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Narrative review

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Neonatal respiratory diseases associated with failure of cardiopulmonary transition: a narrative review

Abstract

The neonatal period is characterized by marked physiological vulnerability and accounts for a substantial proportion of childhood morbidity and mortality, particularly during the first days of life. Cardiopulmonary transition involves complex physiological changes, including lung aeration, clearance of fetal lung fluid, progressive reduction in pulmonary vascular resistance, increased pulmonary blood flow, and functional closure of the fetal shunts. Failure or delay in these processes may result in a spectrum of respiratory, cardiovascular, and systemic disorders. This narrative review summarizes the physiological mechanisms underlying neonatal cardiopulmonary transition. It discusses the main neonatal diseases associated with impaired transition, including transient tachypnea of the newborn, respiratory distress syndrome, meconium aspiration syndrome, and persistent pulmonary hypertension of the newborn. The article discusses their pathophysiological mechanisms, clinical manifestations, radiographic findings, and current management strategies, emphasizing early recognition, appropriate respiratory and hemodynamic support, and evidence-based care.

Keywords: newborn, neonatal diseases, cardiopulmonary transition, respiratory distress, respiratory failure

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Introduction

The neonatal period is marked by significant physiological vulnerability, particularly during the first days of life, when newborns undergo profound cardiopulmonary and systemic changes to adapt to extrauterine life.[1] Failure or maladaptation of these processes is associated with several neonatal morbidities and may contribute to adverse outcomes, including death in severe cases.[2]

Among the physiological changes occurring during the neonatal period, cardiovascular and pulmonary adaptations are particularly important. During fetal life, circulation is characterized by high pulmonary vascular resistance and relatively low systemic vascular resistance, with right-to-left blood flow through the ductus arteriosus and foramen ovale. At birth, lung aeration and the onset of pulmonary gas exchange initiate a complex transition involving a rapid decrease in pulmonary vascular resistance, a marked increase in pulmonary blood flow, changes in ventricular loading conditions, and progressive functional closure of the fetal shunts.[3,4]

Successful cardiopulmonary transition depends on coordinated lung aeration, pulmonary vascular adaptation, cardiac function, and adequate oxygenation and ventilation. Gestational age, pulmonary maturity, perinatal conditions, neurological function, and the quality of care at birth may influence this process.[5,6] Failure or delay in one or more components of this transition may compromise pulmonary gas exchange, cardiac output, and systemic perfusion, contributing to several common neonatal disorders.

This narrative review describes the principal respiratory and cardiovascular consequences associated with impaired cardiopulmonary transition in newborns, focusing on transient tachypnea of the newborn (TTN), respiratory distress syndrome (RDS), meconium aspiration syndrome (MAS), and persistent pulmonary hypertension of the newborn (PPHN), as well as the systemic consequences of perinatal hypoxia.

The newborn without effective spontaneous breathing at birth

At birth, approximately 10% of newborns require some form of assistance to establish effective breathing, approximately 5% to 10% require positive-pressure ventilation, and a much smaller proportion require advanced resuscitation, including chest compressions and/or medications.[7,8] The need for respiratory and resuscitative interventions increases as gestational age and birth weight decrease. In term newborns, cesarean delivery, particularly when performed before the onset of labor, is independently associated with an increased need for respiratory support at birth.[8]

In this context, appropriate delivery-room care plays a central role in ensuring a safe transition from intrauterine to extrauterine life. Neonatal resuscitation is a time-critical intervention, and effective ventilation is the cornerstone of resuscitation for newborns who do not establish adequate spontaneous breathing. Respiratory failure, primarily resulting from inadequate lung aeration and ventilation, is the most important immediate mechanism leading to neonatal deterioration at birth.[9] Most newborns who require respiratory assistance respond to effective positive-pressure ventilation. Rapidly establishing adequate ventilation in the first minutes after birth highlights the importance of appropriately trained healthcare professionals, effective teamwork, and adherence to evidence-based neonatal resuscitation guidelines. International organizations, including the International Liaison Committee on Resuscitation (ILCOR), and national neonatal resuscitation programs provide current recommendations.[10,11]

