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ORIGINAL ARTICLE

Persistent Pulmonary Hypertension in Neonates: Insights from Echocardiography

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ABSTRACT

Background: A serious clinical issue known as persistent pulmonary hypertension of the newborn (PPHN) is typified by severe and refractory hypoxemia brought on by increased pulmonary vascular resistance, which causes deoxygenated blood to be shunted from the right to the left extrapulmonaryly. Infants with severe PPHN have a significant early death rate and are linked to a variety of cardiopulmonary diseases. Infants with PPHN who survive are more likely to experience long-term complications. The physiology of PPHN can be divided into three categories: (1) maladaptation, where pulmonary vessels have normal numbers and structures but exhibit abnormal vasoreactivity; (2) excessive muscularization, where smooth muscle cells are thicker and muscles extend farther to vessels that are typically not muscularized; and (3) underdevelopment, where pulmonary hypoplasia is linked to fewer pulmonary arteries. PPHN is diagnosed by trained neonatal intensivists using bedside structural and functional echocardiography, even though the diagnosis is initially prompted by clinical signs like respiratory or hemodynamic instability in a typical setting (like the ones listed above).
Conclusion: it is beneficial to diagnose PPHN, rule out any underlying congenital cardiac defects (such transposition of the great arteries or total abnormal pulmonary venous return), and then evaluate the hemodynamic condition (hypovolemic or obstructive shock).

Keywords: Persistent pulmonary hypertension, neonates, echocardiography.

INTRODUCTION

When the pulmonary vascular resistance does not sufficiently reduce during the transition to extrauterine life, the infant develops persistent pulmonary hypertension (PPHN). Infants affected have anatomically normal hearts, but because of the pulmonary hypertension, they have substantial right-to-left shunts at the atrial and ductal levels **[1].**

A persistent increase in pulmonary vascular resistance (PVR) is the hallmark of persistent pulmonary hypertension of the neonate (PPHN), a phenomenon that is frequently linked to normal or low systemic vascular resistance (SVR). Labile hypoxemia results from this extrapulmonary shunting from right to left across the patent ductus arteriosus (PDA) and patent foramen ovale (PFO), two persisting fetal channels. Previously known as persistent fetal circulation (PFC), this condition frequently

results from a failed pulmonary transition during delivery **[2].**

A dangerous illness known as persistent pulmonary hypertension of the newborn (PPHN) occurs when a baby's circulatory system is unable to adjust to breathing outside of the womb. Clinicians frequently evaluate lung pressures and vascular resistance using echocardiography, cardiac catheterization, and other diagnostic techniques in order to identify PPHN. The most widely utilized technique is pulmonary artery pressure. By detecting the pressure gradient across the tricuspid valve (tricuspid regurgitation jet), it calculates the pressure in the pulmonary arteries. PPHN is indicated by elevated pulmonary artery pressure **[3].**

Neonates normally have low pulmonary wedge pressure (between 6 and 12 mmHg), as determined by cardiac catheterization. Instead of PPHN, elevated wedge pressure indicates left heart disease. The formula for calculating pulmonary vascular resistance (PVR) is as follows: PVR = (mean pulmonary artery pressure − pulmonary wedge pressure) / cardiac output. Neonates often have a PVR between 0.5 to 2 Wood units. PPHN is linked with increased PVR, which signifies increased resistance in the pulmonary vasculature **[4].**

Mean pulmonary artery pressure of more than 25 mmHg, pulmonary wedge pressure of normal (6–12 mmHg), and high pulmonary vascular resistance, usually more than 2 Wood units, are typical diagnostic results for PPHN **[5].**

The circulatory systems of fetuses and adults differ greatly. This complex system enables the placenta to supply the fetus with nutrients and oxygenated blood. It is made up of the umbilical cord's two umbilical arteries and one umbilical vein as well as the placenta's blood vessels. Through a shunt called the ductus arteriosus, fetal circulation avoids the lungs; the ductus venosus avoids the liver; and the foramen oval allows blood to move from the right atrium to the left atrium. Fetal heart rates typically range from 110 to 160 beats per minute. Fetuses exhibit lower ventricular filling and contractility in comparison to adults **[6].**

After birth, the fetal circulation quickly changes to make room for life outside the womb. Although fetal sheep provided the basis for human knowledge of prenatal circulation, precise information is now available because to fetal ultrasound and magnetic resonance imaging (MRI). According to **Remien et al. [6],** there are clear variations in fetal circulation that, if improperly developed, might result in illnesses in children or adults.

