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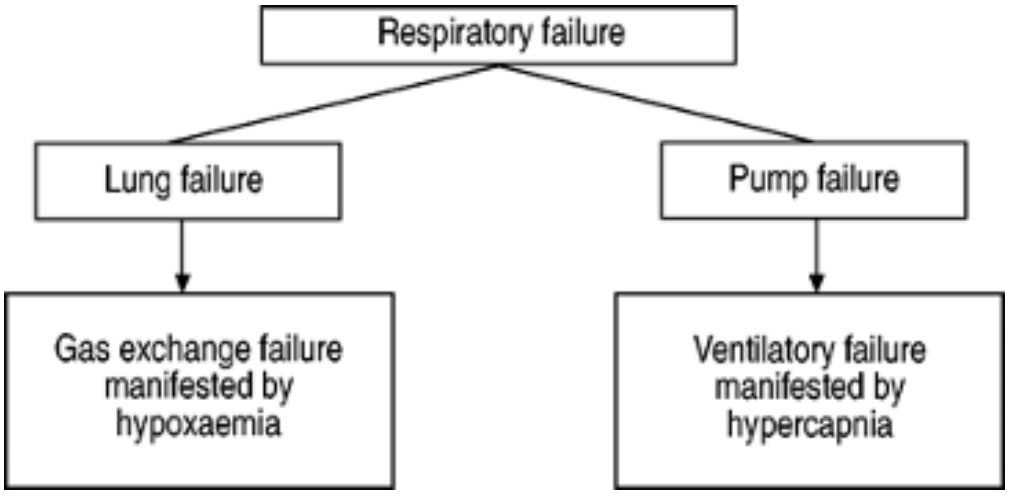
Emergency Department Clinical Guidelines

CCT Respiratory Support Guidelines

Purpose and Guideline Statement
To provide a clinical practice guideline for the management and initiation of respiratory support devices in patients presenting with respiratory failure.

Background
Acute respiratory failure is broadly defined as the inability to provide sufficient gas exchange due to insufficient oxygenation (type I or hypoxemic respiratory failure) or inability to clear carbon dioxide (type II or hypercapnic respiratory failure), or both. It is a common presenting condition treated in the emergency department with a high morbidity and mortality.



The management of acute respiratory failure often requires an escalation from various forms of non-invasive respiratory support devices to invasive ventilation. The type of respiratory support depends largely on the type of acute respiratory failure (although at times this is a mixed picture). The decision to escalate to invasive ventilation/intubation is largely based on clinical judgement and the patient's initial presentation and/or response to treatment.

Respiratory Support Guidelines
Hypoxemic respiratory failure covers a wide range of lung pathology. This guideline divides acute hypoxemic respiratory failure into the most common etiologies encountered in the Emergency Department, and provides initial options for respiratory support:

· Acute Respiratory Distress Syndrome: ARDS represents the most severe form of hypoxemic respiratory failure with a high mortality rate, and is due to diffuse inflammation of the lung parenchyma; the diagnosis of ARDS is based on the Berlin criteria (see below). In most cases, severe ARDS requires intubation, however mild ARDS (as classified by P/F ratio between 200-300) may be managed initially with non-invasive methods e.g. HFNC or NIPPV.

Table 3. The Berlin Definition of Acute Respiratory Distress Syndrome

	Acute Respiratory Distress Syndrome
Timing	Within 1 week of a known clinical insult or new or worsening respiratory symptoms
Chest imaging[a]	Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload Need objective assessment (eg, echocardiography) to exclude hydrostatic edema if no risk factor present
Oxygenation[b]	
Mild	200 mm Hg < PaO_2/FIO_2 ≤ 300 mm Hg with PEEP or CPAP ≥5 cm H_2O[c]
Moderate	100 mm Hg < PaO_2/FIO_2 ≤ 200 mm Hg with PEEP ≥5 cm H_2O
Severe	PaO_2/FIO_2 ≤ 100 mm Hg with PEEP ≥5 cm H_2O

o Non-invasive ventilation: In the setting of mild ARDS as defined by the Berlin criteria, BiPAP or HFNC can be considered. It is important to note that trials of NIPPV should be limited to patients that can be closely monitored with intubation available as needed.

o Initial settings:

- Start at IPAP of 10/EPAP of 5.
- Higher PEEP/EPAP will allow for recruitment of alveoli and reduce shunt physiology.
- Lower driving pressure/pressure support will provide for lung protection

o COVID considerations:

- In the setting of COVID-19, initial management can include awake proning in addition to HFNC or NIPPV, however the patient should be closely monitored with intubation available as needed.

