

The management of respiratory distress in the moderately preterm newborn infant

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ABSTRACT

Respiratory distress in a moderately preterm baby often presents diagnostic and management challenges to the attending paediatrician. Many of these babies will require little or no intervention, but it is known that early intervention in babies with acute respiratory distress often prevents further complications. Most current research evidence relates to extremely preterm newborns, yet moderately preterm infants are numerically far more common. This article explores the differential diagnosis of respiratory distress in this population and presents an evidence based approach to treatment.

INTRODUCTION

You are the paediatrician on call in a district general hospital. You receive a call that a woman has been admitted at 33+0 weeks with significant ante-partum haemorrhage and is going to be taken to theatre for an emergency caesarean section. There has been no time to administer steroids. She is not in established labour and there are no signs of fetal distress. Your intensive care space is occupied with a ventilated baby but you do have a high dependency space set up. The local tertiary intensive care unit is 20 miles away and is full. The nurse in charge wants to know what you think the chances are of this baby needing ventilatory support and if so which kind of ventilatory support she should set up.

What is the chance of needing respiratory support (CPAP or intermittent positive pressure ventilation) at 33 weeks gestation?

As you are pondering this question, the baby is brought from the delivery suite, he is male with a birth weight of 2.0 kg and has a respiratory rate of 80 breaths/min with visible intercostal recession and obvious expiratory grunting. His preductal oxygen saturation (SaO_2) is 84% in room air. He did not require any resuscitation at birth and has been wrapped in a warm towel. As you prepare to stabilise the baby in the incubator you consider two further questions:

What is the differential diagnosis of mild to moderate respiratory distress in a baby at 33 weeks gestation? What is the best way to manage this baby's respiratory distress in order

to maximise the chance of a good outcome for the baby given the resources available?

This is not an uncommon situation faced by paediatricians and neonatologists and the decisions involved can sometimes be more challenging than when the clinician is faced with a critically ill or extremely preterm baby who clearly needs full intensive care, surfactant and respiratory support. This article will explore the differential diagnosis and the management strategies that might best be employed to treat a moderately preterm infant with respiratory distress.

HOW COMMON IS RESPIRATORY DISTRESS IN MODERATELY PRETERM INFANTS?

Many recent studies have rightly focused on the morbidity and mortality associated with extremely preterm births (≤ 28 weeks).¹ However, mild (34–36 weeks) and moderately (32–33 weeks) preterm birth is far more common. In a Canadian cohort, 93.9% of births were at term, 4.9% at 34–36 weeks, 0.8% at 32–33 weeks and just 0.4% before 28 weeks.² Although the clinical course may be less dramatic than that of an extremely preterm baby, moderately preterm birth is not without risk. The mortality in a US cohort was 18.5/1000 live births at 30–34 weeks compared with 6.9/1000 at 35–36 weeks and 2.5/1000 at term.³ Estimating the individual risk of an infant developing respiratory compromise is difficult. The reported incidence of respiratory distress will be highly influenced by the population studied, the proportion of babies with surfactant deficient respiratory distress syndrome (RDS) and the thresholds set for intervention. Changing perinatal practice has had a profound effect on the frequency of RDS and older epidemiological studies may no longer reflect the current picture. In particular, the use of antenatal steroids is associated with a reduction in RDS (RR 0.66) and the need for respiratory support (RR 0.80).⁴ A large population study in Italy showed that 30.8% of 34–36-week infants develop respiratory problems.⁵ The Consortium on Safe Labour recently published the outcomes of 19 334 late preterm infants (34–36 weeks), of whom 24% developed respiratory compromise.⁶ At 34 weeks, RDS occurred in 10.5% and transient tachypnoea of the newborn (TTN) in 6.4%; 8.5% received non-invasive ventilation, 6.6% ventilation and 2.8% high-frequency oscillatory ventilation (HFOV). The risk of developing

RDS approximately doubled for each week of gestation below 37 weeks.⁶ Yorkshire Neonatal Network (2005–2007) data show that the frequency of respiratory support at 32 weeks was 40%, rising to 60% at 31 weeks (B Manktelow, personal communication, 2009). Overall, about a quarter to a third of infants born at 32–34 weeks will require respiratory support and a higher proportion will show signs of respiratory distress. It is clear that despite improved outcomes, significant respiratory morbidity still occurs in moderately preterm infants. There is a need for ongoing surveillance of the health outcomes of this group in large population based studies.