Fetal circulation begins at 22 days, when the fetal heart begins to develop. Until the placenta completely takes over, gas exchange first takes place in the yolk sac. About ten weeks into the pregnancy, this change takes place. Before leaving for the fetus, placental blood, which has less oxygen, combines with maternal oxygenated blood. The fetus is comparatively hypoxic in comparison to the maternal arterial blood as a result of this mixing **[7].**

The cardiovascular system changes rapidly and dramatically once the infant is delivered. As a result of the oxygen that is now in the lungs and the physical process of breathing, the baby's pulmonary vascular resistance significantly decreases with its first breath. After birth, the umbilical cord clamps, increasing systemic vascular resistance and facilitating blood flow to the lungs. Within ten minutes, the ductus arteriosus flows from left to right. In response to the oxygen, the ductus arteriosus' smooth muscle increases calcium channel activity, which results in constriction and, eventually, shunt closure. The foramen oval closes as a result of the elevated systemic resistance, which also

makes the left atrium's pressure greater than the right atrium's **[8].**

William Harvey originally referred to persistent fetal circulation (PFC), also called persistent pulmonary hypertension of the neonate, as "unripe births of mankind" in 1628. But it wasn't until the last half of the 1800s that the syndrome was recognized. When the foramen oval in asphyxiated newborns has either failed to shut or reopened, postnatal maintenance of fetal circulation patterns occurs **[9].**

The failure of the normal circulatory transition at birth is the secondary cause of persistent pulmonary hypertension of the newborn (PPHN). Due to reduced pulmonary blood flow and right-to-left blood shunting, this syndrome, which is characterized by high pulmonary vascular resistance (PVR), results in labile hypoxemia **[1].**

A more sophisticated understanding of the pathophysiology and underlying causes of persistent pulmonary hypertension of the newborn (PPHN) has been incorporated into recent classifications. As diagnostic methods have improved and our understanding of newborn physiology and pathology has expanded, the classification system has changed to reflect these developments. According to **Varghese et al. [10]**, the most recent classification—

- In lung parenchymal illnesses like meconium aspiration syndrome, maladaptation refers to an aberrant pulmonary vascular response.
- Underdeveloped vasculature: reduced pulmonary vasculature, as shown in oligohydramnios or small for gestational age.
- A newborn's idiopathic chronic pulmonary hypertension is most likely brought on by an excessively thick pulmonary vascular smooth muscle **[11].**

Mild respiratory distress to severe hypoxic respiratory failure necessitating extracorporeal membrane oxygenation (ECMO) and mechanical ventilation are among the symptoms. In the early stages of neonatal life, persistent pulmonary hypertension in the infant can be fatal. Neonates must be swiftly identified and given the proper care by the healthcare practitioner **[12].**

Echocardiography Findings:

Echocardiography, which offers comprehensive insights into the architecture, hemodynamics, and function of the heart, is essential in the assessment and treatment of Persistent Pulmonary Hypertension of the Newborn (PPHN). The gold standard for both diagnosis confirmation and tracking the effectiveness of certain treatment approaches is echocardiography. Right-sided pressures and hemodynamic physiology can be determined by measuring the tricuspid regurgitation velocity, the interventricular septum's flatness or left deviation, and the orientation of the ductal and foramen oval shunts while also taking a systemic blood pressure reading **[13].**

Findings of echocardiography include:

