# The Late Preterm
## A Population at Risk

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**KEYWORDS**

• Late preterm infant • Respiratory complications • Hypoglycemia • Neonatal nutrition • Hyperbilirubinemia • Neonatal morbidity • Neurodevelopmental outcomes

**KEY POINTS**

- The most common morbidities experienced by the late preterm infant include respiratory complications, feeding difficulty, hypoglycemia, temperature instability, hyperbilirubinemia, and neurodevelopmental delays.
- The late preterm infant has a higher morbidity and mortality rate compared with their term counterparts.
- Discharge planning and follow-up care is crucial for reducing hospital readmission rates and promoting healthy growth and development.

## INTRODUCTION

Late preterm infants (LPIs) are born between 34 0/7 and 36 6/7 weeks gestational age. From 2014 to 2016, the LPI birth rate rose from 6.82% to 7.09%, accounting for approximately 72% of all preterm births in the United States.[1] The increase in preterm births, has been attributed to the rise in assisted reproductive therapy, improved obstetric surveillance, multiple births, and maternal factors, including advanced maternal age.[2] LPIs are usually larger than premature infants and often mistakenly equated to the term infant. Although they may be close to term, the loss of the last 6 weeks of gestation is vital to their physiologic and metabolic maturity. Because of their physiologic and metabolic immaturity, they have higher morbidity and mortality rates compared with term infants (gestational age $\geq$37 weeks).[3]

Research has shown that the newborn morbidity rate doubles for every gestational week less than 38 weeks.[4] For LPIs, this translates to a morbidity rate as high as 51% in the 34-week infant.[3] LPIs experience higher morbidities during hospitalization and higher readmission rates during their first year of life when compared with the term

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infant.[2,5] The most common morbidities experienced include respiratory complications, feeding difficulty, hypoglycemia, temperature instability, hyperbilirubinemia, and neurodevelopmental delays.[6–8] Given the heightened risks LPIs are exposed to, this article presents an overview of the complications placing them at risk for increased morbidity, mortality, and long-term adverse outcomes (**Table 1**).

## RESPIRATORY COMPLICATIONS

LPIs have a higher incidence and risk of respiratory complications than the term infant. Respiratory complications more commonly encountered include respiratory distress syndrome (RDS), transient tachypnea of the newborn (TTN), and apnea.[5,9] LPIs are born during the saccular and alveolar stages of lung development. These stages of development are characterized by the development and remodeling of the respiratory bronchioles and alveoli, which are important for the process of surfactant synthesis and secretion, and gas exchange.[10,11] An interruption in this development leads to a delay in lung maturation and predisposes the LPI to RDS. Other factors, including the ineffective clearance of fetal lung fluid during the transition to neonatal life, can diminish alveolar gas exchange and is often implicated in TTN.[12] Compounding factors including Cesarean birth without the benefit of labor, and maternal and/or fetal complications contribute to the increased incidence of RDS, TTN, and apnea.

In a large retrospective study, found infants born at 34 weeks' gestation required more oxygen supplementation and delivery of oxygen via bag-mask ventilation in the delivery room than infants born at each advancing week of gestation until 39 weeks.[5] Among this LPI population, RDS was the most common respiratory morbidity followed by TTN. When morbidities were compared across gestational ages, the adjusted odds ratio for RDS and TTN decreased from 34 to 38 weeks' gestation.[13] In another retrospective cross-sectional study, 39.4% of all LPIs admitted to the neonatal intensive care unit (NICU) were admitted for respiratory distress, making this the number one reason for admission. Within this cohort, 34.4% were diagnosed with TTN, 4.4% with pneumonia, and 0.8% with meconium aspiration syndrome.[2]

