Fetal-to-neonatal transition: Part 2

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The transition from fetus to newborn is a process of rapid physiological adaptation that is sometimes delayed by complications. Most newborns complete the process of transition with little or no delay.

These infants may demonstrate normal transitional findings, including tachypnea and tachycardia, a soft heart murmur and fine crackles in the lungs as well as acrocyanosis for varying lengths of time after birth.

Prolonged or exaggerated signs of distress should lead the healthcare provider to carry out a thorough history and physical examination aimed at rapid initiation of interventions. In Part 1 of this series the physiology of transition was reviewed.

In Part 2 we will examine signs and symptoms that may assist the healthcare team identify the infant in need of further evaluation during the period of transition.

Introduction

As outlined in Part 1 of this series, the transition from fetal to neonatal life requires complex physiological changes which must occur in a short period of time.

A small percentage of neonates experience disturbances in the normal transitional process, displaying signs of mild-to-moderate respiratory distress, mild temperature instability or borderline blood sugars.

These infants must be observed closely to ensure that these signs are not indicative of underlying disease. Timely interventions which support thermal stability, adequate blood glucose levels and adequate oxygenation will prevent infants with a complicated transition from experiencing unnecessary morbidity or mortality [11].

This paper discusses common findings in transition that must be evaluated to differentiate infants experiencing a delayed transition from those with underlying disease processes.

Problems in transition

Infants with underlying pathology such as infection, respiratory or cardiac disease may initially exhibit the same symptoms as those with mild transition problems. However, infants with serious illnesses will deteriorate over time and may require increasing levels of support to maintain stability.

Clinical deterioration should raise a red flag with caregivers (see Table 1). Depending on the severity of the symptoms and the infant's risk factors, the healthcare team may elect to initiate medical treatment early in the transition period rather than wait for the symptoms to progress.

- Symptoms of greater than 2 hours' duration
- Worsening distress
- Congenital anomalies
- Abnormal muscle tone
- Central cyanosis
- Apnea in a near-term or term infant
- Moderate-to-severe respiratory distress: grunting, flaring, marked retractions and need for supplemental oxygen beyond 2 hours of age

TABLE 1: Red flags in transition

Maternal
• Diabetes • Hypertension • Cardiac or respiratory disease • Severe anemia • Shock • Infection or febrile illness
Antepartum
• Intrauterine growth restriction • Placenta previa, Abruptio placenta • Fetal-maternal hemorrhage • Malpresentation, • Multiple gestation • Pregnancy induced hypertension • Illicit or prescription drug exposure
Intrapartum
• Chorioamnionitis • Fetal distress • Prolapsed cord • Premature or prolonged rupture of membranes • Narcotic or magnesium sulphate administration • Malpresentation • Shoulder dystocia • Vacuum forceps or cesarean delivery • Presence of meconium stained amniotic fluid
Neonatal complications
• Prematurity • Congenital malformations • Postmaturity • Birth trauma

TABLE 2: Risk factors for problems in transition

The presence of risk factors increases the likelihood that an infant will experience a delayed or complicated transition. These risk factors may be maternal, or occur in the antepartum, intrapartum or immediate postpartum period.

Table 2 outlines some of the factors that place the infant at risk of problems in transition. The clinician must weigh the clinical presentation and history of the baby to determine the degree of intervention required.

Respiratory distress

Signs of respiratory distress include grunting, nasal flaring, retractions and tachypnea. Infants who present with these signs warrant careful examination, including a review of antepartum and intrapartum history.

Table 3 identifies the most common respiratory problems in the newborn. The presence of risk factors for sepsis or underlying respiratory disease suggests the need

for earlier, more aggressive intervention than might be warranted for an infant with no risk factors.

The distinction between normal or difficult transition and more significant respiratory disease is based on the severity and duration of symptoms [6]. Infants with mild Transient Tachypnea of the Newborn (TTN) will demonstrate signs of mild-to-moderate respiratory distress for 1-2 hours following delivery.

These infants may be tachypneic with respiratory rates of up to 100 breaths per minute. Intermittent grunting, acrocyanosis and increased mild work of breathing (nasal flaring and mild retractions) may also be present.

Blood gases usually demonstrate a mild mixed acidosis with carbon dioxide levels of up to 60 mmHg and supplemental oxygen may be required.

