

Queensland Clinical Guidelines

Translating evidence into best clinical practice

Maternity and Neonatal **Clinical Guideline**

Respiratory distress and CPAP

Document title:	Respiratory distress and CPAP
Publication date:	June 2020
Document number:	MN20.3-V8-R25
Document supplement:	The document supplement is integral to and should be read in conjunction with this guideline.
Amendments:	Full version history is supplied in the document supplement.
Amendment date:	Full review of original document published in October 2014
Replaces document:	MN14.3-V7-R25
Author:	Queensland Clinical Guidelines
Audience:	Health professionals in Queensland public and private maternity and neonatal services.
Review date:	June 2025
Endorsed by:	Queensland Clinical Guidelines Steering Committee Statewide Maternity and Neonatal Clinical Network (Queensland)
Contact:	Email: Guidelines@health.qld.gov.au URL: www.health.qld.gov.au/qcg



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.

Disclaimer

This guideline is intended as a guide and provided for information purposes only. The information has been prepared using a multidisciplinary approach with reference to the best information and evidence available at the time of preparation. No assurance is given that the information is entirely complete, current, or accurate in every respect.

The guideline is not a substitute for clinical judgement, knowledge and expertise, or medical advice. Variation from the guideline, taking into account individual circumstances, may be appropriate.

This guideline does not address all elements of standard practice and accepts that individual clinicians are responsible for:

- Providing care within the context of locally available resources, expertise, and scope of practice
- Supporting consumer rights and informed decision making, including the right to decline intervention or ongoing management
- Advising consumers of their choices in an environment that is culturally appropriate and which enables comfortable and confidential discussion. This includes the use of interpreter services where necessary
- Ensuring informed consent is obtained prior to delivering care
- Meeting all legislative requirements and professional standards
- Applying standard precautions, and additional precautions as necessary, when delivering care
- Documenting all care in accordance with mandatory and local requirements

Queensland Health disclaims, to the maximum extent permitted by law, all responsibility and all liability (including without limitation, liability in negligence) for all expenses, losses, damages and costs incurred for any reason associated with the use of this guideline, including the materials within or referred to throughout this document being in any way inaccurate, out of context, incomplete or unavailable.

Recommended citation: Queensland Clinical Guidelines. Respiratory distress and CPAP Guideline No. MN20.3-V8-R25. Queensland Health. 2020. Available from: <http://www.health.qld.gov.au/qcg>

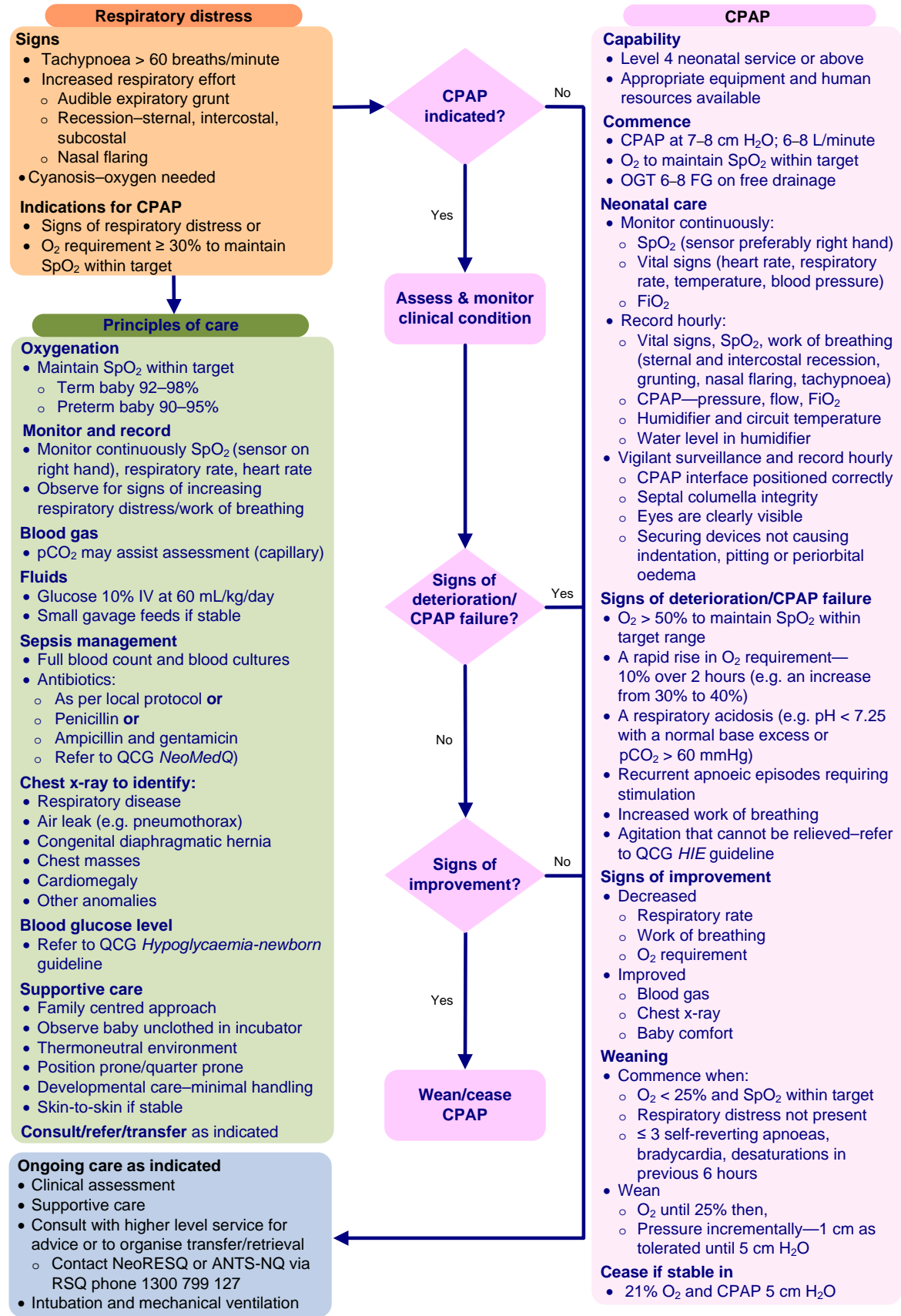
© State of Queensland (Queensland Health) 2020



This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives V4.0 International licence. In essence, you are free to copy and communicate the work in its current form for non-commercial purposes, as long as you attribute Queensland Clinical Guidelines, Queensland Health and abide by the licence terms. You may not alter or adapt the work in any way. To view a copy of this licence, visit <https://creativecommons.org/licenses/by-nc-nd/4.0/deed.en>

For further information, contact Queensland Clinical Guidelines, RBWH Post Office, Herston Qld 4029, email Guidelines@health.qld.gov.au. For permissions beyond the scope of this licence, contact: Intellectual Property Officer, Queensland Health, GPO Box 48, Brisbane Qld 4001, email ip_officer@health.qld.gov.au, phone (07) 3234 1479.

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, FiO₂: fractional inspired oxygen, HIE: Hypoxic-ischaemic encephalopathy, IV: intravenous, NeoRESQ Neonatal Retrieval Emergency Service Southern Queensland, pCO₂: partial pressure of carbon dioxide, QCG: Queensland Clinical Guidelines, RSQ: Retrieval Services Queensland, SpO₂: peripheral capillary oxygen saturation, >: greater than, ≥: greater than or equal to, <: less than; ≤: less than or equal to

Table of Contents

Abbreviations	5
Definitions	5
1 Introduction	6
1.1 Lung development and respiratory physiology	6
1.2 Clinical course of respiratory distress	6
1.3 Clinical standards	7
2 Assessment	8
2.1 Signs	8
2.2 X-rays	9
3 Diagnosis	10
3.1 Differential diagnosis	10
3.2 Respiratory distress syndrome	10
3.3 Transient tachypnoea of the newborn	11
3.4 Pulmonary air leaks	12
3.5 Congenital anomalies	13
3.6 Other respiratory distress	14
3.6.1 Infection, aspiration and interstitial lung disease	14
3.6.2 Persistent pulmonary hypertension of the newborn (PPHN)	15
4 Management	16
4.1 Supportive care	16
5 Continuous positive airway pressure	17
5.1 CPAP physiology	17
5.2 CPAP use	18
5.3 CPAP administration	19
5.4 CPAP settings	20
5.5 Care of baby	21
5.5.1 General care	21
5.5.2 Developmental care and positioning	22
5.5.3 Pressure area care	23
5.6 Complications of CPAP	24
5.7 Clinical course	25
5.8 Weaning CPAP	26
References	27
Appendix A Comparison of respiratory disease	29
Appendix B Pulmonary air leaks	31
Acknowledgements	32

List of Tables

Table 1. Respiratory disease clinical course	6
Table 2. Clinical standards	7
Table 3. Signs	8
Table 4. X-ray interpretation	9
Table 5. Differential diagnosis	10
Table 6. Respiratory distress syndrome	10
Table 7. Transient tachypnoea of the newborn	11
Table 8. Pulmonary air leaks	12
Table 9. Congenital anomalies	13
Table 10. Infection and aspiration	14
Table 11. PPHN	15
Table 12. Supportive care	16
Table 13. CPAP physiology	17
Table 14. CPAP use	18
Table 15. CPAP administration	19
Table 16. CPAP settings	20
Table 17. Care of baby on CPAP	21
Table 18. Developmental care	22
Table 19. Pressure area care	23
Table 20. CPAP complications	24
Table 21. CPAP clinical course	25
Table 22. Weaning CPAP	26