Neonatal consequences associated with failure of cardiopulmonary transition

Major physiological events in neonatal cardiopulmonary adaptation include lung aeration, clearance of fetal lung fluid, reduced pulmonary vascular resistance via pulmonary vasodilation, establishment of functional residual capacity, increased pulmonary

blood flow, and progressive functional closure of the foramen ovale and ductus arteriosus.[12,13] Adequate lung aeration reduces pulmonary vascular resistance, increases pulmonary blood flow, and facilitates effective gas exchange. At the same time, changes in pulmonary and systemic vascular resistance alter cardiac loading conditions and promote the transition from fetal to neonatal circulation.[12,14]

Failure or delay in this adaptation may impair oxygenation, increase work of breathing, alter pulmonary vascular resistance, reduce cardiac output, and compromise systemic tissue perfusion. The resulting clinical spectrum ranges from transient respiratory disorders to severe respiratory and cardiovascular failure.

From a respiratory perspective, the principal disorders associated with impaired postnatal pulmonary adaptation include TTN, RDS, and MAS. PPHN is an important cardiovascular consequence of persistently elevated pulmonary vascular resistance and may occur as a primary disorder or secondary to pulmonary disease, hypoxia, or systemic conditions.[14,15] Severe or prolonged hypoxemia may also contribute to perinatal asphyxia and hypoxic-ischemic encephalopathy.[16,17]

The initial assessment of a newborn with respiratory distress should include a detailed perinatal history and physical examination, continuous oxygen saturation monitoring (preferably with preductal and postductal measurements when PPHN is suspected), and blood gas analysis when clinically indicated. Chest radiography may help differentiate common neonatal respiratory disorders. Lung ultrasonography can provide additional information in selected clinical settings. Echocardiography is essential when congenital heart disease or PPHN is suspected, particularly when hypoxemia is persistent or disproportionate.

Characteristics of the main cardiopulmonary diseases associated with impaired postnatal adaptation

Transient tachypnea of the newborn

TTN is one of the most common causes of respiratory distress in term and late-preterm newborns and occurs particularly frequently after cesarean delivery without labor. Its pathophysiology primarily involves delayed clearance of fetal lung fluid after birth.[18] Failure or delay in pulmonary fluid clearance increases interstitial fluid and, in some cases, leads to persistence of fluid within the alveolar spaces. This may decrease pulmonary compliance and increase airway resistance, contributing to tachypnea and increased work of breathing.[19]

Clinical manifestations usually develop within the first few hours after birth. They are characterized primarily by tachypnea, which may be accompanied by mild to moderate respiratory distress, including nasal flaring, grunting, and chest retractions. Chest radiography typically shows pulmonary hyperinflation, prominent perihilar interstitial or vascular markings, and fluid within the interlobar fissures (Figure 1).

Management is primarily supportive and includes clinical monitoring, maintenance of adequate temperature and glucose levels, and supplemental oxygen as needed. Noninvasive respiratory support, particularly nasal CPAP, may be used when respiratory distress or oxygen requirements are significant. Invasive mechanical ventilation is rarely necessary. The clinical course is generally self-limited, with substantial improvement typically occurring within 24 to 72 hours.[20] Routine administration of antibiotics, surfactant, diuretics, or bronchodilators is not recommended for uncomplicated TTN. Antibiotic therapy may nevertheless be considered when the clinical presentation does not allow early-onset neonatal infection to be reasonably excluded.

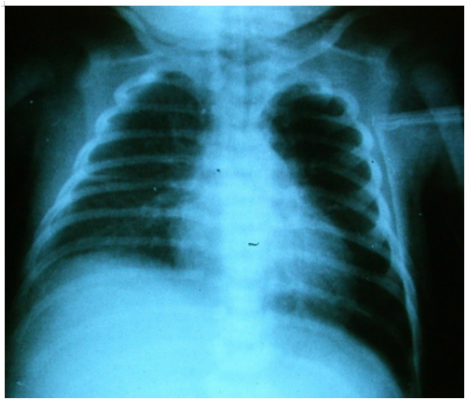

Figure 1 Chest radiograph in transient tachypnea of the newborn. The radiograph demonstrates pulmonary hyperinflation, prominent perihilar vascular and interstitial markings, and fluid within the interlobar fissures, findings compatible with transient tachypnea of the newborn.

Source: Authors' personal collection.

