Queensland Clinical Guidelines

Translating evidence into best clinical practice

Maternity and Neonatal **Clinical Guideline**

Respiratory distress and CPAP

Queensland Government

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Cultural acknowledgement

We acknowledge the Traditional Custodians of the land on which we work and pay our respect to the Aboriginal and Torres Strait Islander Elders past, present and emerging.

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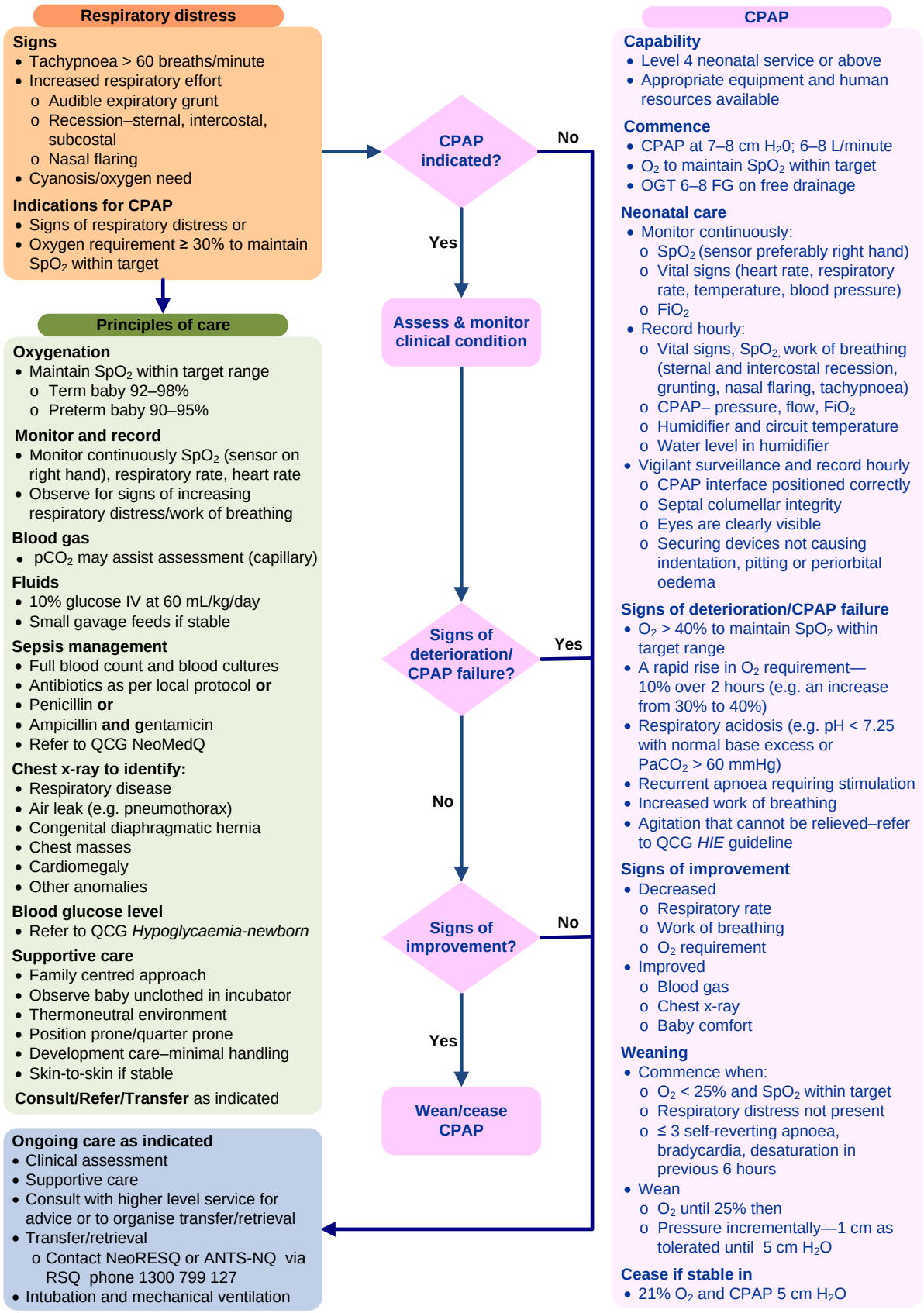
Flow Chart: Neonatal respiratory distress and CPAP in babies ≥ 32 weeks gestation



ANTS-NQ: Advanced Neonatal Transport Service-North Queensland, **CPAP:** continuous positive airway pressure, **FG** French gauge, **FiO_2:** fractional inspired oxygen, **HIE:** Hypoxic-ischaemic encephalopathy, **IV:** intravenous, **NeoRESQ** Neonatal Retrieval Emergency Service Southern Queensland, **pCO_2:** partial pressure of carbon dioxide, **QCG:** Queensland Clinical Guidelines, **RSQ:** Retrieval Services Queensland, **SpO_2:** peripheral capillary oxygen saturation, **>:** greater than, **≥:** greater than or equal to, **<:** less than; **≤**: less than or equal to

Flowchart: F20.3-1-V8-R25

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Abbreviations

AP	Antero-posterior
BPD	Broncho-pulmonary dysplasia
CPAP	Continuous positive airway pressure
CSCF	Clinical Services Capability Framework
DIP	Diabetes in pregnancy
FiO_2	Fractional inspired oxygen
FRC	Functional residual capacity
IV	Intravenous
OGT	Orogastric tube
$PaCO_2$	Partial pressure of arterial carbon dioxide
PaO_2	Partial pressure of arterial oxygen
pCO2	Partial pressure of carbon dioxide
pO2	Partial pressure of oxygen (unreliable on venous or capillary samples)
PIE	Pulmonary interstitial emphysema
PPHN	Persistent pulmonary hypertension of the newborn
RDS	Respiratory distress syndrome
RSQ	Retrieval Services Queensland
SpO_2	Peripheral capillary oxygen saturation
SSRI	Selective serotonin reuptake inhibitors
SNRI	Serotonin norepinephrine reuptake inhibitor
TTN	Transient tachypnoea of the newborn

Definitions

Acrocyanosis	Peripheral cyanosis around the mouth and extremities (palms of hands and soles of feet and is benign in the absence of central cyanosis).
Functional residual capacity	Volume of air in the lungs at the end of passive expiration.
Hypoxaemia	PaO_2 less than 60 mmHg.
Hypercarbia	$PaCO_2$ greater than 45 mmHg.
Tachypnoea	Respiratory rate greater than 60 breaths per minute.
Pulse pressure	Difference between the systolic and diastolic blood pressure.
Ventilation-perfusion mismatch	Poor perfusion to well oxygenated area and poor perfusion to poorly oxygenating areas.
Vital signs	Temperature, heart rate, respiratory rate, blood pressure.
Work of breathing	Tachypnoea, chest recession (sternal, intercostal, subcostal); nasal flaring; expiratory grunt.