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
pCO ₂	Partial pressure of carbon dioxide
pO ₂	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 deficiency²	<ul style="list-style-type: none"> • Pulmonary surfactant: <ul style="list-style-type: none"> ○ Reduces alveolar surface tension ○ Facilitates alveolar expansion ○ Reduces the likelihood of atelectasis from alveolar collapse ○ 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: <ul style="list-style-type: none"> ○ Generation of increased negative intrathoracic pressure to expand poorly compliant lungs resulting in severe sternal and intercostal recession⁴ ○ High surface tension leading to instability of the lung at end-expiration, low lung volume and decreased compliance ○ Pulmonary oedema and increased airway resistance resulting from lung inflammation and respiratory epithelial injury • Risk factors and causes include: <ul style="list-style-type: none"> ○ Preterm birth ○ Elective caesarean section⁵ ○ Maternal diabetes in pregnancy (DIP)—poorly controlled ○ Meconium aspiration syndrome (MAS)⁶ ○ Infection ○ Genetic surfactant dysfunction disorder²
Lung function²	<ul style="list-style-type: none"> • Changes in lung function cause ventilation-perfusion mismatch due to: <ul style="list-style-type: none"> ○ Collapse of portions of the lungs (atelectasis) ○ 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
Hypoxaemia²	<ul style="list-style-type: none"> • Caused by: <ul style="list-style-type: none"> ○ Ventilation perfusion mismatch from intrapulmonary shunting— influenced by relative size of under ventilated lung ○ Extrapulmonary shunting across foramen ovale and patent ductus arteriosus
Acidosis²	<ul style="list-style-type: none"> • Poor ventilation identified from: <ul style="list-style-type: none"> ○ Elevated pCO₂ 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	<ul style="list-style-type: none"> • 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: <ul style="list-style-type: none"> ○ Risk of moderate to severe RDS⁸ ○ Incidence of transient tachypnoea of the newborn (TTN) ○ Need for surfactant¹⁰ • Refer to Queensland Clinical Guidelines <i>Preterm labour and birth</i> guideline¹¹
Principles of care	<ul style="list-style-type: none"> • Commence continuous positive airways pressure (CPAP) if: <ul style="list-style-type: none"> ○ Signs of respiratory distress or ○ Oxygen requirement to maintain oxygen saturation (SpO₂) is greater than or equal to 30% • Refer to Queensland Clinical Guidelines Standard care¹² 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)	<ul style="list-style-type: none"> • Consider the clinical service capability of the facility <ul style="list-style-type: none"> ○ Refer to Clinical Services Capability Framework for neonatal services¹³ • 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 <ul style="list-style-type: none"> ○ If level 2–3 neonatal service and baby has respiratory distress <ul style="list-style-type: none"> ▪ Oxygen greater than or equal to 30% to maintain SpO₂ 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 ○ If level 4 neonatal service and baby requires CPAP <ul style="list-style-type: none"> ▪ Commencing when baby is greater than 24 hours of age ▪ Oxygen requirement is greater than 40% to maintain SpO₂ within target range [refer to Table 3. Signs] ▪ Oxygen requirement rapidly increases ▪ Blood gas partial pressure arterial carbon dioxide (PaCO₂) 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 ○ If level 5 neonatal service <ul style="list-style-type: none"> ▪ CPAP commencing when baby is greater than 24 hours of age ▪ Oxygen requirement is greater than 40% to maintain SpO₂ within target range [refer to Table 3. Signs] ▪ Blood gas PaCO₂ 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	<ul style="list-style-type: none"> Pale <i>or</i> Cyanosis (excluding acrocyanosis)—central and/or peripheral due to right-to-left intra- and extrapulmonary shunting
SpO₂	<ul style="list-style-type: none"> Pre-ductal measure on right hand <ul style="list-style-type: none"> Follow manufacturer's instructions Targets after 10 minutes of age*: <ul style="list-style-type: none"> Term baby: 92–98% Preterm: 90–95% Hypoxaemia usually due to ventilation perfusion mismatch² Left to right shunting of blood occurs in areas of poorly ventilated lungs²
Cardiovascular	<ul style="list-style-type: none"> Peripheral pulses diminished² Prolonged capillary refill time (greater than two seconds) May be tachycardic May be having bradycardic episodes and/or apnoeas² Extra-pulmonary shunting may occur across the foramen ovale and patent ductus arteriosus²
Urine output	<ul style="list-style-type: none"> May be low in first 24–48 hours² Hyponatraemia may be present If baby recovering from RDS, spontaneous diuresis occurs on second to fourth day of life and precedes improved pulmonary function²
Respiratory	<ul style="list-style-type: none"> Tachypnoea <ul style="list-style-type: none"> More than 60 breaths per minute¹ Most common presenting sign⁷ Due to hypercarbia, hypoxaemia or acidosis¹⁵ Breath sounds on auscultation are decreased² Increased respiratory effort: <ul style="list-style-type: none"> Expiratory grunt¹—results from exhalation through a partially closed glottis; slows the decrease in end expiratory lung volume Recession¹—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 Nasal flaring¹ due to use of accessory respiratory muscles <ul style="list-style-type: none"> Compensatory sign that increases upper airway diameter and lower total respiratory system resistance and work of breathing¹⁵
Respiratory acidosis	<ul style="list-style-type: none"> 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] pCO₂ may assist assessment <ul style="list-style-type: none"> pCO₂ greater than or equal to 50 mmHg and pH less than 7.35 indicates respiratory acidosis¹⁶ Degree of hypoxaemia¹⁵ (as shown by a low pO₂) 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 sufficient¹⁷
 - Take chest x-ray at four hours of age unless earlier indication
- Assists with diagnosis¹⁷
- Checks position of tubes and lines—including orogastric tube (OGT)¹⁷
- Assists assessment of pulmonary inflation¹⁷
- Excludes undiagnosed congenital abnormality¹⁷
- Excludes pulmonary air leaks^{17,18}

Table 4. X-ray interpretation

Aspect	Consideration
Normal	<ul style="list-style-type: none"> • 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 shadow¹⁷)
RDS	<ul style="list-style-type: none"> • Low lung volume • Diffuse reticulogranular ground glass appearance • Air bronchograms (more severe) • Confluent alveolar shadowing¹⁷ • Radiographic pattern results from: <ul style="list-style-type: none"> ○ Alveolar atelectasis contrasting with aerated airways ○ Pulmonary oedema may contribute to diffuse appearance
TTN	<ul style="list-style-type: none"> • Normal or slightly overinflated lung fields • Increased streaky shadowing with perihilar densities⁷ 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	<ul style="list-style-type: none"> • Pneumothorax and other air leaks: <ul style="list-style-type: none"> ○ Common in initial x-ray ○ More often observed when lung compliance improves ○ Consider air leak if acute deterioration in baby's condition • Pneumothorax: <ul style="list-style-type: none"> ○ Air in pleural space, atelectasis with flattening of the diaphragm on the affected side, shift of mediastinum away from the pneumothorax¹⁸ • Pneumomediastinum: <ul style="list-style-type: none"> ○ Radiolucent halo over the cardiac outline or retrosternal or mediastinal radiolucency (on lateral view)^{17,18} ○ Thymus gland lifted giving 'angel wing' or 'spinnaker sail' appearance^{17,18} • Pneumopericardium: <ul style="list-style-type: none"> ○ Air in the pericardial space ○ May cause cardiac tamponade ○ In antero-posterior (AP) view air surrounds the heart shadow within the pericardium¹⁸ • Refer to 3.4 Pulmonary air leaks
Pulmonary interstitial emphysema (PIE)	<ul style="list-style-type: none"> • Interstitial air appearing as cyst-like or linear translucencies¹⁸
Congenital diaphragmatic hernia	<ul style="list-style-type: none"> • Mediastinal shift • Absence of diaphragm—usually left sided • Visible bowel gas in chest cavity¹⁹
MAS	<ul style="list-style-type: none"> • 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	<ul style="list-style-type: none"> • Consider: <ul style="list-style-type: none"> ○ Baby's history—gestational age and postnatal age ○ Maternal history ○ Clinical features ○ Chest x-ray ○ Capillary/venous blood gas • Refer to Appendix A Comparison of respiratory disease
Non-pulmonary causes⁷	<ul style="list-style-type: none"> • Consider: <ul style="list-style-type: none"> ○ Hyperthermia from overheating baby resulting in tachycardia, tachypnoea and increased oxygen consumption²¹ ○ Sepsis including meningitis ○ Cardiac disease¹ ○ Metabolic acidosis from inborn error of metabolism ○ Hypoglycaemia—may cause or aggravate tachypnoea ○ Polycythaemia or anaemia ○ Hydrocephalus ○ Intracranial haemorrhage ○ Maternal narcotic use causing respiratory depression

