Approach to acute respiratory failure for frontline clinicians

Opening Vignette

A 70-year-old man with chronic obstructive pulmonary disease and hypertension was brought by family members to the emergency department for severe dyspnoea. You were the doctor on duty; therefore, you obtained the patient's history of worsening fever and productive cough over the past 3 days. His vital signs were as follows: respiratory rate 30 breaths/min, heart rate 110 beats/min, blood pressure 130/80 mmHg and peripheral oxygen saturation 95% in room air. On examination, he had normal mental status, a normal capillary refill time of two seconds and bilateral bronchial breath sounds. A chest X-ray showed bilateral consolidation consistent with multi-lobar pneumonia. You quickly obtained blood cultures, administered antibiotics and started nebulised bronchodilators. After an hour of monitoring, you noticed that the patient was getting more tired and breathless. Peripheral oxygen saturation fell to 85% in room air.

IDENTIFICATION OF ACUTE RESPIRATORY FAILURE

The patient had acute hypoxaemic respiratory failure (also known as Type I respiratory failure), which is defined as an acute reduction of arterial oxygen partial pressure to <60 mmHg. This level of arterial oxygen is equivalent to arterial oxygen saturation (SaO_2) <90%, and is reflected by a peripheral oxygen saturation measured by pulse oximetry (SpO_2) <90%. This is a potentially life-threatening condition since the oxygen content of blood supplied may be insufficient to satisfy end-organ demand, leading to tissue hypoxia (i.e. low tissue oxygen content). A concomitant increase in arterial carbon dioxide partial pressure ($PaCO_2$) to >45 mmHg and a decrease in blood pH to <7.35 (i.e. acidaemia) may also occur, leading to acute hypercapnic respiratory failure (also known as Type II respiratory failure).

Acute respiratory distress syndrome (ARDS) is a combination of four features: (1) acute hypoxaemic respiratory failure; (2) onset of lung injury within one week of a known clinical insult or new or worsening respiratory symptoms; (3) bilateral radiographic opacities; and (4) respiratory failure not fully explained by cardiac failure or fluid overload.[1] Additionally, ARDS can only be diagnosed under conditions of positive pressure ventilation, with a minimum positive end-expiratory pressure (PEEP) of 5 cmH_2O delivered either non-invasively or invasively. Mortality becomes particularly high if ARDS occurs. Treatments for acute respiratory failure (ARF) largely apply to ARDS. To summarise the terminology, ARF is a term that encompasses acute hypoxaemic respiratory failure, acute hypercapnic respiratory failure and ARDS.

GENERAL APPROACH TO ACUTE RESPIRATORY FAILURE

Step 1: Check

The management of ARF entails a three-step cyclical loop, comprising the Check-Treat-Review steps [Figure 1]. The first step involves recognising possible ARF given an abnormal level of consciousness or an abnormal respiratory rate. Both hypoxaemia and hypercapnia can cause decreased mentation. In response to hypoxemia or hypercapnia, respiratory rate may be elevated (e.g. >20 breaths/min), while a decreased respiratory rate can lead to hypoventilation, hypoxaemia and hypercapnia. Confirming hypoxaemic respiratory failure using pulse oximetry (elaborated as Key Clinical Tool 1), ascertaining hypercapnia using arterial blood gas analysis (elaborated as Key Clinical Tool 2) and thoroughly evaluating ARF's causes must be done.

The aetiology of ARF can be divided into the six pathophysiological causes of hypoxaemia and the three causes of hypercapnia. The two common causes of hypoxaemia and hypercapnia are as follows:

1. Ventilation-perfusion mismatch (i.e. an imbalance of pulmonary blood flow and alveolar ventilation, in turn due to decreased pulmonary perfusion, heart failure, pulmonary vascular disease, parenchymal lung disease or obstructive lung disease)

