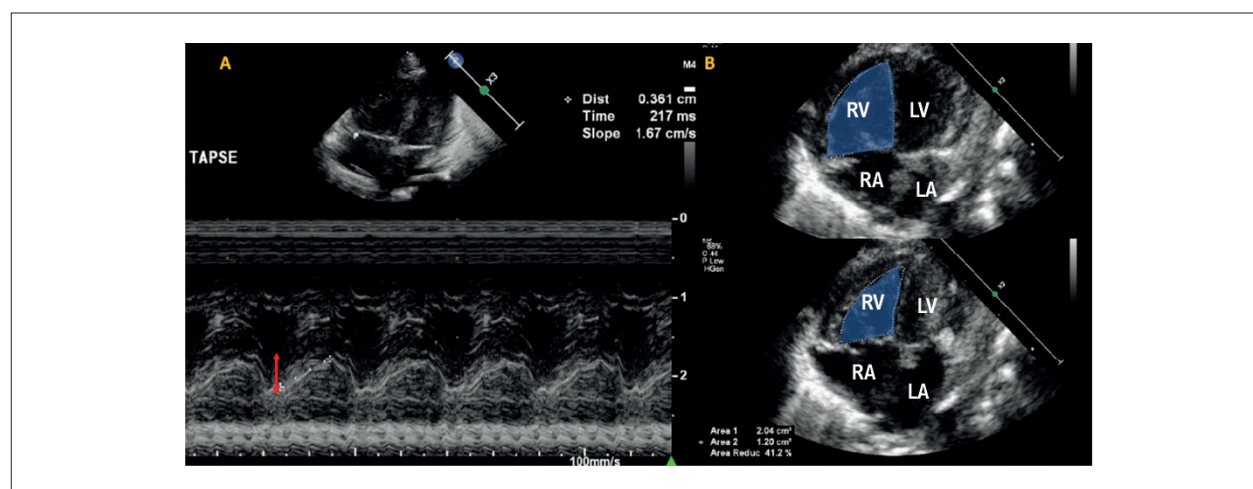


Figure 5 – A: Apical 4-chamber view showing the M-mode of the RV free wall, the red arrow shows the tricuspid annular plane systolic excursion (TAPSE). B: RV fractional area change (FAC). RV: Right ventricle; LV: left ventricle; AD: Right atrium; LA: Left atrium.

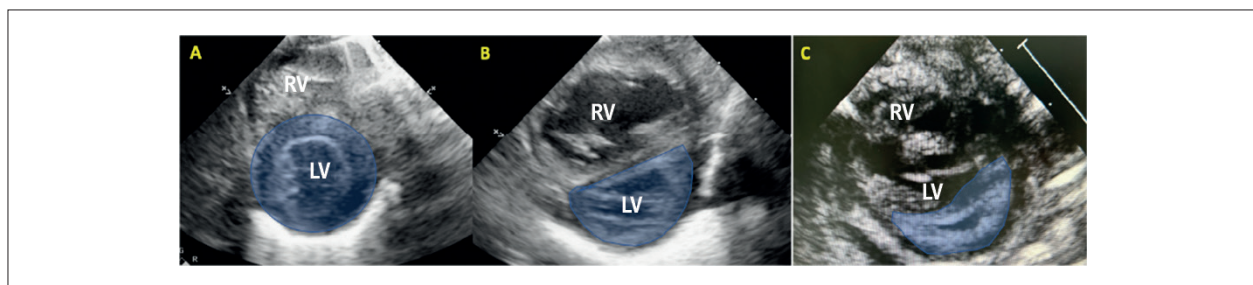


Figure 6 – Echocardiogram on the short axis, showing the shape of the LV. A: “O” shape, estimate of RVP <50% of LVP; B: NB with pulmonary hypertension, “D” shape, estimate of RVP 50-100% of LVP; C: NB with diaphragmatic hernia and significant PH, “crescent” shape, estimated RVP > 100% of LVP. LV: left ventricle; RVP: Right ventricular pressure; LVP: Left ventricular pressure; NB: newborn; PH: Pulmonary hypertension.