3.2 Respiratory distress syndrome

Table 6. Respiratory distress syndrome

Aspect	Consideration
Physiology	<ul style="list-style-type: none"> • Caused by: <ul style="list-style-type: none"> ○ Under developed lungs ○ Surfactant deficiency and or dysfunction²²
Presentation	<ul style="list-style-type: none"> • Presents more commonly if: <ul style="list-style-type: none"> ○ Baby is less than 34 weeks gestation⁷ ○ Maternal DIP⁷ • Baby has respiratory distress within first minutes after birth² <ul style="list-style-type: none"> ○ Worsens over the next 48 hours² • Baby generally improves by the third to fourth day of life with onset of diuresis¹⁵
Signs	<ul style="list-style-type: none"> • Increased work of breathing¹⁵ [refer to Definitions • Cyanosis and hypoxia • Breath sounds diminished; coarse rhonchi in bilateral lung fields¹⁵ • Bell shaped chest • Refer to Table 3. Signs • Chest x-ray—refer to Table 4. X-ray interpretation • Blood gas—hypoxaemia and respiratory acidosis⁷ <ul style="list-style-type: none"> ○ If baby has metabolic acidosis and history suggestive of hypoxaemic ischaemic encephalopathy (HIE) refer to Queensland Clinical Guideline <i>Hypoxic ischaemic encephalopathy (HIE)</i>²³
Management	<ul style="list-style-type: none"> • Refer to Section 16 Management • Early CPAP is usually indicated²⁴ • May require exogenous surfactant Curosurf® (poractant alfa) or Survanta® (beractant) with/without intermittent positive pressure ventilation <ul style="list-style-type: none"> ○ 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	<ul style="list-style-type: none"> • Parenchymal lung disorder more commonly seen in term or late preterm babies¹ • Occurs due to pulmonary oedema resulting from delayed reabsorption and clearance of fetal alveolar fluid⁷
Presentation	<ul style="list-style-type: none"> • Common cause of respiratory distress in the newborn baby^{1,7} <ul style="list-style-type: none"> ○ Occurs two to three times more often in babies of women with diabetes¹ • Onset is usually at the time of birth and within two hours of age^{1,7} <ul style="list-style-type: none"> ○ Symptoms usually last for 12–24 hours, but may last up to 72 hours • May occur with RDS^{1,7} • More common if¹: <ul style="list-style-type: none"> ○ DIP ○ Born by caesarean section ○ Precipitous birth⁷ ○ Small for gestational age or large for gestational age/macrosomia ○ Maternal obesity¹ ○ Maternal asthma ○ Born before 39+0 weeks
Signs	<ul style="list-style-type: none"> • Increased work of breathing¹ [refer to Definitions <ul style="list-style-type: none"> ○ Tachypnoea—higher respiratory rate associated with longer duration of TTN⁷ • Cyanosis¹ • 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: <ul style="list-style-type: none"> ○ Refer to Table 4. X-ray interpretation • Blood gas—hypoxaemia, hypercapnia or respiratory acidosis⁷
Management	<ul style="list-style-type: none"> • Supportive measures⁷ <ul style="list-style-type: none"> ○ 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
Context⁷	<ul style="list-style-type: none"> • 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: <ul style="list-style-type: none"> ○ Complication of RDS ○ An isolated presentation ○ Associated with another underlying disorder • Diagnosis is made from chest x-ray² and clinical assessment <ul style="list-style-type: none"> ○ Refer to Table 4. X-ray interpretation
Pneumothorax¹⁸	<ul style="list-style-type: none"> • Air escapes into the pleural space • Suspect in baby with unexpected deterioration in oxygenation, ventilation or cardiovascular status • Signs: <ul style="list-style-type: none"> ○ Increased work of breathing [refer to Definitions ○ Positive chest transillumination (if available) <ul style="list-style-type: none"> ▪ 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 pneumothorax¹⁸	<ul style="list-style-type: none"> • Increases intrathoracic pressure, increases central venous pressure and decreases venous return • Signs: <ul style="list-style-type: none"> ○ Acute deterioration in baby's condition ○ Chest asymmetry with enlargement on the affected side ○ Decreased breath sounds on affected side ○ Shift of heart sound away from the affected side ○ Hypotension (with narrow pulse pressure), bradycardia, oxygen desaturation and poor perfusion ○ Sudden increase in oxygen requirement to maintain SpO₂ within target range [refer to Table 3. Signs] ○ Positive chest transillumination (if available) <ul style="list-style-type: none"> ▪ Not reliable in term or large baby • Requires emergency management • Refer to Appendix B Pulmonary air leaks
Pneumo-mediastinum¹⁸	<ul style="list-style-type: none"> • Air escapes into the mediastinal space • Usually caused by high peak pressures during IPPV²⁸ • Signs: <ul style="list-style-type: none"> ○ Usually asymptomatic—may be detected by cardiac auscultation (distant heart sounds)
Pneumo-pericardium²⁹	<ul style="list-style-type: none"> • 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
PIE¹⁸	<ul style="list-style-type: none"> • 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: <ul style="list-style-type: none"> ○ 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 babies⁷ 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 disease⁷	<ul style="list-style-type: none"> • Usually presents with less respiratory distress unless persistent pulmonary hypertension is also present <ul style="list-style-type: none"> ○ 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 condition²
Pulmonary hypoplasia⁷	<ul style="list-style-type: none"> • May occur in isolation or be associated with other congenital anomalies, (e.g. congenital diaphragmatic hernia¹⁷) • Unilateral: <ul style="list-style-type: none"> ○ Chest x-ray characterised a small lung with reduced vascularity and ipsilateral heart displacement¹⁷ • Bilateral: <ul style="list-style-type: none"> ○ Associated with oligohydramnios and prolonged preterm rupture of membranes ○ Chest x-ray may show bell shaped thorax, reduced inflation increased pulmonary shadowing and pulmonary air leaks¹⁷
Diaphragmatic hernia	<ul style="list-style-type: none"> • 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 hernia¹⁹ • Refer to Table 4. X-ray interpretation
Airway	<ul style="list-style-type: none"> • Obstruction: <ul style="list-style-type: none"> ○ Pierre Robin sequence ○ Choanal atresia⁷ • Malformation: <ul style="list-style-type: none"> ○ Tracheo-oesophageal fistula⁷ ○ Oesophageal atresia ○ Laryngeal web
Other	<ul style="list-style-type: none"> • Congenital cystic adenomatoid malformation <ul style="list-style-type: none"> ○ 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	<ul style="list-style-type: none"> • Bacterial pneumonia⁷ • X-ray demonstrates patchy opacities and/or lobar consolidation • Blood cultures assist diagnosis • Refer to Table 12. Supportive care
MAS	<ul style="list-style-type: none"> • History: <ul style="list-style-type: none"> ○ Meconium stained liquor or evidence of meconium staining on examination ○ Baby frequently small for gestational age or postmature ○ Intrauterine pathologic processes²⁰—asphyxia, infection • May lead to: <ul style="list-style-type: none"> ○ Airway obstruction ○ Chemical irritation ○ Inflammation ○ Infection ○ Increased inactivation of surfactant²⁰ ○ Persistent pulmonary hypertension of the newborn²⁰ ○ Increased incidence of pneumothorax ○ Hypoxaemia due to ventilation-perfusion mismatch • Signs include¹: <ul style="list-style-type: none"> ○ Marked tachypnoea and cyanosis ○ Intercostal and substernal recession due to reduced lung compliance and use of accessory muscles ○ Grunting and nasal flaring²⁰ ○ Barrel-shaped chest ○ Rales and rhonchi on auscultation ○ X-ray—refer to Table 4. X-ray interpretation ○ Blood gas—respiratory acidosis^{30,31} • Management: <ul style="list-style-type: none"> ○ Monitor preductal and postductal SpO₂ ○ Refer to Table 12. Supportive care ○ Intermittent positive pressure ventilation may be indicated ○ Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required
Other aspiration	<ul style="list-style-type: none"> • May be caused by aspiration of: <ul style="list-style-type: none"> ○ 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	<ul style="list-style-type: none"> • Interstitial and diffuse lung diseases including²: <ul style="list-style-type: none"> ○ Genetic disorders of surfactant dysfunction ○ Lung growth abnormalities ○ Pulmonary interstitial glycogenosis