The incidence of central, obstructive, and mixed apnea is higher in the LPI than the term infant. The etiology of apnea is multifactorial, reflecting an immature neurologic system and the physiologic immaturity of the respiratory system. Preterm infants have a decreased ventilatory response to increased carbon dioxide levels and a biphasic ventilatory response to hypoxia.[14] The very compliant chest wall and upper airway of the preterm infant also plays a role in the propensity toward apnea.[13,15] The incidence of apnea within the literature has varied based on the definition, acquisition, and documentation clarity of the event. In a meta-analysis of studies, the incidence of apnea to be 0.9% in the LPI and 0.05% in the term infant.[14] A decrease in apnea was observed with increasing gestational age. LPIs can also experience apnea associated with feedings because of a lack of coordination among sucking, swallowing, and breathing.[16]

LPIs require special attention given the risk of respiratory complications. LPIs should be monitored for increased rate and work of breathing, especially during transition. When clinically stable, skin-to-skin should be implemented to minimize infant stress, optimize respiration and oxygenation, and safeguard from hypothermia-induced apnea.[16] Parents should be educated on their LPI's risk of respiratory complications and the signs and symptoms of distress. The family also should be advised of the LPI's increased risk for asthma, respiratory infection, and rehospitalization during the first year of life.[17] Methods to avoid respiratory infections, including proper

**Table 1**
**Late preterm development and common morbidities**

| Clinical Manifestation | LPI Development | Indications for Care |
|---|---|---|
| Respiratory | | |
| RDS | • LPIs experience an interruption in the development and remodeling of the respiratory bronchioles and alveoli altering surfactant synthesis and secretion, and gas exchange. | • Monitor rate and work of breathing especially during transition.<br>• Promote skin-to-skin when infant is medically stable. |
| TTN | • LPIs can experience ineffective clearance of fetal fluid. | • Signs of TTN can last up to 72 h. |
| Apnea | • LPIs have decreased ventilatory response to increased carbon dioxide levels.<br>• LPIs have a biphasic ventilatory response to hypoxia.<br>• LPIS have a very compliant chest wall and upper airway. | • Evaluate for other causes of apnea, ie, sepsis.<br>• Apnea spells typically resolve at approximately 36–37 wk postmenstrual age. |
| Fluid, Electrolytes, Nutrition, and Gastrointestinal | | |
| Hypoglycemia | • LPIs have immature breakdown of glycogen in the liver (glycogenolysis), adipose tissue lipolysis, hormonal dysregulation, and decreased hepatic gluconeogenesis, and ketogenesis. | • Become familiar with the most up-to-date hypoglycemia guidelines including that of the AAP, ABM, and PES, and your hospital policy.<br>• Monitor the LPI's glucose closely for at least 24 h. |
| Feeding difficulties | • LPIs have immature brain development leading to low oromotor tone, immature suck-swallow reflex.<br>• LPIs have a higher incidence of gastroesophageal reflux. | • Monitor enteral intake, growth, wet diapers, and number of stools.<br>• Consider increasing caloric density to help optimize growth.<br>• Provide lactation support when necessary.<br>• Supplement with vitamin D and iron for optimal bone mineralization and brain growth and development. |
| Hematology | | |
| Hyperbilirubinemia | • LPIs have increased hemoglobin breakdown resulting in an increased bilirubin load.<br>• LPIs have increased enterohepatic circulation or gastrointestinal bilirubin reabsorption.<br>• LPIs have decreased albumin levels. | • Promote frequent feedings.<br>• Follow bilirubin levels.<br>• Discharged infant should have a bilirubin level check 24–48 h after discharge. |

(*continued on next page*)

**Table 1**
**(*continued*)**

| Clinical Manifestation | LPI Development | Indications for Care |
|---|---|---|
| Neurologic | | |
| Poor neurologic outcomes | • LPIs have immature nervous systems. | • Developmental follow-up should be considered for those infants who are very ill during hospitalization to minimize sequalae. |
| Other | | |
| Thermal instability/cold stress | • LPIs have less subcutaneous fat than term infants.<br>• LPIs have nonkeratinized thin skin.<br>• LPIs have an immature response to temperature receptors.<br>• LPIs have a higher surface area–to–body mass ratio.<br>• LPIs have less brown adipose tissue. | • Provide a neutral thermal environment.<br>• Encourage skin-to-skin in the medically stable infant.<br>• Bathing should be postponed until the infant exhibits thermal, respiratory, and cardiovascular stability.<br>• Educate parents on methods to prevent cold stress. |

*Abbreviations:* AAP, American Academy of Pediatrics; ABM, Academy of Breastfeeding Medicine; LPI, late preterm infant; PES, Pediatric Endocrine Society; RDS, respiratory distress syndrome; TTN, transient tachypnea of the newborn.

hand hygiene, maintaining current immunizations, and avoiding large crowds and sick people, should be reinforced.