WHAT IS THE DIFFERENTIAL DIAGNOSIS OF MILD TO MODERATE RESPIRATORY DISTRESS IN A BABY AT 33 WEEKS GESTATION?

The differential diagnosis is outlined in box 1.

Surfactant deficient RDS

RDS develops within 4 h of birth and worsens over the first 24–36 h before improving over the next 1–2 days, often coinciding with marked diuresis. RDS is primarily due to a lack of alveolar surfactant production in association with immature lung architecture. This causes atelectasis, poor gas exchange and ultimately respiratory failure. RDS is confirmed by a 'ground glass' appearance on chest x-ray (CXR), usually with air bronchograms (see figure 1). The Vermont Oxford Network criteria additionally require $\text{PaO}_2 < 6.6$ kPa (50 mm Hg) in air or need for supplemental oxygen. The main risk factor for RDS is low gestational age. Surfactant production starts at 24–25 weeks and is mature by 36–37 weeks. Corticosteroids boost production and are recommended in all threatened preterm labour between 24 and 34 weeks and should be considered at 35–36 weeks.⁷

A proportion of women delivering at 32–34 weeks will not have received two doses and these infants remain at significant risk of RDS. Maternal diabetes mellitus, hypothyroidism, male sex, Caucasian race, prelabour delivery and hypothermia all significantly increase the risk of RDS.

Transient tachypnoea of the newborn

Increased fetal epinephrine concentration during labour normally reduces lung fluid production and activates sodium channels leading to re-absorption. Failure to properly clear lung fluid leads to TTN. Risk factors include prelabour delivery, delivery by caesarean section, delivery prior to 38 weeks and male sex. TTN is associated with maternal asthma.⁸ TTN can initially be clinically difficult to distinguish from RDS, although the x-ray appearance is different with 'streaky' lung opacities, fluid in the transverse fissure and often cardiomegaly. The appearance is similar to heart failure and sepsis (see figure 2). TTN occurs in approximately 5/1000 term infants and may



Figure 1 Acute respiratory distress syndrome with widespread ground glass opacity and air bronchograms.

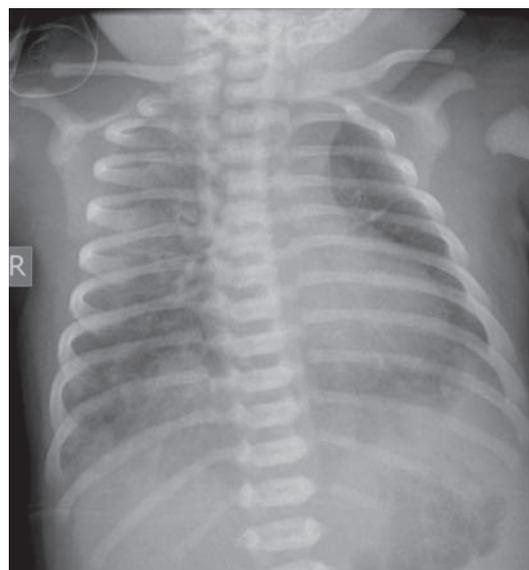


Figure 2 Transient tachypnoea of the newborn with fluid in the transverse fissure and some hyperexpansion.

Box 1 Causes of respiratory distress in moderately preterm infants

- ▶ Respiratory distress syndrome
- ▶ Transient tachypnoea of the newborn
- ▶ Congenital pneumonia
- ▶ Intrapartum asphyxia
- ▶ Meconium aspiration syndrome
- ▶ Pulmonary hypoplasia
- ▶ Pneumothorax or pneumomediastinum
- ▶ Pulmonary haemorrhage
- ▶ Persistent pulmonary hypertension of the newborn
- ▶ Congenital lung malformation
 - ▶ Congenital diaphragmatic hernia
 - ▶ Cystic adenomatous malformation
- ▶ Pleural effusion (isolated or with hydrops fetalis)
- ▶ Congenital heart disease
- ▶ Aspiration of secretions (oesophageal atresia), blood or milk
- ▶ Neuromuscular disease (eg, congenital myotonic dystrophy)
- ▶ Airway obstruction
- ▶ Inborn error of metabolism
- ▶ Thyrotoxicosis

coexist with RDS in preterm infants. TTN presents with marked tachypnoea (up to 100/min) and hyperinflation, but grunting is less common. In the preterm infant, respiratory failure requiring ventilation may occur. TTN is essentially a diagnosis of exclusion – RDS, sepsis and heart failure must first be ruled out.

