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Assessment Of Fetal Lung Maturity Using Pulmonary Artery Doppler Indices: A Review Article

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ABSTRACT

This study highlights the role of fetal pulmonary artery Doppler as a reliable, non-invasive technique for assessing fetal lung maturity and predicting the risk of neonatal respiratory distress syndrome (NRDS). Conventional invasive procedures, such as amniocentesis, carry procedural risks, whereas Doppler evaluation offers a safe alternative by analyzing hemodynamic changes and vascular impedances. The main pulmonary artery acceleration time to ejection time (At/Et) ratio demonstrates a strong association with gestational age, lung maturity, and neonatal outcomes, with lower ratios indicating immaturity and higher RDS risk. Additional indices, including systolic/diastolic ratio, pulsatility index, resistance index, and peak systolic velocity, further strengthen its diagnostic performance. While the at/Et ratio alone may not achieve complete specificity across all gestational ages, combining it with other clinical parameters enhances predictive accuracy. By enabling early detection of at-risk fetuses, pulmonary artery Doppler can guide timely interventions, reduce neonatal morbidity and mortality, and improve overall perinatal care.

Keywords: *Fetal lung maturity, Acceleration time/ejection time (At/Et) ratio, Pulmonary artery Doppler, Respiratory distress syndrome (RDS), Non-invasive prediction.*

INTRODUCTION

Fetal lung maturity is a vital developmental milestone that ensures independent breathing after birth. It involves structural and biochemical changes that prepare the lungs for gas exchange. Assessing lung development is clinically important, especially in premature delivery (Antsaklis and Theodora 2021). NRDS is a leading cause of respiratory compromise in infants, often appearing within hours of birth. It primarily affects preterm infants and, less frequently, term neonates (Yadav, et al. 2020). The underlying cause is surfactant deficiency, which predisposes alveoli to collapse at end-expiration. Clinical signs include cyanosis, tachypnea, intercostal retractions, and expiratory grunting (Wu, et al. 2020). Early prediction of RDS can significantly improve outcomes and reduce economic burden. Several biochemical and imaging approaches have been investigated for this purpose. Lung ultrasound, once considered unsuitable, is now widely studied and offers advantages over chest X-ray, including absence of radiation, simplicity, cost-effectiveness, repeatability, and bedside applicability (Alsheikh, et al. 2021). In addition, Doppler velocimetry provides a rapid, non-invasive assessment of fetal pulmonary circulation. Analysis of blood flow in the main pulmonary artery and its branches shows that the ratio of At/Et correlates with gestational age and lung maturity, as confirmed by amniotic fluid testing (Bedeer, et al. 2024).

Anatomy of the Fetal Lung

Adaptation from an intrauterine fluid environment to permanent air breathing is the critical step for extrauterine survival, and its failure necessitates neonatal intensive care. Lung development starts at 4– 6 weeks of pregnancy as a ventral outpouching of the primitive foregut (laryngotracheal groove).

Posterior progenitor cells form paired bronchial tubes, which give rise to all distal epithelial structures (Korraa 2023). Distal gas exchange surfaces appear by 20 weeks and are largely formed by term. Proximal airway branching ends at 16–18 weeks at the bronchoalveolar duct junctions, with ~5 alveolar ducts arising distally from each junction. Each duct gives rise to alveoli, with up to 5 daughter alveoli budding from each primary sac (Warburton 2017). At the duct end, 5 interconnected alveoli open into the lumen, with alveolar shape constrained by pleural and fascial planes (Warburton and Biology 2021).

Embryology of the Lung

Embryonic Stage (3–6 weeks):

The respiratory diverticulum seems to be in the primitive foregut endoderm. By week 4, the trachea bifurcates into right and left primary bronchial buds, which invade splanchnopleuric mesenchyme (Downey and Samra 2020).