· Pneumonia: infection and subsequent interstitial edema creates a large area of dead space and shunt physiology.

o Non-invasive ventilation: HFNC reduces the work of breathing and allows for clearance of more secretions when compared to BiPAP, and may reduce mortality rates and days spent on mechanical ventilation.

o Initial Settings:

- Initiate and titrate flow rate against the patient's work of breathing in increments of 10 L/minute up to 60 L/minute, titrate FiO2 against oxygen saturation to target saturation.

· Congestive Heart Failure Exacerbation/Flash Pulmonary Edema: In the setting of acute flash pulmonary edema, respiratory failure occurs when excess interstitial and alveolar fluid prevents appropriate gas exchange by creating an area of dead space and shunt physiology.

- o Non-invasive ventilation: For heart failure, CPAP is as effective as BiPAP; BiPAP may be used in patients with a mixed picture of hypoxemic and hypercapnic respiratory failure.
- o Initial Settings: A high ePAP will maintain high intrathoracic pressures, reducing both preload and afterload, and reducing intrapulmonary shunting.
  - Start at IPAP of 10/EPAP of 5
- o Escalation is based on patient need, work of breathing, oxygenation and hemodynamics. Avoid exceeding pressures greater than 20 cmH20 to decrease aspiration risk.

· Pulmonary embolism:

- o Oxygen can decrease RV afterload. Patients with RV dysfunction/failure due to significant PE and who are hypoxemic should have oxygen applied, however management should be geared towards hemodynamics, clot management, and avoidance of positive pressure when possible as transition to positive pressure ventilation can lead to worsening RV dysfunction/failure.

· Pneumothorax/Pleural effusion:

- o Management: acute respiratory failure due to pleural effusion or tension pneumothorax may require pleural drainage.

Hypercapnic respiratory failure results from pump failure and can be divided into two general etiologies:

· Asthma:

- o Non-invasive ventilation: For asthma, driving pressure provides mechanical support for breathing and offloads the work of the respiratory muscles especially in the setting of fatigue. However, if driving pressure does not reduce respiratory effort, gas trapping can occur. Higher EPAP keeps the airway open during exhalation potentially offsetting Auto-PEEP.
- o Initial Settings:
  - Start at IPAP of 10/EPAP of 5
- o Escalation is based on patient need, work of breathing, oxygenation and hemodynamics. Avoid exceeding pressures greater than 20 cmH20 to decrease aspiration risk.

· Chronic obstructive pulmonary disease: acute bronchospasm in the setting of chronic emphysema/bronchitis results in significant muscle fatigue and respiratory acidosis, resulting in pump failure and subsequent hypercapnia

- o Non-invasive ventilation: For COPD, the driving pressure (IPAP – EPAP) provides support during initiation of every breath
- o Initial Settings
  - Start at IPAP of 10/EPAP of 5
  - Titrate FiO2 to saturation 88-92%
- o Escalation is based on patient need, work of breathing, oxygenation and hemodynamics. Avoid exceeding pressures greater than 20 cmH20 to decrease aspiration risk.

· Acute and Acute on Chronic hypoventilation syndrome: due to neuromuscular disease including but not limited to muscular dystrophy, and myasthenia gravis, or chest wall injury resulting in diaphragmatic weakness/paralysis; Chronic hypoventilation syndrome (e.g. obesity hypoventilation):

- o Non-invasive ventilation: Similar to COPD/asthma, the key in acute hypoventilation syndrome is driving pressure, which provides mechanical support for each breath
  - Start at IPAP of 10/EPAP of 5
- o Escalation is based on patient need, work of breathing, oxygenation and hemodynamics. Avoid exceeding pressures greater than 20 cmH20 to decrease aspiration risk.
- o High ePAP is required to overcome extra-thoracic pressures.