3.6.2 Persistent pulmonary hypertension of the newborn (PPHN)

Table 11. PPHN

Aspect	Consideration
Physiology	<ul style="list-style-type: none"> Physiological adaptation after birth has not occurred⁷ resulting in persistent or delayed transition of fetal circulation
Signs	<ul style="list-style-type: none"> SpO₂ difference of greater than 5% between preductal and postductal measures due to right-to-left shunting through the patent ductus arteriosus³² <ul style="list-style-type: none"> Right-to-left shunting can also occur through the foramen ovale and will have an absent oxygen saturation gradient³² Measure on right hand/wrist (preductal), and left hand/wrist or either foot (postductal) Arterial blood gas—normal PaCO₂ (if no lung disease present) and PaO₂ below 100 mmHg in 100 percent inspired oxygen³² Barrel-shaped chest
Causes^{32,33}	<ul style="list-style-type: none"> Idiopathic Abnormalities of pulmonary vasculature <ul style="list-style-type: none"> Underdevelopment (e.g. fetal growth restriction, diaphragmatic hernia) Maldevelopment (e.g. congenital heart disease, meconium aspiration syndrome) 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	<ul style="list-style-type: none"> If PPHN suspected: <ul style="list-style-type: none"> Aim to keep oxygen saturation at greater than 97% Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required Minimise noise, light and handling baby 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	<ul style="list-style-type: none"> Give oxygen to maintain SpO₂ within target ranges <ul style="list-style-type: none"> Refer to Table 3. Signs Monitor delivered oxygen concentration continuously If greater than or equal to 30% oxygen is required, consider CPAP <ul style="list-style-type: none"> Refer to Table 14. CPAP use] If PPHN suspected—refer to Table 11. PPHN
Fluids	<ul style="list-style-type: none"> 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 <ul style="list-style-type: none"> If the baby's respirations are greater than 80 breaths per minute do not feed
Temperature	<ul style="list-style-type: none"> Nurse baby in incubator or on open care system Refer to Queensland Clinical Guideline <i>Stabilisation for retrieval—neonata</i>³³ Maintain temperature within normal range <ul style="list-style-type: none"> Axilla 36.8–37.2 °C Monitor and record temperature every 4 hours
Blood glucose	<ul style="list-style-type: none"> Refer to Queensland Clinical Guideline <i>Hypoglycaemia—newborn</i>³⁴
Infection screening	<ul style="list-style-type: none"> If clinically indicated, surface swabs from baby Full blood count Blood cultures—check result at 24 hours: <ul style="list-style-type: none"> If negative and respiratory distress resolved—administer 24 hour antibiotic dose and then cease (36 hour coverage provided) If delay in processing blood cultures—continue antibiotics until 48 hours of treatment Recheck result at 48 hours 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*	<ul style="list-style-type: none"> Empirical antibiotics are given prophylactically until cultures and clinical course known benzylpenicillin OR amoxicillin/ampicillin AND gentamicin Refer to NeoMedQ neonatal monographs for benzylpenicillin³⁵ ampicillin³⁶ and gentamicin³⁷ Refer to Queensland Clinical Guideline <i>Early onset Group B Streptococcal disease</i>³⁸
Monitoring	<ul style="list-style-type: none"> Monitor and record every hour (or if change in baby's condition)⁷: <ul style="list-style-type: none"> Vital signs—temperature, heart rate, respirations, blood pressure and perfusion Oxygen saturations—preferably on right hand/wrist (pre-ductal) <ul style="list-style-type: none"> If PPHN suspected, refer to Table 11. PPHN Work of breathing [refer to Definitions Check blood gases as clinically indicated Check blood glucose levels [refer to Queensland Clinical Guideline <i>Hypoglycaemia—newborn</i>^{23,39}]
Blood gas	<ul style="list-style-type: none"> Determine frequency based on baby's clinical condition and support required <ul style="list-style-type: none"> Not routinely required if previous blood gas within normal limits If baby is stable, leaving baby undisturbed may be indicated
Pain management	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> • 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 cycle⁴¹ • Prevents atelectasis • Enhances gas exchange⁴² • Rationale for use: <ul style="list-style-type: none"> ○ Stents the airways and maintains functional residual capacity (FRC)
CPAP physiology	<ul style="list-style-type: none"> • Improves lung compliance⁴³ and uniformity of ventilation by preventing: <ul style="list-style-type: none"> ○ De-recruitment of alveoli ○ Collapse of small airways during exhalation in the surfactant deficient lung ○ Increases functional residual capacity and improves oxygenation⁴⁴ • 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: <ul style="list-style-type: none"> ○ PaCO₂ lowers ○ Progressing arterial hypoxaemia is prevented ○ 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 movements⁴⁴

5.2 CPAP use

Table 14. CPAP use

Aspect	Consideration
Prophylactic	<ul style="list-style-type: none"> • Insufficient evidence to compare prophylactic CPAP to oxygen therapy and other supportive care²⁴ 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 surfactant²⁴
Indications	<ul style="list-style-type: none"> • Correct existing respiratory failure in babies with RDS⁴ <ul style="list-style-type: none"> ○ Baby has signs of respiratory distress [refer to Table 3. Signs] • Treat airway obstruction⁴ • Prevent respiratory failure (e.g. apnoea of prematurity)⁴
Contraindications	<ul style="list-style-type: none"> • Nasal CPAP is contraindicated in babies with a range of congenital abnormalities or surgical conditions, including <ul style="list-style-type: none"> ○ Certain cleft palates ○ Bi-lateral choanal atresia <ul style="list-style-type: none"> ▪ Single naso-pharyngeal tube may be used in unilateral choanal atresia ○ Tracheo-oesophageal atresia ○ Congenital diaphragmatic hernia ○ Gastroschisis or omphalocele ○ Necrotising enterocolitis (suspected or confirmed) ○ Significant loss of skin or septal integrity following use of bi-nasal CPAP ○ Congenital lung/airway lesions (e.g. congenital lobar emphysema, congenital pulmonary airway malformation) ○ 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}	<ul style="list-style-type: none"> • 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 <ul style="list-style-type: none"> ○ May decrease central apnoea due to regular breathing pattern • Reduces oxygen requirements • Reduces risk of bronchopulmonary dysplasia
Potential harms⁴³	<ul style="list-style-type: none"> • 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—CSCF¹³
- 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 staff¹³ 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	<ul style="list-style-type: none"> • CPAP system provides stable pressures at required levels with humidity and supplemental oxygen⁴⁷ • 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 closure⁴⁷ • Mean airway pressure delivered to a baby by nasal CPAP is affected by: <ul style="list-style-type: none"> ○ Seal of the interface ○ Loss of pressure through the mouth ○ Airway resistance^{40,51}
Nasal CPAP	<ul style="list-style-type: none"> • Improves oxygenation and maintains lung volume • Increases FRC, distends the larynx, reduces subglottic airway resistance⁵² • Standard respiratory treatment over invasive intubation⁷
Bi-nasal prongs	<ul style="list-style-type: none"> • Standard of care for babies of all gestations who breathe spontaneously and require continued respiratory support⁴⁵ • Better than single prong to deliver CPAP to preterm baby for early treatment of RDS⁴⁴ <ul style="list-style-type: none"> ○ Lower oxygen requirements (than single prong) ○ Incidence of BPD, pulmonary air leaks, ventilator associated pneumonia and neurocognitive disorders is lower than with mechanical ventilation⁴⁵ ○ Longer term outcomes and medium-term outcomes (e.g. chronic lung disease at 36 weeks corrected age) not identified⁴⁴ • Associated with increased resistance to air flow • Correct fitting of device minimises perinasal leak and nasal trauma⁵³ <ul style="list-style-type: none"> ○ Refer to Table 19. Pressure area care
Nasal mask	<ul style="list-style-type: none"> • 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 nares⁴⁵ • May relieve trauma from prongs⁵¹ <ul style="list-style-type: none"> ○ Evidence from a systematic review and meta-analysis reported a lower incidence of nasal trauma with very low certainty evidence⁵⁴ • May reduce CPAP failure⁵³
Bubble device versus ventilator	<ul style="list-style-type: none"> • Bubble device versus ventilator to deliver CPAP <ul style="list-style-type: none"> ○ No difference in physiological parameters in the short-term^{47,52}

5.4 CPAP settings

Record CPAP pressure, gas flow, fractional inspired oxygen (FiO₂) and humidifier temperature hourly and when any changes made to settings.

Table 16. CPAP settings

Aspect	Consideration
Pressure	<ul style="list-style-type: none"> Commence at 7–8 cm H₂O
Flow	<ul style="list-style-type: none"> Generally, 6–8 L per minute⁵⁵ depending on CPAP device being used Use lowest level possible to deliver desired pressure <ul style="list-style-type: none"> Refer to manufacturer's instructions
Oxygen	<ul style="list-style-type: none"> Deliver FiO₂ to maintain the baby's oxygen saturations within target levels Refer to Table 3. Signs and Table 12. Supportive care
Humidification	<ul style="list-style-type: none"> Humidify and warm gases before delivery to baby Set temperature⁴¹ according to manufacturer's instructions—generally: <ul style="list-style-type: none"> Interface with baby 37 °C Humidifier 40 °C
Device management	<ul style="list-style-type: none"> Insert circuit through lowest insertion port on incubator to: <ul style="list-style-type: none"> Assist drainage of condensation away from the baby^{45,52} 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	<ul style="list-style-type: none"> To determine observations and assessment required, refer to Table 3. Signs and Table 12. Supportive care
Fluids and feeding	<ul style="list-style-type: none"> Commence/continue IV therapy [refer to Table 12. Supportive care] Insert a size 6–8 FG OGT to minimise gastric distension^{21,45} <ul style="list-style-type: none"> If significant abdominal distension the larger (8 FG) OGT may be required OGT: <ul style="list-style-type: none"> Leave on free drainage to vent stomach⁴⁵ Cap for 30 minutes after a feed Aspirate air every 4–6 hours and as required Non-nutritive sucking on a pacifier⁵⁶ while on CPAP is beneficial: <ul style="list-style-type: none"> Promotes physiological stability Reduces stress Relaxes the baby during invasive procedures Improves preparation for oral feeding Improves oxygenation Reduces air leak from mouth
Suctioning	<ul style="list-style-type: none"> Keep airway clear of secretions <ul style="list-style-type: none"> Routine suctioning not required Avoid deep suctioning⁴⁵ Risks from suctioning include: <ul style="list-style-type: none"> Infection, bradycardia, laryngospasm, blood pressure fluctuations, hypoxaemia, increased intracranial pressure, cardiac arrhythmia, nasal injury⁵⁷ 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	<ul style="list-style-type: none"> Minimise unnecessary noxious procedures Assess and record pain every 4 hours using validated pain management tool²¹ Can be alleviated non-pharmacologically by: <ul style="list-style-type: none"> Modifying the environmental and sensory stimuli (e.g. noise and light) Sucrose 25% orally⁵⁸ Positioning of baby [refer to Table 18. Developmental care] Non-nutritive sucking⁵⁹

