



## CLINICAL PRACTICE GUIDELINE

Guideline coverage includes NICU KEMH, NICU PMH and NETS WA

# Respiratory Distress Syndrome (RDS)

This document should be read in conjunction with the [Disclaimer](#)

Respiratory distress persisting beyond 4 hours of age, in infants with characteristic radiographic findings of bilateral air bronchograms with a ground glass or reticulogranular appearance in the lung fields.

## Epidemiology

- A common neonatal respiratory disorder most frequently seen in preterm infants. The incidence of RDS in preterm infants < 32 weeks gestation is reported as high as 71%.
- Risk factors include: male sex, Caucasian, maternal diabetes, elective caesarean section, multiple pregnancy, perinatal asphyxia.
- Protective factors include: antenatal corticosteroids, chronic foetal stress (maternal drug abuse, chronic congenital infections, prolonged rupture of membranes).

## Pathophysiology

RDS is primarily a function of surfactant deficiency. Spontaneous or mechanical ventilation of the surfactant-deficient lung causes epithelial injury, protein leakage and pulmonary oedema which is prevented with exogenous surfactant treatment.

Lack of surfactant results in collapse of alveoli (decreased surface tension) and hence decreased lung compliance, increased resistance, and decreased lung volume. Decreased alveolar ventilation impairs effective gas exchange. An increased mean airway pressure is required to open up the lung (critical opening pressure) and to establish effective tidal volumes.

There is often a marked right to left shunt in the range of 50-90% of cardiac output, although this effect is limited by hypoxic vasoconstriction in poorly ventilated, but over perfused areas of the lung.

## Clinical Presentation

- RDS usually presents immediately after birth, but may be up to 4 hours after birth, with grunting respirations, subcostal and sternal retractions, nasal flaring, bilateral poor air entry on auscultation and cyanosis in room air.
- The natural history of HMD is one of deterioration over the first 24-36 hours of age, followed by stabilisation for about 24 hours and later continued improvement often accompanied by diuresis. Resolution of disease is often a more protracted process in less mature infants.
- Complications prior to the advent of surfactant included a high rate of air leak.

## Investigations

- Baseline observations and SaO<sub>2</sub>.
- Arterial blood gas (hypoxemia, hypercarbia and sometimes a mild metabolic acidosis).
- FBC and U&Es, glucose.
- Septic screen.
- ECG/cardiac USS if suspecting CHD.
- CXR (AP and Lateral) will demonstrate increased density of both lung fields with reticulo-granular (ground glass) appearance, air bronchograms and elevation of the diaphragm.

## Management

- Management should always be discussed with the on-call consultant/senior registrar. Almost all lung disease affecting the neonate can develop within the first 4 hours of life and the most difficult diagnostic problem at all gestations is differentiating RDS from congenital pneumonia. Furthermore, sepsis and RDS may co-exist thus all infants with respiratory distress should be investigated appropriately and treated with antibiotics.
- In those who have mild respiratory distress, a trial of CPAP is warranted.
- The commonest approach is to intubate and use surfactant early as rescue treatment. The use of exogenous surfactant has dramatically changed the natural course of the disease and is associated with a rapid decrease in oxygen and ventilation requirements, and a decrease in the incidence of gas leaks. In infants < 28 weeks the earlier the surfactant is given the better. This can be in the delivery ward as prophylactic treatment - but must be discussed with the consultant before the birth of the infant. Infants who need to be transported post-delivery with a tracheal tube **in situ** should be treated with exogenous surfactant as per the WANTS guidelines.

## Indications for Intubation

- A rising PaCO<sub>2</sub> to > 60 mmHg + pH < 7.25.
- Recurrent apnoea requiring stimulation and resuscitation.
- Incipient collapse.
- Avoidance of high tidal volumes is essential for prevention of air leak syndromes, especially in the period of rapid increase in compliance following surfactant administration. Volume guarantee should be commenced as soon as the infant is placed on a ventilator equipped with flow monitoring.
- The initial starting ventilation parameters are dependent on the size of the infant.

## Ventilation: Starting Guidelines


### Preterm Neonate

- Mode SIPPV; with the infant managed on Volume Guarantee (4-5 mL/kg tidal volume) prior to and after surfactant given.
- PIP=18 cmH<sub>2</sub>O      PEEP=5 cmH<sub>2</sub>O      Inspiratory Time=0.3 s.

- Rate=40 breaths/min (Ventilator rate may be increased if infant does not spontaneously breathe above the backup ventilator rate).
- Pressure support ventilation (PSV) may be used as an alternative, using a lower flow (5-6 L/min), as synchronisation with both onset and end of inspiration may reduce airleak, and improve patient comfort. Lower flows during PSV lengthen the duration of the spontaneous inspiratory time, and promote more even and gentle inflation of the lung. When PSV is used, a backup inspiratory time approximately 1.5-2 times the spontaneous inspiratory time should be set.

### Term Neonate

- Mode=SIMV or SIPPV; with the infant managed on Volume Guarantee (4.5-6 mL/kg) prior to and after surfactant is given.
- PIP=22 cmH<sub>2</sub>O      PEEP= 5 cmH<sub>2</sub>O      Inspiratory Time=0.4 s.
- Rate=30 breaths/min

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