1 Introduction

1.1 Lung development and respiratory physiology

The transition to potentially viable lungs occurs at the canalicular stage of fetal development (at 16–25 weeks gestation). At this stage, respiratory bronchioles and alveolar ducts develop in the gas exchange region of the lung. From 24 weeks gestation, large and primitive forms of alveoli have developed and provide potential for viability.

Surfactant production is related to the developmental stage of the fetus. After 20 weeks gestation, lamellar bodies develop, indicating that surfactant is being produced from glycogen, and being stored. The protein content of surfactant in the preterm baby's lung is low relative to the surfactant lipid. Surfactant is not usually produced in sufficient amounts until 32–34 weeks gestation. The most common cause of respiratory distress syndrome (RDS) from surfactant deficiency is preterm birth.

Maturation of the preterm fetal lung is enhanced when antenatal corticosteroids are administered to the woman. These improve lung function by enhancing maturational changes in lung development and inducing enzymes that stimulate phospholipid synthesis and surfactant production.[1-3]

Newborn babies have a compliant chest wall which leads to an increased susceptibility to more severe manifestations of respiratory disease. The degree of chest wall compliance combined with decreased lung compliance increases the severity of respiratory distress with decreased gestation.[2,4]

1.2 Clinical course of respiratory distress

Table 1. Respiratory disease clinical course

Aspect	Consideration
Pulmonary surfactant deficiency[2]	• Pulmonary surfactant: o Reduces alveolar surface tension o Facilitates alveolar expansion o Reduces the likelihood of atelectasis from alveolar collapse o If deficient causes RDS • Deficiency may be in the quantity and/or quality of surfactant causing RDS when surfactant is consumed or inactivated resulting in: o Generation of increased negative intrathoracic pressure to expand poorly compliant lungs resulting in severe sternal and intercostal recession[4] o High surface tension leading to instability of the lung at end-expiration, low lung volume and decreased compliance o Pulmonary oedema and increased airway resistance resulting from lung inflammation and respiratory epithelial injury • Risk factors and causes include: o Preterm birth o Elective caesarean section[5] o Maternal diabetes in pregnancy (DIP)–poorly controlled o Meconium aspiration syndrome (MAS)[6] o Infection o Genetic surfactant dysfunction disorder[2]
Lung function[2]	• Changes in lung function cause ventilation-perfusion mismatch due to: o Collapse of portions of the lungs (atelectasis) o Abnormal fluid absorption (due to inefficient clearing of liquid in damaged lung) resulting in pulmonary oedema that impedes gas exchange due to reduced functional residual capacity
Hypoxaemia[2]	• Caused by: o Ventilation perfusion mismatch from intrapulmonary shunting—influenced by relative size of under ventilated lung o Extrapulmonary shunting across foramen ovale and patent ductus arteriosus
Acidosis[2]	• Poor ventilation identified from: o Elevated pCO_2 and resultant respiratory acidosis • Metabolic acidosis caused by lactic acid production from anaerobic metabolism due to hypoxaemia and poor tissue perfusion

1.3 Clinical standards

Table 2. Clinical standards

Aspect	Consideration
Prevention	• Antenatal corticosteroids reduce the risk of RDS in the preterm baby less than 35+0 weeks gestation by enhancing the maturation of the lungs and improving lung function[1,2,7-9] • Late preterm (34+0–36+6 weeks gestation) corticosteroid administration is associated with decreased risk of serious neonatal morbidity risk including reduced: o Risk of moderate to severe RDS[8] o Incidence of transient tachypnoea of the newborn (TTN) o Need for surfactant[10] • Refer to Queensland Clinical Guidelines *Preterm labour and birth* guideline[11]
Principles of care	• Commence continuous positive airways pressure (CPAP) if: o Signs of respiratory distress **or** o Oxygen requirement to maintain oxygen saturation (SpO_2) is greater than or equal to 30% • Refer to Queensland Clinical Guidelines *Standard care*[12] guideline for routine aspects of clinical care • Humidified high-flow support is not recommended as first-line management of babies with RDS outside of a neonatal intensive care unit
Clinical Services Capability Framework (CSCF)	• Consider the clinical service capability of the facility o Refer to Clinical Services Capability Framework for neonatal services[13] • Care of a baby requiring ongoing CPAP requires level 4 or higher neonatal service[13,14] • Consult with neonatologist via Retrieval Services Queensland (RSQ) for advice or retrieval of baby to a higher level of service as required o If level 2–3 neonatal service and baby has respiratory distress ▪ Oxygen greater than or equal to 30% to maintain SpO_2 within target range [refer to Table 3. Signs] ▪ Oxygen requirement rapidly increases (10% or more over two hours or less) ▪ Baby is less than 35 weeks gestational age o If level 4 neonatal service and baby requires CPAP ▪ Commencing when baby is greater than 24 hours of age ▪ Oxygen requirement is greater than 40% to maintain SpO_2 within target range [refer to Table 3. Signs] ▪ Oxygen requirement rapidly increases ▪ Blood gas partial pressure arterial carbon dioxide ($PaCO_2$) is greater than 60 mmHg or pH is less than 7.25 ▪ Baby's birth weight is less than 1500 g ▪ Baby is less than 32 weeks gestational age o If level 5 neonatal service ▪ CPAP commencing when baby is greater than 24 hours of age ▪ Oxygen requirement is greater than 40% to maintain SpO_2 within target range [refer to Table 3. Signs] ▪ Blood gas $PaCO_2$ is greater than 60 mmHg or pH is less than 7.25 ▪ Baby's birth weight is less than 1000 g ▪ Baby is less than 29 weeks gestational age

2 Assessment

Assess the baby's clinical signs, and radiographic and pathology results. Also consider the maternal and neonatal history. Assess changes in the baby's clinical status over time, including increasing respiratory rate or increasing oxygen requirement to maintain oxygen saturations. Refer to Table 3. Signs