In general patients requiring High Flow Nasal Cannula, non-invasive and/or invasive mechanical ventilation should have respiratory therapy available for titration of therapies and ventilator settings.

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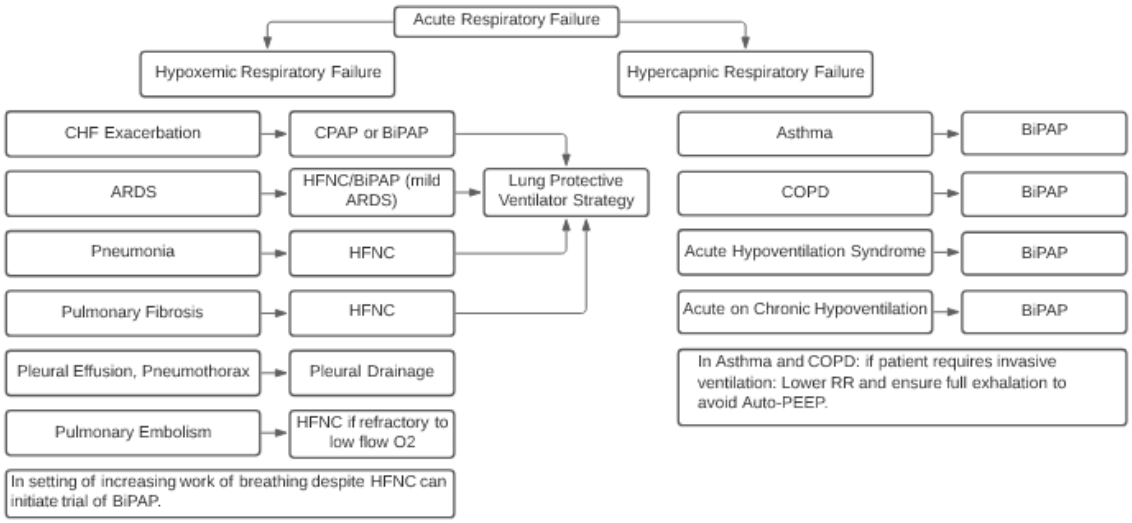
Guidelines for Respiratory Support in Acute Respiratory Failure