Table 1 – RVP estimate based on LV configuration

LV configuration	RVP estimate
“O” shape	<50% of LVP
“D” shape	50-100% of LVP
“crescent” shape	>100% of LVP

Modified from WP from Boode et al., 2018. RVP: Right ventricular pressure; LV: left ventricle; LVP: Left ventricular pressure.

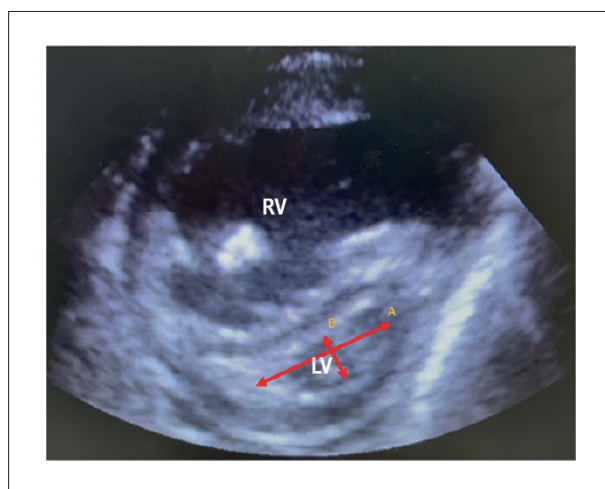


Figure 7 – Echocardiogram in the parasternal short axis view at the level of the papillary muscle at the end of ventricular systole. EI= A/B. EI: Eccentricity index.

5. Communications between the systemic and pulmonary bed: A problem or a relief?

In situations of normal pulmonary pressure, shunts such as the ASD and the PDA present flow directed from the left to the right chambers. Increased pressure in the right chambers causes flow from the AD to the left atrium (LA) through the ASD. The inversion of flow in the ductus arteriosus (DA) translates into important PH since the pulmonary pressure exceeds the systemic one. Pulmonary pressure can be estimated as discussed above. In cases of severe PH, the LV preload from the pulmonary venous return may be greatly

reduced, the flow coming from the right side through the communications can help complement this preload and increase the systemic cardiac output.

Importance of assessing the DA in preterm infants

PDA is a common problem in the NICU and can have deleterious effects on both the severely ill full-term newborn and the PTNB.

A failure to close the DA associated with a drop in postpartum pulmonary vascular resistance results in a left-to-right shunt. Consequences may include pulmonary hyperflow and/or systemic hypoperfusion, both of which are associated with increased morbidity. Clinical impact depends on the magnitude of the flow, existing comorbidities and the ability of the neonate to initiate compensatory mechanisms. The increase in pulmonary flow and the accumulation of interstitial fluid secondary to the large patent ductus contribute to a decrease in lung compliance and an increase in LV DP₂, which can be more pronounced in preterm infants, since ventricular compliance is lower.

In PTNB, a hemodynamically significant channel is associated with an increased risk of pulmonary edema, pulmonary hemorrhage, bronchopulmonary dysplasia, and increased duration of pulmonary ventilation. Although the presence of a wide duct supports increased cardiac output, post-ductal blood flow is reduced due to the left-to-right shunt, resulting in reduced oxygen delivery and perfusion to vital organs, thus contributing to an increased risk of intra- and periventricular hemorrhage, necrotizing enterocolitis, renal injury, and hypotension.¹²

Clinical findings may develop earlier in those patients treated with surfactant because the reduction in pulmonary vascular resistance associated with improved lung function results in increased left-to-right shunting.

Surgical or pharmacological closure should be postponed in patients with severe PH due to the risk of right heart failure. The assessment of ventricular function should be performed using qualitative and quantitative methods in these cases, always comparing to previous exams, if available.