2.1 Signs

Table 3. Signs

Aspect	Consideration
Colour	• Pale *or* • Cyanosis (excluding acrocyanosis)—central and/or peripheral due to right-to-left intra- and extrapulmonary shunting
SpO_2	• Pre-ductal measure on right hand o Follow manufacturer's instructions • Targets after 10 minutes of age*: o Term baby: 92–98% o Preterm: 90–95% • Hypoxaemia usually due to ventilation perfusion mismatch[2] • Left to right shunting of blood occurs in areas of poorly ventilated lungs[2]
Cardiovascular	• Peripheral pulses diminished[2] • Prolonged capillary refill time (greater than two seconds) • May be tachycardic • May be having bradycardic episodes and/or apnoeas[2] • Extra-pulmonary shunting may occur across the foramen ovale and patent ductus arteriosus[2]
Urine output	• May be low in first 24–48 hours[2] • Hyponatraemia may be present • If baby recovering from RDS, spontaneous diuresis occurs on second to fourth day of life and precedes improved pulmonary function[2]
Respiratory	• Tachypnoea o More than 60 breaths per minute[1] o Most common presenting sign[7] o Due to hypercarbia, hypoxaemia or acidosis[15] • Breath sounds on auscultation are decreased[2] • Increased respiratory effort: o Expiratory grunt[1]—results from exhalation through a partially closed glottis; slows the decrease in end expiratory lung volume o Recession[1]—sternal/suprasternal, intercostal, subcostal due to the highly compliant rib cage that is drawn in during inspiration by the increased negative intrathoracic pressures required to expand poorly compliant lungs o Nasal flaring[1] due to use of accessory respiratory muscles ▪ Compensatory sign that increases upper airway diameter and lower total respiratory system resistance and work of breathing[15]
Respiratory acidosis	• Blood gas not routinely required after initial capillary or venous sample if within normal limits and baby is stable (to minimise pain and handling) • Measure blood gas depending on individual baby's clinical presentation or if there are signs of deterioration or CPAP failure [refer to Table 21. CPAP clinical course] • pCO_2 may assist assessment o pCO2 greater than or equal to 50 mmHg and pH less than 7.35 indicates respiratory acidosis[16] • Degree of hypoxaemia[15] (as shown by a low pO_2) is unreliable on a capillary or venous sample

*In the absence of good quality evidence, Queensland Neonatal Services Advisory Group (QNSAG) endorsed, by consensus, these recommended oxygen saturation targets for babies after 10 minutes of age (2018).

2.2 X-rays

- Standard anteroposterior (AP) chest x-ray is sufficient[17]
  - Take chest x-ray at four hours of age unless earlier indication
- Assists with diagnosis[17]
- Checks position of tubes and lines—including orogastric tube (OGT)[17]
- Assists assessment of pulmonary inflation[17]
- Excludes undiagnosed congenital abnormality[17]
- Excludes pulmonary air leaks[17,18]

Table 4. X-ray interpretation

Aspect	Consideration
Normal	• Symmetrically aerated lung fields • Diaphragm is at level of the 8th ribs posteriorly and 6th ribs anteriorly • Heart size—transverse cardiothoracic ratio less than 60% (may be difficult to evaluate due to the thymic shadow[17])
RDS	• Low lung volume • Diffuse reticulogranular ground glass appearance • Air bronchograms (more severe) • Confluent alveolar shadowing[17] • Radiographic pattern results from: o Alveolar atelectasis contrasting with aerated airways o Pulmonary oedema may contribute to diffuse appearance
TTN	• Normal or slightly overinflated lung fields • Increased streaky shadowing with perihilar densities[7]extending into peripheral lung fields • Normal or mildly enlarged heart • Fluid in the horizontal fissure and small pleural effusions may be present[1,17]
Air leaks	• Pneumothorax and other air leaks: o Common in initial x-ray o More often observed when lung compliance improves o Consider air leak if acute deterioration in baby's condition • Pneumothorax: o Air in pleural space, atelectasis with flattening of the diaphragm on the affected side, shift of mediastinum away from the pneumothorax[18] • Pneumomediastinum: o Radiolucent halo over the cardiac outline or retrosternal or mediastinal radiolucency (on lateral view)[17,18] o Thymus gland lifted giving 'angel wing' or 'spinnaker sail' appearance[17,18] • Pneumopericardium: o Air in the pericardial space o May cause cardiac tamponade o In antero-posterior (AP) view air surrounds the heart shadow within the pericardium[18] • Refer to 3.4 Pulmonary air leaks
Pulmonary interstitial emphysema (PIE)	• Interstitial air appearing as cyst-like or linear translucencies[18]
Congenital diaphragmatic hernia	• Mediastinal shift • Absence of diaphragm–usually left sided • Visible bowel gas in chest cavity[19]
MAS	• Asymmetric patches of opacification and streaky linear densities • Hyperinflation of lungs • Flattening of the diaphragm[6,20]

3 Diagnosis

3.1 Differential diagnosis

Table 5. Differential diagnosis

Aspect	Consideration
Diagnosis	• Consider: o Baby's history—gestational age and postnatal age o Maternal history o Clinical features o Chest x-ray o Capillary/venous blood gas • Refer to Appendix A Comparison of respiratory disease
Non-pulmonary causes[7]	• Consider: o Hyperthermia from overheating baby resulting in tachycardia, tachypnoea and increased oxygen consumption[21] o Sepsis including meningitis o Cardiac disease[1] o Metabolic acidosis from inborn error of metabolism o Hypoglycaemia—may cause or aggravate tachypnoea o Polycythaemia or anaemia o Hydrocephalus o Intracranial haemorrhage o Maternal narcotic use causing respiratory depression

3.2 Respiratory distress syndrome

Table 6. Respiratory distress syndrome

Aspect	Consideration
Physiology	• Caused by: o Under developed lungs o Surfactant deficiency and or dysfunction[22]
Presentation	• Presents more commonly if: o Baby is less than 34 weeks gestation[7] o Maternal DIP[7] • Baby has respiratory distress within first minutes after birth[2] o Worsens over the next 48 hours[2] • Baby generally improves by the third to fourth day of life with onset of diuresis[15]
Signs	• Increased work of breathing[15] [refer to Definitions • Cyanosis and hypoxia • Breath sounds diminished; coarse rhonchi in bilateral lung fields[15] • Bell shaped chest • Refer to Table 3. Signs • Chest x-ray–refer to Table 4. X-ray interpretation • Blood gas–hypoxaemia and respiratory acidosis[7] o If baby has metabolic acidosis and history suggestive of hypoxaemic ischaemic encephalopathy (HIE) refer to Queensland Clinical Guideline *Hypoxic ischaemic encephalopathy (HIE)*[23]
Management	• Refer to Section 16 Management • Early CPAP is usually indicated[24] • May require exogenous surfactant Curosurf® (poractant alfa) or Survanta® (beractant) with/without intermittent positive pressure ventilation o Refer to NeoMedQ[25,26] for surfactant indications and administration

3.3 Transient tachypnoea of the newborn

Table 7. Transient tachypnoea of the newborn

Aspect	Consideration
Physiology	• Parenchymal lung disorder more commonly seen in term or late preterm babies[1] • Occurs due to pulmonary oedema resulting from delayed reabsorption and clearance of fetal alveolar fluid[7]
Presentation	• Common cause of respiratory distress in the newborn baby[1,7] o Occurs two to three times more often in babies of women with diabetes[1] • Onset is usually at the time of birth and within two hours of age[1,7] o Symptoms usually last for 12–24 hours, but may last up to 72 hours • May occur with RDS[1,7] • More common if[1]: o DIP o Born by caesarean section o Precipitous birth[7] o Small for gestational age or large for gestational age/macrosomia o Maternal obesity[1] o Maternal asthma o Born before 39+0 weeks
Signs	• Increased work of breathing[1] [refer to Definitions o Tachypnoea–higher respiratory rate associated with longer duration of TTN[7] • Cyanosis[1] • Increased AP chest diameter • Breath sounds clear without rales or rhonchi[1,7] • Barrel-shaped chest • Oxygen requirement usually less than 40%[1] • Chest x-ray: o Refer to Table 4. X-ray interpretation • Blood gas—hypoxaemia, hypercapnia or respiratory acidosis[7]
Management	• Supportive measures[7] o Refer to Table 12. Supportive care • Fluid restriction may be beneficial[7,27]

3.4 Pulmonary air leaks

Pulmonary air leaks occur more often in the neonatal period than at any other time of life.[18] Refer to Appendix B Pulmonary air leaks.