Congenital pneumonia and sepsis

The frequency of congenital pneumonia and sepsis increases with prematurity, especially if there is prolonged preterm rupture of the membranes. Group B *Streptococcus* is the commonest pathogen, but *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Listeria* and Gram negative bacteria can be responsible. In the moderately preterm baby with respiratory distress, it is impossible to exclude infection since the presentation may mimic RDS and sepsis may be co-existent with RDS. For this reason empirical treatment with antibiotics is mandatory.

Birth depression

Intrapartum asphyxia may lead to respiratory distress either by inducing rapid, deep respirations in an attempt to blow off CO₂ generated by anaerobic metabolism or by heart failure secondary to myocardial dysfunction. There is usually a history of fetal distress and acidotic umbilical cord gases. Inborn errors of metabolism, thyrotoxicosis and cerebellar disorders (Joubert's syndrome) can present with tachypnoea, but other diagnostic clues are usually present.⁹

Pneumothorax

The relative lack of surfactant at 32–34 weeks renders the immature lung less compliant, increasing the risk of pneumothorax and pneumomediastinum. Air leak is rare at these gestations without severe RDS; the incidence at 34 weeks in a recent series was 0.8%.⁶ Air leaks may occur spontaneously or secondary to positive pressure ventilation. The move from self-inflating bags to pressure limited T-piece circuits may reduce the iatrogenic

incidence of pneumothorax, but extreme care should be taken not to adjust the flow rate once positive end-expiratory pressure (PEEP) is set, or very high PEEP may be generated.¹⁰ Small pneumothoraces may resolve spontaneously. Historically, 100% oxygen has sometimes been used as treatment for smaller pneumothoraces, however the evidence for its efficacy is poor and given the added concern of oxygen toxicity in the preterm infant, we would not recommend this. Hypoxia, respiratory failure and tension pneumothorax are indications to insert a chest drain. Increasingly, in our centre we use pigtail catheters inserted using the Seldinger technique. These are quicker and easier to insert and cause less scarring than traditional chest tubes¹¹ (see figure 3).

Congenital lung malformation and congenital pleural effusion

Congenital diaphragmatic hernia, cystic adenomatous malformation and tracheo-oesophageal fistula should all be managed in the same way as at term, with additional early administration of surfactant if there is RDS. These diagnoses should be suspected if an anomaly scan has been missed. The diagnosis is usually obvious on x-ray.

Congenital heart disease

Congenital heart disease does not typically present with respiratory distress at birth. However, cardiac pathology should be suspected when there is persistent cardiomegaly, abnormal pulses or a postductal drop in SaO₂. A high pCO₂ and parenchymal changes on CXR make a respiratory pathology more likely, but anomalous pulmonary venous drainage can mimic RDS and is difficult to diagnose.

Aspiration

Meconium aspiration is rare in preterm infants and green liquor should raise the suspicion of chorioamnionitis, *Listeria* infection or high intestinal atresia (bile).¹² Aspiration of milk is unlikely so