The quantification of the size of the left cardiac chambers reflects the chronic effect of the left ventricular volume

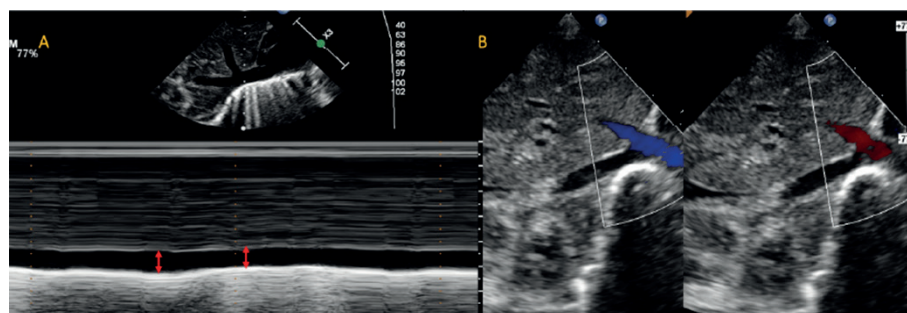


Figure 8 – IVC M-mode, the red arrows show the respiratory variation of the IVC diameter; B: Color flow mapping of the hepatic vein showing forward (blue) and reverse (red) flow. IVC: Inferior vena cava.

overload due to the left-right shunt through the duct and, therefore, is not an early parameter of hemodynamic repercussions.¹³

1. Anatomical assessment of the DA

The first echocardiographic assessment should always be comprehensive to exclude congenital heart defects. The following plans are used in DA assessment:

- On the parasternal short axis, the channel can be seen lateral to the left pulmonary branch and connecting to the descending thoracic aorta at the opposite end. With a slight inclination, it is possible to elongate the aorta and obtain a better image of the canal;
- In the high left parasternal short axis, also called the canal cut, it is possible to evaluate the entire path of the DA. This cut is obtained by placing the transducer in the left infraclavicular area, between the sternal notch and the conventional parasternal plane. This plane allows a more precise measurement of the size of the defect, normally acquired at its narrowest point, close to the left pulmonary artery (LPA). Prefer this measurement to the two-dimensional one, since the color Doppler tends to overestimate the DA size (Figure 9); and
- Suprasternal long-axis view angled toward the LPA allows a wide view of the DA.

The echocardiographer must provide information regarding the morphology, measurements of the diameters of the pulmonary and aortic extremities, total length and site of greatest canal narrowing, especially in cases that are candidates for percutaneous closure. An assertive determination of the diameter is extremely important in the sense of a probable hemodynamic impact, and a diameter ≤ 1.5 mm is associated with a risk of slight clinical repercussions, a diameter between 1.5 and 3.0 mm with moderate hemodynamic repercussions, and above 3.0 mm with significant repercussions.¹⁴

Krichenko et al.¹⁵ described isolated PDA as seen by angiography in five main groups, using the site of canal narrowing as a reference:

- Group A or conical ductus: narrow pulmonary end and hourglass at the aortic end;
- Group B or window-type ductus: short and narrow channel in the aortic region, wide in the pulmonary region;
- Group C: comprises the tubular ductus without constriction at the aortic and pulmonary end;
- Group D or saccular: the ductus has a wide central region with aortic and pulmonary constriction;
- Group E: channel is elongated, with constriction at the lung end; and
- Group F (fetal): in premature neonates who do not fit any of the morphologies previously described, generally with wide and tortuous canals, without significant stenoses (Figure 10).

2. Doppler assessment of the canal: new paradigms

Color flow mapping improves visualization of the DA, especially small caliber ones (<1 mm), and allows visualization of the site of greatest flow acceleration,

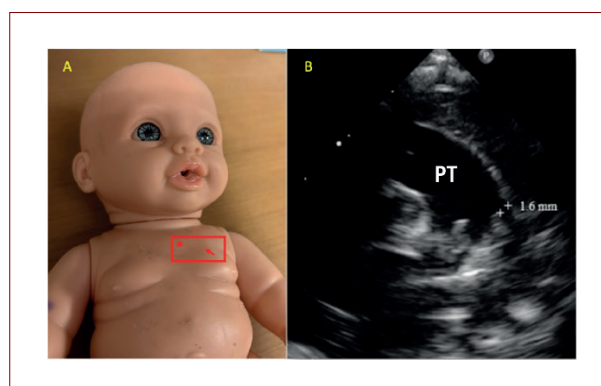


Figure 9 – High left parasternal view. A: Representation on the dummy of the site of the upper left parasternal cut ("canal cut"). B: Echocardiogram in the upper left parasternal plane demonstrating the location of the ductus arteriosus measurement, as well as its relationship with the left pulmonary artery, pulmonary trunk, and aorta. PT: pulmonary trunk; PDA: patent ductus arteriosus; Ao: descending aorta.