Table 8. Pulmonary air leaks

Aspect	Consideration
Context[7]	• Air escapes from the lungs into the extra-alveolar space • Results from overdistension of alveoli due to uneven distribution of gas or generalised air trapping • May be: o Complication of RDS o An isolated presentation o Associated with another underlying disorder • Diagnosis is made from chest x-ray[2] and clinical assessment o Refer to Table 4. X-ray interpretation
Pneumothorax[18]	• Air escapes into the pleural space • Suspect in baby with unexpected deterioration in oxygenation, ventilation or cardiovascular status • Signs: o Increased work of breathing [refer to Definitions o Positive chest transillumination (if available) ▪ May require chest x-ray to confirm (refer to Table 4. X-ray interpretation) ▪ Not reliable in term or large baby ▪ May be incidental finding
Tension pneumothorax[18]	• Increases intrathoracic pressure, increases central venous pressure and decreases venous return • Signs: o Acute deterioration in baby's condition o Chest asymmetry with enlargement on the affected side o Deceased breath sounds on affected side o Shift of heart sound away from the affected side o Hypotension (with narrow pulse pressure), bradycardia, oxygen desaturation and poor perfusion o Sudden increase in oxygen requirement to maintain SpO_2 within target range [refer to Table 3. Signs] o Positive chest transillumination (if available) ▪ Not reliable in term or large baby • Requires emergency management • Refer to Appendix B Pulmonary air leaks
Pneumo-mediastinum[18]	• Air escapes into the mediastinal space • Usually caused by high peak pressures during IPPV[28] • Signs: o Usually asymptomatic—may be detected by cardiac auscultation (distant heart sounds)
Pneumo-pericardium[29]	• Air escapes from the mediastinum or pleural space into the pericardial sac • If untreated, the air causes a tamponade with shock and subsequent cardiac arrest • Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required
PIE[18]	• Air is trapped in the perivascular space resulting in decreased compliance and increased airway resistance • More common in very low birth weight ventilated babies • Signs: o Increasing hypoxaemia and hypercapnia usually presenting within 96 hours of birth

3.5 Congenital anomalies

Congenital anomalies are less common causes of respiratory distress in newborn babies[7] and are usually diagnosed antenatally. Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required.

Table 9. Congenital anomalies

Aspect	Consideration
Congenital heart disease[7]	• Usually presents with less respiratory distress unless persistent pulmonary hypertension is also present o Refer to Table 11. PPHN • Chest x-ray characterised by **absence** of diffuse reticulogranular ground glass appearance with air bronchograms • Respiratory support and surfactant do not improve the baby's clinical condition[2]
Pulmonary hypoplasia[7]	• May occur in isolation or be associated with other congenital anomalies, (e.g. congenital diaphragmatic hernia[17]) • Unilateral: o Chest x-ray characterised a small lung with reduced vascularity and ipsilateral heart displacement[17] • Bilateral: o Associated with oligohydramnios and prolonged preterm rupture of membranes o Chest x-ray may show bell shaped thorax, reduced inflation increased pulmonary shadowing and pulmonary air leaks[17]
Diaphragmatic hernia	• Usually diagnosed antenatally by ultrasound scan • Signs include scaphoid abdomen, diminished breath sounds ipsilateral to the side of the hernia, displacement of heart sounds contralateral to the side of the hernia[19] • Refer to Table 4. X-ray interpretation
Airway	• Obstruction: o Pierre Robin sequence o Choanal atresia[7] • Malformation: o Tracheo-oesphageal fistula[7] o Oesophageal atresia o Laryngeal web
Other	• Congenital cystic adenomatoid malformation o Chest x-ray characterised by large isolated cystic area above diaphragm

3.6 Other respiratory distress

3.6.1 Infection, aspiration and interstitial lung disease

Table 10. Infection and aspiration

Aspect	Consideration
Infection	• Bacterial pneumonia[7] • X-ray demonstrates patchy opacities and/or lobar consolidation • Blood cultures assist diagnosis • Refer to Table 12. Supportive care
MAS	• History: o Meconium stained liquor or evidence of meconium staining on examination o Baby frequently small for gestational age or postmature o Intrauterine pathologic processes[20]–asphyxia, infection • May lead to: o Airway obstruction o Chemical irritation o Inflammation o Infection o Increased inactivation of surfactant[20] o Persistent pulmonary hypertension of the newborn[20] o Increased incidence of pneumothorax o Hypoxaemia due to ventilation-perfusion mismatch • Signs include[1]: o Marked tachypnoea and cyanosis o Intercostal and substernal recession due to reduced lung compliance and use of accessory muscles o Grunting and nasal flaring[20] o Barrel-shaped chest o Rales and rhonchi on auscultation o X-ray–refer to Table 4. X-ray interpretation o Blood gas–respiratory acidosis[30,31] • Management: o Monitor preductal and postductal SpO_2 o Refer to Table 12. Supportive care o Intermittent positive pressure ventilation may be indicated o Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required
Other aspiration	• May be caused by aspiration of: o Blood, liquor or water (if water birth) • If baby has commenced feeds and aspirates milk, consider trachea-oesophageal fistula or oesophageal atresia
Interstitial lung disease	• Interstitial and diffuse lung diseases including[2]: o Genetic disorders of surfactant dysfunction o Lung growth abnormalities o Pulmonary interstitial glycogenosis

3.6.2 Persistent pulmonary hypertension of the newborn (PPHN)

Table 11. PPHN

Aspect	Consideration
Physiology	• Physiological adaptation after birth has not occurred[7] resulting in persistent or delayed transition of fetal circulation
Signs	• SpO2 difference of greater than 5% between preductal and postductal measures due to right-to-left shunting though the patent ductus arteriosus[32] o Right-to-left shunting can also occur through the foramen ovale and will have an absent oxygen saturation gradient[32] o Measure on right hand/wrist (preductal), and left hand/wrist or either foot (postductal) • Arterial blood gas—normal $PaCO_2$ (if no lung disease present) and PaO_2 below 100 mmHg in 100 percent inspired oxygen[32] • Barrel-shaped chest
Causes[32,33]	• Idiopathic • Abnormalities of pulmonary vasculature o Underdevelopment (e.g. fetal growth restriction, diaphragmatic hernia) o Maldevelopment (e.g. congenital heart disease, meconium aspiration syndrome) o Maladaptation (e.g. post-term birth, caesarean birth, large for gestational age, perinatal asphyxia, sepsis) • Associated with maternal use of selective serotonin reuptake inhibitors (SSRI) or serotonin norepinephrine reuptake inhibitors (SNRI) in late pregnancy (greater than or equal to 33+0 weeks gestation)[32,33]
Management	• If PPHN suspected: o Aim to keep oxygen saturation at greater than 97% o Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required o Minimise noise, light and handling baby o Minimise baby crying • Refer to Table 12. Supportive care

4 Management

4.1 Supportive care

Observe the unclothed baby (with nappy only), and only disturb the baby when absolutely necessary (minimal handling). Refer to Table 17. Care of baby on CPAP, Table 18. Developmental care and Table 19. Pressure area care for other aspects of care.