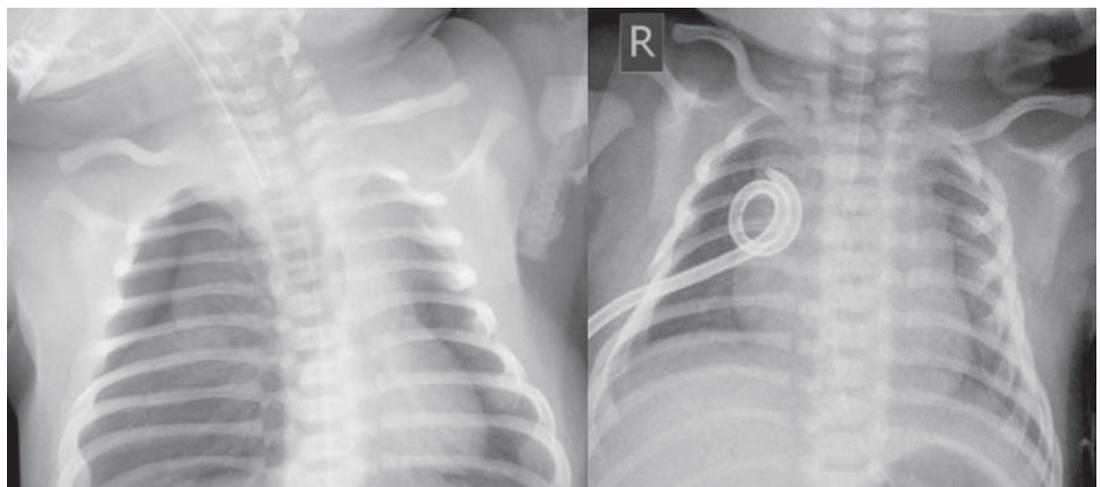


Figure 3 (A) Right sided pneumothorax. (B) Insertion of pigtail catheter showing resolution of the pneumothorax.

soon after birth, but perinatal aspiration of blood or vernix causes respiratory distress. The diagnosis is made by a combination of patchy consolidation on x-ray and aspiration of abnormal material from the trachea after intubation. A nasogastric tube should always be passed prior to x-ray to exclude oesophageal atresia.

Neuromuscular disorders

Neuromuscular disease such as congenital myotonic dystrophy and congenital myasthenia gravis may present coincidentally at moderately preterm gestation. Diagnostic clues include abnormal neurology in the mother, polyhydramnios, reduced fetal movements and thin, osteopenic ribs on CXR. Ventilation is offered while a careful assessment is made to establish the long term prognosis.¹³

Airway obstruction

Severe airway obstruction will manifest soon after birth with obvious intercostal and sternal recession during inspiration. Stridor suggests an upper airway abnormality (vocal cord palsy, laryngeal abnormality or haemangioma). Intermittent apnoea and cyanosis suggest choanal atresia (improves when the baby cries) or micrognathia (improves with prone position or jaw thrust). In many cases airway positioning, or a nasopharyngeal or Guedel airway, can prevent the need for ventilation. There is evidence that propranolol can rapidly shrink symptomatic airway haemangiomas.¹⁴

What is the best way to manage this baby's respiratory distress in order to maximise the chance of a good outcome for the baby given the resources available?

Moderately preterm babies require admission to a neonatal or transitional care unit. A full perinatal history including administration of antenatal steroids, maternal infection and mode of delivery should be obtained. Detailed examination and regular monitoring of temperature, pulse, respiration and blood glucose are necessary to identify those babies who need additional intervention.

Investigations

Babies with tachypnoea or increased work of breathing (recession, grunting) should initially be investigated with:

- ▶ SaO₂
- ▶ Capillary or arterial blood gas
- ▶ CXR
- ▶ Partial septic screen (blood culture, C reactive protein, full blood count).

Further specific investigations (eg, echocardiogram) may be required, but in the interests of minimising handling, these should be limited to those which will alter subsequent management.

Intervention

Although moderately preterm birth is much more common than extreme prematurity, evidence

from the modern era on how best to manage respiratory distress in this population is not always available. Optimal management depends on the diagnosis and the degree of prematurity, but the need to provide adequate oxygenation, support ventilation and minimise iatrogenic injury is universal.

We would advocate a stepwise approach to respiratory support with frequent reassessment and a low threshold for escalation of treatment (see figure 4).

Liaison with other network centres and arrangements for possible transfer will depend on locally agreed clinical pathways and the facilities available at the unit in question. Babies developing unexpectedly severe symptoms or those who do not respond to appropriate management should be discussed with the lead network centre.