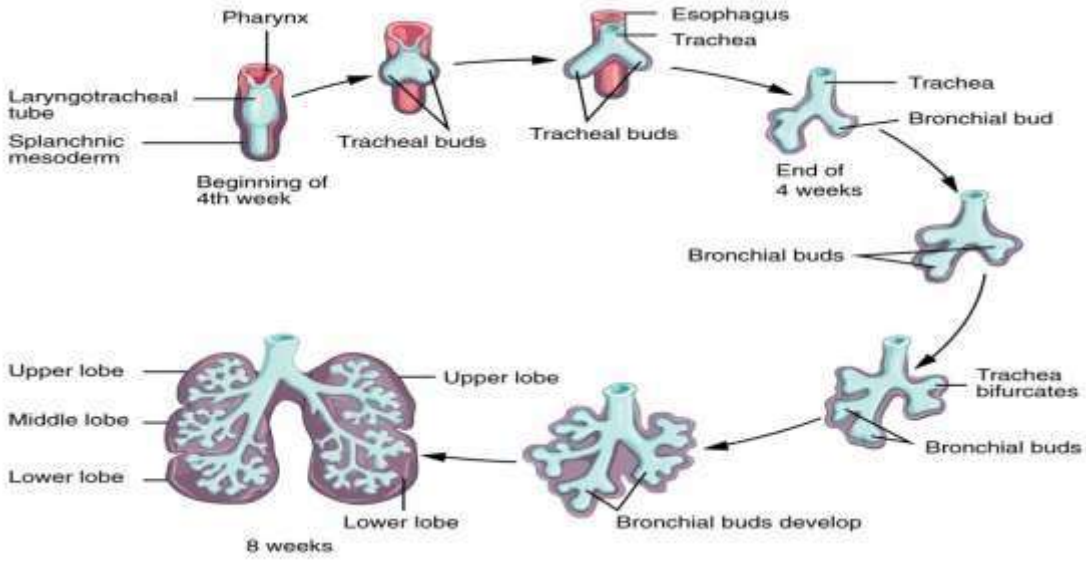

Figure 1: Development of the Lower Respiratory System.

By week 5, primary buds divide asymmetrically into 2 left and 3 right secondary buds (future lobes). By week 6, tertiary buds form bronchopulmonary segments (Rehman and Bacha 2023). Parietal and visceral pleura arise from mesoderm (weeks 5–7), sealing the thoracic cavity. At this stage, the larynx, trachea, lung primordia, lobes, and segments are complete (Mahabadi, et al. 2018).

Pseudoglandular Stage (5–17 weeks):

The cuboidal epithelium gives the lungs a gland-like appearance. By week 16, branching produces ~20 generations of conducting airways (Rehman and Bacha 2023). Differentiation of columnar epithelium produces cilia, while mesoderm develops intrapulmonary arteries, cartilage, and smooth muscle. Development ends at terminal bronchioles; infants born now cannot survive due to absence of respiratory bronchioles (Rehman and Bacha 2023).

Canalicular Stage (16–25 weeks):
Terminal bronchioles elongate, forming acini with 3–6 alveolar ducts each. Angiogenesis creates a dense capillary network and the blood-air barrier (Rehman and Bacha 2023). 20 weeks, lamellar bodies appear in type II pneumocytes, storing surfactant proteins. Limited differentiation into type I pneumocytes begins. Although minimal respiration is possible, survival is poor due to insufficient surface area and surfactant (Swarr, et al. 2023).

Saccular Stage (24 weeks–birth):
Terminal airways expand into saccules, separated by thick septa containing a double capillary layer.
Distinguishing type II from type I pneumocytes creates thin gas-exchange surfaces (Caldeira, et al. 2021). Surfactant production starts at 24 weeks, but only reaches protective levels at 32 weeks, improving survival significantly after this point (Tana, et al. 2023).

Alveolar Stage (36 weeks–8 years):
Immature alveoli bud from saccules, and secondary septa divide sacculi into alveoli (Campbell and Sapra 2023). Most septation occurs in the first 6 months, continuing until 3 years. Capillary fusion reduces diffusion distance, enhancing gas exchange. Lung growth until 3 years is due to increased alveoli number; afterward, both number and size increase, with full maturity reached at ~8 years (Campbell and Sapra 2023).

Function of the Lung
The pulmonary system extracts oxygen for cellular respiration and expels carbon dioxide and other byproducts. Oxygen is essential for ATP generation, while carbon dioxide is eliminated via breathing (Brinkman, et al. 2018). The respiratory tract includes the nose, throat, oral cavity, bronchi, trachea, and lungs. The lungs are separated into 5 lobes—3 right and 2 left—composed of numerous alveoli, where gas exchange occurs by diffusion into arterioles (Brinkman, et al. 2018).

PHYSIOLOGY OF THE LUNG

Pulmonary Hypoplasia
Pulmonary hypoplasia results from insufficient lung fluid through pregnancy. Lung fluid is secreted by epithelial cells from six weeks, contributing to lung expansion and development through fetal breathing movements and peristaltic shifts (Szoták-Ajtay, et al. 2020). Low fluid volume reduces airway branching and terminal sac formation, impairing lung structure. Causes include limited pleural cavity expansion, congenital diaphragmatic hernia, chest wall defects, and oligohydramnios due to renal agenesis (Yaremenko, et al. 2024).

Surfactant Function and Deficiency
Surfactant deficiency is the leading cause of NRDS. Surfactants, secreted by type II pneumocytes, prevent alveolar collapse by lowering surface tension. It is 90% phospholipids (phosphatidylcholine, phosphatidylglycerol, phosphatidylethanolamine) and 10% proteins. Surfactant proteins A and D (SPA, SP-D) regulate innate immunity (Zhuo, et al. 2021), while proteins B and C (SP-B, SP-C) stabilize tubular myelin, reducing surface tension (Floros, et al. 2021).
Surfactant production begins at 24 weeks but reaches adequate levels only in late gestation. NRDS risk is inversely related to gestational age: ~60% at <24 weeks, declining to <5% beyond 34 weeks. Clinical signs within the first hours of life include nasal flaring, tachypnea, cyanosis, hypoxia, hypercapnia, and respiratory difficulty (Norman, et al. 2022).