## HYPOGLYCEMIA

Hypoglycemia affects newborn infants of all gestational ages who have not had any form of exogenous nutrition while still managing the sudden loss of maternal glucose.[17] Hypoglycemia occurs most commonly in infants with risk factors including prematurity, small for gestational age, large for gestational age, infants of diabetic mothers, maternal tocolytic therapy, genetic syndromes, and significant stressors, such as perinatal asphyxia, hypothermia, and resuscitation, and late preterm.[18–20] Preterm infants have immature breakdown of glycogen in the liver (glycogenolysis), adipose tissue lipolysis, hormonal dysregulation, decreased hepatic gluconeogenesis, and ketogenesis.[17] Usually, blood glucose concentrations among preterm infants decrease to a nadir 1 hour after birth, then rise and stabilize 3 hours after birth until metabolic pathways assume control or glucose is provided through feedings or intravenous dextrose.[17,21]

Although there is no consensus for the definition of hypoglycemia, studies have shown that a glucose concentration of less than 47 mg/dL offers the greatest predictive power.[22] In 2004, Wang and colleagues[23] showed a 15% incidence of hypoglycemia in the LPI population. When low plasma glucose levels are prolonged or recurrent, the results can be acute systemic effects and neurologic sequelae.[22] The clinical signs of hypoglycemia include an abnormal cry, poor feeding, hypothermia, diaphoresis, tremors and jitteriness, hypotonia, irritability, lethargy, seizures, cyanosis, pallor, tachypnea, apnea, and cardiac arrest.[18,22] Treatment should be based on clinical presentation and not by glucose concentration alone.[18,22]

The health care team should be aware of the most recent hypoglycemia guidelines, including the American Academy of Pediatrics (AAP), Pediatric Endocrine Society, and Academy of Breastfeeding Medicine, as well as individual hospital protocols.[18,20,24] Because LPIs are more vulnerable to low glucose concentrations, they require close monitoring for at least 24 hours.[18,22] It is also important to closely monitor LPIs' temperatures to prevent hypothermia, because cold stress can lead to worsened hypoglycemia among LPIs.[25]

## NUTRITION/GASTROINTESTINAL

Feeding challenges in LPIs predispose them to longer hospital stays and hospital readmissions. LPIs have fewer awake-alert periods, which can result in decreased nutritional intake, and when combined with high energy demands, can lead to dehydration, and/or poor growth.[25] In 2004, Wang and colleagues[23] showed that 27% of all LPIs received intravenous fluid (IV) due to various clinical conditions, including poor feeding, compared with 5% of term infants.[23] Gastrointestinal immaturity is another factor that leads to feeding problems and impacts weight gain in LPIs. Preterm infants have a higher incidence of gastroesophageal reflux (GER) due to transient relaxation of the lower esophageal sphincter.[26] GER can have a cascading effect, leading to dehydration and hypernatremia in the initial weeks after birth.[13]

Immature brain development, including low oromotor tone, is another reason LPIs have feeding challenges, lack adequate oral intake, and are predisposed to dehydration, poor growth, and hyperbilirubinemia.[17] During the final weeks of gestation, oral motor skills become more coordinated, and movements become smoother, but LPIs miss this crucial period of development.[27] Additionally, LPIs have immature suck-swallow reflexes that can lead to difficulties with latching during breastfeeding and inadequate intake during bottle feeding.[25]