improving the positioning and alignment of the Doppler sample. Flow direction can be left-right, bidirectional or right-left, as seen in severe PH with suprasystemic pulmonary pressure and in heart diseases with DA-dependent systemic flow (Figure 11). Flow velocities below 1m/s should be recorded with pulsed Doppler. Continuous Doppler with the sample in the DA allows quantifying the pulmonary artery pressure using the modified Bernoulli equation. This measurement correlates with the instantaneous peak gradient between the aorta and the pulmonary artery by cardiac catheterization. Thus, by measuring the patient's systemic systolic pressure using conventional methods during the echocardiogram and subtracting it by the peak gradient obtained by continuous Doppler, it is possible to estimate the PASP. In the case of right-left flow, the pulmonary systolic pressure is estimated by adding the pressure gradient obtained by the Bernoulli equation to the systemic systolic pressure measured at the time of the test. If the Doppler flow pattern is bidirectional, with right-left flow occupying less than 30% of the cardiac cycle, the pulmonary pressure is probably lower than the systemic one. This bidirectional flow time measurement can also be obtained using the color M-mode in doubtful cases. Additional care must be taken with continuous Doppler flow contamination of structures adjacent to the canal, such as the LPA, aorta, and pulmonary systemic collaterals.

The appearance and speed of the Doppler curve also

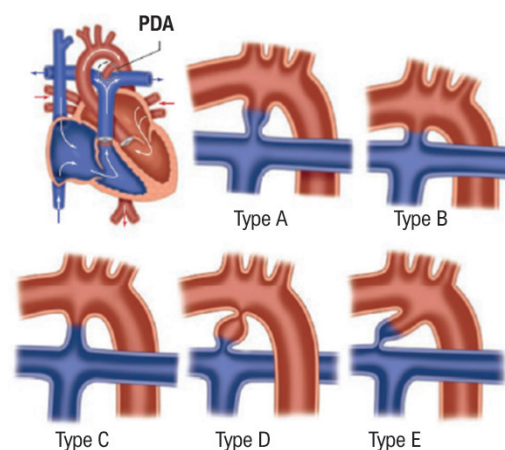


Figura 10 – Classificação de Krichenko para canal arterial isolado Adaptado de: DeFaria Yeh, Doreen; Bhatt, Ami (2018). [In Clinical Practice] Adult Congenital Heart Disease in Clinical Practice || Patent Ductus Arteriosus. , (Chapter 7), 91–105. doi:10.1007/978-3-319-67420-9_7

already give us important signals about the degree of flow restriction. In a small, restrictive DA without hemodynamic repercussions, the Doppler curve shows a high continuous flow velocity, both in systole and diastole, with a maximum