Respiratory distress syndrome

RDS is primarily caused by pulmonary surfactant deficiency and is most strongly associated with prematurity. Surfactant reduces alveolar surface tension and helps maintain alveolar stability at low lung volumes. Both quantitative and qualitative surfactant deficiency increase alveolar surface tension, promoting alveolar collapse, diffuse atelectasis, reduced pulmonary compliance, and impaired functional residual capacity. These abnormalities lead to ventilation-perfusion mismatch, hypoxemia, and increased work of breathing.[21] RDS is particularly common in very and extremely preterm infants, and its incidence increases as gestational age decreases. Antenatal corticosteroid administration in women at risk of preterm birth reduces the risk.[22,23] In addition to prematurity, infants born to mothers with diabetes mellitus may be at increased risk of RDS. Fetal hyperinsulinemia has long been proposed as a mechanism contributing to delayed pulmonary maturation and impaired surfactant production.[24]

Clinical manifestations typically develop shortly after birth and may already be evident in the delivery room. Typical findings include tachypnea, expiratory grunting, nasal flaring, and intercostal and subcostal retractions. As the disease progresses, oxygen requirements rise, and respiratory failure may develop. [22] The typical chest radiographic appearance includes diffuse ground-glass opacities, air bronchograms, and reduced lung volume (Figure 2).

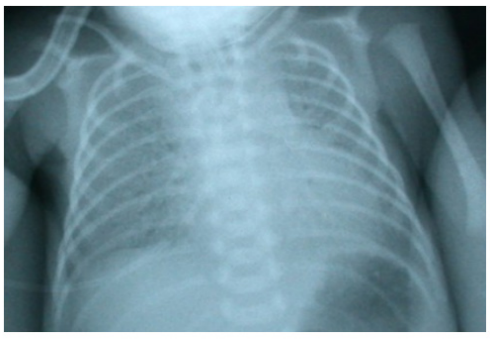

Figure 2 Chest radiograph in respiratory distress syndrome. Chest radiograph demonstrating a diffuse reticulogranular pattern associated with air bronchograms and reduced lung volume, characteristic of neonatal respiratory distress syndrome.

Source: Authors' personal collection.

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Initial respiratory management should prioritize early noninvasive respiratory support, particularly nasal CPAP, when clinically appropriate. In preterm infants, early CPAP can reduce the need for invasive mechanical ventilation. Carefully titrate oxygen to target saturation ranges to prevent both hypoxemia and excessive oxygen exposure.[25]

Exogenous surfactant is indicated when clinical and respiratory parameters suggest significant surfactant deficiency despite noninvasive respiratory support. Current European guidelines recommend considering surfactant administration in preterm infants with worsening respiratory distress and increasing oxygen requirements while receiving CPAP, with an oxygen concentration of about 30% as one criterion in appropriate clinical contexts.[25] Lung ultrasonography has emerged as a useful adjunct for identifying surfactant deficiency and predicting the need for surfactant therapy. However, its use should complement rather than replace clinical assessment and established respiratory criteria.[26] For spontaneously breathing preterm infants receiving CPAP, surfactant administration via a thin catheter is currently preferred to routine intubation and mechanical ventilation when the clinical condition permits. This approach is commonly referred to as less invasive surfactant administration (LISA) or minimally invasive surfactant therapy (MIST).

Additional measures include appropriate thermal management, early nutritional support, and caffeine therapy for preterm infants at risk of apnea. Current respiratory management strategies emphasize noninvasive support, lung-protective ventilation when invasive ventilation is necessary, and minimizing exposure to excessive oxygen concentrations.[25]

Meconium aspiration syndrome

MAS is associated with meconium in the amniotic fluid and with the development of respiratory disease in the newborn. Fetal hypoxemia may increase intestinal peristalsis and relax the anal sphincter, leading to meconium passing into the amniotic fluid. In severe fetal compromise, gasping respiratory movements may contribute to aspiration of meconium-stained amniotic fluid.[27,28] MAS is generally defined as respiratory distress in a newborn delivered through meconium-stained amniotic fluid, in the absence of a more likely explanation, with radiographic findings consistent with aspiration.[27] The severity of disease depends on several factors, including the amount and characteristics of aspirated meconium, the degree of airway obstruction, pulmonary inflammation, surfactant dysfunction, and the presence and severity of perinatal hypoxia.