Management of LPIs should include educating parents on infants' sleep-wake cycles, feeding cues, and promoting postural stability while feeding.[25] Postural stability, which means ensuring hips are flexed and head and neck are in alignment with the trunk, improves feeding success in the LPI.[27] The LPI's immature brain development is often overlooked because LPIs are considered stable when compared with their extremely premature counterparts. However, the caregiver must pay special attention to provide safe and effective oral feedings. Close monitoring of adequate enteral intake in the early neonatal period is of utmost importance, and if the mother is breastfeeding, lactation support is critical because of the increased risk of difficulty in establishing effective breastfeeding.[22] If growth is not adequately maintained, calorie fortification should be considered.[22] LPIs also should be supplemented with vitamin D, for bone mineralization, and iron, which are essential for the growth and development of the brain and nervous system.[22]

## THERMAL INSTABILITY/COLD STRESS

LPIs are at increased risk of thermal instability, particularly cold stress, due to their physiologic and metabolic immaturity. An understanding of the LPI's limitations can minimize morbidity and mortality. Term infants experiencing cold stress use several mechanisms to conserve and generate body heat. After the activation of temperature-specific receptors, term infants constrict their peripheral blood vessels, increase muscle flexion and activity, and metabolize brown adipose tissue (BAT), also known as nonshivering thermogenesis (NST).[28] These processes allow the term infant to maintain blood and heat in the core of the body, decrease the surface area available for heat loss, and generate heat and energy through muscle movement

and fat breakdown. LPIs are more likely to experience thermal instability than the term infant because of their deficiency in subcutaneous fat, nonkeratinized thin skin, immature response to temperature receptors, high surface area–to–body mass ratio, and deficiency in BAT.[13,28,29]

NST through BAT metabolism is the major mechanism of heat production in infants. BAT cells begin to differentiate at approximately 25 to 26 weeks' gestation, and brown adipocytes can be seen at approximately 29 weeks and continue to increase until a few weeks after birth.[30] In the term infant, BAT accounts for 1% of the infant's body weight and can increase heat production by 100% or more above basal level.[13,28] In response to cold stress, norepinephrine stimulates the nerve endings on the brown fat resulting in metabolization and heat production. Blood is warmed as it passes through the various areas of BAT metabolization and, subsequently, warms the body. NST is limited in LPIs due to insufficient BAT mass.[13]

Cold stress can have deleterious effects on the LPI. In the setting of cold stress, there is a release of norepinephrine, which causes various systemic effects, each of which can increase infant morbidity and mortality. Norepinephrine release in LPIs increases their metabolic rate, oxygen consumption, and glucose utilization. To maintain an increased metabolic rate, the infant must consume more oxygen. Higher oxygen consumption can lead to hypoxemia, and, subsequently, decreased oxygen delivery to the tissues, or hypoxia. Higher metabolic rates also require higher glucose utilization, which can lead to hypoglycemia. In a population that already has a higher risk of respiratory distress and hypoglycemia, cold stress can exacerbate these conditions and increase their risk of morbidity and mortality.

Several tactics can be implemented to minimize heat loss and avoid cold stress. The provision of a neutral thermal environment is ideal for the LPI.[28] Providers and caretakers must be aware that environments that may be comfortable for an adult may require increased metabolic efforts to maintain a normal temperature in the infant. Skin-to-skin in the stable LPI should be encouraged. Most infants are able to maintain their temperature during skin-to-skin, especially if the infant is wearing a hat and a blanket.[16,28] Bathing should be postponed until the infant exhibits thermal, respiratory, and cardiovascular stability. Consider a partial rather than a full body bath, dry infant immediately after bathing, and cover the infant's head with a dry hat. Most importantly, the family should be educated on methods of heat loss (**Table 2**) and taught techniques to prevent cold stress.