5.5.2 Developmental care and positioning

Table 18. Developmental care

Aspect	Consideration
Supportive care	<ul style="list-style-type: none"> • If baby is fragile or compromised, perform cares by two clinicians, for example if: <ul style="list-style-type: none"> ○ Has acute lung disease ○ Weighs less than 1000 g ○ Requires greater than 7 cmH₂O CPAP ○ 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 visiting⁴⁵ <ul style="list-style-type: none"> ○ Encourage parent/s to assist with eye and mouth cares and head massage with assistance from clinician (to maintain CPAP) ○ Encourage skin to skin contact as soon as clinical condition allows⁶⁰
Positioning	<ul style="list-style-type: none"> • Position baby to avoid: <ul style="list-style-type: none"> ○ Inadvertent tension to the interface⁴⁵ ○ Accumulation of condensate to the nares • Use pacifier or chin strap to keep baby's mouth closed (to maintain CPAP) <ul style="list-style-type: none"> ○ 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 aspiration⁴⁵ • 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 <ul style="list-style-type: none"> ○ Positive effect on respiratory mechanics by increasing oxygenation, improving ribcage-abdominal synchrony and better consistency of breathing pattern¹² ○ Disadvantages include developmental delays, corneal lesions and loss of vascular access in long term use⁶¹ • Prone quarter turn position beneficial⁶¹⁻⁶³ for stabilising respiratory rate and increasing oxygen saturation <ul style="list-style-type: none"> ○ Place a small neck roll under the baby to prevent neck flexion and airway obstruction⁶⁰ ○ Nest baby by supporting hips, uppermost arm and trunk with roll ○ Position knees and feet in neutral alignment with hips
Developmental care	<ul style="list-style-type: none"> • Developmental positioning is important to prevent pressure injury and deformational plagiocephaly⁴² • 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 <ul style="list-style-type: none"> ○ Involve parents in the baby's cares ○ Minimal handling—disturb baby only when absolutely necessary ○ If baby positioned supine during cares, use of a peanut pillow to help maintain head in mid-line position ○ Refer to Table 12. Supportive care • Comfort and natural development provided by: <ul style="list-style-type: none"> ○ Gentle touch and containment ○ Developmental positioning ○ Age-appropriate stimulation ○ Cycled lighting ○ Decreased environmental stimuli including noise ○ Family bonding time⁴²—encourage parents to be present during painful procedures⁶⁴ • Benefits of skin-to-skin positioning when clinically suitable are physiological, psychological, behavioural, neurobehavioral and psychosocial⁴²

5.5.3 Pressure area care

Table 19. Pressure area care

Aspect	Consideration
Context	<ul style="list-style-type: none"> • Constant pressure on the nares, nasal septum and forehead can lead to reduced skin integrity and injury⁶⁵ causing pressure injury (ulcers) and friction shear from respiratory tubing⁴² • Nasal trauma: <ul style="list-style-type: none"> ○ Most common complication of nasal CPAP⁶⁶—incidence 15–60% ○ Occurs commonly on medial aspect of nostrils ○ Pressure caused by poorly fitting nasal prongs or carrying crossbar exerting pressure on septum ○ 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} ○ May progress to facial scarring⁴² and/or permanent deformity • Risk factors for skin breakdown, development of nasal pressure ulcers, compression necrosis, and/or nasal deformities⁶⁹: <ul style="list-style-type: none"> ○ Nasal CPAP device ○ Length of therapy ○ Age, gestation and size of baby ○ Poor perfusion ○ Environmental humidity and temperature
Assessment	<ul style="list-style-type: none"> • Provide vigilant skin assessments and skin resting time⁷⁰ • First sign of skin breakdown is nasal erythema⁶⁹ • Assess baby regularly (with cares) for signs of pressure injury <ul style="list-style-type: none"> ○ Nasal: redness, blanching, skin breakdown, necrosis, bruising, indentation, bleeding, altered shape ○ Ears: creases, folds, pressure areas ○ Forehead: if using midline device (mask or prongs) ○ Nasal bridge: mid-facial indentation (mask) ○ 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	<ul style="list-style-type: none"> • 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 prevented⁴⁵ <ul style="list-style-type: none"> ○ Position binasal prongs 2 mm from the nares ○ Avoid contact with the septal columella ○ Fit firmly without blanching the skin to decrease movement⁴⁵ ○ Avoid pressure on the nasolabial sulcus and philtrum ○ Avoid propping the cot as baby sliding will cause pressure on the septum ○ 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}: <ul style="list-style-type: none"> ○ Reduces nasal injuries—use soft silicone-based skin protection over vulnerable areas <ul style="list-style-type: none"> ▪ Assess regularly for moisture⁷², and change at least every 12 hours ▪ Remove during trial off CPAP as baby is unable to nasal flare [refer to Table 3. Signs] ○ 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 strap⁴⁵
Management	<ul style="list-style-type: none"> • If pressure area(s) present: <ul style="list-style-type: none"> ○ Review interface, delivery mechanisms and pressure injury reduction measures⁴⁵ and reassess size of prongs or mask ○ Consider alternating between binasal prongs and mask

5.6 Complications of CPAP

Table 20. CPAP complications

Aspect	Consideration
Pulmonary air leaks	<ul style="list-style-type: none"> Continuous distending pressure (CPAP) may increase the risk of pneumothorax^{18,73}
Pain	<ul style="list-style-type: none"> CPAP treatment and associated procedures (e.g. heel pricks⁶⁴, suctioning) are painful or unpleasant^{21,74} due to: <ul style="list-style-type: none"> Flow of gas through nose and mouth Restricted head movements Obstructed vision Suctioning Refer to Table 17. Care of baby on CPAP
Abdominal insufflation	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> Caused by excessive pressure that results in: <ul style="list-style-type: none"> Increased work of breathing⁷⁵ [refer to Definitions Reduced cardiac output secondary to impaired venous return⁷⁵ Refer to Table 16. CPAP settings for recommended CPAP in babies
Pressure injury	<ul style="list-style-type: none"> Refer to Table 19. Pressure area care

5.7 Clinical course

Table 21. CPAP clinical course

Aspect	Consideration
Signs of improvement	<ul style="list-style-type: none"> • Reduction in the baby's work of breathing as shown by reduction in: <ul style="list-style-type: none"> ○ Respiratory rate (typically by 10–20 breaths per minute) ○ Expiratory grunting ○ Chest recession ○ Nasal flaring • Stabilised or reduced oxygen requirement to maintain SpO₂ within target range • Improvement in blood gas (if taken) • Improvement in appearance and lung volumes on chest x-ray • More settled baby
Weaning/ceasing	<ul style="list-style-type: none"> • Refer to Table 22. Weaning CPAP
Signs of deterioration	<ul style="list-style-type: none"> • 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: <ul style="list-style-type: none"> ○ Respiratory signs not improving or worsening ○ Oxygen requirement increasing or greater than 40% to maintain target SpO₂ ○ Apnoeic episodes ○ Blood gas showing increasing respiratory acidosis
CPAP failure⁷⁶	<ul style="list-style-type: none"> • 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} • Signs⁷⁶: <ul style="list-style-type: none"> ○ FiO₂ greater than 40% (or according to local protocols) to maintain target SpO₂ with 8 cmH₂O CPAP ○ Rapid increase of oxygen requirement including a rise of 10% over 2 hours or less ○ Respiratory acidosis—pH less than 7.25 with normal base excess or PaCO₂ greater than 60 mmHg ○ Recurrent apnoeic episodes requiring stimulation ○ Increase in work of breathing [refer to Definitions] ○ Development of pneumothorax [refer to Table 8. Pulmonary air leaks] ○ Baby agitated and not settled by comfort measures [refer to Table 18. Developmental care] <ul style="list-style-type: none"> ▪ Review respiratory status ▪ Assess for other causes and manage [refer to Table 17. Care of baby on CPAP] • If there are signs of failure: <ul style="list-style-type: none"> ○ Consult with neonatologist via RSQ for advice or retrieval of baby to a higher level of service as required ○ Assess baby (by medical officer/nurse practitioner) immediately ○ Urgent chest x-ray ○ Transilluminate chest to detect signs of pulmonary air leak <ul style="list-style-type: none"> ▪ 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] ○ Check blood gas for hypercarbia or severe metabolic acidosis <ul style="list-style-type: none"> ▪ If metabolic acidosis refer to Queensland Clinical Guideline <i>Hypoxic ischaemic encephalopathy (HIE)</i>²³ ○ Refer to Queensland Clinical Guideline <i>Stabilisation for retrieval—neonata</i>³