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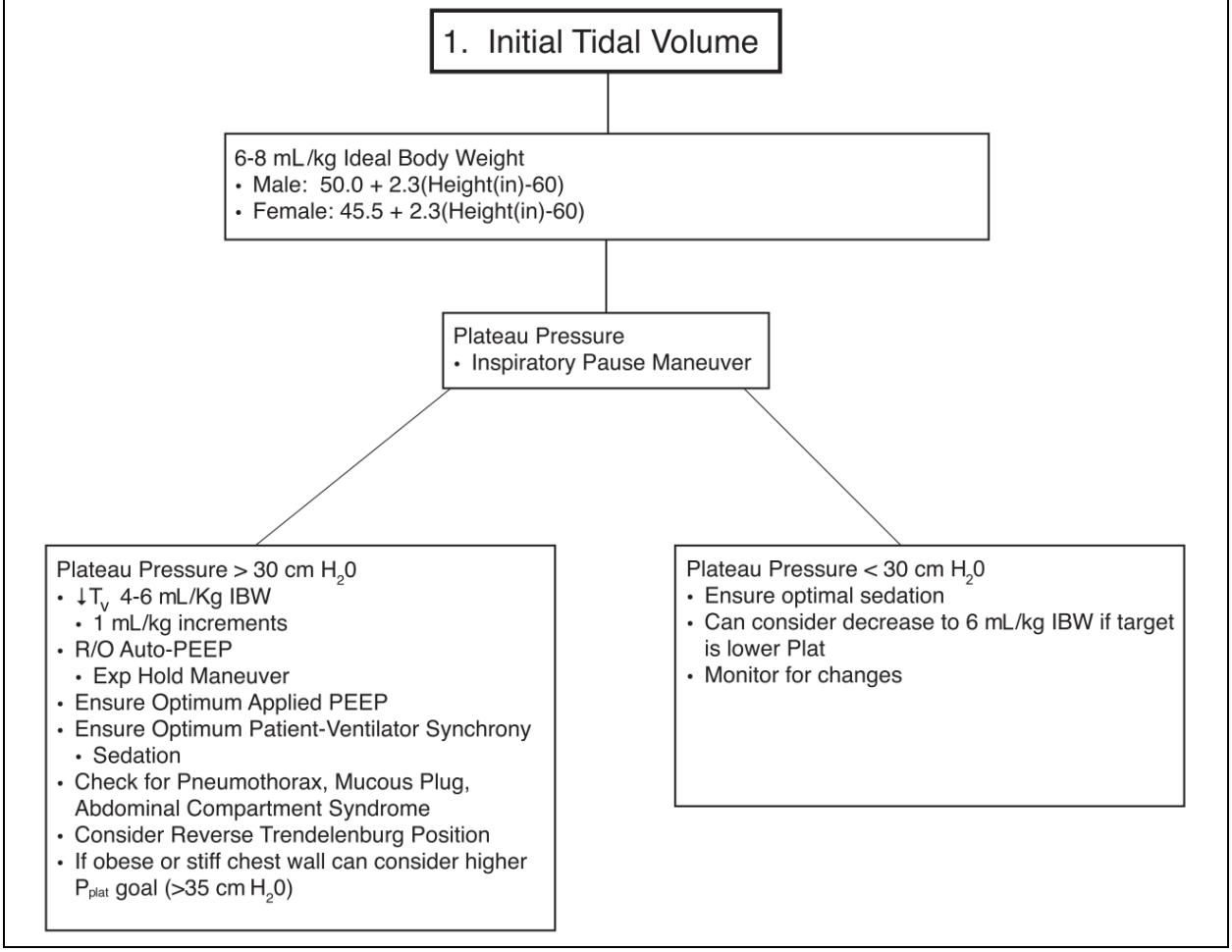
Guidelines for Initiation of Mechanical Ventilation in ARDS



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2. Respiratory Rate

Initial RR: 14-18
- Will depend heavily on metabolic demand, pH and $Paco_2$

Continuous End Tidal CO_2 ($ETco_2$) available?

No

- Estimate V_m based on NIV
- Pre-intubation RR
- Check ABG 30 min after intubation
  - Get exhaled V_m (total breaths x T_V) during ABG

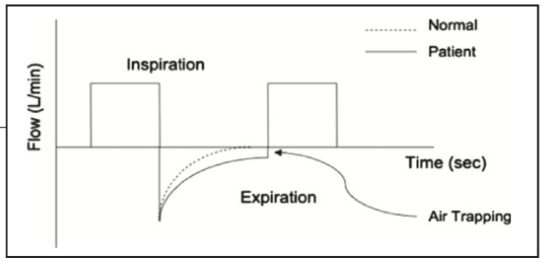
- Avoid Hypoventilation if using Lung Protective Ventilation/Low T_V Ventilation
- Avoid Overcorrection in COPD and OSA/OHS
- $V_m = T_V$ x RR
- Minimum V_m is 100 mL/kg in "normal" individual
  - If patient is on NIV prior to intubation can used measured Minute Ventilation as a guide
  - Estimate on pre-intubation RR and depth of Breaths
- Figure out goal $Paco_2$
  - Brain Injury Patients: $Paco_2$ 35-45
  - Otherwise can tolerate permissive hypercapnea provided pH is acceptable
- Adjust RR to Goal V_m
  - New $V_m = V_m$ x (actual $Paco_2$/desired $Paco_2$)
- Recheck 1 hour after ventilator adjustment, clinical change (especially if there is a change in rate of spontaneous breathing), then Q8-24 hours

- When increasing RR, ensure that there is not Auto-PEEP or air trapping
- Can examine flow waveform to ensure that expiratory flow returns to baseline
- Maneuvers
  - Increase Expiratory time by:
    - Increasing Inspiratory Flow Rate
    - Adjust I:E ratio
  - Decrease Tidal Volume
  - Decrease Resistance
    - Bronchodilators
    - Pulmonary Toilet