Oxygen

Very mild cases of respiratory distress may be successfully managed with incubator or nasal cannula oxygen. Optimising oxygenation allows efficient use of respiratory muscles. The optimum SaO₂ target in moderately preterm infants is not universally agreed, but a target of 88–92% is commonly accepted. Retinopathy of prematurity is less likely in babies born after 32 weeks, but hyperoxia has other deleterious effects and should be avoided.¹⁵

Any sign of increased work of breathing or increasing oxygen requirement suggests the need for early institution of positive pressure support.¹⁶ Babies should not be allowed to become significantly acidotic (pH<7.25) without escalating support.

Invasive or non-invasive pressure support?

This will depend on the severity and likely cause of respiratory distress. There has been a trend towards managing moderately preterm infants with non-invasive ventilation, typically nasal continuous positive airflow pressure (NCPAP). Mechanical ventilation is strongly associated with the development of bronchopulmonary dysplasia (BPD) and also carries a risk of local trauma and infection.¹⁷ While BPD is more common following extreme prematurity, the moderately preterm lung is still vulnerable. Although it has not been shown conclusively that avoiding intubation prevents BPD, using non-invasive support where possible and minimising the duration of ventilation are likely to reduce lung injury.¹⁸

Non-invasive ventilation

Continuous positive airflow pressure

NCPAP provides end-expiratory pressure which reduces atelectasis, maintains higher functional residual capacity and improves lung function by reducing workload and minimising ventilation/perfusion (V/Q) mismatch. NCPAP reduces