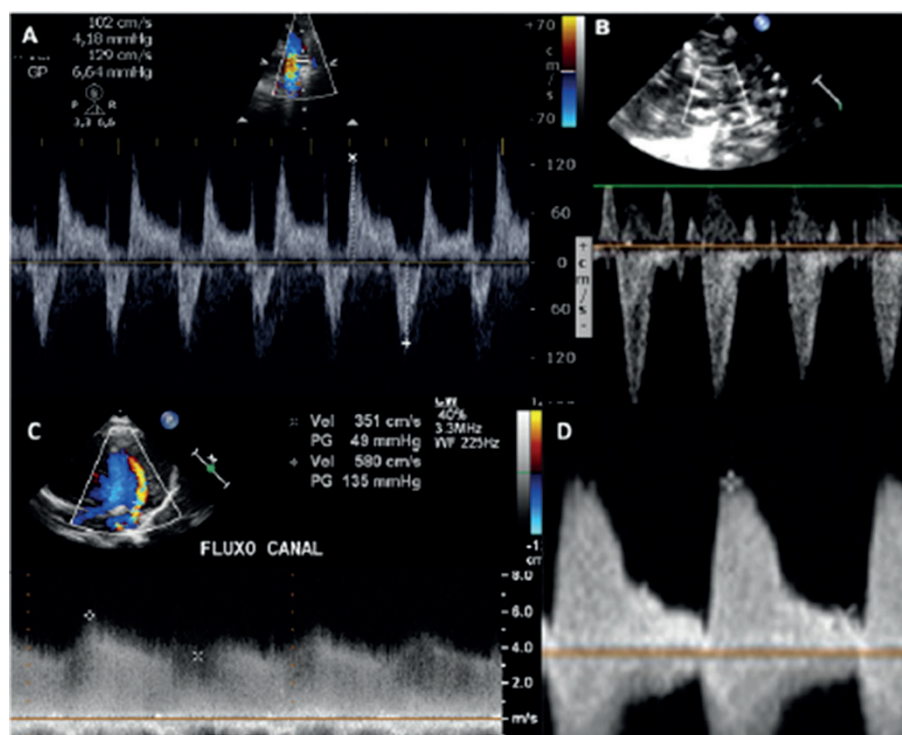


Figure 11 – Doppler pattern of the ductus arteriosus. A: Bidirectional pulsatile flow through the ductus arteriosus. B: right-left pulsatile flow through the ductus arteriosus. C: Flow curve with a continuous left-right pattern with diastolic velocity greater than 50% of the systolic velocity, suggestive of a restrictive canal. D: Flow curve tending to pulsatile, with diastolic velocity < 50% of the systolic velocity.

diastolic velocity greater than 2.0 m/s. Wide channels, without flow restriction, with important hemodynamic repercussions, present a flow curve tending more toward pulsatile flow, with a diastolic component with a velocity lower than 1.0 m/s and a wide difference in systolic and diastolic velocity. Channels with moderate hemodynamic repercussions have a pulsatile, non-restrictive flow model and maximum diastolic velocity lower than 2.0 m/s (Figure 11).

When analyzing the maximum velocity of the canal at the end of systole, it is possible to classify as a small canal those with velocity above 2.5m/s, moderate between 1.5 and 2.5m/s, and large with velocity below 1.5m /s.

It is important to differentiate the Doppler patterns of the pulmonary artery from the DA with a right-to-left shunt. The spectral Doppler of the pulmonary artery begins at the beginning of systole and reaches maximum amplitude quickly, while the flow from the DA with a right-to-left shunt begins at a later stage of systole and reaches maximum amplitude between mid- and late-systole.

In some cases, a characteristic serration on the pulsed Doppler curve of the pulmonary valve may be observed, raising the suspicion of a small DA not visualized previously on the exam (Figure 12).

The diastolic antegrade flow of the LPA has been considered a marker of pulmonary hyperflow in neonates. This measurement is obtained with the marker in the proximal third of the LPA. Some authors have demonstrated that a mean LPA velocity of 0.42m/s and/or an end-diastolic velocity of 0.2m/s are predictive of a Q_p/Q_s above 2, with high sensitivity and specificity (Figure 12-B).¹⁶

Evaluation of the E/A ratio on pulsed Doppler of the mitral valve may show signs of increased left heart filling pressure, with an E/A ratio greater than 1.5 in large ducts and an E/A ratio of 1 to 1.5 in moderate channels (Figure 13).

3. And does the Q_p/Q_s measurement really matter in preterm newborns?

In congenital heart diseases with increased pulmonary blood flow, it is common to use measures to compare pulmonary cardiac output with systemic output (Q_p/Q_s) to estimate the degree of the shunt. Echocardiography can estimate this relationship with good correlation with the data obtained during the hemodynamic study. In the DA, the calculation of Q_p/Q_s is performed differently from other congenital heart diseases such as ASD and ventricular septal defect. The flow through the pulmonary valve corresponds