## HYPERBILIRUBINEMIA

Hyperbilirubinemia is the most common reason for readmission among LPIs.[31,32] Sixty percent of term infants and almost all preterm infants develop hyperbilirubinemia.[33] LPIs are 2.4 times more likely to develop hyperbilirubinemia than term infants, and it lasts longer and is more pronounced in LPIs compared with term infants.[17,34] In healthy term infants, bilirubin levels peak at 5 to 7 mg/dL at approximately 3 to 5 days of life and decline by 7 to 10 days.[33] In preterm infants, total serum bilirubin (TSB) levels peak at 10 to 12 mg/dL by the fifth day of life.[33] Infants who are breastfed have higher bilirubin levels than bottle-fed infants.[34]

Hyperbilirubinemia occurs as a result of increased bilirubin production, decreased metabolism and elimination, or a combination of the two.[13] Newborns have a higher volume of red blood cells (RBCs) per kilogram and a higher proportion of RBCs with a shorter lifespan, yielding a source for greater bilirubin production.[13,33] The lifespan

**Table 2**
**Methods of heat loss**

| Method | Definition | Example |
|---|---|---|
| Conduction | The transfer of heat from the body surface to an object in contact with the body. | That is, heat loss to a cold scale or unwarmed mattress. |
| Convection | The loss of heat molecules through the skin or mucous membranes into the surrounding air. It can occur through the skin and/or the respiratory tract. | That is, heat loss from an intubated patient to a cool ventilator circuit or a naked infant to a cool room. |
| Evaporation | The loss of heat as moisture on the skin vaporizes. This process is always accompanied by a cooling effect. | That is, heat loss in the delivery room immediately after birth (amniotic fluid) or after a bath. |
| Radiation | The transfer of heat between solid surfaces that are not in direct contact with each other. | That is, loss to a cold window or incubator wall. |

of RBCs in term newborns is 80 to 100 days, whereas the RBC lifespan in preterm infants is 60 to 80 days, predisposing the preterm infant to greater breakdown of hemoglobin and an increased bilirubin load.[33] LPIs have decreased activity of the UGT gene that is required by the liver enzyme glucuronyl transferase to conjugate indirect bilirubin to direct bilirubin, a water-soluble, excretable form of bilirubin, leading to an increased indirect bilirubin load.[13,33]

LPIs have immature gastrointestinal function and feeding difficulties that predispose them to increased enterohepatic circulation, or gastrointestinal bilirubin reabsorption.[17] These factors contribute to hyperbilirubinemia, decreased stool frequency, and dehydration.[17] Additionally, newborns have decreased levels of albumin compared with older infants and adults, and the levels are even lower in preterm and LPIs.[33] Albumin carries bilirubin to the liver to be conjugated, and decreased albumin means there are fewer binding sites for bilirubin, resulting in an increased amount of free bilirubin, which causes neurotoxicity in infants.[33,35]

According to AAP guidelines, to prevent adverse outcomes, clinicians should promote frequent and successful breastfeeding.[33,36] Facilities should have protocols in place to identify infants at risk for hyperbilirubinemia, and clinicians should measure a bilirubin level on jaundiced infants in the first 24 hours, and treat infants when appropriate.[36] It is important, as well, to provide verbal and written information to parents on neonatal hyperbilirubinemia.[36]

Phototherapy is the most widely used therapeutic modality in infants with hyperbilirubinemia, followed by exchange transfusion, and intravenous immune globulin.[37] Twenty-five percent of LPIs will require phototherapy.[23] The goal of treatment is to prevent severe neonatal hyperbilirubinemia, acute bilirubin encephalopathy, and ultimately, kernicterus.[36] The signs of acute bilirubin encephalopathy include hypertonia, arching, retrocollis, opisthotonos, fever, and a high-pitched cry.[36] The possible causes of hyperbilirubinemia should be explored in infants receiving phototherapy or whose TSB levels are rising rapidly.[36] Conditions that can complicate and prolong hyperbilirubinemia include Rh isoimmunization, ABO incompatibility, and genetic disorders, such as glucose-6-phosphate dehydrogenase deficiency.[32]

## NEUROLOGIC DEVELOPMENT

When compared with term infants, preterm infants have an immature central nervous system. The preterm infant's brain size is approximately two-thirds the size of a full-term infant's brain.[6,22] The preterm brain has fewer sulci and gyri, and the weight of the brain at 34 weeks is 65% of that of a term infant.[6,22] It is between 34 and 40 weeks' gestational age that cortical volume increases by 50%, and 25% of cerebellar development takes place.[38,39] These factors place preterm infants at an increased risk for altered brain development, which may influence long-term neurodevelopmental outcomes.[22,40]