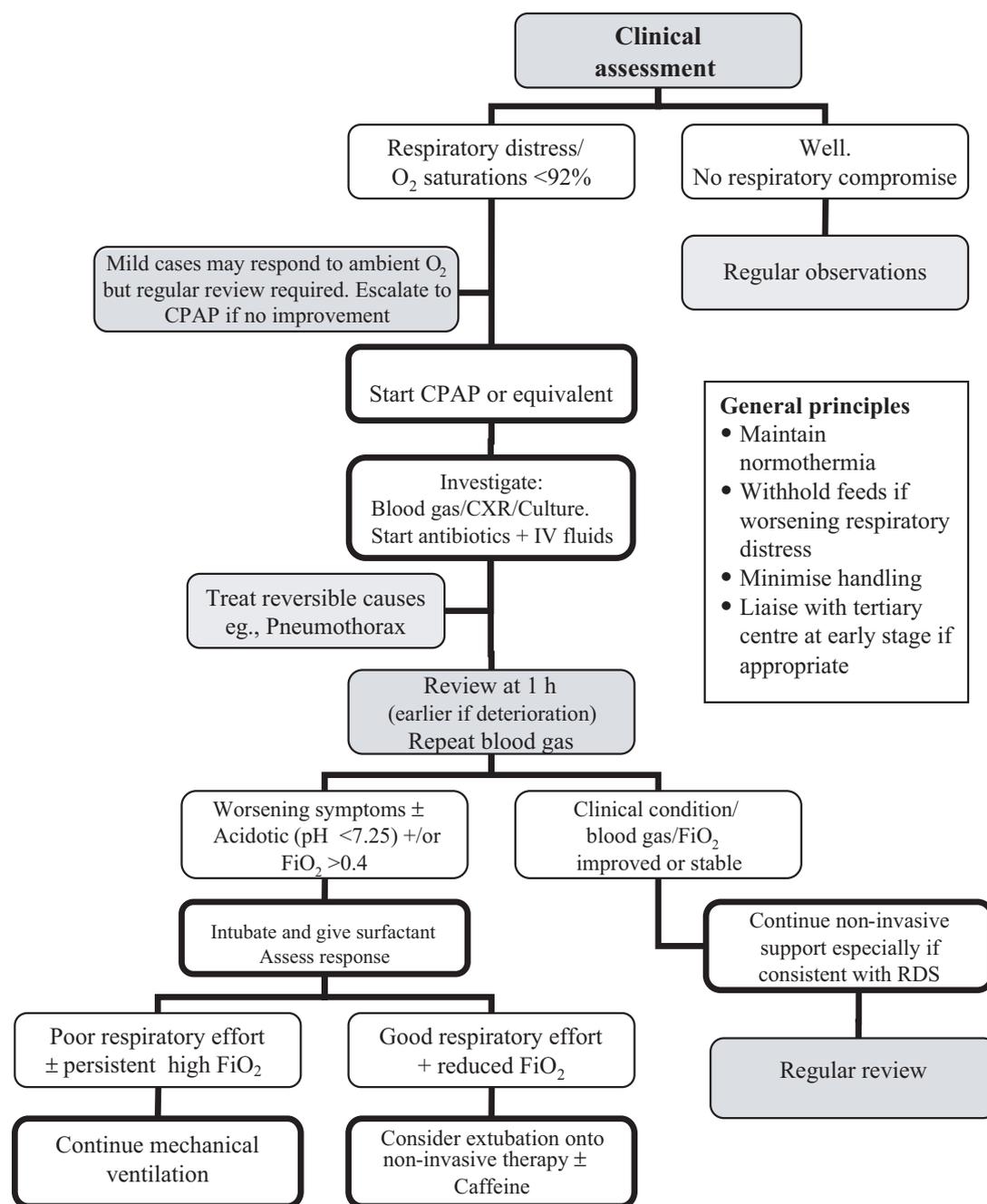


Figure 4 Algorithm for the management of respiratory distress in the moderately preterm infant in the newborn period. CPAP, continuous positive airflow pressure; CXR, chest x-ray; RDS, respiratory distress syndrome.

obstructive and central apnoea and improves synchronisation of respiratory movements.^{17 19} If there are developing signs of respiratory distress, NCPAP is best used early rather than waiting for babies to deteriorate, and has been associated with a significant reduction in the need for intubation.¹⁶ Evidence from older studies of the benefit of CPAP over ambient oxygen shows CPAP is associated with reduced respiratory failure and lower mortality. Increased air leaks were reported, but this predated antenatal steroids, surfactant and more sophisticated CPAP circuits.²⁰ There are many ways of delivering CPAP and no clear evidence as to which is ideal. Bubble CPAP leads to improved gas exchange, but this has not

been studied via the nasal route.¹⁷ Short bi-nasal prongs are more effective at delivering pressure than a nasopharyngeal tube but have the potential to cause nasal trauma.¹⁹ Nasal masks can be used to limit existing nasal trauma but have not been subjected to rigorous comparison. CPAP of 5–7 cm H₂O is recommended.^{15 17} The non-compliant lung in RDS is likely to need considerably higher PEEP than a baby with TTN or sepsis. Excessive PEEP theoretically increases the risk of pneumothorax and may potentially compromise cardiac output.²⁰

Increasing PEEP or O₂ requirements greater than 40% suggest failure of NCPAP and the need for further escalation of therapy.

Non-invasive positive pressure ventilation

Non-invasive positive pressure ventilation (NIPPV, also known as bilevel positive airway pressure, BiPAP) uses nasal prongs or a mask to deliver time-cycled ventilation breaths superimposed onto CPAP. These breaths are synchronised with the baby's respiratory effort. NIPPV may improve ventilation by maintaining patency of the upper airway and by promotion triggering respiratory reflexes.¹⁷ Smaller studies demonstrate that NIPPV may be an alternative to intubation in premature babies who have deteriorated despite standard NCPAP therapy.²¹ There is a suggestion of reduced BPD in extremely preterm infants treated with NIPPV, but there are as yet no randomised trials to support this.²²

Humidified high flow nasal cannula therapy

Humidified high flow nasal cannula therapy (HHFNC) systems (Vapotherm, Optiflow, Humicare) allow delivery of warmed, humidified gases via small calibre nasal cannulae at flow rates greater than 1 l/min without causing the excessive airway drying, mucosal damage, bleeding and increased risk of infection that can complicate conventional high flow nasal oxygen. HHFNC seems to be well tolerated with less reported airway trauma, reduced noise exposure and much easier nursing than NCPAP.²³ HHFNC generates increased pharyngeal pressure, similar to CPAP, and may flush CO₂ from the nasopharyngeal dead space. Unlike CPAP, it is impossible to measure or limit the pressure delivered. The pressure generated can be variable and is related to both flow rate and infant size.²⁴ There has been concern that potentially dangerous levels of positive pressure could arise.²⁵ It is vital that the nasal cannulae diameter is small in relation to the nostrils to allow a natural leak to occur.