Neonatal Respiratory Distress Syndrome

Respiratory distress is a common neonatal problem, with about 10% requiring assistance at birth and 1% needing extensive resuscitation. It occurs in ~7% of neonates. Respiratory disorders are the leading cause of early newborn mortality (zero to seven days) and admission to special care units, with affected neonates being two to four times more likely to die than those with no distress (Enyew, et al. 2022). Symptoms involve tachypnea, cyanosis, nasal flaring, grunting, and retractions, while apnea, irregular (seesaw) breathing, bradypnea, wheeze, stridor, and hypoxia may also occur. Causes include developmental defects such as tracheoesophageal fistula, bronchopulmonary sequestration, bronchogenic cysts, congenital diaphragmatic hernia, or pulmonary hypoplasia from oligohydramnios (Afify, et al. 2024). Transitional diseases include RDS, TTN, MAS, newborn pneumonia, and PPHN, as lungs continue maturing until 2–5 years (Danaoui, et al. 2023).

Bronchopulmonary dysplasia (BPD) influences up to thirty-two percent of preterm and fifty percent of very-low-birth-weight babies due to arrested alveolarization and oxygen/mechanical ventilation exposure (Tracy and Berkelhamer 2019).

CAUSES OF RDS

Pulmonary Surfactant Deficiency

The main cause of RDS is surfactant deficiency. Surfactant appears around 20 weeks' gestation, reducing surface tension and preventing alveolar collapse. Preterm birth is the most common cause, but mutations in SP-C, SP-B, and ABCA3 genes may also impair surfactant function (Nogee 2019).

Composition of Surfactant

Surfactant is 90% lipids (mainly phospholipids) and 10% proteins (Ji, et al. 2021).

Lipids: About 70% are phosphatidylcholine, of which 60% is dipalmitoylphosphatidylcholine, the main surface tension-lowering agent (Fan, et al. 2023).

Proteins: Four specific proteins—SP-A, SP-C, SP-B, and SP-D—have distinct roles (Depicolzuane, et al. 2022).

SP-A, SP-D: Hydrophilic collectins, key in host defense and macrophage-mediated clearance; SP-A levels rise with corticosteroids (Korkmaz and Traber 2023).

SP-B: Essential for lipid absorption; deficiency is rare but lethal. Present in therapeutic surfactants (Acosta-Rivera, et al. 2021).

SP-C: Deficiency causes progressive pulmonary fibrosis and emphysema; mice models confirm later interstitial lung disease (Acosta-Rivera, et al. 2021).

SP-D: Regulates immunity; absence leads to lipid accumulation and emphysema. Recombinant SP-D reduces lung inflammation in preterm lambs (Casals, et al. 2018).

SP-B and SP-C act together to optimize lipid spreading and alveolar stability (Acosta-Rivera, et al. 2021).

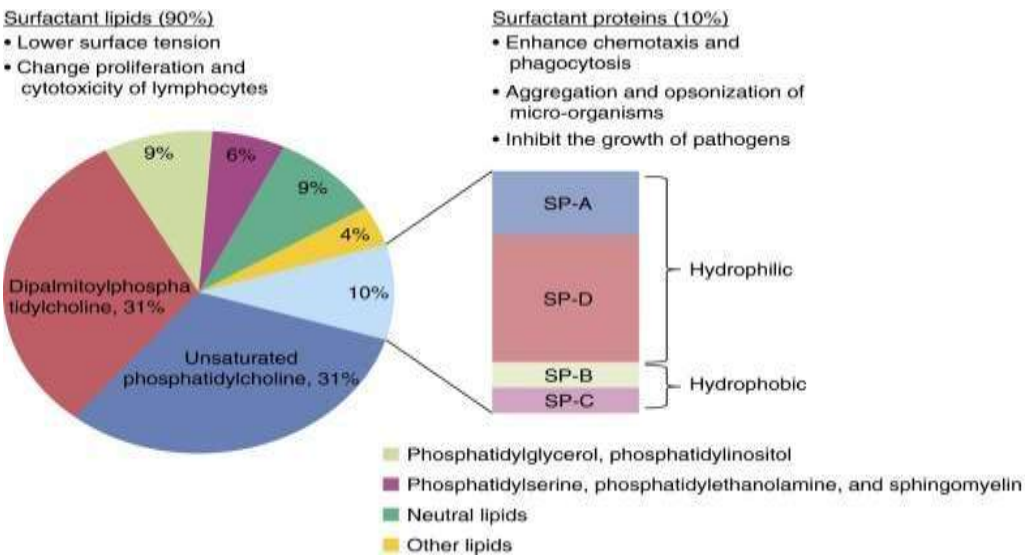

Figure 2: Surfactant composition and function—90% lipid, 10% protein (Han and Mallampalli 2015).