LPIs have a threefold increased risk of cerebral palsy compared with term infants.[6] In 2016, Prachi and colleagues[41] concluded that children born in the late preterm period demonstrate poorer performance on tests of early school readiness, spatial abilities, and verbal reasoning in early childhood, poorer educational achievement at age 5, and poorer school performance at age 7. In 2015, Dusing and Tripathi[40] concluded LPIs are at risk of having reduced long-term neurodevelopmental outcomes, with cognition being at the highest risk and persisting the longest. The review by Dusing and Tripathi[40] also concluded there is an increased risk of neurodevelopmental delay in LPIs up to 18 years of age when compared with infants born at term. LPIs account for 72% of all preterm births, and even the smallest increases in adverse outcomes could translate to a large public health burden.[42]

## MORBIDITY

LPIs are often viewed as functionally mature when, indeed, they are not. This population was previously referred to as "near term," erroneously giving the connotation that nothing more than routine surveillance was required. However, "near term" was changed to "late preterm" to channel the susceptibility of LPIs. An LPI birth is complicated by the arrest in growth and development of vital systems,[6] and is associated with significant short-term and long-term morbidities. In fact, LPIs have a sixfold to sevenfold greater morbidity rate than term infants, a rate that rises with other neonatal morbidity risk factors.[43,44]

Increased neonatal morbidities have been associated with maternal prenatal complications. Conditions, including chorioamnionitis, premature rupture of membranes, hypertension, preeclampsia, diabetes, and maternal smoking, are common maternal complications seen with LPI births.[6] The neonatal complication of intrauterine growth restriction is also more common in the LPI. Although most LPIs will have a benign neonatal course, LPIs continue to have a higher risk of morbidity than the term infant. Morbidities, including respiratory problems requiring mechanical ventilation, apnea, sepsis, feeding problems, jaundice, hypoglycemia, temperature instability, and intraventricular hemorrhage, occur more frequently in the LPI population.[43,44] Higher rates of emergency department visits for apnea/apparent life-threatening events, feeding problems, dehydration, jaundice, and sepsis have also been reported.[44]

## MORTALITY

Although the relative risk of mortality in the LPI compared with the term infant is small, the mortality rate is 4 times higher in the LPI.[5] In 2016, Bulut and colleagues[2] reported the number one cause of death in the LPI was respiratory distress. This same study also reported a higher rate of death from respiratory distress,

pneumonia, perinatal asphyxia, and sepsis in the LPI compared with the term infant.[2] Mortality was also increased with elective Cesarean deliveries, placental complications, newborn bacterial sepsis, antepartum hemorrhage, and hypertensive disorders.[6] Several studies have reported an increased risk of neonatal death with decreasing gestational age.[2,45] In a population of LPIs and term infants, a mortality rate as high as 8.2 per 1000 live births was reported at 34 weeks' gestation compared with 0.5 per 1000 live births for term infants born at 40 weeks' gestation.[45]

## COST OF CARE

The cost of care for LPIs is higher than that of the term infant and can be attributed to many factors. The 2006 Institute of Medicine report *Preterm births: Causes, Consequences and Prevention* estimated the total annual cost of preterm births to be more than $26 billion per year, attributing nearly two-thirds of the cost to medical care.[46] Although this figure was inclusive of all preterm infants less than 37 weeks' gestational age, several studies have attempted to capture the cost of the LPI.[4,47,48] To gather a complete picture of the total cost of the LPI, it is necessary to include antepartum management, delivery cost, cost of care during the neonatal period, and long-term needs, including medical, educational, and social services. Although it may not be easily quantified, the impact of preterm birth on family psychological health and stress levels also should be considered.