HHFNC is increasingly being used as an alternative to NCPAP at all gestations. Despite its emerging popularity, the evidence that HHFNC is as effective as NCPAP is largely anecdotal or retrospective. Different studies have used a variety of flow rates and indications.²³ There has not yet been a rigorous assessment of the effects of HHFNC on duration of oxygen therapy, subsequent ventilation, BPD or survival. As a result, some advise against using HHFNC until formal randomised controlled trial data are available.²⁶

Invasive ventilation

Threshold for intubation

While many preterm babies can be successfully managed with non-invasive ventilation, it does not follow that avoiding intubation is in the best long term interests of every baby. Babies with severe RDS can continue to deteriorate despite non-invasive ventilation and there is potential for harm with very high PEEP or inspired oxygen levels. Ongoing signs of distress in a baby requiring more than 7 cm H₂O of PEEP, an oxygen requirement >40% or a pH<7.25 are generally accepted as triggers for escalation of treatment. Some argue

earlier intervention is preferable and may result in a shorter period of invasive ventilation.^{15 17 27}

Surfactant in moderately preterm infants: late rescue or early prophylaxis?

Surfactant is a safe and effective therapy for the treatment of RDS. The focus of many trials has been on those infants of <30 weeks gestation, but prior to antenatal steroid use many moderately and late preterm babies received surfactant for severe RDS with a proven reduction in mortality and BPD.²⁸ Aside from RDS, relative surfactant deficiency may occur in congenital pneumonia, sepsis and persistent pulmonary hypertension of the newborn. The role for exogenous surfactant in these conditions is uncertain and it not recommended routinely.²⁸ While in ventilated extremely preterm newborns, it is best practice to administer prophylactic surfactant as soon after birth as possible, it is more difficult to determine when it is indicated in more mature babies as 60% of these infants will recover without invasive ventilation.²⁹ Routine intubation for prophylactic surfactant administration is generally considered inappropriate at this gestation, particularly if the infant is asymptomatic.²⁷ Surfactant is however most effective when given early¹⁵ and when oxygen requirements are lower (<45%).³⁰ Waiting for babies to fail non-invasive ventilation reduces the effectiveness of surfactant therapy and may result in longer periods of mechanical ventilation.^{15 31} Guidelines suggest continuous monitoring and regular blood gas analysis in babies requiring non-invasive support, with a low threshold for surfactant if there are signs of ongoing respiratory compromise.^{15 27 31}

InSurE approach

A potential solution to the conflict between early surfactant use and a desire for non-invasive ventilation is the InSurE (intubate, surfactant, extubate) approach. Babies with RDS are electively intubated, given surfactant and extubated rapidly onto non-invasive therapy. Prompt extubation can be facilitated by avoiding premedication, using short acting sedative drugs or reversing the action of opiates with naloxone. Babies receiving InSurE have less need for mechanical ventilation, fewer pneumothoraces and less BPD,³⁰ but evidence of long term benefit is limited.²⁸ Since moderately preterm babies are likely to have greater respiratory drive and more effective respiratory effort than extremely preterm infants, the InSurE technique may offer the opportunity to administer surfactant at the first sign of RDS and then successfully extubate to NCPAP. This needs to be balanced against the greater sedation required for intubation in more mature babies.

Mechanical ventilation

A degree of lung damage may be unavoidable when positive pressure ventilation is applied to the immature lung, but there are strategies which reduce injury and support early extubation. These include:

- ▶ Synchronised ventilation
- ▶ Volume targeted ventilation
- ▶ Appropriate inspiratory time
- ▶ Optimal PEEP
- ▶ Permissive hypercapnia.