Surfactant Life Cycle

Alveolar type II cells (˜7% of epithelial surface) synthesize surfactant from dietary substrates. It is processed in the ER (2), Golgi (3), and stored in lamellar bodies (4). Secretion occurs via exocytosis (5), forming tubular myelin (6) and spreading as a surfactant monolayer (7). Lipids are recycled (8′) or degraded (8), while SP-A, SP-B, and SP-D are also produced in club cells (Han and Mallampalli 2015).

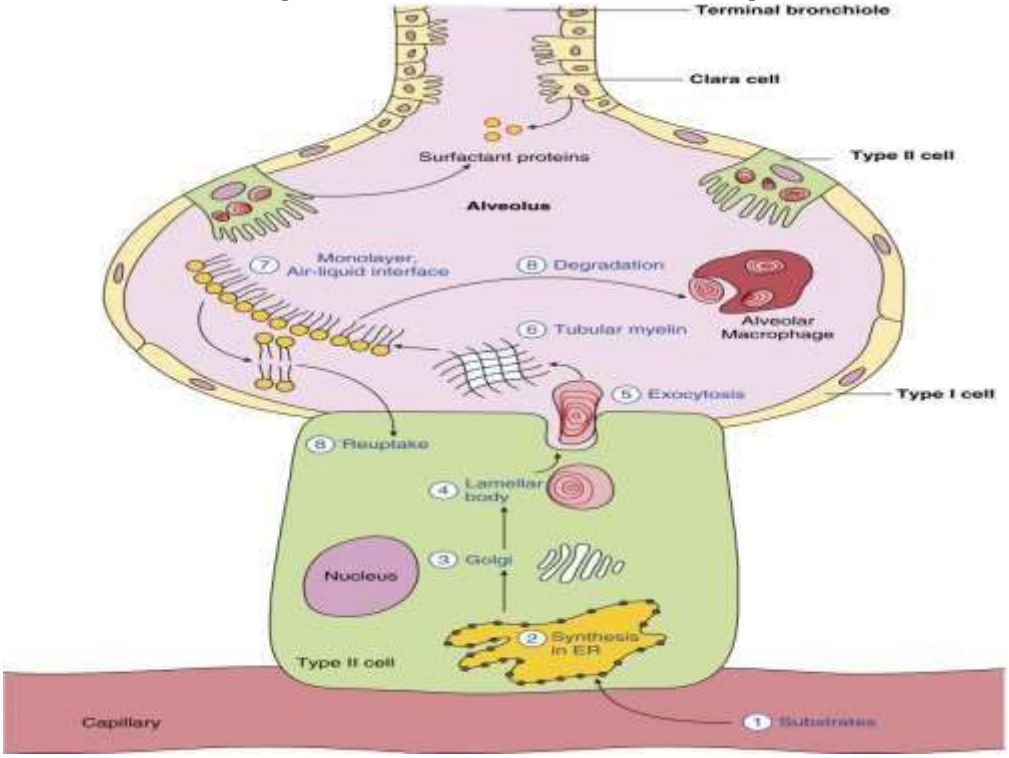

Figure 3: Surfactant synthesis, secretion, and recycling process.

Function of Surfactant

Surfactant has 3 main functions: lowering surface tension at the air–liquid interface to avoid alveolar collapse, killing or inhibiting pathogens, and modulating immune responses (Ji, et al. 2021). At endexpiration, alveolar surface tension must remain <2 mN/m to avoid collapse. This is achieved by DPPC, which reduces surface tension to <1 mN/m during compression. These biophysical properties form the basis of exogenous surfactant therapy, which improves neonatal RDS outcomes (Babbs 2019).

FACTORS INFLUENCING SURFACTANT IN NEWBORNS

Phospholipids and Gestational Age

Surfactant deficiency, particularly in phospholipids, is the main cause of RDS. Phosphatidylcholine (PC) is insufficient before 35 weeks, explaining the high incidence of RDS in preterms (Wang, et al. 2021). After 35 weeks, lecithin levels rise in amniotic fluid, while phosphatidylglycerol (PG), normally appearing after 36 weeks, is absent in preterm infants with RDS. Although survival is possible before 35 weeks, RDS in term infants is rare (Dumpa, et al. 2023).

Genetic Factors

Monozygotic twins show higher RDS risk compared to dizygotic twins, suggesting genetic predisposition.
Rare recessive mutations in SP-B cause lethal neonatal RDS, while SP-C mutations (0.1% prevalence) lead to interstitial lung disease beyond the first month. Deletions in ABCA3, found in ~4% of the population, are also linked, though the fatal RDS incidence is unclear (Yadav, et al. 2020).