Table 12. Supportive care

Aspect	Consideration
Oxygenation	• Give oxygen to maintain SpO_2 within target ranges o Refer to Table 3. Signs • Monitor delivered oxygen concentration continuously • If greater than or equal to 30% oxygen is required, consider CPAP o Refer to Table 14. CPAP use] • If PPHN suspected–refer to Table 11. PPHN
Fluids	• Insert intravenous (IV) cannula and commence glucose 10% IV at 60mL/kg/day • If peripheral IV cannulation is difficult (not achieved after three attempts), consider umbilical venous catheter • If the baby's respiratory status is stable, consider commencing small gavage feeds o If the baby's respirations are greater than 80 breaths per minute do not feed
Temperature	• Nurse baby in incubator or on open care system • Refer to Queensland Clinical Guideline *Stabilisation for retrieval–neonatal*[23] • Maintain temperature within normal range o Axilla 36.8–37.2 °C • Monitor and record temperature every 4 hours
Blood glucose	• Refer to Queensland Clinical Guideline *Hypoglycaemia–newborn*[34]
Infection screening	• If clinically indicated, surface swabs from baby • Full blood count • Blood cultures–check result at 24 hours: o If negative and respiratory distress resolved—administer 24 hour antibiotic dose and then cease (36 hour coverage provided) o If delay in processing blood cultures—continue antibiotics until 48 hours of treatment o Recheck result at 48 hours o If positive (at any time), consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required
Antibiotics*	• Empirical antibiotics are given prophylactically until cultures and clinical course known benzylpenicillin **OR** amoxicillin/ampicillin **AND** gentamicin • Refer to NeoMedQ neonatal monographs for benzylpenicillin[35] ampicillin[36] and gentamicin[37] • Refer to Queensland Clinical Guideline *Early onset Group B Streptococcal disease*[38]
Monitoring	• Monitor and record every hour (or if change in baby's condition)[7]: o Vital signs—temperature, heart rate, respirations, blood pressure and perfusion o Oxygen saturations—preferably on right hand/wrist (pre-ductal) ▪ If PPHN suspected, refer to Table 11. PPHN o Work of breathing [refer to Definitions • Check blood gases as clinically indicated • Check blood glucose levels [refer to Queensland Clinical Guideline *Hypoglycaemia–newborn*[23,39]]
Blood gas	• Determine frequency based on baby's clinical condition and support required o Not routinely required if previous blood gas within normal limits o If baby is stable, leaving baby undisturbed may be indicated
Pain management	• Refer to Table 17. Care of baby on CPAP

*Refer to an Australian pharmacopoeia for complete drug information

5 Continuous positive airway pressure

Preterm babies are prone to lung collapse due to a lack of surfactant. Disparity between chest wall and lung compliance is heightened in babies with surfactant deficiency.[4] CPAP works by maintaining expansion of the alveoli by providing a constant positive pressure to the lungs. This prevents atelectasis and allows gas exchange.[4]

5.1 CPAP physiology

Table 13. CPAP physiology

Aspect	Consideration
Context	• Works by delivering continuous distending pressure to the lungs using an air/oxygen mix • Continuous flow (e.g. bubble)[40] is generated using continuous gas flow during the respiratory cycle[41] • Prevents atelectasis • Enhances gas exchange[42] • Rationale for use: o Stents the airways and maintains functional residual capacity (FRC)
CPAP physiology	• Improves lung compliance[43] and uniformity of ventilation by preventing: o De-recruitment of alveoli o Collapse of small airways during exhalation in the surfactant deficient lung o Increases functional residual capacity and improves oxygenation[44] • Previously constricted blood vessels dilate when atelectasis is reversed, and this leads to increased pulmonary blood flow • Ventilation-perfusion mismatch improves and intra-abdominal pulmonary shunting decreases: o $PaCO_2$ lowers o Progressing arterial hypoxaemia is prevented o Respiratory muscle fatigue is decreased[4,43,45] • Surfactant is conserved by reducing the protein leak associated with atelectasis[4,40] • Dilates the pharynx, reduces subglottic airway resistance, lessens the incidence of obstructive apnoea • Improves the synchrony of thoracoabdominal movements[44]

5.2 CPAP use

Table 14. CPAP use

Aspect	Consideration
Prophylactic	• Insufficient evidence to compare prophylactic CPAP to oxygen therapy and other supportive care[24] in term babies • Prophylactic CPAP in the very preterm baby provides a small but clinically significant reduction in broncho-pulmonary dysplasia (BPD) and death, and need for mechanical ventilation and use of surfactant[24]
Indications	• Correct existing respiratory failure in babies with RDS[4] o Baby has signs of respiratory distress [refer to Table 3. Signs] • Treat airway obstruction[4] • Prevent respiratory failure (e.g. apnoea of prematurity)[4]
Contraindications	• Nasal CPAP is contraindicated in babies with a range of congenital abnormalities or surgical conditions, including o Certain cleft palates o Bi-lateral choanal atresia ▪ Single naso-pharyngeal tube may be used in unilateral choanal atresia o Tracheo-oesphageal atresia o Congenital diaphragmatic hernia o Gastroschisis or omphalocele o Necrotising enterocolitis (suspected or confirmed) o Significant loss of skin or septal integrity following use of bi-nasal CPAP o Congenital lung/airway lesions (e.g. congenital lobar emphysema, congenital pulmonary airway malformation) o Post-abdominal surgery • Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required
Benefits[43,46]	• May improve lung compliance and stabilise the compliant chest wall • Improves thoracoabdominal synchrony • Reduces work of breathing [refer to Definitions • Upper airway splinting reduces obstructive apnoea o May decrease central apnoea due to regular breathing pattern • Reduces oxygen requirements • Reduces risk of bronchopulmonary dysplasia
Potential harms[43]	• Risk of pulmonary air leaks due to airway distending pressure • Facial trauma including loss of skin or nasal integrity from the CPAP interface [refer to Table 19. Pressure area care] • Abdominal distension [refer to Table 17. Care of baby on CPAP]