Yes

$ETco_2$ above goal $Paco_2$ (>10)
- Increase RR until $ETco_2$ approaches goal $Paco_2$
- Get ABG to calculate gap between $ETco_2$ and $Paco_2$ and pH
- Adjust RR to goal Vm using $ETco_2$ approximation of actual $Paco_2$

$ETco_2$ at or near goal $Paco_2$
- Check ABG to calculate gap between $ETco_2$ and $Paco_2$ and pH
- $ETco_2$ closely approximates $Paco_2$?
  - Adjust RR to goal V_m using $ETco_2$ approximation of actual $Paco_2$
- Large discrepancy between $ETco_2$ and $Paco_2$?
  - High Dead Space and/or Shock
  - Will need to recheck ABGs frequently based on ventilator adjustments and clinical changes to ensure that maneuvers have desired clinical response



3. Fraction of Inspired Oxygen (Fio_2)

- If acceptable pulse oximetry waveform is present titrate down to lowest possible Fio_2 required for goal Oxygen Saturation (> 95%)
  - COPD, OSA/OHS: 88-92%
- If no lung pathology (ie intubated for airway protection) can do rapidly
- Otherwise ≈ ↓10% Q 10 minutes
- Get ABG to confirm once at goal
- If you can't get Fio_2 less than 50%, see PEEP table below
  - Consider ARDS and other causes of Shunt Physiology and Severe Respiratory Failure

4. Positive End Expiratory Pressure

- Ensure patient is volume resuscitated and monitored
- Adjust PEEP per table to lowest Fio_2 and PEEP value to maintain adequate Spo_2
- Monitor for appropriate response
  - If paradoxical response, ie Oxygen Saturation drops with higher PEEP or initiation of MV consider cardiac shunts and unilateral lung disease and shunt
    - Chest xray and/or Lung US
    - ECHO bubble study
    - Decrease PEEP and consider other treatments for hypoxemia
- If PEEP > 8 is needed consider ARDS and other causes of Shunt Physiology and Severe Respiratory Failure

Fio_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	18-24

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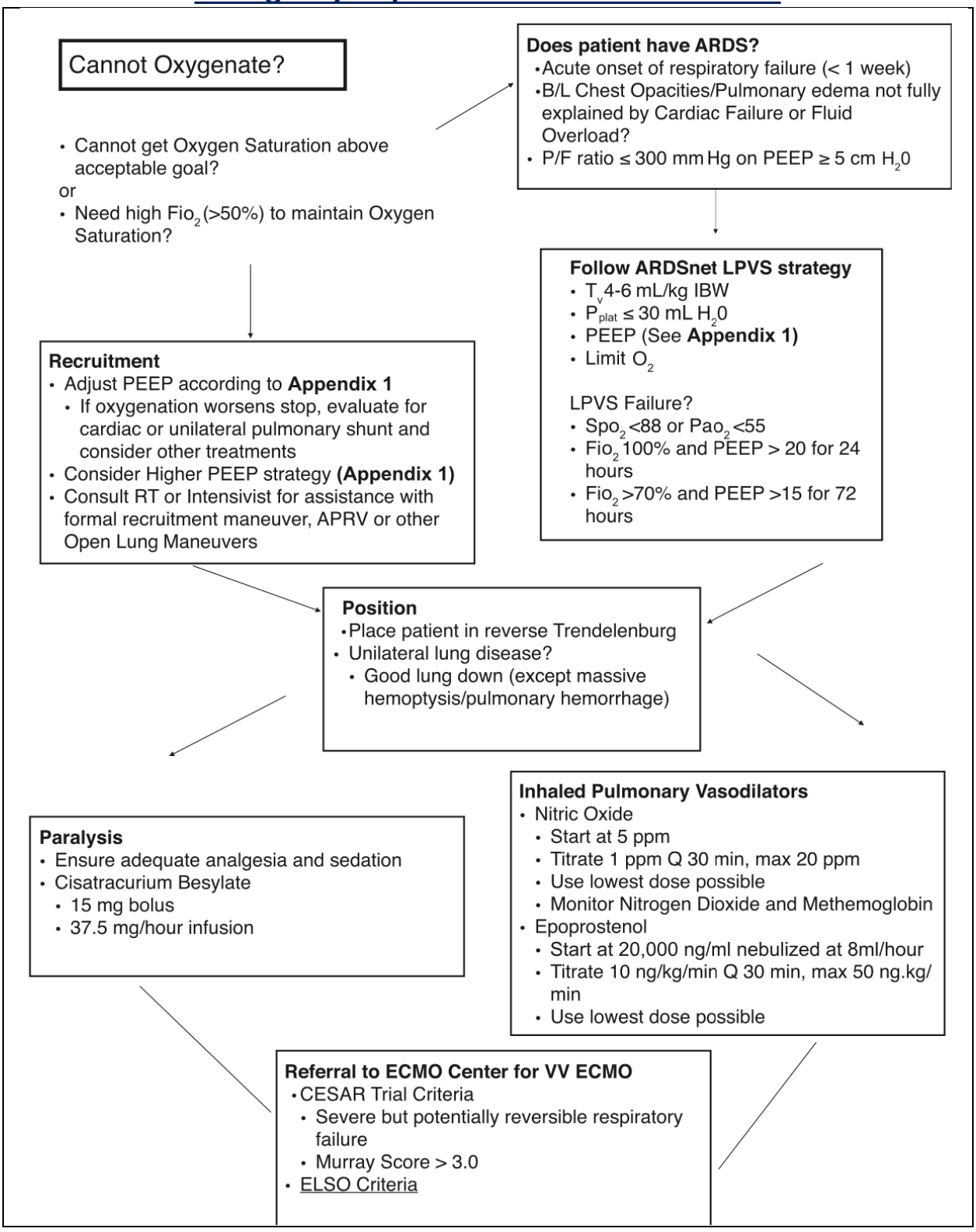