Incidence of Neonatal RDS

RDS is the most common respiratory disease in preterm babies, affecting ~24,000 infants annually in the U.S. It is a major complication of prematurity, with risk factors including prematurity, white race, low birth weight, maternal diabetes, male sex, late-preterm delivery, perinatal hypoxia/ischemia, and cesarean delivery without labor (Li, et al. 2019).
The incidence is inversely related to gestational age: 98% at 24 weeks, 5% at 34 weeks, and <1% at 37 weeks (Wondie, et al. 2023).

Pathophysiology of Neonatal RDS

RDS results from surfactant deficiency, which increases alveolar surface tension and reduces lung compliance. According to LaPlace law (P=2T/R), where T = surface tension, P = pressure, and R = alveolar radius, higher surface tension demands greater pressure to keep alveoli open. Insufficient surfactant causes widespread atelectasis, impaired gas exchange, and hypoxemia (Yadav, et al. 2020). Atelectasis triggers epithelial damage, cytokine-mediated inflammation, and pulmonary edema, with protein-rich fluid inactivating surfactant. Mechanical ventilation, though lifesaving, can worsen damage via alveolar overdistension, oxidative stress, protein oxidation, and lipid peroxidation (Carvallo and Stevenson 2022).
Consequently, hypoxemia arises from hyperventilation, diffusion defects, V/Q mismatch, and shunting. Tissue hypoperfusion leads to anaerobic metabolism and lactic acidemia (Lim, et al. 2023).

Clinical Manifestations

Respiratory distress syndrome (RDS) is a developmental disorder due to surfactant deficiency, presenting within minutes to hours after birth, and worsening over the first 48 h if untreated. Infants are usually preterm and exhibit tachypnea, nasal flaring, expiratory grunting, chest retractions, and cyanosis due to right-to-left

shunting. Breath sounds are reduced, pulses weak, urine output low in the first 24–48 h, and edema may occur (Abdelbaseer et al., 2023).

Grading of Severity

Severity is evaluated by Silverman-Anderson score (Table 1) and Downes' score (Table 2). A score >6 indicates impending respiratory failure. Increasing FiO_2 requirements to maintain SpO_2 90–92% (preterm) or 94–96% (term) is a sensitive marker (Hedstrom, et al. 2018).

Table 1. Silverman Anderson retraction score (Silverman and Andersen 1956).

Score	Upper chest retraction	Lower chest retraction	Xiphoid retraction	Nasal dilatation	Grunt
0	Synch	None	None	None	None
1	Lag on inspiration	Just visible	Just visible	Minimal	Stethoscope only
2	See-Saw	Marked	Marked	Marked	Naked ear

A score of >6 is indicative of impending respiratory failure.

Table 2. Downes' score (Wood, et al. 1972)

Score	Respiratory rate	Cyanosis	Air entry	Grunt	Retraction
0	<60/min	Nil	Normal	None	Nil
1	60-80/min	In room air	Mild	Ausc with stethoscope	Mild
2	>80/min	In >40%	Marked	Audible with naked ear	Moderate

A score of >6 is indicative of impending respiratory failure.

Diagnosis

Diagnosis is based on progressive respiratory failure in preterm infants, rising oxygen demand, and chest imaging showing diffuse reticulogranular ground glass appearance with air bronchograms (Bulimba, et al. 2022).

Investigations

Essential tests include chest radiograph, ABG (Table 3), sepsis screen (CRP, ESR, WBC, smear, cultures), blood glucose, calcium, and hematocrit (Yadav, et al. 2020). ABG scoring >3 indicates need for CPAP/ventilation; pH <7.2 with $PaCO_2$ >60 mmHg or PaO_2 <50 mmHg suggests respiratory failure. Additional findings: hypoxemia, hypercarbia, and occasionally hyponatremia (Silveira Neves, et al. 2024).

Table 3. ABG score (Mathai, et al. 2007).

	Points			
	0	1	2	3
paO2 mmHg	> 60	50-60	< 50	< 50
pH	> 7.3	7.20-7.29	7.1-7.19	< 7.1
paCO2 mmHg	< 50	50-60	61-70	> 70

Score of > 3 suggestive of ventilatory support requirement

Chest Imaging

Chest X-ray shows low lung volume, diffuse ground glass with air bronchograms, and sometimes edema; pneumothorax occurs later (Hassan et al., 2025). Ultrasound demonstrates lung consolidation with air bronchograms, pleural line abnormalities, B-lines, and effusions in 15–20% cases (Khalil et al., 2025).

Prevention

Risk declines with advancing gestation until 38 wks; at 37 wks risk is threefold higher than 39–40 wks. Antenatal corticosteroids (24–34 wks, sometimes 35–36 wks) reduce RDS, while limiting elective cesarean helps (Dixon, et al. 2018).

Management

Management combines antenatal corticosteroids, respiratory support, surfactant therapy, and supportive care (Costa, et al. 2024):

Monitoring: Maintain PaO_2 50–80 mmHg, $PaCO_2$ 40–55 mmHg, pH >7.25; SpO_2 target 90–95%.

CPAP: Early CPAP reduces mortality, BPD, and need for surfactant.

NIPPV: Superior to CPAP in reducing extubation failure.

HFNC: Inferior to CPAP.

Mechanical ventilation: Indicated for pH <7.2, $PaCO_2$ >60–65 mmHg, PaO_2 <50 mmHg, or FiO_2 >0.40.

Surfactant therapy: Intratracheal surfactant (beractant, poractant alfa, calfactant, or synthetic) within 30–60 min improves outcomes; FiO_2 thresholds: >0.3 (immature) and >0.4 (mature). Less invasive surfactant administration (LISA) lowers BPD and mortality.

Supportive care: Caffeine for apnea, optimal fluids/electrolytes, nutrition, thermoregulation, transfusions, PDA and infection management (Sleem et al., 2025).

Fetal Lung Maturity Testing

Fetal lung maturity (FLM) is a crucial milestone determining neonatal survival, reflecting structural and biochemical lung development required for independent respiration. Assessment guides clinical decisions, especially in preterm delivery (Antsaklis and Theodora 2021).

Fetal Lung Development

Human lung development begins at 3 weeks of gestation and continues until 8 years. Alveolarization starts between weeks 24–38, when type II pneumocytes begin surfactant synthesis. Development has four stages: glandular (5–16 weeks, bronchi/bronchioles formation), canalicular (16–24 weeks, bronchioles and alveolar ducts), terminal sac (24 weeks–childhood, terminal sacs and capillary proliferation), and alveolar (birth–8 years, capillary–alveolar thinning) (Jung and Fraser 2020). Surfactant appears in lamellar bodies at 20–24 weeks, but sufficient levels accumulate later, explaining the risk of RDS in premature infants (Possmayer, et al. 2023).

Pulmonary Surfactant

Produced by type II pneumocytes and stored in lamellar bodies, surfactant secretion into amniotic fluid increases from week 32. It lowers surface tension at the air–liquid interface, preventing alveolar collapse (Soncini, et al. 2023). Deficiency causes neonatal RDS with atelectasis, hypoxia, hypercapnia, and acidosis (Thandaveshwara, et al. 2024).

Fetal Lung Maturity Tests

Accurate evaluation of lung maturity is essential for prognosis and reducing unnecessary corticosteroid exposure, which carries risks such as neonatal hypoglycemia and reduced birth size. Tests assess amniotic fluid or imaging findings (Ahmed, et al. 2021).

Biochemical Tests

L/S ratio: L/S > 2.0 indicates maturity.
Phosphatidylglycerol assay: late surfactant marker, less affected by contamination.
Lamellar Body Count (LBC): automated, quantitative, supported by CLSI C58.
Fluorescent Polarization (FLM): surfactant/albumin ratio, precise, automated. Clements' test: qualitative, fast, inexpensive, but high false-negative rate

Imaging

QuantusFLM ultrasound: machine learning–based texture analysis, non-invasive, promising. MRI: detailed but costly and impractical for routine use (Ahmed, et al. 2021).

Table 4: Summary of Fetal lung maturity Testing

Method	Sample Type	Principle	Advantages	Limitations
Lecithin-Sphingomyelin Ratio	Amniotic fluid	Ratio of surfactant phospholipids	Wellestablished, predictive	Requires amniocentesis, affected by contamination
Phosphatidylglycerol Assay	Amniotic fluid	Presence of late surfactant phospholipid	Less affected by contamination	Requires amniocentesis
Lamellar Body Count	Amniotic fluid	Quantification of surfactantcontaining bodies Surfactant to albumin ratio via fluorescence	Rapid, automated	Requires standardized protocols
Fluorescent Polarization Test	Amniotic fluid, vaginal fluid		Automated, precise	Affected by blood/meconium
Clements' Test	Amniotic fluid	Qualitative surfactant detection	Fast, inexpensive	High false negatives
Quantitative Ultrasound (QuantusFLM)	Ultrasound imaging	Texture analysis of fetal lungs	Non-invasive, rapid	New, requires further validation
Magnetic Resonance Imaging	Imaging	Detailed lung structure visualization	Detailed imaging	Expensive, less practical

Clinical Guidelines

FLM testing before 32 weeks or when early delivery is mandated is not recommended. It is indicated only before scheduled delivery <39. In diabetic mothers, RDS incidence is higher; however, with good glycemic control and accurate dating, testing is not routinely required (Ginoudis, et al. 2024).

Clinical Value and Outcomes

Despite diagnostic sensitivity, FLM testing has declined since it does not consistently improve neonatal outcomes. Advances in antenatal corticosteroids and surfactant therapy reduced RDS-related mortality, though preterm infants remain at risk of NICU admission and higher care costs (Bhakta, et al. 2023).

Fetal Pulmonary Artery Doppler

Predicting fetal lung maturity (FLM) is essential to anticipate complications such as neonatal respiratory distress syndrome (RDS) or death (Fasad et al., 2025). The standard method, amniocentesis, carries a 0.7% risk of complications, including preterm labor, PPROM, placental abruption, and fetomaternal hemorrhage. A non-invasive alternative is desirable. Studies have demonstrated a correlation between gestational age (GA), pulmonary artery Doppler waveforms (acceleration time/ejection time, At/Et), and FLM in amniotic fluid, suggesting its predictive value (Antsaklis and Theodora 2021).

Doppler in Pregnancy

Doppler sonography evaluates fetal well-being and circulation, identifying at-risk fetuses and reducing morbidity and mortality. It is particularly valuable in IUGR, alloimmunization, multiple pregnancies, and pre-eclampsia prediction when used in the 2nd trimester (Souidan and Abdel Salam 2022).

Fetal Pulmonary Artery Doppler

RDS is a major neonatal cause of morbidity and mortality. Doppler evaluation of the fetal main pulmonary artery (MPA) assesses vascular impedance, which correlates with GA, amniotic fluid FLM tests, and neonatal outcomes. The At/Et ratio specifically links to GA and FLM results (Keshuraj, et al. 2022). Reference values exist for systolic/diastolic velocities, PI, RI, acceleration, and ejection times across gestation (Sosa-Olavarria, et al. 2019).

In neonates and adults, Doppler estimation of pulmonary artery pressure has shown strong accuracy. Dabestani et al. proposed the formula: Pulmonary artery pressure = 90 – (0.62 × acceleration time), with excellent correlation (R = 0.98) to invasive measurement (Dabestani, et al. 1987).

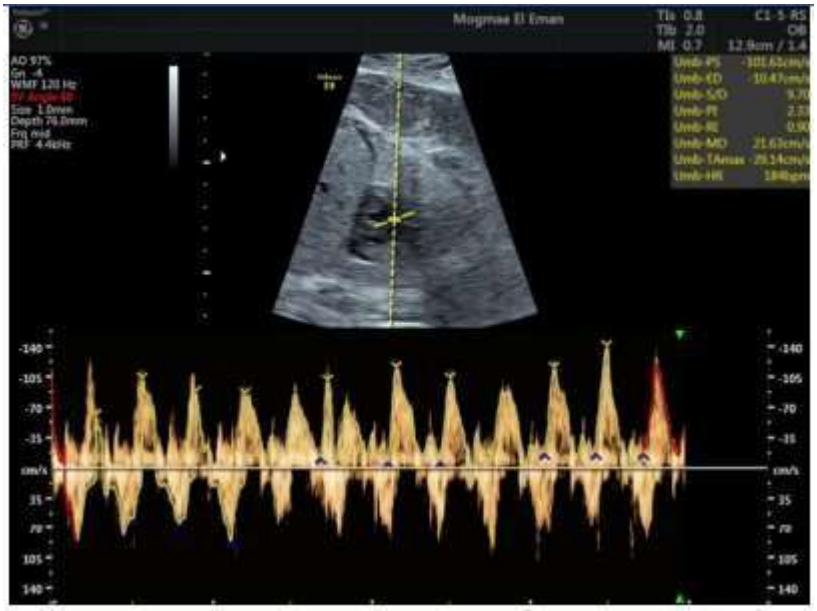

Figure 6: fetal pulmonary artery Doppler (Alkashty, et al. 2021).

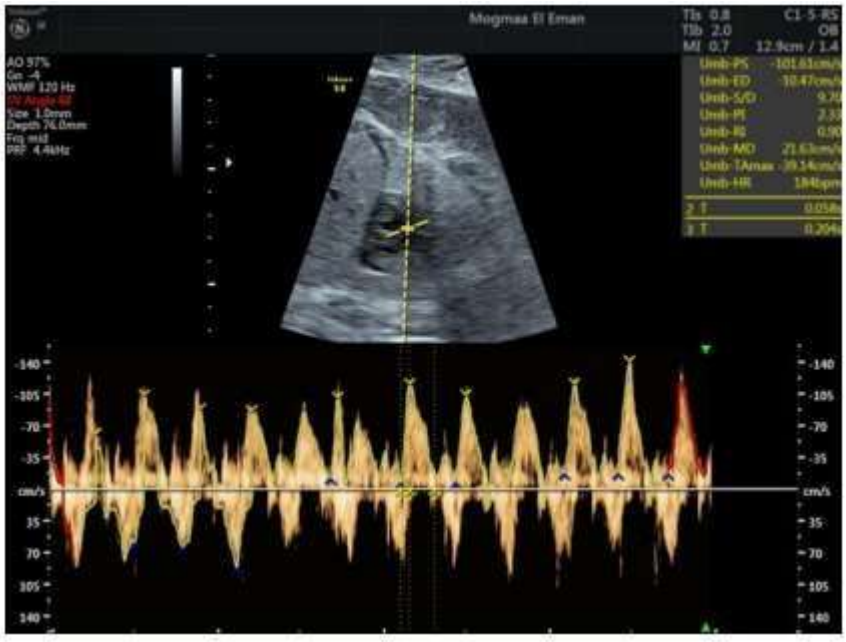

Figure 7: fetal pulmonary artery AT/ET (Alkashty, et al. 2021).