Synchronised ventilation is now standard practice and is associated with fewer air leaks and a shorter duration of ventilation.³² It is known that lung injury is most directly related to excessive tidal volumes and, conversely that an inadequate tidal volume increases work of breathing, promotes atelectasis and V/Q mismatch.³³ Lung compliance changes rapidly with surfactant administration and volume targeted ventilation reduces the frequency of excessive tidal volumes, decreasing inadvertent hyperventilation. Short term benefits include reduced duration of mechanical ventilation, less pneumothoraces and less frequent severe intraventricular haemorrhage. There has not however been conclusive evidence of improved long term outcomes.³⁴ Short inspiratory times (<0.5 s) should be used in preterm infants. This reduces the incidence of air leak in babies with less compliant lungs.³⁵ Flow sensors incorporated into the breathing circuit of modern ventilators can help avoid excessive inspiratory times. Optimal ventilation requires equal diffusion of gas throughout the lung and sufficient PEEP is necessary to achieve this. Determining the PEEP necessary for individual patients is difficult, but generally PEEP <5 cm H₂O should be avoided.³³ One approach to minimise ventilator induced lung injury is to tolerate higher levels of pCO₂ (permissive hypercapnia) allowing the use of lower tidal volumes. Unfortunately, prospective trials have not demonstrated a reduction in BPD.³⁶ Low pCO₂ is known to decrease cerebral blood flow. There is a proven association between hypocapnia, neonatal brain injury and subsequent cerebral palsy.³⁷ Hypocapnia should therefore be avoided in ventilated infants wherever possible.³⁷

Additional measures

Antibiotics

Infection can be a major contributory factor in preterm lung injury. The index of suspicion must be particularly high in moderately and late preterm babies (32–36 weeks) with respiratory distress, since we would otherwise expect many of these infants to have only mild lung disease. In addition, subclinical maternal chorioamnionitis may have triggered preterm labour. Given the difficulty in diagnosis and the potential consequences of untreated infection, commencing intravenous antibiotics is mandatory in any preterm newborn with respiratory illness. Penicillin (or amoxicillin) and gentamicin, or a cephalosporin, are usually chosen. Blood cultures should be taken, but culturing lung secretions is not possible without intubation. Antibiotics must be used rationally and stopped as soon as infection has been excluded.

Caffeine

Apnoea of prematurity is less common with advancing gestational age. Recurrent apnoea in

more mature infants should raise suspicion of infection, hypoxia, respiratory failure or intracranial pathology. If it does occur, caffeine has been found to be an effective treatment in babies up to 37 weeks gestation.³⁸ Early caffeine use is effective at reducing the duration of respiratory support³⁹ and there is evidence that caffeine can improve neuro-development.⁴⁰ While there is not enough evidence to recommend routine use of caffeine in babies born at 32–34 weeks gestation, it may benefit those with recurrent apnoeas and those weaning from respiratory support.

Thermoregulation

Hypothermia increases metabolic requirements and oxygen demand and is known to increase mortality in preterm infants.¹⁵ Temperature should be maintained between 36.5°C and 37.3°C with an incubator or heated mattress.

Fluid/nutrition

In the acute phase, intravenous fluids are required. Full volume enteral feeding is usually postponed until severe respiratory distress has settled, but minimal enteral nasogastric feeding can be continued. Fluid balance should be monitored. There is no evidence to support either fluid restriction or diuretics in the management of TTN or RDS.¹⁵ Effective nutrition is essential as good muscle development and bone mineralisation are required to support effective breathing.

SUMMARY

The moderately preterm infant can present particular challenges to the paediatrician in the first hours of life. It is important to anticipate respiratory distress and act promptly to support respiratory function. We recommend an expectant strategy with close monitoring and an immediate escalation in therapy dependent on clinical signs (figure 4). Evidence of any significant respiratory distress should prompt the early use of non-invasive pressure support; NCPAP is known to be effective. High flow therapy offers a well-tolerated potential alternative, but more evidence is required to prove its benefit. If RDS is present, a rising O₂ requirement >40% or signs of respiratory acidosis (pH<7.25) on non-invasive support should prompt intubation and surfactant administration. The InSurE technique appears to be useful in this age group. Ventilation should be synchronised and volume targeted (tidal volume 4–5 ml/kg). Hyperoxia and hypocapnia must be avoided. Consider caffeine if apnoea is evident or to aid extubation. Supportive therapy with thermoregulation, nutrition, antibiotics and careful fluid balance is essential. It is important to remember that the majority (about 70%) of babies born at 32–34 weeks will have little or no respiratory disease and the overall prognosis for survival with a normal outcome is excellent. If we can anticipate, identify and carefully manage those who do develop respiratory distress, we can further improve this outcome.

Competing interests None.

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