Meconium aspiration can cause airway obstruction, chemical pneumonitis, alveolar inflammation, surfactant dysfunction, and ventilation-perfusion mismatch. The resulting pulmonary disease can range from mild respiratory distress to severe respiratory failure.[27]

Chest radiography typically shows bilateral, patchy, and heterogeneous pulmonary opacities, with alternating areas of hyperinflation and atelectasis. Air-leak syndromes, particularly pneumothorax, may result from uneven airway obstruction and increased ventilatory pressures. PPHN is also a major complication of severe MAS.[29]

Management is primarily supportive and should be guided by the severity of respiratory and cardiovascular compromise. Supplemental oxygen may suffice in mild cases, whereas more severe disease may require noninvasive or invasive mechanical ventilation. Routine tracheal suctioning of vigorous or non-vigorous infants born through meconium-stained amniotic fluid is not recommended solely because of meconium. Instead, initiate respiratory support based on the infant's clinical condition and response.[9,10]

Surfactant therapy may be considered for severe MAS, particularly when substantial oxygen requirements and impaired pulmonary compliance are present. Evidence suggests that surfactant therapy may reduce respiratory disease severity and the need to escalate respiratory support in selected patients. The routine use of antibiotics in MAS remains controversial. Consider antibiotics when clinical or laboratory findings suggest early-onset neonatal infection or bacterial pneumonia, rather than administering them solely because of meconium-stained amniotic fluid.

In severe cases, consider high-frequency ventilation. Inhaled nitric oxide may be indicated when significant PPHN accompanies MAS and hypoxemia persists despite optimized ventilation and lung recruitment (Figure 3).[30,31]

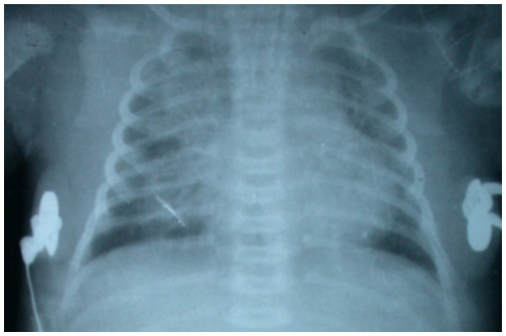

Figure 3 Chest radiograph in meconium aspiration syndrome. Chest radiograph demonstrating bilateral heterogeneous pulmonary opacities with alternating areas of hyperinflation and atelectasis, consistent with meconium aspiration syndrome.

Source: Authors' personal collection.

Persistent pulmonary hypertension of the newborn

PPHN results from failure of the normal postnatal decline in pulmonary vascular resistance, leading to persistent elevation of pulmonary arterial pressure and right-to-left shunting through fetal channels, including the ductus arteriosus and foramen ovale. This causes impaired pulmonary blood flow and significant hypoxemia.[32]

The pathophysiology of PPHN may involve pulmonary vascular hypoplasia, abnormal pulmonary vascular development, or maladaptation of a structurally developed pulmonary vascular bed. In addition to functional abnormalities in pulmonary vascular reactivity, structural remodeling may occur, including increased muscularization of the pulmonary arterioles.[32] PPHN may occur as a primary disorder or as a secondary condition. Major associated conditions include MAS, neonatal sepsis, pneumonia, respiratory distress syndrome, pulmonary hypoplasia, and perinatal asphyxia.[33]

Clinically, affected newborns may present with respiratory distress, labile oxygenation, severe or refractory hypoxemia, and a preductal-postductal oxygen saturation difference. However, the absence of a significant saturation difference does not exclude PPHN.[33]

Chest radiographic findings largely depend on the underlying pulmonary condition. In newborns with PPHN without substantial parenchymal lung disease, the lung fields may appear relatively clear, with reduced pulmonary vascular markings. Cardiac size may be normal or enlarged.[34] Echocardiography is the key diagnostic examination for suspected PPHN. It allows indirect assessment of

pulmonary pressures, right ventricular function, shunt direction, and the presence of congenital heart disease or other structural abnormalities that may mimic the clinical presentation.