5.3 CPAP administration

- Manage the baby on CPAP in a level 4 or higher neonatal service—CSCF[13]
- The nurse:patient ratio is generally 1:2 depending on acuity
- If the baby is unstable the ratio is 1:1
- Completion of a neonatal CPAP training program by clinical staff[13] is recommended
- Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required

Table 15. CPAP administration

Aspect	Consideration
Context	• CPAP system provides stable pressures at required levels with humidity and supplemental oxygen[47] • Early versus delayed initiation of CPAP reduces the requirement for mechanical ventilation and surfactant[48,49] and the incidence of BPD[49,50] • Goal of CPAP delivery device is to prevent atelectasis and airway closure[47] • Mean airway pressure delivered to a baby by nasal CPAP is affected by: o Seal of the interface o Loss of pressure through the mouth o Airway resistance[40,51]
Nasal CPAP	• Improves oxygenation and maintains lung volume • Increases FRC, distends the larynx, reduces subglottic airway resistance[52] • Standard respiratory treatment over invasive intubation[7]
Bi-nasal prongs	• Standard of care for babies of all gestations who breathe spontaneously and require continued respiratory support[45] • Better than single prong to deliver CPAP to preterm baby for early treatment of RDS[44] o Lower oxygen requirements (than single prong) o Incidence of BPD, pulmonary air leaks, ventilator associated pneumonia and neurocognitive disorders is lower than with mechanical ventilation[45] o Longer term outcomes and medium-term outcomes (e.g. chronic lung disease at 36 weeks corrected age) not identified[44] • Associated with increased resistance to air flow • Correct fitting of device minimises perinasal leak and nasal trauma[53] o Refer to Table 19. Pressure area care
Nasal mask	• If staff are competent providing nasal mask CPAP, may be used in non-tertiary facility as an alternative interface to nasal prongs • Choose correct size mask—small enough to be flush with side of baby's face and not slide out of position • Ensure mask does not slide up and occlude the nares[45] • May relieve trauma from prongs[51] o Evidence from a systematic review and meta-analysis reported a lower incidence of nasal trauma with very low certainty evidence[54] • May reduce CPAP failure[53]
Bubble device versus ventilator	• Bubble device versus ventilator to deliver CPAP o No difference in physiological parameters in the short-term[47,52]

5.4 CPAP settings

Record CPAP pressure, gas flow, fractional inspired oxygen (FiO_2) and humidifier temperature hourly and when any changes made to settings.

Table 16. CPAP settings

Aspect	Consideration
Pressure	• Commence at 7–8 cm H_20
Flow	• Generally, 6–8 L per minute[55] depending on CPAP device being used • Use lowest level possible to deliver desired pressure o Refer to manufacturer's instructions
Oxygen	• Deliver FiO_2 to maintain the baby's oxygen saturations within target levels • Refer to Table 3. Signs and Table 12. Supportive care
Humidification	• Humidify and warm gases before delivery to baby • Set temperature[41] according to manufacturer's instructions–generally: o Interface with baby 37 °C o Humidifier 40 °C
Device management	• Insert circuit through lowest insertion port on incubator to: o Assist drainage of condensation away from the baby[45,52] o Reduce aspiration risk • Position inspiratory limb (blue) above expiratory limb (white)

5.5 Care of baby

5.5.1 General care

Table 17. Care of baby on CPAP

Aspect	Consideration
Assessment and monitoring	• To determine observations and assessment required, refer to Table 3. Signs and Table 12. Supportive care
Fluids and feeding	• Commence/continue IV therapy [refer to Table 12. Supportive care] • Insert a size 6–8 FG OGT to minimise gastric distension[21,45] o If significant abdominal distension the larger (8 FG) OGT may be required • OGT: o Leave on free drainage to vent stomach[45] o Cap for 30 minutes after a feed o Aspirate air every 4–6 hours and as required • Non-nutritive sucking on a pacifier[56] while on CPAP is beneficial: o Promotes physiological stability o Reduces stress o Relaxes the baby during invasive procedures o Improves preparation for oral feeding o Improves oxygenation o Reduces air leak from mouth
Suctioning	• Keep airway clear of secretions o Routine suctioning not required o Avoid deep suctioning[45] • Risks from suctioning include: o Infection, bradycardia, laryngospasm, blood pressure fluctuations, hypoxaemia, increased intracranial pressure, cardiac arrhythmia, nasal injury[57] • Provide comfort measures including facilitated tucking, swaddling and oral sucrose to reduce associated pain • Consider use of peanut pillow to maintain midline position of head during suctioning
Pain management	• Minimise unnecessary noxious procedures • Assess and record pain every 4 hours using validated pain management tool[21] • Can be alleviated non-pharmacologically by: o Modifying the environmental and sensory stimuli (e.g. noise and light) o Sucrose 25% orally[58] o Positioning of baby [refer to Table 18. Developmental care] o Non-nutritive sucking[59]

5.5.2 Developmental care and positioning

Table 18. Developmental care

Aspect	Consideration
Supportive care	• If baby is fragile or compromised, perform cares by two clinicians, for example if: o Has acute lung disease o Weighs less than 1000 g o Requires greater than 7 cmH_2O CPAP o Requires greater than 25% oxygen • Minimise time off CPAP • Provide oxygen in the incubator at the same concentration as CPAP in case device dislodges • Encourage family collaboration[21,45] and unrestricted visiting[45] o Encourage parent/s to assist with eye and mouth cares and head massage with assistance from clinician (to maintain CPAP) o Encourage skin to skin contact as soon as clinical condition allows[60]
Positioning	• Position baby to avoid: o Inadvertent tension to the interface[45] o Accumulation of condensate to the nares • Use pacifier or chin strap to keep baby's mouth closed (to maintain CPAP) o If the baby's mouth habitually remains open use a chin strap • Position the strap under the bony portion of the chin and not over the fleshy portion of the hypopharynx region—may cause airway obstruction and increase risk of aspiration[45] • Avoid lateral positioning of baby with acute lung disease as this may reduce effective bilateral lung expansion and increase the risk of atelectasis • Prone positioning o Positive effect on respiratory mechanics by increasing oxygenation, improving ribcage-abdominal synchrony and better consistency of breathing pattern[12] o Disadvantages include developmental delays, corneal lesions and loss of vascular access in long term use[61] • Prone quarter turn position beneficial[61-63] for stabilising respiratory rate and increasing oxygen saturation o Place a small neck roll under the baby to prevent neck flexion and airway obstruction[60] o Nest baby by supporting hips, uppermost arm and trunk with roll o Position knees and feet in neutral alignment with hips
Developmental care	• Developmental positioning is important to prevent pressure injury and deformational plagiocephaly[42] • Release bonnet for a few minutes with each routine handling and care episode—assess skin including scalp and ears • Perform care as indicated by baby's clinical condition—usually 4–6 hourly by one or two people o Involve parents in the baby's cares o Minimal handling—disturb baby only when absolutely necessary o If baby positioned supine during cares, use of a peanut pillow to help maintain head in mid-line position o Refer to Table 12. Supportive care • Comfort and natural development provided by: o Gentle touch and containment o Developmental positioning o Age-appropriate stimulation o Cycled lighting o Decreased environmental stimuli including noise o Family bonding time[42]–encourage parents to be present during painful procedures[64] • Benefits of skin-to-skin positioning when clinically suitable are physiological, psychological, behavioural, neurobehavioral and psychosocial[42]