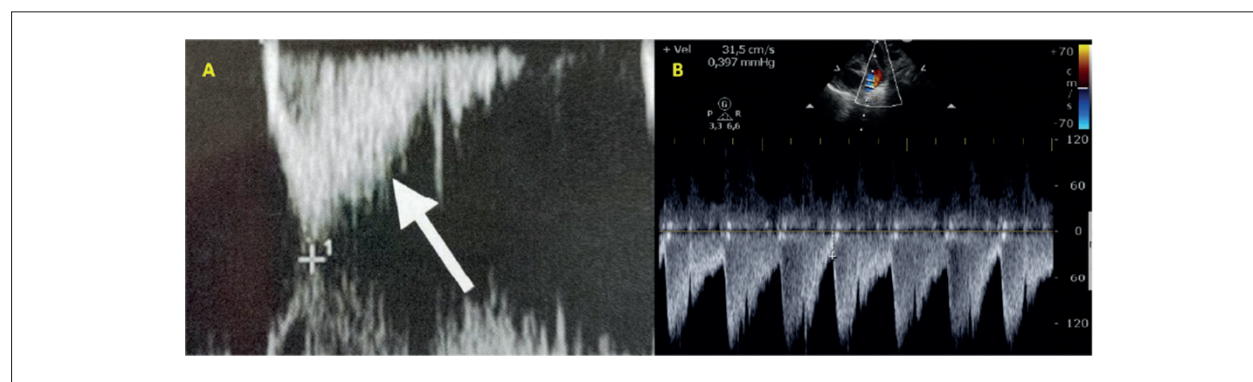


Figura 12 – A: Pulsed Doppler of the pulmonary valve demonstrating a serration in the ascending part of the curve as shown by the arrow, suggestive of contamination of flow from the ductus arteriosus. **B:** Doppler flow pattern in the left pulmonary artery demonstrating an end-diastolic component with high velocity predictive of increased pulmonary hyperflow.

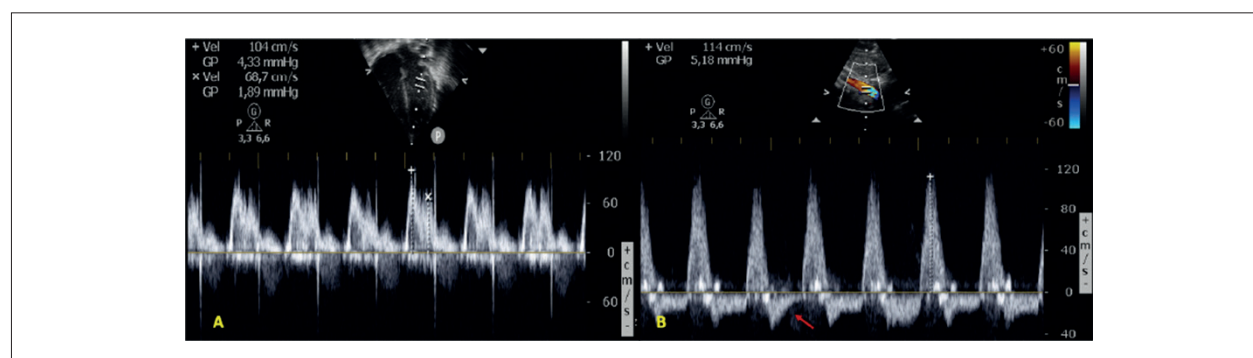


Figura 13 – A: Doppler pattern of the mitral valve in the apical 4-chamber view demonstrating alteration in the E/A wave ratio in preterm newborns with wide ductus arteriosus. **B:** Doppler pattern of the descending abdominal aorta, arrow shows the presence of holodiastolic reverse flow.

to the systemic flow (Q_s) and the flow through the aortic valve represents the effective pulmonary flow (sum of Q_s with the flow from the DA, causing overload of the left heart chambers). The presence of a PFO in the neonate, ASDs greater than 3mm, as well as alterations in the flow from the vena cava, can alter the estimation of the right ventricular output and, consequently, the systemic flow.¹⁷

4. Are signs of systemic flow steal easily assessed?

A large AC with significant left-to-right flow shunt will result in significant retrograde flow from the thoracic and abdominal aorta. The amount of retrograde diastolic flow can be >50% of the total aortic flow in neonates with a large AC, contributing to decreased systemic perfusion (renal, intestinal, and even coronary). Various indices based on pulmonary, aortic, and peripheral arterial flow velocity patterns have been proposed as objective methods for assessing the magnitude of ductal steal. The qualitative evaluation of the pulsed Doppler tracing of the descending aorta allows identifying the presence of reverse holodiastolic flow in the abdominal aorta and superior mesenteric artery, both evaluated in the subcostal or sagittal abdominal plane (Figure 13-B). The presence of holodiastolic reverse flow suggests moderate flow steal, which is associated with a Q_p/Q_s greater than 1.6, being one of the most specific signs of hemodynamic repercussion. Reverse flow in the abdominal aorta can occur in other congenital heart diseases that must be exhaustively investigated and ruled out.