Management of PPHN requires a multifaceted approach that includes optimizing oxygenation and ventilation, maintaining adequate lung recruitment, stabilizing hemodynamics, correcting metabolic abnormalities, and treating the underlying disease. Inhaled nitric oxide (iNO) is a selective pulmonary vasodilator and an important therapy for term and near-term newborns with hypoxemic respiratory failure associated with PPHN. Nitric oxide diffuses into pulmonary vascular smooth muscle cells, activates soluble guanylate cyclase, and increases cyclic guanosine monophosphate (cGMP) concentrations. This pathway decreases intracellular calcium availability and promotes vascular smooth muscle relaxation and pulmonary vasodilation.[31] The use of iNO should be guided by the clinical and echocardiographic context. Its role in extremely preterm infants remains more selective because evidence for routine use in this population is limited.[31]

For refractory hypoxemic respiratory failure despite optimized conventional therapy, high-frequency ventilation and other advanced cardiopulmonary support strategies may be considered. Patients with PH may require inotropes and other vasodilators. A detailed discussion of the indications for these medications is beyond the scope of this review. Extracorporeal membrane oxygenation (ECMO) may be required in selected patients with severe, refractory respiratory and cardiovascular failure when available and eligibility criteria are met.[31] Correction of potentially reversible factors, including metabolic acidosis, hypothermia, hypoglycemia, systemic hypotension, and inadequate oxygenation or ventilation, is an essential component of management.[35]

Conclusion

Neonatal cardiopulmonary transition is a complex, highly coordinated physiological process essential for successful adaptation to extrauterine life. Failure or delay can lead to respiratory, cardiovascular, and systemic complications, ranging from transient respiratory disorders to severe hypoxemic respiratory and circulatory failure. TTN, RDS, MAS, and PPHN are important clinical manifestations of impaired neonatal adaptation, although their underlying mechanisms differ substantially. Recognizing these mechanisms is essential for appropriate diagnosis and management.

Effective delivery-room care, particularly timely and technically adequate ventilation when indicated, remains fundamental to successful neonatal transition. Early identification of respiratory and hemodynamic abnormalities, appropriate use of noninvasive and invasive respiratory support, optimized oxygenation and perfusion, and treatment of underlying conditions are essential to improve outcomes.

Implementing evidence-based protocols, simulation-based training, and continuous professional education may substantially improve neonatal care quality and reduce neonatal morbidity and mortality. Table 1 summarizes the key characteristics of neonatal diseases associated with impaired cardiopulmonary transition.

Table 1 Main characteristics of neonatal diseases associated with impaired cardiopulmonary transition

Disease	Main pathophysiology	Typical onset	Main clinical features	Typical chest radiograph	Main management
TTN	Delayed clearance of fetal lung fluid	First hours of life	Tachypnea; mild-to-moderate respiratory distress	Hyperinflation; prominent perihilar markings; fluid in interlobar fissures	Supportive care; oxygen; CPAP when needed
RDS	Surfactant deficiency and alveolar instability	Early after birth	Progressive respiratory distress; increasing oxygen requirement	Diffuse reticulogranular/ground-glass pattern; air bronchograms; reduced lung volume	CPAP; surfactant when indicated; lung-protective ventilation
MAS	Meconium-induced airway obstruction, inflammation, and surfactant dysfunction	At birth or shortly thereafter	Variable respiratory distress; hypoxemia; severe cases may develop respiratory failure and PPHN	Bilateral heterogeneous opacities; alternating hyperinflation and atelectasis	Respiratory support; consider surfactant in severe disease; iNO when associated with significant PPHN
PPHN	Persistently elevated pulmonary vascular resistance and right-to-left shunting	First hours of life	Severe/labile hypoxemia; respiratory distress; possible preductal-postductal saturation difference	Normal or nonspecific findings, depending on underlying pulmonary disease	Optimize ventilation and hemodynamics; iNO in appropriate patients; advanced support/ECMO in refractory cases

Abbreviations: TTN, transient tachypnea of the newborn; RDS, respiratory distress syndrome; MAS, meconium aspiration syndrome; PPHN, persistent pulmonary hypertension of the newborn; CPAP, continuous positive airway pressure; iNO, inhaled nitric oxide; ECMO, extracorporeal membrane oxygenation.

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Conflicts of interest

The authors declare no conflicts of interest.

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