The neonate with more serious underlying pathology such as sepsis, meconium aspiration, pneumothorax, respiratory distress syndrome or persistent pulmonary hypertension will demonstrate more significant symptoms or symptoms which persist for a longer period of time.

This baby may have significant tachypnea with rates of up to 120 breaths per minute accompanied by grunting and moderate-to-severe retractions. In some cases, the infant may have slow respirations or episodes of apnea. Persistent grunting and central cyanosis, pallor or an underlying gray color may be present.

The need for supplemental oxygen will persist beyond the first few hours of life [6]. Other signs of significant illness include systemic hypotension, poor perfusion, hypotonia [9], severe metabolic or mixed acidosis, temperature instability, hypoglycemia and signs of cardiac compromise.

Caution is warranted in distinguishing those infants with temporary transitional problems from those with infection caused by Group B Streptococcal (GBS) pneumonia. Careful attention to risk factors and close

Disorder	Pathology	Risk factors
Transient tachypnea of the newborn (TTN)	Retained fetal lung fluid	Late preterm infant Delivery by cesarean section, especially with no laborInfants of diabetic mothers
Respiratory distress syndrome (RDS)	Surfactant deficiency and anatomic immaturity	Prematurity
Meconium aspiration	Chemical pneumonitis secondary to meconium Surfactant deactivation Ball and valve obstruction leading to air trapping	Term or postterm History of meconium stained amniotic fluid May accompany signs of fetal intolerance of labor
Pneumonia	Initiation of inflammatory cascade Secondary surfactant deficiency Systemic illness	Preterm Prolonged rupture of membranes Maternal Group B strep colonization Maternal urinary tract infection or febrile illness
Persistent pulmonary hypertension of the newborn (PPHN)	Failure of pulmonary vascular resistance to lower after birth, leading to continued right-to-left shunting, severe hypoxemia and acidosis	Late preterm/term infant History of meconium aspiration, sepsis, RDS, congenital diaphragmatic hernia and congenital heart diseas

TABLE 3: Newborn respiratory disorders

monitoring ensure prompt treatment of infants at risk as the clinical signs of GBS pneumonia may be quite subtle and difficult to distinguish from less serious problems such as TTN [1].

Persistent pulmonary hypertension of the newborn (PPHN) is a special case of a cardiopulmonary disorder occurring in term or near-term infants that is triggered by an insult such as hypoxia, hypotension or hypercarbia.

PPHN develops when the expected drop in pulmonary vascular resistance does not occur after birth. Pressure within the pulmonary vasculature remains elevated, leading to continued shunting of blood away from the lungs and across the foramen ovale and ductus arteriosus.

This results in hypoxia, which further aggravates constriction of the pulmonary vessels and ongoing shunting. A number of clinical conditions are known to precipitate PPHN. These include: meconium aspiration, sepsis, RDS, congenital diaphragmatic hernia and congenital heart disease. For a significant number of infants, the cause is unknown [6].

Infants with PPHN present with profound respiratory distress, acidosis and cardiovascular instability. Significant and rapidly progressive hypoxemia is present. For these infants, rapid intervention to reverse the cycle of hypoxia and hypertension is critical to the infant's recovery.

Extrapulmonary signs

In a newborn, non-pulmonary disorders often present with respiratory symptoms. In addition there may be specific symptoms such as cyanosis, pallor or a murmur that suggest the presence of a cardiovascular or neurologic problem.

Acrocyanosis, bluish discoloration of the extremities, is a normal finding in the first 24 hours of life [4]. Transient dusky episodes during crying are also considered normal in the period immediately following delivery.

By contrast, persistent central cyanosis (blue color of lips and mucous membranes), oxygen saturation that remains below 85 % in room air or the need for supplemental oxygen beyond 2 hours require immediate assessment [6].

The presence of central cyanosis signifies hypoxemia that may originate either in the lungs with inadequate delivery of oxygen to the alveoli or inadequate perfusion of the functioning gas exchange units (ventilation-perfusion mismatching) or as a result of cardiac dysfunction.

Cardiovascular problems that lead to cyanosis include congenital heart defects, shock, systemic hypotension or significant anemia that prevents adequate levels of oxygen from being delivered to the body tissues. Cyanosis may also result from non-cardiopulmonary causes such as methemoglobinemia, central nervous system depression or neuromuscular diseases.