5.5.3 Pressure area care

Table 19. Pressure area care

Aspect	Consideration
Context	• Constant pressure on the nares, nasal septum and forehead can lead to reduced skin integrity and injury[65] causing pressure injury (ulcers) and friction shear from respiratory tubing[42] • Nasal trauma: o Most common complication of nasal CPAP[66]–incidence 15–60% o Occurs commonly on medial aspect of nostrils o Pressure caused by poorly fitting nasal prongs or carrying crossbar exerting pressure on septum o Most common nasal injuries–necrosis, scab formation, excoriation of the septum, nasal hyperaemia and disfigurement of the size and shape, nasal erosion, functional airway obstruction[67,68] o May progress to facial scarring[42] and/or permanent deformity • Risk factors for skin breakdown, development of nasal pressure ulcers, compression necrosis, and/or nasal deformities[69]: o Nasal CPAP device o Length of therapy o Age, gestation and size of baby o Poor perfusion o Environmental humidity and temperature
Assessment	• Provide vigilant skin assessments and skin resting time[70] • First sign of skin breakdown is nasal erythema[69] • Assess baby regularly (with cares) for signs of pressure injury o Nasal: redness, blanching, skin breakdown, necrosis, bruising, indentation, bleeding, altered shape o Ears: creases, folds, pressure areas o Forehead: if using midline device (mask or prongs) o Nasal bridge: mid-facial indentation (mask) o Head: especially on bony prominences–remove hat to assess) • Documentation of pressure area injury includes location, nature and extent–clinical photos may assist assessment of progression and recovery
Prevention	• Measure and size the interface device for each baby • If prongs fitted correctly pressure is avoided on high risk areas, and excess rubbing and movement are prevented[45] o Position binasal prongs 2 mm from the nares o Avoid contact with the septal columella o Fit firmly without blanching the skin to decrease movement[45] o Avoid pressure on the nasolabial sulcus and philtrum o Avoid propping the cot as baby sliding will cause pressure on the septum o Resizing may be required as the nares increase in size • If mask is used ensure it covers entire nose–do not fit mask tightly and avoid indentations and pressure on nasal bridge • Consider use of septum skin protector[67,71]: o Reduces nasal injuries–use soft silicone-based skin protection over vulnerable areas ▪ Assess regularly for moisture[72], and change at least every 12 hours ▪ Remove during trial off CPAP as baby is unable to nasal flare [refer to Table 3. Signs] o Helps to provide seal around device • Avoid hydrocolloid nasal dressing under binasal prongs within first 15 minutes of insertion as it inhibits visualisation of nares and septal columella, and pressure is injury most likely to occur during this time • Avoid tight fitting hat over forehead, ears and bony prominences • Position the baby's hand under their chin to help keep the mouth closed without the use of a chin strap[45]
Management	• If pressure area(s) present: o Review interface, delivery mechanisms and pressure injury reduction measures[45] and reassess size of prongs or mask o Consider alternating between binasal prongs and mask

5.6 Complications of CPAP

Table 20. CPAP complications

Aspect	Consideration
Pulmonary air leaks	• Continuous distending pressure (CPAP) may increase the risk of pneumothorax[18,73]
Pain	• CPAP treatment and associated procedures (e.g. heel pricks[64], suctioning) are painful or unpleasant[21,74] due to: o Flow of gas through nose and mouth o Restricted head movements o Obstructed vision o Suctioning • Refer to Table 17. Care of baby on CPAP
Abdominal insufflation	• Caused by delivered gas entering the stomach and gastrointestinal tract[21,45] • May cause feed intolerance[45,75] • Use lowest possible flow of gas to achieve required CPAP [refer to Table 16. CPAP settings] • Refer to Table 17. Care of baby on CPAP
Hyperinflation of lungs	• Caused by excessive pressure that results in: o Increased work of breathing[75] [refer to Definitions o Reduced cardiac output secondary to impaired venous return[75] • Refer to Table 16. CPAP settings for recommended CPAP in babies
Pressure injury	• Refer to Table 19. Pressure area care

5.7 Clinical course

Table 21. CPAP clinical course

Aspect	Consideration
Signs of improvement	• Reduction in the baby's work of breathing as shown by reduction in: o Respiratory rate (typically by 10–20 breaths per minute) o Expiratory grunting o Chest recession o Nasal flaring • Stabilised or reduced oxygen requirement to maintain SpO_2 within target range • Improvement in blood gas (if taken) • Improvement in appearance and lung volumes on chest x-ray • More settled baby
Weaning/ceasing	• Refer to Table 22. Weaning CPAP
Signs of deterioration	• If baby's condition deteriorates, consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required • Signs of deterioration include: o Respiratory signs not improving or worsening o Oxygen requirement increasing or greater than 40% to maintain target SpO_2 o Apnoeic episodes o Blood gas showing increasing respiratory acidosis
CPAP failure[76]	• Defined as the need for endotracheal intubation and mechanical ventilation due to severe respiratory distress and high oxygen requirement within 72 hours of nasal CPAP initiation[53,77] • Signs[76]: o FiO_2 greater than 40% (or according to local protocols) to maintain target SpO_2 with 8 cmH_2O CPAP o Rapid increase of oxygen requirement including a rise of 10% over 2 hours or less o Respiratory acidosis—pH less than 7.25 with normal base excess or $PaCO_2$ greater than 60 mmHg o Recurrent apnoeic episodes requiring stimulation o Increase in work of breathing [refer to Definitions o Development of pneumothorax [refer to Table 8. Pulmonary air leaks] o Baby agitated and not settled by comfort measures [refer to Table 18. Developmental care] ▪ Review respiratory status ▪ Assess for other causes and manage [refer to Table 17. Care of baby on CPAP • If there are signs of failure: o Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required o Assess baby (by medical officer/nurse practitioner) immediately o Urgent chest x-ray o Transilluminate chest to detect signs of pulmonary air leak ▪ Negative transillumination may not be meaningful in a baby greater than or equal to 32 weeks gestational age [refer to Table 8. Pulmonary air leaks] o Check blood gas for hypercarbia or severe metabolic acidosis ▪ If metabolic acidosis refer to Queensland Clinical Guideline *Hypoxic ischaemic encephalopathy (HIE)*[23] o Refer to Queensland Clinical Guideline *Stabilisation for retrieval–neonatal*[23]