5. DA assessment proposal based on a scorecard

A recent study by a group from Iowa¹⁸ proposes a useful echocardiographic score to assess the hemodynamic repercussions of DA through signs of pulmonary hyperflow and systemic hypoflow. The score is obtained with the sum of the points of the variables listed in the table below, added to the canal diameter divided by the weight (Table 2). Treatment has been considered when score ≥ 6 .

Soon after DA ligation, some PTNB become hemodynamically unstable due to acute changes in pre- and afterload, with oxygenation failure and systemic hypoperfusion, particularly in the first 8 to 12 hours after DA ligation. Thus, an early echocardiographic evaluation is necessary to optimize the treatment of these patients.

6. Does percutaneous treatment have its role?

More recently, several studies have reported experiences with the percutaneous closure technique, using various devices for closing the canal in PTNB. Comparison with surgical ligation revealed a positive impact on post-procedure pulmonary outcome. Currently, percutaneous closure of the DA in extremely low-weight premature patients weighing more than 700 grams is a safe procedure, with high efficacy, low complication rates, and is proven to be associated with improved prognosis in well-selected patients. TTE is the exam of choice both for selecting favorable cases and for guiding the procedure, thus reducing the amount of contrast used. Canals with a diameter < 4.0mm and length > 3.0mm are the most appropriate for the devices currently used.¹⁹

Table 2 – IOWA Score: echocardiographic evaluation of PDA severity

PDA IOWA SCORE	0	1	2
Mitral E velocity (cm/s)	< 45	45 - 80	> 80
IVRT (ms)	> 50	30 - 50	< 30
VD PV velocity	< 0.3	0.3 - 0.5	> 0.5
LA/Ao	< 1.3	1.3 - 2.2	> 2.2
LVEF:RVEF	≤ 1	1 - 1.7	> 1.7
Aorta/peripheral flow	Anterograde		Reverse

PDA: patent ductus arteriosus; IVRT: isovolumetric relaxation time; PV: pulmonary vein; LA: left atrium; Ao: aorta; LVEF: left ventricular ejection fraction; RVEF: right ventricular ejection fraction. Adapted from John M. Dagle, Matthew A. Rysavy, Stephen K. Hunter, Tarah T. Colaizy, Timothy G. Elgin, Regan E. Giesinger, Steve J. McElroy, Heidi M. Harmon, Jonathan M. Klein, Patrick J. McNamara, *Cardiorespiratory management of infants born at 22 weeks' gestation: The Iowa approach, Seminars in Perinatology, Volume 46, Issue 1, 2022*

New perspectives in assessing DA and PH in preterm newborns

More recent methods for assessing myocardial contractility were incorporated into the echocardiogram with the aim of assessing LV and RV function regardless of their geometry or ventricular filling.²⁰ One of these methods, speckle tracking (ST) by TTE, assesses myocardial fiber deformation during the cardiac cycle, allowing earlier diagnosis of myocardial dysfunction, before a drop in ejection fraction occurs. Levy et al.²¹ demonstrated in a prospective study that it is possible to perform global longitudinal strain (GLS) of the RV using the ST method in PTNB patients with reliable reproducibility of right ventricular function and hemodynamic variations. Almeida et al.²² confirmed that the analysis of myocardial deformation through the ST technique by TTE is feasible and reproducible in PTNB ≤ 34 weeks of gestational age. It was also shown that radial and circumferential strain and GLS measurements are significantly higher in PTNB with DA with hemodynamic repercussions, possibly as a means of compensating for the left ventricular volume overload imposed by the large duct.

Acknowledgments

To dear Dr. Adriana Mello and Dr. Gabriela Leal, who contributed by sharing experience and bibliography.

Author Contributions

Conception and design of the research: Sawamura KSS; acquisition of data, analysis and interpretation of the data, writing of the manuscript and critical revision of the manuscript for intellectual content: Sawamura KSS, Brito MM.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