Cyanosis is a late sign of distress as the infant may have a significant degree of hypoxemia before cyanosis becomes apparent [5]. Cyanosis only becomes visible in the presence of at least 4 mg/dL of unoxygenated (desaturated) hemoglobin; therefore polycythemic babies may look cyanotic at higher (even normal) O_2 saturation levels because they have more hemoglobin, while an anemic baby with low total hemoglobin concentration may be quite hypoxic before becoming visibly cyanotic.

Pallor and poor peripheral circulation may reflect shock, systemic hypotension or anemia. Acute or chronic blood loss is suggested by a history of maternal bleeding. Silent blood loss resulting from a retro-placental clot or fetal-maternal hemorrhage should also be considered.

Pallor may signify anemia resulting from intrauterine hemolysis or chronic blood loss or may be the result of peripheral vasoconstriction occurring because of infection, hypothermia or perinatal asphyxia.

Capillary refill time is a quick and simple test that can assist in evaluating the effectiveness of systemic

circulation. Capillary refill time is checked by gently pressing the skin over a firm surface such as the chest wall and counting the number of seconds for the normal skin color to return.

Capillary refill of greater than 3 seconds or blood pressure that falls outside the standards for the infant's gestational age indicate serious underlying conditions such as sepsis, hypoxia, cardiovascular compromise or central nervous system injury [6].

A heart murmur is a common finding in the newborn period, particularly in the first 24 hours following delivery. In asymptomatic infants, turbulent blood flow resulting from the closure of fetal shunts results in a functional or innocent murmur.

Any murmur accompanied by cyanosis, respiratory distress or signs of congestive heart failure cannot be assumed to be innocent and warrants urgent investigation. It is important to note that many infants with significant congenital heart disease are asymptomatic in the immediate newborn period and therefore ongoing vigilance is necessary.

Recent studies have suggested that it may be beneficial to screen all newborns with pulse oximetry to identify those with congenital heart disease [7, 12].

Abnormalities in heart rate and rhythm suggest compromised cardiovascular function. With hypoxia or low cardiac output, the adult heart normally compensates by increasing heart rate and/or stroke volume.

Because of the small, relatively thick, non-compliant neonatal heart, the baby is unable to increase stroke volume; therefore, to increase cardiac output, the baby must increase heart rate [8]. Persistent heart rates above 160 bpm may signal anemia, hypovolemia respiratory problems or congestive heart failure [4].

Mild heart-rhythm disturbances are not unusual in the immediate newborn period; however, infants with persistent or marked abnormalities in heart rhythm or those where rhythm disturbances are accompanied by other symptoms of distress merit an electrocardiogram to determine the clinical significance of the rhythm disturbance.

Neonates who present with persistent bradycardia (heart rate less than 80 bpm) may have a complete heart block as a result of maternal systemic lupus erythematous (SLE) [13]. If the bradycardia occurs mainly during periods of rest or deep sleep, it may be an indication of intrauterine hypoxia or sepsis [2].

Neurologic and metabolic abnormalities may also be present in cases of underlying disease. Abnormal tone is an important red flag in the newborn and may result from a wide range of problems, including hypoglycemia, sepsis, narcotic exposure or birth trauma.

Hypoxic-ischemic encephalopathy (HIE) resulting from a reduction in fetal oxygenation is manifested in a number of neurologic findings, including hypo- or hypertonia, seizures or coma [10].

Neonatal seizure disorders may present immediately after birth with abnormalities in tone and muscle activity as well as overt seizure activity. In neonates the symptoms of seizures are quite subtle and include apnea, eye deviations or blinking, staring, lip smacking, repetitive sucking or tongue movements. Arching or stiffening may be seen along with changes in heart rate and blood pressure.

Hypoglycemia is an important complication of transition which must be recognized and treated immediately to prevent morbidity. Classic symptoms of hypoglycemia include: jitteriness, diaphoresis, poor feeding, lethargy, apnea and, as a late sign, seizures.

However, hypoglycemic neonates are not always symptomatic; not infrequently they can be profoundly hypoglycemic with no symptoms. Vigilant attention should be given to risk factors such as hypothermia, hypoxia, intrauterine growth restriction, macrosomia, prematurity and maternal diabetes and appropriate screening measures put in place for at-risk infants [3].

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