Tricuspid Regurgitation

One common and important echocardiographic finding in newborns with PPHN is tricuspid regurgitation (TR). Blood can flow backward into the right atrium during systole when the tricuspid valve, which separates the right atrium from the right ventricle, malfunctions. TR can offer crucial diagnostic and prognostic data in the context of PPHN. Elevated pulmonary vascular resistance, which raises right ventricular pressure, is a hallmark of PPHN. Because of the increased pressure load, the right ventricle dilates, and the tricuspid valve may not work properly, which can lead to regurgitation **[14].**

The right ventricle enlarges because of the ongoing pressure overload. TR may develop from this dilatation's ability to stretch the tricuspid valve annulus, which

would hinder the leaflets' correct coaptation. The right ventricle's ability to contract may be hampered by the increased workload. The degree of tricuspid regurgitation may worsen if the right ventricle contracts less forcefully **[15].**

Doppler echocardiography, more especially color flow Doppler and continuous-wave Doppler, is used to evaluate tricuspid regurgitation. The retrograde flow of blood from the right ventricle to the right atrium is visualized by Doppler as a turbulent jet that passes above the tricuspid valve. The regurgitant jet's breadth and length, the continuous-wave Doppler signal's density and shape, and the right atrium's size are all used to gauge how severe TR is. These metrics aid in the mild, moderate, and severe classification of TR. The pressure differential between the right ventricle and right atrium can be estimated using the tricuspid regurgitant jet's velocity. This gradient can be used to predict the right ventricular systolic pressure (RVSP) using the modified Bernoulli equation. RVSP is a rough estimate of pulmonary artery systolic pressure (PASP) when there is no blockage of the right ventricular outflow tract. In PPHN, elevated PASP is a clear sign of elevated pulmonary vascular resistance **[16].**

Assessment of Ductal Patency

To effectively manage PPHN, echocardiography assesses the ductus arteriosus's patency and flow characteristics. Continued right-to-left shunting is made possible by persistent patency, which may exacerbate hypoxemia **[17].**

Right Ventricular Function

Echocardiography frequently shows evidence of right ventricular (RV) strain and dysfunction in PPHN. Increased pulmonary vascular resistance puts the right ventricle under more pressure overload, which can cause dilatation and hypertrophy. This manifests as right ventricular hypertrophy, decreased contractility, and compromised systolic function on echocardiography. These results can reveal the severity of PPHN and represent the increased stress on the RV. Systemic perfusion and total cardiac output may also be impacted by abnormalities in RV function **[18].**

Pulmonary Artery Pressure

Estimating pulmonary artery pressure (PAP) is one of echocardiography's main functions in PPHN. PPHN is characterized by elevated PAP, which can be measured indirectly using echocardiograms **[19].** The most popular technique uses Doppler ultrasonography to measure the tricuspid regurgitation velocity. This makes it possible to estimate the pressure differential between the right atrium and the right ventricle. The severity of PPHN is correlated with elevated tricuspid regurgitant jet velocity, which is a sign of elevated pulmonary artery pressure **[20].**

Right-to-Left Shunting

The existence and severity of right-to-left shunting through the ductus arteriosus and foramen ovale can also be evaluated by echocardiography. Color Doppler imaging can show the right-to-left shunting of blood across these fetal channels in PPHN, which is caused by increased pulmonary vascular resistance. Treatment choices can be influenced by the degree of shunting, which is a crucial sign of the severity of PPHN. More severe hypoxemia and the requirement for aggressive therapy are generally correlated with increased right-to-left shunting **[21].**

Structural Anomalies

Echocardiography can identify a number of structural cardiac abnormalities that may be linked to PPHN. Patent ductus arteriosus (PDA), which can be noticeable in newborns with PPHN, is a common occurrence. Echocardiography can also detect additional congenital cardiac abnormalities that could worsen or cause pulmonary hypertension. For thorough management and better results,

it is essential to recognize and correct these structural irregularities **[22].**

Associated Cardiac Remodeling

Echocardiography can show structural changes in the heart caused by persistently high pulmonary pressures in PPHN. Ventricular remodeling, such as right ventricular dilatation and hypertrophy, is one of these alterations. Increased pressure gradients between the heart's chambers and changed blood flow patterns can also be seen on echocardiographic imaging. These results aid in determining how PPHN affects heart function and in directing treatment strategies **[23].**

Septal Wall Deviation

Under high pulmonary pressures, the interventricular septum may deviate toward the left ventricle; this is referred to as paradoxical septal motion or septal flattening. This illustrates the difference in pressure between the left and right ventricles. Because of the high resistance of the pulmonary vasculature in PPHN, the right ventricle experiences elevated pressure **[24].**

The interventricular septum is forced toward the left ventricle as the right ventricular pressure rises. Because the septum is a flexible structure that adapts to the pressure dynamics inside the ventricles, this displacement takes place. The septum shifts into the left ventricular chamber as a result of force exerted by the elevated pressure in the right ventricle. This deviation reflects the severity of pulmonary hypertension and is suggestive of severe right ventricular pressure overload **[25].**

Standard echocardiographic methods, including as Doppler investigations and two-dimensional (2D) imaging, are used to assess septal wall deviation. Several perspectives, such as the parasternal, apical, and subcostal views, can be used to visualize the deviation. During systole, the interventricular septum shifts toward the left ventricle, as seen in 2D echocardiography **[26].**

Both qualitative and quantitative methods can be used to evaluate the degree of septal wall deviation. Echocardiographic pictures qualitatively demonstrate the extent of displacement and its effect on left ventricular function. The degree of deviation and its impact on the size and function of the left ventricular cavity can be evaluated quantitatively. A higher septal deviation can serve as a gauge for the severity of PPHN and is linked to more severe right ventricular pressure overload **[19, 24].**

Left Ventricular Function

One important component of cardiac function that can be greatly impacted in newborns with PPHN is left ventricular (LV) function. The right ventricle experiences an elevated pressure burden due to PPHN, which may have an indirect effect on the left ventricle's operation. Assessing LV function helps direct management methods for impacted infants and offers insights into overall cardiac functioning **[27].**

The interventricular septum may diverge toward the left ventricle as a result of elevated right ventricular pressure brought on by PPHN. The amount of space available for left ventricular filling during diastole is decreased by this divergence. Heart output and left ventricular stroke volume may be impacted by the reduced filling volume. Systemic hypotension and insufficient perfusion of critical organs may occur from the ensuing decreased left ventricular performance. By lowering the left ventricular end-diastolic volume (LVEDV), the septum's shift towards the left ventricle can also affect diastolic filling. Lower preload results from decreased LVEDV, which may affect left ventricular function and total cardiac output. Clinical instability and systemic hypoperfusion may

be worsened by this decrease in preload **[28].**

The left ventricle's capacity to provide sufficient cardiac output may be impacted by the strain that results from PPHN's increased workload on the right ventricle. Hypotension reduces systemic perfusion, and inadequate oxygen supply to tissues can result from the left ventricle's impaired function. Optimizing both left and right ventricular performance is frequently necessary for the management of PPHN to guarantee appropriate perfusion **[27].**

Visual evaluation of the size, shape, and motion of the left ventricle is possible with two-dimensional echocardiography. It aids in assessing how the left ventricle is affected by right ventricular pressure overload. Changes in the interventricular septum, anomalies in wall motion, and changes in the size of the LV chamber are important findings. The speed and direction of blood flow across the heart's chambers and valves are measured using Doppler investigations. Wave-pulsed Mitral inflow velocities and other left ventricular filling patterns can be evaluated by Doppler, which can reveal details regarding diastolic function. Variations in these metrics may be a sign of decreased heart function and filling **[26].**

By measuring cardiac velocities and strain, Doppler imaging provides information on the left ventricle's contractile activity. By monitoring myocardial velocities during contraction and relaxation, TDI is able to assess left ventricular systolic and diastolic function. Myocardial deformation can be quantitatively evaluated by strain imaging. It provides comprehensive data on both systolic and diastolic function by measuring the contraction and stretching of the cardiac fibers. Reduced strain values could be a sign of PPHN-related left ventricular dysfunction **[26].**

Tissue Doppler imaging in Pulmonary Hypertension of the Newborn (PPHN)

Infants with Persistent Pulmonary Hypertension of the Newborn (PPHN) have their myocardial function evaluated using Tissue Doppler echocardiography (TDE). Through the measurement of myocardial tissue movement velocities, TDE offers vital information about PPHN-specific heart mechanics. At crucial locations like the septal and lateral mitral annulus, it enables physicians to assess metrics like systolic velocity (S'), early diastolic velocity (E'), and late diastolic velocity (A'). By evaluating cardiac function and identifying any localized irregularities in myocardial motion, these velocities can assist identify strain or dysfunction brought on by high pulmonary pressures. **[29].**

There is a substantial link between the amounts of B-type natriuretic peptide (BNP) in plasma and the echocardiographic findings of ventricular strain. This correlation has been demonstrated repeatedly in studies. For instance, higher BNP levels are associated with increased left ventricular mass and wall thickness seen on echocardiography in situations of left ventricular hypertrophy brought on by systemic hypertension or aortic stenosis. Likewise, elevated BNP levels in PPHN and other right ventricular strain circumstances correspond to RV dilatation and hypertrophy observed on echocardiography **[30].**

Response to Therapy

In order to track how well therapeutic procedures like inhaled nitric oxide (iNO), surfactant therapy, and mechanical ventilation changes are working, serial echocardiography is crucial. Over time, improvements in shunting patterns, RV function, and pulmonary pressures can be seen **[31].**

Long-Term Follow-Up

Long-term follow-up is guided by echocardiography to monitor heart function and determine whether PPHN-related problems have

resolved. Dilatation and persistent RVH could be signs of chronic pulmonary hypertension or PPHN sequelae that have an impact on long-term results. Both the left and right ventricles are strained by high pulmonary pressures in PPHN. Through the measurement of the right ventricle's myocardial movement velocity, TDE is able to assess right ventricular function. Reduced systolic velocities or changed diastolic patterns can be signs of impaired right ventricular function. Likewise, it is possible to evaluate left ventricular function; results like decreased systolic velocities or impaired diastolic filling might reveal details about the overall effect of PPHN **[14].**

When determining the extent of ventricular strain brought on by elevated pulmonary pressures, TDE is useful. TDE directs the treatment of PPHN and aids in assessing the degree of functional impairment by monitoring cardiac deformation. Significant right ventricular pressure overload and possible systemic perfusion compromise may be indicated by abnormal strain patterns. TDE makes it possible to evaluate how therapeutic interventions affect myocardial function in real time. Clinicians can modify therapy depending on improvements in myocardial velocities and overall cardiac function by assessing the effects of medications like sildenafil, inhaled nitric oxide, or other pulmonary vasodilators **[32].**

Since measures of E' and A' velocities help diagnose and track diastolic dysfunction, TDE is especially helpful in PPHN for evaluating diastolic function. This is important because PPHN frequently results in elevated right ventricular afterload, which changes the dynamics of myocardial relaxation and filling. TDE also aids in tracking how the right ventricle reacts to treatments such systemic vasodilators and inhaled nitric oxide (iNO) that lower pulmonary vascular resistance. Clinicians can evaluate the efficacy of treatment and modify management techniques to maximize heart function and improve outcomes for these critically unwell neonates by monitoring changes in myocardial velocities over time **[33].**

Notwithstanding its advantages, TDE in PPHN necessitates experience because of technical difficulties with neonatal picture capture and interpretation. The accuracy of velocity measurements can be affected by variables such as patient size and the caliber of acoustic windows. To help with early diagnosis, continuous monitoring, and directing therapeutic interventions to lessen the effects of pulmonary hypertension on cardiac function, TDE is still a useful and non-invasive method for thoroughly evaluating myocardial mechanics in infants with PPHN **[34].**

Conflict of Interest: None
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