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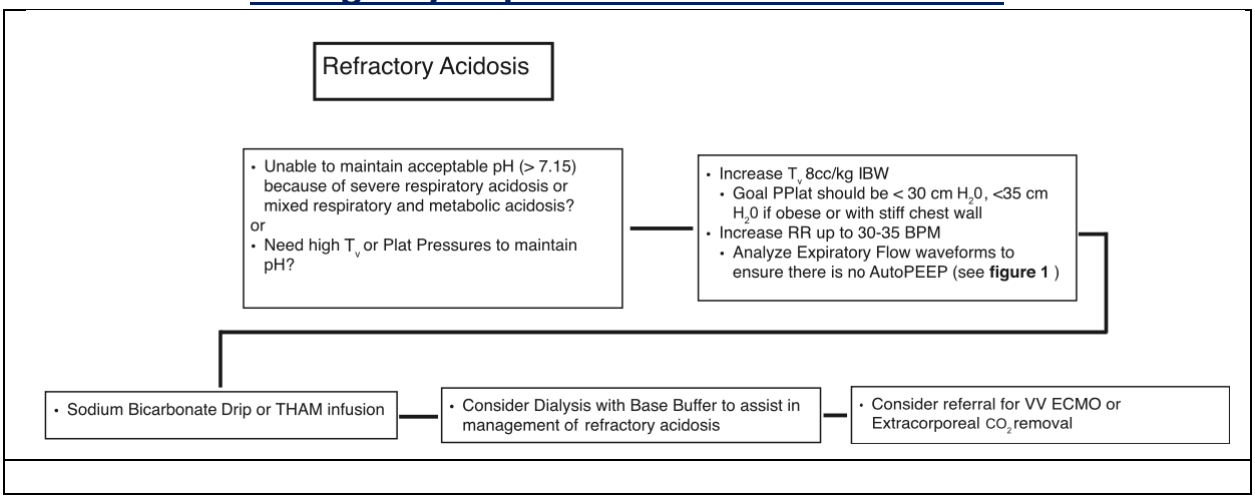