5.8 Weaning CPAP

Table 22. Weaning CPAP

Aspect	Consideration
Context	<ul style="list-style-type: none"> • Lung compliance changes occur between 48 and 96 hours of age due to surfactant secretion • If CPAP is discontinued prematurely, risk of⁷⁸: <ul style="list-style-type: none"> ○ Atelectasis ○ Apnoea and bradycardia • If CPAP is continued unnecessarily, risk of complications include⁷⁸: <ul style="list-style-type: none"> ○ Gastric distension; nasal trauma; agitation ○ Alveolar over distension resulting in longer term consequences⁷⁸ (e.g. BPD)
Weaning	<ul style="list-style-type: none"> • Reduce FiO₂ before reducing the CPAP pressure <ul style="list-style-type: none"> ○ Maintain SpO₂ levels in target range⁶⁰ [refer to Table 3. Signs] • Commence gradual CPAP weaning when: <ul style="list-style-type: none"> ○ Oxygen requirement less than 25% ○ Respiratory distress improved <ul style="list-style-type: none"> ▪ Respiratory rate less than 60 breaths per minute ▪ Chest recessions absent ○ Apnoeas are no longer than 20 seconds duration and are self-reverting ○ Bradycardic episodes are not lower than 100 beats per minute ○ Average SpO₂ greater than 95%⁷⁹ for the previous 6 hours • Wean incrementally by 1 cmH₂O as tolerated until 5 cmH₂O • Cease if baby is stable in 21% oxygen and CPAP 5 cmH₂O • Signs of CPAP being ceased too early include: <ul style="list-style-type: none"> ○ Increasing apnoea frequency ○ Increased oxygen requirement ○ Increased work of breathing [refer to Definitions] • Weaning too early may result in a need to restart CPAP or intubation and ventilation⁷⁶ • Weaning CPAP to a predefined level and then stopping completely, results in: <ul style="list-style-type: none"> ○ Less total time on CPAP ○ Shorter durations of oxygen ○ Reduced length of hospital stay⁷⁶
Duration	<ul style="list-style-type: none"> • Clinical criteria to reduce, wean or discontinue CPAP: <ul style="list-style-type: none"> ○ Gestational and chronological age of baby ○ Current weight ○ Clinical markers of respiratory stability • Assess clinical signs indicating the baby is not tolerating CPAP reduction or cessation⁴³ • 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 weaning⁴³ • Prolonged use of CPAP (as indicated) reduces the overall supplemental oxygen requirement⁴³
Recommencing CPAP	<ul style="list-style-type: none"> • Before recommencing CPAP assess baby's clinical condition to exclude complications requiring additional management (e.g. pneumothorax, apnoea of prematurity) • Recommence if: <ul style="list-style-type: none"> ○ Work of breathing increases [refer to Definitions] ○ SpO₂ falls • Recommence at 6–8 cmH₂O and adjust accordingly

References

1. Johnson K. Transient tachypnea of the newborn. [Internet]. Waltham MA: UpToDate Inc; 2018 [cited 2019 May 30]. Available from: <https://www.uptodate.com>.
2. Martin R. Pathophysiology, clinical manifestations, and diagnosis of respiratory distress syndrome in the newborn. [Internet]. Waltham MA: UpToDate Inc; 2018 [cited 2019 May 30]. Available from: <https://www.uptodate.com>.
3. Schittny JC. Development of the lung. *Cell and Tissue Research* 2017;367(3):427-44.
4. Alexiou S, Panitch HB. Physiology of non-invasive respiratory support. *Seminars in Fetal and Neonatal Medicine* 2016;21(3):174-80.
5. The Royal Australian and New Zealand College of Obstetricians and Gynaecologists. Timing of elective caesarean section at term. *College Statement C-Obs 23*. [Internet]. 2018 [cited 2019 November 18]. Available from: <https://ranzcoog.edu.au>.
6. Garcia-Pratts J. Clinical features and diagnosis of meconium aspiration. [Internet]. Waltham MA: UpToDate Inc; 2019 [cited 2019 October 3]. Available from: <https://www.uptodate.com>.
7. Hermansen CL, Mahajan A. Newborn respiratory distress. *American Family Physician* 2015;92(11):994-1002.
8. Mirzamoradi M, Hasani Nejhad F, Jamali R, Heidar Z, Bakhtiyari M. Evaluation of the effect of antenatal betamethasone on neonatal respiratory morbidities in late preterm deliveries (34–37 weeks). *The Journal of Maternal-Fetal & Neonatal Medicine* 2019;1-8.
9. Roberts D, Brown J, Medley N, Dalziel S. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. *Cochrane Database of Systematic Reviews*. [Internet]. 2017 [cited 2019 December 12]; Issue 3. Art. No.: CD004454 DOI:10.1002/14651858.CD004454.pub3.
10. Gyamfi-Bannerman C. Antenatal late preterm steroids (ALPS): a randomized trial to reduce neonatal respiratory morbidity. *American Journal of Obstetrics and Gynecology* 2016;214(1, Supplement):S2.
11. Queensland Clinical Guidelines. Preterm labour and birth. Guideline No. MN20.6-V9-R25. [Internet]. Queensland Health. 2020. [cited 2020 June 30]. Available from: <https://www.health.qld.gov.au/gcg>
12. Queensland Clinical Guidelines. Standard care. Guideline No. MN18.50-V1-R23. [Internet]. Queensland Health. 2018. [cited 2019 September 30]. Available from: <https://www.health.qld.gov.au/gcg>
13. Queensland Health. Clinical Services Capability Framework Neonatal Services V3.2. [Internet]. 2014 [cited 2019 August 29]. Available from: <https://www.health.qld.gov.au>.
14. Queensland Health. Clinical Services Capability Framework for Public and Licensed Private Health Facilities v3.2. [Internet]. 2014 [cited 2019 August 29]. Available from: <https://www.health.qld.gov.au>.
15. Reuter S, Moser C, Baack M. Respiratory distress in the newborn. *Pediatrics in Review* 2014;35(10):417-29.
16. Victoria Government. Blood gas interpretation for neonates. [Internet]. 2018 [cited 2019 December 12]. Available from: <https://www.bettersafercare.vic.gov.au>.
17. Arthur R. The neonatal chest x-ray. *Paediatric Respiratory Reviews* 2001;2(4):311-23.
18. Fernandes C. Pulmonary air leak in the newborn. [Internet]. Waltham MA: UpToDate Inc; 2019 [cited 2019 June 6]. Available from: <https://www.uptodate.com>.
19. Longoni M, Pober B, High F. Congenital diaphragmatic hernia overview. In: Adam M, Ardinger H, Pagon R, editors. *GeneReviews*. [Internet]. Seattle: University of Washington; 2019 [cited 2019 June 5]. Available from: <https://www.ncbi.nlm.nih.gov>.
20. Garcia-Pratts J. Prevention and management of meconium aspiration syndrome. [Internet]. Waltham MA: UpToDate Inc; 2018 [cited 2019 September 11]. Available from: <https://www.uptodate.com>.
21. Fraser D. Nursing care. In: Goldsmith J, Karotkin E, Kesler M, Suresh G, editors. *Assisted Ventilation of the Neonate*. Philadelphia: Elsevier; 2017. p. 310-21.e3.
22. Sweet DG, Carnielli V, Greisen G, Hallman M, Ozek E, te Pas A, et al. European consensus guidelines on the management of respiratory distress syndrome—2019 update. *Neonatology* 2019;115(4):432-50.
23. Queensland Clinical Guidelines. Hypoxic ischaemic encephalopathy. Guideline No. MN16.11-V9-R21. [Internet]. Queensland Health. 2018. [cited 2019 December 12]. Available from: <https://www.health.qld.gov.au/gcg>
24. Subramaniam P, Ho JJ, Davis PG. Prophylactic nasal continuous positive airway pressure for preventing morbidity and mortality in very preterm infants. *Cochrane Database of Systematic Reviews*. 2016; (Issue 6. Art. No.: CD001243.) DOI:10.1002/14651858.CD001243.pub3.
25. Queensland Clinical Guidelines. NeoMedQ Survanta. Guideline No. NMedQ20.039-V1-R25. [Internet]. Queensland Health. 2020. [cited 2020 May 29]. Available from: <https://www.health.qld.gov.au/gcg>
26. Queensland Clinical Guidelines. NeoMedQ Curosurf. Guideline No. NMedQ20.040-V1-R25. [Internet]. Queensland Health. 2020. [cited 2020 May 29]. Available from: <https://www.health.qld.gov.au/gcg>
27. Stroustrup A, Trasande L, Holzman I. Randomized controlled trial of restrictive fluid management in transient tachypnea of the newborn. *Journal of Pediatrics* 2012;160(1):38-43.e1.
28. Mohamed ISI, Lee Y-H, Yamout SZ, Fakir S, Reynolds AM. Ultrasound guided percutaneous relief of tension pneumomediastinum in a 1-day-old newborn. *BMJ case reports*. [Internet]. 2009 [cited 2019 December 18]; bcr2006114322 DOI:<https://doi.org/10.1136/adc.2006.114322>.
29. Roychoudhury S, Kaur S, Soraisam A. Neonatal pneumopericardium in a nonventilated term infant: a case report and review of the literature. *Case Reports in Pediatrics*. [Internet]. 2017 [cited 2020 February 25]; 2017 DOI:<https://doi.org/10.1155/2017/3149370>.
30. Brouillette R, Waxman D. Evaluation of the newborn's blood gas status. *Clinical Chemistry* 1997;43(1):215-21.
31. Gardner S, Hines ME, Nyp M. Respiratory diseases. In: Gardner SL, Hernandez JA, Gardner SL, Carter BS, Hines ME, Hernandez JA, editors. *Merenstein and Gardner's Handbook of Neonatal Intensive Care*. Eighth ed. St. Louis, Missouri: Elsevier; 2016. p. 565-643.
32. Stark A, Eichenwald E. Persistent pulmonary hypertension of the newborn. UpToDate Inc. [Internet]. 2019 [cited 2019 December 3]. Available from: <https://www.uptodate.com>.
33. Grigoriadis S, VonderPorten EH, Mamisashvili L, Tomlinson G, Dennis C-L, Koren G, et al. Prenatal exposure to antidepressants and persistent pulmonary hypertension of the newborn: systematic review and meta-analysis. *British Medical Journal* 2014;348:f6932.
34. Queensland Clinical Guidelines. Hypoglycaemia-newborn. Guideline No. MN19.8-V6-R24. [Internet]. Queensland Health. 2019. [cited 2019 October 30]. Available from: <https://www.health.qld.gov.au/gcg>
35. Queensland Clinical Guidelines. NeoMedQ Benzylpenicillin. Guideline No. NeoMedQ20.013-V1-R25. [Internet]. Queensland Health. 2020. [cited 2020 March 2]. Available from: <https://www.health.qld.gov.au/gcg>
36. Queensland Clinical Guidelines. NeoMedQ Ampicillin. Guideline No. NeoMedQ19.012-V1-R24. [Internet]. Queensland Health. 2019. [cited 2019 August 28]. Available from: <https://www.health.qld.gov.au/gcg>
37. Queensland Clinical Guidelines. NeoMedQ Gentamicin. Guideline No. NeoMedQ20.038-V1-R25. [Internet]. Queensland Health. 2020. [cited 2020 March 02]. Available from: <https://www.health.qld.gov.au/gcg>
38. Queensland Clinical Guidelines. Early onset Group B Streptococcal disease. Guideline No. MN16.20-V3-R21. [Internet]. Queensland Health. 2016. [cited 2019 August 28]. Available from: <https://www.health.qld.gov.au/gcg>
39. Queensland Clinical Guidelines. Newborn hypoglycaemia. Guideline No. MN13.8-V5-R18. [Internet]. Queensland Health. 2015. [cited 2016 February 26]. Available from: <https://www.health.qld.gov.au/gcg>
40. Gupta S, Donn SM. Continuous positive airway pressure: physiology and comparison of devices. *Seminars in Fetal and Neonatal Medicine* 2016;21(3):204-11.
41. de Klerk A. Physiology of humidification in critically ill neonates. In: Esquinas A, editor. *Humidification in the Intensive Care Unit*. Heidelberg: Springer-Verlag; 2012. p. 253-66.
42. Flanagan K, Ann Msn R-N. Noninvasive ventilation in premature neonates. *Advances in Neonatal Care* 2016;16(2):91-8.
43. Bamat N, Jensen EA, Kirpalani H. Duration of continuous positive airway pressure in premature infants. *Seminars in Fetal and Neonatal Medicine* 2016;21(3):189-95.
44. Paoli A, P D, Faber B, Morley CJ. Devices and pressure sources for administration of nasal continuous positive airway pressure (NCPAP) in preterm neonates. *Cochrane Database of Systematic Reviews*. 2008; Issue 1. Art. No.: CD002977 DOI:<https://doi.org/10.1002/14651858.cd002977>.
45. Guay J, Carvi D, Raines D, Luce W. Care of the neonate on nasal continuous positive airway pressure: a bedside guide. *Neonatal Network* 2018;37(1):24-32.
46. Schmöler GM, Kumar M, Pichler G, Aziz K, O'Reilly M, Cheung P-Y. Non-invasive versus invasive respiratory support in preterm infants at birth: systematic review and meta-analysis. *British Medical Journal* 2013;347:f5980.
47. Faal G, Eghbal F. Effect of bubble and ventilator-derived continuous positive airway pressure on the management of respiratory distress syndrome in premature neonates. *Iranian Journal of Neonatology* 2018;9(4):22-7.