Technique

Ultrasound was performed in supine pregnant women, assessing fetal biometry, amniotic fluid index, and cardiac views (three-vessel, outflow tracts, four-chamber). The MPA was sampled midway between pulmonary valve and bifurcation, with gate size 3 mm and insonation angle 15°. The MPA Doppler waveform showed a "spike and dome" pattern with systolic peak, small diastolic notch, and minimal reversed flow. Measurements were averaged over three cycles. Variables included S/D ratio, PSV, PI, RI, and At/Et ratio, calculated as acceleration time (At) ÷ ejection time (Et) (Bedeer, et al. 2024).

Diagnosis of Neonatal RDS

At birth, delivery mode and sex were recorded. Neonatal birth weight and Apgar scores (1 and 5 min) were noted. RDS diagnosis required tachypnea, retractions, or nasal flaring, oxygen ≥0.4 for ≥24 h, and typical chest X-ray findings (reticulogranular pattern, air bronchograms, ground glass). NICU admission duration was also recorded (Eldeeb, et al. 2023).

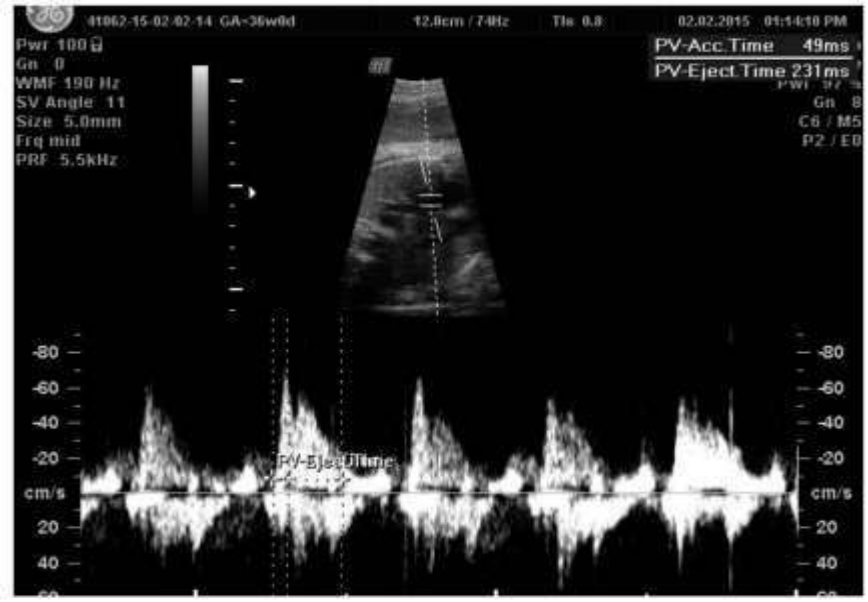

Figure 8. Measurement of the main pulmonary artery (MPA) acceleration time and ejection time. The acceleration time/ejection time (At/Et) ratio can then be calculated (Moety, et al. 2015).

Predictive Value

Fetal pulmonary artery indices demonstrate high sensitivity and specificity in predicting FLM and RDS. The At/Et ratio effectively distinguished RDS from non-RDS cases; however, using it alone reduces specificity across gestational ages. It is best applied alongside other predictors (Khalifa, et al. 2021).

CONCLUSION

Fetal pulmonary artery Doppler is a valuable, non-invasive tool for predicting fetal lung maturity and assessing the risk of neonatal respiratory distress syndrome (RDS). Unlike amniocentesis, which carries procedural risks, Doppler evaluation safely measures pulmonary vascular impedance and hemodynamic changes, providing insight into fetal well-being. The acceleration time to ejection time (At/Et) ratio of the main pulmonary artery correlates strongly with gestational age, lung maturity, and neonatal outcomes; a lower ratio indicates immaturity and higher RDS risk, while higher ratios reflect better pulmonary function. Additional indices such as resistance index, pulsatility index, systolic/diastolic ratio, and peak systolic velocity further enhance diagnostic value. Although the At/Et ratio alone may not provide complete specificity across all gestational ages, when combined with other clinical predictors, it significantly improves diagnostic accuracy. By enabling early identification of at-risk fetuses, fetal pulmonary artery Doppler can guide timely interventions, reduce neonatal morbidity and mortality, and ultimately optimize perinatal outcomes.

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