Gestational age and cost of care have an inverse relationship. In 2010, Loftin and colleagues[8] reported the average cost for a single infant born at 34, 35, and 36 weeks' gestation was estimated to be $7200, $4200, and $2600, respectively.[8] When the total number of births was annualized, the cost translated to $41.4 million, $41.1 million, and $42.8 million at 34, 35, and 36 weeks, respectively.

LPIs have higher rates of morbidity immediately after birth and higher readmission rates during infancy and their first year of life.[4,48] It has been reported that LPIs between 34 0/7 and 34 6/7 weeks' gestation have as high as a ninefold increased risk of long-term morbidity and continual use of health care resources.[8] LPIs often have a longer length of hospital stay after birth. In a retrospective study of commercially insured infants in the United States, LPIs averaged 8.8 days in the hospital with an average cost of $26,054 versus 2.2 hospital days and an average cost of $2061 for term infants.[4] In 2009, McLaurin and colleagues[4] reported hospital readmission rates to be 2 to 3 times greater for LPIs compared with term infants. During the first year after discharge, medical cost was reported as much as 3 times higher in LPIs compared with term infants.[43] This was echoed in a study of Arkansas Medicaid claims data, which reported that LPIs had higher outpatient and inpatient Medicaid expenses during the first year compared with the term infant.[48]

## DISCHARGE PLANNING/FOLLOW-UP

There is a high number of readmissions in the LPI population, and this population is particularly at risk for readmission related to hyperbilirubinemia, apparent life-threatening events or apnea, feeding problems, possible sepsis, hypothermia, and respiratory problems.[49] LPIs warrant specific medical monitoring and nutritional practices to optimize their growth and positive outcomes.[22] Careful discharge planning for the LPI may help prevent hospital readmissions and lead to positive long-term outcomes (**Box 1**).

**Box 1**
**Discharge planning for the late preterm infant**

- Infants must have a carefully documented assessment of gestational age.
- Before discharge, infants must demonstrate an established feeding pattern with adequate volumes and calories to promote growth and prevent dehydration.
- Infants must be able to maintain temperatures of 36.5 to 37.4° Celsius (97.7–99.3°F) in an open crib without excessive clothing.
- If apnea of prematurity is identified as a diagnosis, the infant must have a documented period free of apnea based on the institution's policy.
- Perform a systematic assessment on all infants before discharge for the risk of severe hyperbilirubinemia.
- Provide parents with written and verbal information about newborn jaundice.
- Provide appropriate hyperbilirubinemia follow-up based on the time of discharge and the risk assessment.
- A primary care provider must be identified before discharge, and the first appointment must be scheduled within 24 to 48 hours after discharge.
- A car seat safety study must be completed by a trained professional with documentation the infant passed.
- Newborn metabolic screenings have been submitted according to requirements mandated by each state.
- The infant has received the first Hepatitis B vaccine, or arrangements have been made for the infant to get the vaccine in the pediatrician's office.
- Late preterm infants (LPIs) of less than 36 weeks' gestational age should be considered at risk for infection and managed according to current guidelines for prevention of group B streptococcal infection.
- Educate parents on RSV (respiratory syncytial virus), most common from fall to spring, RSV prophylaxis, and preventing the spread of RSV.
- Educate parents on avoiding exposure of infant to people with active upper respiratory tract infections or other viral infections.
- Educate parents on avoiding exposure of infant to second-hand and third-hand smoke.
- To help prevent sudden infant death syndrome, educate parents on infants sleeping alone and on their backs with no additional bedding.
- LPIs are generally not scheduled for developmental follow-up; however, developmental surveillance is important given the risk of adverse long-term developmental outcomes in this population.

## SUMMARY

Most LPIs will fare well, however, this population is met with challenges unique to this age group. They often experience morbidities that can prolong hospital stays and lead to hospital readmissions, often related to feeding problems, dehydration, hypothermia, jaundice, and apparent life-threatening events. The LPI population also experiences mortality rates 4 times higher than the rates for term infants.[5] With diligent surveillance by the health care team and caregivers, challenges in the LPI population can be addressed before they become life-threatening or lead to long-term adverse outcomes that could translate to a large public health burden.

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