48. Behnke J, Lemyre B, Czernik C, Zimmer K-P, Ehrhardt H, Waitz M. Non-invasive ventilation in neonatology. *Deutsches Arzteblatt International* 2019;116(11):177-83.
49. Celik M, Bulbul A, Uslu S, Dursun M, Guran O, Kiray Bas E, et al. A comparison of the effects of invasive mechanic ventilation/surfactant therapy and non-invasive nasal-continuous positive airway pressure in preterm newborns. *The Journal of Maternal-Fetal and Neonatal Medicine* 2018;31(24):3225-31.
50. Fischer HS, Bührer C. Avoiding endotracheal ventilation to prevent bronchopulmonary dysplasia: a meta-analysis. *Pediatrics* 2013;132(5):e1351.
51. Green EA, Dawson JA, Davis PG, De Paoli AG, Roberts CT. Assessment of resistance of nasal continuous positive airway pressure interfaces. *Archives of Disease in Childhood–Fetal and Neonatal Edition* 2019;104(5):F535.
52. Guerin C. Randomized control trial comparing physiologic effects in preterm infants during treatment with nasal continuous positive airway pressure (NCPAP) generated by bubble NCPAP and ventilator NCPAP: a pilot study. *Journal of Perinatal Medicine* 2016;44(6):655-61.
53. Jasani B, Ismail A, Rao S, Patole S. Effectiveness and safety of nasal mask versus binasal prongs for providing continuous positive airway pressure in preterm infants—a systematic review and meta-analysis. *Pediatric Pulmonology* 2018;53(7):987-92.
54. King BC, Gandhi BB, Jackson A, Katakam L, Pammi M, Suresh G. Mask versus prongs for nasal continuous positive airway pressure in preterm infants: a systematic review and meta-analysis. *Neonatology* 2019;116(2):100-14.
55. Fisher and Paykel Healthcare. Bubble CPAP system set-up guide. [Internet]. 2011 [cited 2019 November 11]. Available from: <https://www.fphcare.com/au>.
56. Ahmadpour-Kacho M, Zahed Pasha Y, Hahdinejad Z, Khafri S. The effect of non-nutritive sucking on transcutaneous oxygen saturation in neonates under the nasal continuous positive airway pressure (CPAP). *International Journal of Pediatrics* 2017;5(3):4511-9.
57. Sweet M, Armbruster D, Bainbridge E, Reiner B, Tan A, Chipps E. A pilot study of responses to suctioning among neonates on bubble nasal continuous positive airway pressure. *Advances in Neonatal Care* 2017;17(6):E3-E11.
58. Liaw J-J, Yang L, Lee C-M, Fan H-C, Chang Y-C, Cheng L-P. Effects of combined use of non-nutritive sucking, oral sucrose, and facilitated tucking on infant behavioural states across heel-stick procedures: a prospective, randomised controlled trial. *International Journal of Nursing Studies* 2013;50(7):883-94.
59. Thakkar P, Arora K, Goyal K, Das RR, Javadekar B, Aiyyer S, et al. To evaluate and compare the efficacy of combined sucrose and non-nutritive sucking for analgesia in newborns undergoing minor painful procedure: a randomized controlled trial. *Journal of Perinatology* 2016;36(1):67-70.
60. Sahni R, Schiaratura M, Polin RA. Strategies for the prevention of continuous positive airway pressure failure. *Seminars in Fetal and Neonatal Medicine* 2016;21(3):196-203.
61. Montgomery K, Choy NL, Steele M, Hough J. The effectiveness of quarter turn from prone in maintaining respiratory function in premature infants. *Journal of Paediatrics and Child Health* 2014;50(12):972-7.
62. Utario Y, Rustina Y, Waluyanti F. The quarter prone position increases oxygen saturation in premature infants using continuous positive airway pressure. *Comprehensive Child and Adolescent Nursing* 2017;40(S1):95-101.
63. Yin T, Yuh Y-S, Liaw J-J, Chen Y-Y, Wang K-WK. Semi-prone position can influence variability in respiratory rate of premature infants using nasal CPAP. *Journal of Pediatric Nursing* 2016;31(2):e167-e74.
64. Courtois E, Cimerman P, Dubuche V, Goiset M-F, Orfèvre C, Lagarde A, et al. The burden of venipuncture pain in neonatal intensive care units: EPIPPAIN 2, a prospective observational study. *International Journal of Nursing Studies* 2016;57:48-59.
65. Newnam KM, McGrath JM, Estes T, Jallo N, Salyer J, Bass WT. An integrative review of skin breakdown in the preterm infant associated with nasal continuous positive airway pressure. *Journal of Obstetric, Gynecologic & Neonatal Nursing* 2013;42(5):508-16.
66. Chen C-Y, Chou A-K, Chen Y-L, Chou H-C, Tsao P-N, Hsieh W-S. Quality improvement of nasal continuous positive airway pressure therapy in neonatal intensive care unit. *Pediatrics & Neonatology* 2017;58(3):229-35.
67. Badr L, Zeineddine M, Abbas H, Charafeddine L. NeoSeal to prevent nasal injury in preterm infants receiving oxygen therapy. *Neonatal Network* 2017;35(4):228-33.
68. Chao JW, Raveendran JA, Sauerhammer TM, Rogers GF, Oh AK, Boyajian M. Columellar reconstruction after nasal continuous positive airway pressure associated necrosis. *The Journal of Craniofacial Surgery* 2017;28(4):928-30.
69. Newnam KM, McGrath JM, Salyer J, Estes T, Jallo N, Bass WT. A comparative effectiveness study of continuous positive airway pressure-related skin breakdown when using different nasal interfaces in the extremely low birth weight neonate. *Applied Nursing Research* 2015;28(1):36-41.
70. Ottinger D, Hicks J, Wilson S, Sperber K, Power K. The pressure is on! Neonatal skin and nasal continuous positive airway pressure. *Advances in Neonatal Care* 2016;16(6):420-3.
71. Imbulana D, Owen L, Dawson JA, Bailey J, Davis PG, Manley B. A randomized controlled trial of a barrier dressing to reduce nasal injury in preterm infants receiving binasal noninvasive respiratory support. *Journal of Pediatrics* 2018;201:34-9.
72. McCoskey L. Nursing care guidelines for prevention of nasal breakdown in neonates receiving nasal CPAP. *Advances in Neonatal Care* 2008;8(2):116-24.
73. Ho JJ, Subramaniam P, Davis PG. Continuous distending pressure for respiratory distress in preterm infants. *Cochrane Database of Systematic Reviews*. [Internet]. 2015 [cited 2019 June 7]; Issue 7. Art. No.: CD002271(7) DOI:<https://doi.org/10.1002/14651858.cd002271.pub2>
74. Rodrigues L, Nesargi SV, Fernandes M, Shashidhar A, Rao SPN, Bhat S. Analgesic efficacy of oral dextrose and breast milk during nasopharyngeal suctioning of preterm infants on CPAP: a blinded randomized controlled trial. *Journal of Tropical Pediatrics* 2017;63(6):483-8.
75. American Association for Respiratory Care. Application of continuous positive airway pressure to neonates via nasal prongs, nasopharyngeal tube, or nasal mask—2004 revision & update. *Respiratory Care* 2004;49(9):1100-8.
76. Jardine LA, Inglis GDT, Davies MW. Strategies for the withdrawal of nasal continuous positive airway pressure (NCPAP) in preterm infants. *Cochrane Database of Systematic Reviews*. 2011; (2) DOI:<https://doi.org/10.1002/14651858.cd006979.pub2>
77. Al-Lawama M, Alkhatib H, Wakileh Z, Elqaisi R, AlMassad G, Badran E, et al. Bubble CPAP therapy for neonatal respiratory distress in level III neonatal unit in Amman, Jordan: a prospective observational study. *International Journal of General Medicine* 2018;12:25-30.
78. Eze N, Murphy D, Dhar V, Rehan VK. Comparison of sprinting vs non-sprinting to wean nasal continuous positive airway pressure off in very preterm infants. *Journal of Perinatology* 2017;38:164.
79. Todd DA, Wright A, Broom M, Chauhan M, Meskell S, Cameron C, et al. Methods of weaning preterm babies < 30 weeks gestation off CPAP: a multicentre randomised controlled trial. *Archives of Disease in Childhood–Fetal and Neonatal Edition* 2012;97(4):F236.

Appendix A Comparison of respiratory disease

Type	Gestation	Onset after birth	Risk factors	Clinical features	X-ray features
		Cause			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	Risk factors	Clinical features	X-ray features
		Cause			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	Intrauterine and perinatal asphyxia SSRI exposure PPROM	Worsening respiratory distress and cyanosis despite high FiO ₂	Normal or reflects underlying respiratory disease
		Abnormalities of pulmonary vasculature <ul style="list-style-type: none"> • Underdevelopment (e.g. diaphragmatic hernia) • Maldevelopment (e.g. CHD, MAS) • Maladaptation (e.g. post-term birth, caesarean birth, LGA) 			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: <ul style="list-style-type: none"> • 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

Acknowledgements

Queensland Clinical Guidelines gratefully acknowledge the contribution of Queensland clinicians and other stakeholders who participated throughout the guideline development process particularly:

Working Party Clinical Lead

Ms Seija Argyros, Neonatal Nurse Practitioner, Royal Brisbane and Women's Hospital
Dr Kirsty Devine, Neonatologist, The Townsville Hospital

QCG Program Officer

Ms Stephanie Sutherns

Working Party Members

Dr Gary Alcock, Neonatologist, The Townsville Hospital, Townsville
Mrs Maxine Ballinger, Clinical Nurse Consultant, Rockhampton Hospital, Rockhampton
Miss Hannah Bettison, Acting Clinical Midwife Consultant, Thursday Island Hospital, Thursday Island
Ms Lyn Chapple, Clinical Nurse, Royal Brisbane and Women's Hospital, Brisbane
Ms Li-an Collie, Nurse Educator, NeoRESQ, Brisbane
Ms Deborah Collins, Clinical Facilitator, Logan Hospital, Logan
Mr Greg Coulson, Neonatal Nurse Practitioner, Sunshine Coast University Hospital, Sunshine Coast
Ms Amanda Curley, Clinical Nurse, Logan Hospital, Brisbane
Dr Joy Domingo-Bates, Neonatal Fellow, Mater Mother's Hospital/Royal Brisbane and Women's Hospital NeoRESQ, Brisbane
Ms Anne-Marie Feary, Practice Development Nurse, Gold Coast University Hospital, Gold Coast
Mrs Anndrea Flint, Neonatal Nurse Practitioner, Redcliffe Hospital, Redcliffe
Mrs Ariadne Forman, Principal Medical Education Officer/Senior Pharmacist, Gold Coast University Hospital, Gold Coast
Mrs Nicol Franz, Registered Nurse, Caboolture Hospital, Caboolture
Dr Deborah Gilmour, Neonatologist, Mater Mothers' Hospital, Brisbane
Mrs Tina Gray, Registered Nurses, Gladstone Hospital, Gladstone
Mrs Linda Hackett, Clinical Nurse, Bundaberg Hospital, Bundaberg
Ms Joanne Hardcastle, Clinical Nurse/Midwife/Clinical Nurse Educator, The Wesley Hospital, Brisbane
Dr Shivanand Hebbandi, Paediatrician, Redland Hospital, Cleveland
Mrs Julianne Hite, Clinical Nurse/Nurse Educator, Rockhampton Hospital, Rockhampton
Mrs Jodie Hole, Registered Nurse/Midwife, Buderim Private Hospital, Sunshine Coast
Ms Karen Hose, Nurse Practitioner, Royal Brisbane and Women's Hospital, Brisbane
Dr Judy Hough, Physiotherapist Consultant, Mater Health, South Brisbane
Miss Merbie Babu Kurian, Registered Nurse, Royal Brisbane and Women's Hospital, Brisbane
Miss Christine Latimer, Clinical Nurse Consultant, The Townsville Hospital, Townsville
Mrs Katharine Lawlor, Clinical Educator, Logan Bayside, Logan
Mrs Kate McFarlane, Clinical Nurse, Hervey Bay Hospital, Hervey Bay
Mrs Colette McIntyre, Clinical Nurse Consultant, Royal Brisbane and Women's Hospital, Brisbane
Mrs Melanie McKenzie, Director/Consumer Representative, Harrison's Little Wings Inc, Logan
Mrs Lisa McKeown, Registered Nurse, Royal Brisbane and Women's Hospital, Brisbane
Ms Amanda Merchant, Clinical Nurse Consultant, Mater Mothers' Hospital, Brisbane
Ms Hayley Mongta, Registered Nurse/ Registered Midwife, Hervey Bay Hospital, Hervey Bay
Mrs Megan Murphy, Nurse Practitioner, The Townsville Hospital, Townsville
Ms Jacqueline Plazina, Clinical Nurse, Royal Brisbane and Women's Hospital, Brisbane
Dr Prasanna Shirkhedkar, Paediatrician, Caboolture Hospital, Caboolture
Miss Divya Siva Das, Speech Pathologist, Gold Coast University Hospital, Gold Coast
Mrs Melinda Stevenson, Registered Midwife, Redland Hospital, Brisbane
Mr Christopher Swan, Consumer Representative, Preterm Infant Parents Association (PIPA), Brisbane
Mrs Elizabeth Upton, Clinical Pharmacist, Sunshine Coast University Hospital, Sunshine Coast
Dr Lizelle Weber, Neonatologist, Sunshine Coast University Hospital, Sunshine Coast
Dr Katherine White, Neonatologist, Royal Brisbane and Women's Hospital, Brisbane
Mrs Deborah Wright, Clinical Nurse, Sunshine Coast University Hospital, Sunshine Coast

Queensland Clinical Guidelines Team

Associate Professor Rebecca Kimble, Director
Ms Jacinta Lee, Manager
Ms Stephanie Sutherns, Clinical Nurse Consultant
Ms Cara Cox, Clinical Nurse Consultant
Ms Emily Holmes, Clinical Nurse Consultant
Steering Committee

Funding

This clinical guideline was funded by Healthcare Improvement Unit, Queensland Health