5.8 Weaning CPAP

Table 22. Weaning CPAP

Aspect	Consideration
Context	• Lung compliance changes occur between 48 and 96 hours of age due to surfactant secretion • If CPAP is discontinued prematurely, risk of[78]: o Atelectasis o Apnoea and bradycardia • If CPAP is continued unnecessarily, risk of complications include[78]: o Gastric distension; nasal trauma; agitation o Alveolar over distension resulting in longer term consequences[78] (e.g. BPD)
Weaning	• Reduce FiO_2 before reducing the CPAP pressure o Maintain SpO_2 levels in target range[60] [refer to Table 3. Signs] • Commence gradual CPAP weaning when: o Oxygen requirement less than 25% o Respiratory distress improved ▪ Respiratory rate less than 60 breaths per minute ▪ Chest recessions absent o Apnoeas are no longer than 20 seconds duration and are self-reverting o Bradycardic episodes are not lower than 100 beats per minute o Average SpO_2 greater than 95%[79] for the previous 6 hours • Wean incrementally by 1 cmH_2O as tolerated until 5 cmH_2O • Cease if baby is stable in 21% oxygen and CPAP 5 cmH_2O • Signs of CPAP being ceased too early include: o Increasing apnoea frequency o Increased oxygen requirement o Increased work of breathing [refer to Definitions] • Weaning too early may result in a need to restart CPAP or intubation and ventilation[76] • Weaning CPAP to a predefined level and then stopping completely, results in: o Less total time on CPAP o Shorter durations of oxygen o Reduced length of hospital stay[76]
Duration	• Clinical criteria to reduce, wean or discontinue CPAP: o Gestational and chronological age of baby o Current weight o Clinical markers of respiratory stability • Assess clinical signs indicating the baby is not tolerating CPAP reduction or cessation[43] • Babies born at lower gestations compared to more mature babies (equivalent corrected age) require a longer total duration and are more likely to fail CPAP weaning[43] • Prolonged use of CPAP (as indicated) reduces the overall supplemental oxygen requirement[43]
Recommencing CPAP	• Before recommencing CPAP assess baby's clinical condition to exclude complications requiring additional management (e.g. pneumothorax, apnoea of prematurity) • Recommence if: o Work of breathing increases [refer to Definitions o SpO_2 falls • Recommence at 6–8 cmH_2O and adjust accordingly

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Appendix A Comparison of respiratory disease

Type	Gestation	Onset after birth Cause	Risk factors	Clinical features	X-ray features Blood gas
TTN	Late preterm/ term	Immediate to within 2 hours of birth	Caesarean birth Precipitous birth Macrosomia Male Maternal asthma Maternal diabetes	Cyanosis Increased work of breathing* Increased AP chest diameter Barrel shaped chest Breath sounds clear	Normal or slightly overinflated lung fields Increased streaky shadowing with perihilar densities extending into peripheral lung fields Normal or mildly enlarged heart Small pleural effusions may be present
		Delayed reabsorption and clearance of fetal lung fluid			Hypoxaemia Respiratory acidosis
RDS	Preterm	Immediate	Male Caucasian Maternal diabetes	Increased work of breathing* Hypoxia Cyanosis Bell shaped chest Diminished breath sounds	Ground glass appearance Air bronchograms Decreased lung volumes
		Deficiency of surfactant Under-developed lungs			Hypoxaemia Acidosis
Meconium aspiration	Term/post-term	Immediate	Birth through meconium stained liquor	Cyanosis Increased work of breathing* Barrel-shaped chest Rales and rhonchi on auscultation	Asymmetric patches of opacification and streaky linear densities Hyperinflation of lungs Flattening of the diaphragm
		Lung irritation Airway obstruction (ball valve effect)			Hypoxaemia Hypercarbia Respiratory acidosis
Infection	Any	Early: 24–72 hours of Delayed: 5–14 days	PROM Maternal Group B streptococcal colonisation Maternal fever	Overlap with RDS Non-specific (e.g. poor feeding)	Overlap with RDS
		Placental transmission Infected amniotic fluid aspiration			

*Increased work of breathing–tachypnoea, chest recession (sternal, intercostal, lower costal); nasal flaring; audible expiratory grunt

Comparison of respiratory disease (continued)

Type	Gestation	Onset after birth Cause	Risk factors	Clinical features	X-ray features Blood gas
Congenital diaphragmatic hernia	Any	Immediate (usually diagnosed antenatally)–dependent on type and severity		Barrel shaped chest Scaphoid appearing abdomen Absence of breath sounds on ipsilateral side–may have displaced heart beat (left sided)	Absence of diaphragm–usually left sided Visible bowel gas in chest cavity No visible aerated lung on affected side
Persistent pulmonary hypertension	Late preterm, term, post-term	Within 24 hours of birth Abnormalities of pulmonary vasculature • Underdevelopment (e.g. diaphragmatic hernia) • Maldevelopment (e.g. CHD, MAS) • Maladaptation (e.g. post-term birth, caesarean birth, LGA)	Intrauterine and perinatal asphyxia SSRI exposure PPROM	Worsening respiratory distress and cyanosis despite high F_iO_2	Normal or reflects underlying respiratory disease Severe hypoxaemia Normal/mild hypercarbia Metabolic acidosis

Appendix B Pulmonary air leaks

Type	Gestation	Description	Risk factors	Clinical features	X-ray features/blood gas
Pneumothorax	Any	Occurs any time Extra pleural pressure exceeds intrapleural pressure	Infection Aspiration Lung deformity Ventilation causing barotrauma	May be asymptomatic Unexpected deterioration in oxygenation, ventilation or cardiovascular status On affected side: • Chest asymmetry • Decreased breath sounds • Shift of heart sounds away	Air in pleural space, atelectasis with flattening of the diaphragm on the affected side, shift of mediastinum away from the pneumothorax
Tension pneumothorax	Any	Occurs any time Increases intrathoracic and central venous pressures Decreases venous return	IPPV	Rapid clinical deterioration Hypotension Bradycardia	Air in pleural space, atelectasis with flattening of the diaphragm on the affected side, shift of mediastinum away from the pneumothorax Hypoxaemia
Pneumo-mediastinum	Any	Air in the mediastinal space	IPPV with high peak airway pressure	Usually asymptomatic—may be detected by cardiac auscultation (distant heart sounds)	Radiolucent halo over the cardiac outline or retrosternal or mediastinal radiolucency (on lateral view Thymus gland lifted giving ‘angel wing’ or ‘spinnaker sail’ appearance
Pneumopericardium	Any	Air surrounding the pericardium	Active resuscitation Assisted ventilation in preterm baby	May cause cardiac tamponade	In AP view air surrounds the heart shadow within the pericardium
PIE	Any (commonly preterm)	Occurs usually within 96 hours of birth Air is trapped in the perivascular space	Preterm	Increasing hypoxaemia and hypercapnia	More common in very low birth weight ventilated babies

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