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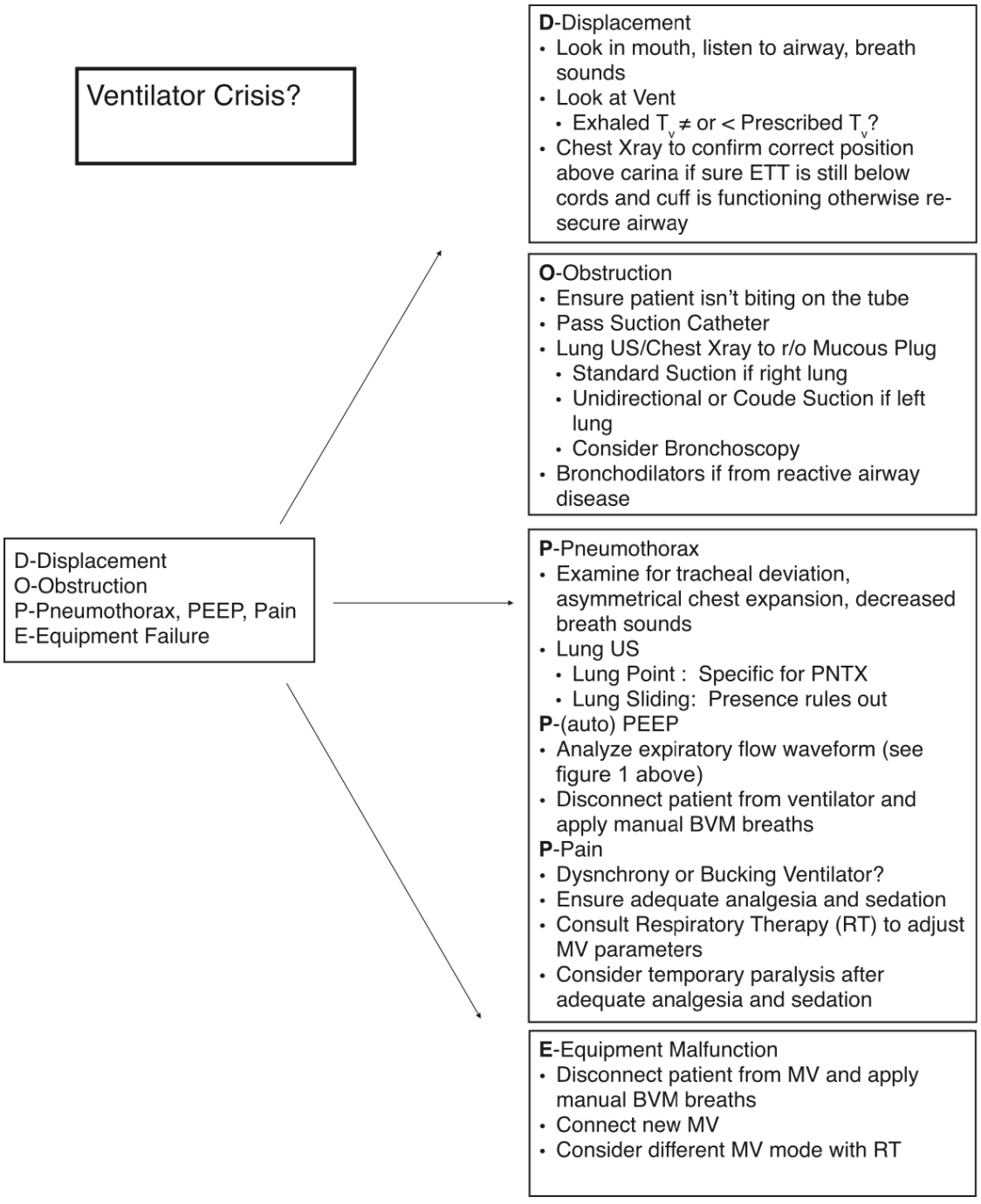
Algorithm for Ventilator Crisis Management



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APPENDIX

There are several devices available for non-invasive ventilation, detailed below:

High flow nasal cannula (HFNC):

AIRVO2

Optiflow

Non-Invasive Positive Pressure Ventilation (NIPPV):

LTV1200

Versamed IVent

Philips V60

Revel

Effective Date: