Clinical Guidance

Paediatric Critical Care: Acute Respiratory Distress Syndrome (ARDS)

Summary
Guidance for the management of patients with Acute Respiratory Distress Syndrome.

Document Detail	
Document type	Clinical Guideline
Document name	Paediatric Critical Care: Acute Respiratory Distress Syndrome (ARDS)
Document location	GTi Clinical Guidance Database & Evelina London Website
Version	V2.0
Effective from	*14th October 2022*
Review date	*14th October 2025*
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Approved by, date	Evelina London Clinical Guideline Committee, October 2022
Superseded documents	V1.0
Related documents	VV ECMO, ECMO Goals, Sepsis, Asthma, Bronchiolitis
Keywords	ARDS, acute respiratory distress syndrome, ventilation, respiratory, hypoxia, ECMO, PICU, child, Evelina
Relevant external law, regulation, standards	This clinical guideline has been produced by the South Thames Retrieval Service (STRS) at Evelina London for nurses, doctors and ambulance staff to refer to in the emergency care of critically ill children. This guideline represents the views of STRS and was produced after careful consideration of available evidence in conjunction with clinical expertise and experience. The guidance does not override the individual responsibility of healthcare professionals to make decisions appropriate to the circumstances of the individual patient.

Date	Change details, since approval	Approved
September 2022	Update on PALICC guidance about diagnosing ARDS. Initial management steps, including emergency resuscitation. Consideration of pulmonary embolus. Updated advice on ventilation strategy, recruitment measures, VV ECMO with attached guidance.	October 2022

Glossary:
OI: oxygenation index, OSI Oxygen Saturation Index, SIADH: syndrome of inappropriate ADH
TAPVD: total anomalous pulmonary venous drainage, PH: Pulmonary Hypertension, RV: right ventricle
iNO: inhaled nitric oxide, Ti: inspiratory time, V-Q: ventilation-perfusion, CTPA: CT pulmonary angiogram

Paediatric Critical Care

Paediatric Acute Respiratory Distress Syndrome (PARDS)

Diagnostic criteria of PARDS

- Clinical syndrome of hypoxaemia associated with pulmonary infiltrates secondary to oedema (Table)[1]. Degree of hypoxaemia is used to classify severity:
  - **OI** (uses PaO_2) ***OR***
  - **OSI** (SpO_2) if no arterial gas – removes barrier to diagnosis & allows earlier initiation of correct management strategy.
- PARDS affects 3% of PICU patients internationally

Mortality >30% in severe PARDS[2]

Age	Exclude patients with perinatal lung disease		
Timing	Within 7 days of known insult		
Origin of oedema	Respiratory failure not fully explained by cardiac failure of fluid overload		
Chest imaging	Chest imaging findings of new infiltrate(s) consistent with acute pulmonary parenchymal disease		
Severity	**Mild**	**Moderate**	**Severe**
OI*	4–8	8–16	≥16
OSI**	5–7.5	7.5–12.3	≥12.3
Special populations	Not explained by cyanotic heart disease, chronic lung disease and/or left ventricular dysfunction		

*OI = (FiO_2 in % x mean airway pressure) / PaO_2 in mmHg
**OSI = (FiO_2 in % x mean airway pressure) / SpO_2

Major causes of PARDS[2]:

- Pneumonia (63%, mortality 12%)
- Sepsis (19%, mortality 30%)
- Aspiration (8%, mortality 22%)

Baseline investigations:

- FBC, CRP, procalcitonin (PCT), biochemistry, coagulation, blood culture, Group and save, blood gas
- Respiratory secretion panel (ideally BAL)
- Daily CXR

Additional investigations:

- Bedside US: discriminate ARDS vs effusions
- Echocardiogram: ventricular function; CHD e.g. ASD to explain hypoxia or TAPVD, pulmonary hypertension (PH)

Management:

Airway & Breathing

- Cuffed ETT of age-appropriate size
- Anticipate cardio-respiratory collapse during induction of anaesthesia – difficult to match negative pressure self-ventilation, with positive pressure mechanical ventilation
- Use Ayre's T-piece to provide PEEP until ETT placed
- Use Ketamine/Fentanyl/Rocuronium for induction

Cardiovascular

- Anticipate CVS instability: fluid boluses & adrenaline infusion
- Pre-load may be affected by high-pressure ventilation, and in sepsis - low threshold for inotropic support.
- Place arterial line

Neurology

- Ensure adequate sedation – IV morphine infusion & clonidine enteral boluses or IVI recommended
- Muscle relaxation helpful in severe ARDS/ high pressure ventilation to optimise synchronicity

Renal/ Fluids

- High likelihood SIADH
- Pulmonary oedema will worsen ventilation status
- Aim for negative balance – fluid-restriction

Sepsis

- Presume infection – broad spectrum antibiotics initially
- Fluid resuscitate +/- inotropic support as needed

Consider pulmonary embolus *(especially in COVID-19 ARDS):*

- *High suspicion if hypoxia out of keeping with clinical state*
- *Perform echocardiogram (right heart strain) +/- CTPA[15]*

"Open lung" ventilation strategy for PARDS[1,3,4]

- Take care to exclude dynamic hyperinflation (obstructive airways disease) prior to initiating this strategy - if hyperinflation: use Asthma/ Bronchiolitis guidance[5]
- **Targets:**
  - SpO_2 ≥85% (>90% if pulmonary hypertension)
  - pH ≥7.2 (pH ≥7.3 if pulmonary hypertension)
  - Appropriate sized cuffed ETT for ventilation in PARDS
- **Mode:**
  - Conventional ventilation preferred to HFOV[6]
  - HFOV for consideration if respiratory ECMO not available AND peak plateau pressure >28
- **Parameters:**
  - Aim for adequate ventilation with minimal Ventilator-Induced Lung Injury (VILI)
  - High PEEP (10–14 cm H_2O[1]) for alveolar recruitment
  - Target low tidal volumes of 3–6 mL/kg[1] (ideal body weight)
  - Ensure peak plateau pressure capped at 32cmH_2O
  - Be mindful of driving pressure (*peak plateau – PEEP)* independently associated with length of ventilation & VILI[13]
  - Increase Ti to 1–1.2s depending on set rate[1]
  - iNO if PH or RV dysfunction and trial this if hypoxia refractory to above measures
- **Monitoring:**
  - SpO_2, $ETCO_2$, arterial gases, OI trend
  - Regular CXR; CT chest not routinely indicated

Ancillary therapy

- Prone positioning for 18-24 hours has been shown to improve outcomes[7]
  - Recruitment of atelectatic lung
  - Improves V-Q mismatch – better ventilation & oxygenation
  - Less over-distension in non-dependent lung regions[14]
- Suctioning to keep the ETT clear is essential, but caution to minimise the risk of derecruitment – consider inline vs open[1]
- There is no current evidence to support routine physiotherapy or use of surfactant/ corticosteroid treatment in PARDS. In severe cases, these therapies usually trialed/considered[1]

Extra-corporeal membranous oxygenation (ECMO)

- Initiate ECMO discussion if OI ≥25
- ECMO indicated if OI ≥40, disease process is reversible and no contraindications[10]
- Optimal time to initiate is unknown[11]. Worse outcomes if:
  - pH<7.2, Cardiac arrest prior to ECMO[12], >7 days since onset of disease[10]
- Standard of care is veno-venous ECMO[10] – ELCH guidance:
  - VV ECMO
  - ECMO Goals
- Counsel parents (stroke, bleeding, infection)[10]

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