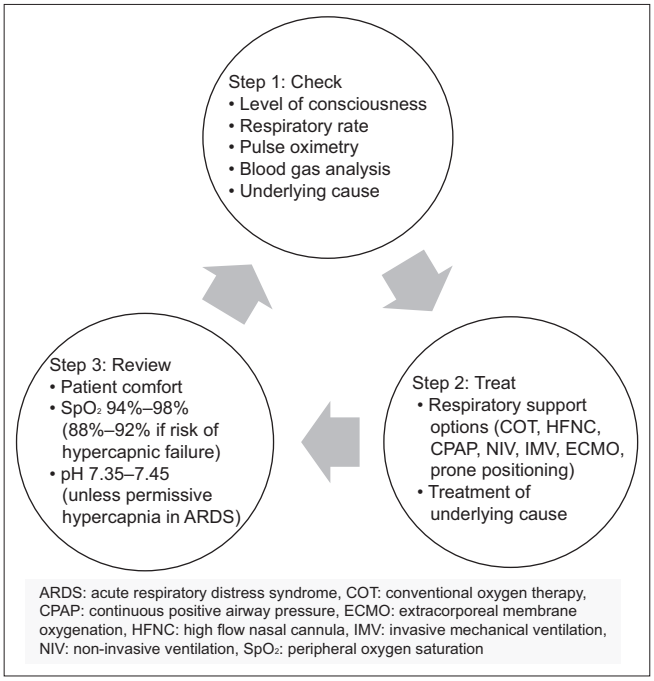

Figure 1: Chart shows the acute respiratory failure management cycle.

Table 1. Methods of respiratory support.

Method of respiratory support	Contra-indications	Initiation	Adjustment	Failure criteria
COT	Inability to protect the airway; hypercapnic respiratory failure	Oxygen flow via nasal prongs/face mask 5 L/min or Venturi mask 35%	Titrate the oxygen flow or Venturi mask to achieve the SpO_2 target.*	Severe respiratory distress e.g., respiratory rate >40 breaths per min; cardiovascular decompensation; SpO_2 <90% despite maximum settings; pH <7.35 despite maximum settings†
HFNC	Inability to protect the airway; hypercapnic respiratory failure	Flow 30 L/min; FIO_2 30%	Increase flow by 10 L/min increments and FIO_2 by 10% increments to achieve the SpO_2 target.*	
CPAP	Inability to protect the airway; hypercapnic respiratory failure; inability to tolerate mask interface	PEEP 5 cmH_2O; FIO_2 30%	Increase PEEP by 2 cmH_2O increments and FIO_2 by 10% increments to achieve the SpO_2 target.*	
NIV	Inability to protect the airway; inability to tolerate mask interface	Spontaneous/timed (S/T) mode; IPAP 10 cmH_2O; EPAP 5 cmH_2O; FIO_2 30%; backup rate 12 breaths/min	Increase EPAP by 2 cmH_2O increments and FIO_2 by 10% increments to achieve the SpO_2 target.* Increase IPAP to keep tidal volume 4-8 ml/kg PBW. Adjust respiratory rate to keep pH within the range.	
IMV	None	AC/PC or AC/VC mode; PEEP 5 cmH_2O; FIO_2 30%; respiratory rate 12 breaths/min	Increase FIO_2 by a 10% increment to achieve the SpO_2 target*. Set PEEP according to the PEEP-FIO_2 table [Table 2]. For AC/PC mode, adjust inspiratory pressure above PEEP to keep tidal volume 4-8 ml/kg PBW. For AC/VC mode, adjust tidal volume to 4-8 ml/kg PBW and inspiratory flow to maintain patient comfort. Lower tidal volume to keep plateau pressure <30 cmH_2O. Adjust the respiratory rate to keep the pH within the range.†	
ECMO	None	Use low tidal volume ventilation	Adjust ECMO blood flow, sweep gas flow and FIO_2 as required to maintain target SpO_2, $PaCO_2$ and pH.	

*Target SpO_2 should be 94%-98% for most acutely ill patients, and 88%-92% for patients at risk of hypercapnic respiratory failure. †For patients with acute respiratory distress syndrome, to facilitate low tidal volume ventilation and lung protection during IMV, $PaCO_2$ may be allowed to rise with pH falling as low as 7.15 (i.e. permissive hypercapnia). AC/PC: assist-control pressure-control, AC/VC: assist-control volume-control, COT: conventional oxygen therapy, CPAP: continuous positive airway pressure, ECMO: extracorporeal membrane oxygenation, EPAP: expiratory positive airway pressure, FIO_2: inspired oxygen fraction, HFNC: high flow nasal cannula, IMV: invasive mechanical ventilation, IPAP: inspiratory positive airway pressure, NIV: non-invasive ventilation, $PaCO_2$: partial pressure of arterial carbon dioxide, PBW: predicted body weight. For men, PBW (kg)=50+ (0.91 × [height (cm) − 152.4]). For women, PBW (kg)=45.5+ (0.91 × [height (cm) − 152.4]). PEEP: positive end-expiratory pressure, SpO_2: peripheral oxygen saturation

2. Alveolar hypoventilation (which in turn can be due to decreased central respiratory drive, neuromuscular impairment or ribcage restriction). Provided no significant atelectasis has occurred, alveolar hypoventilation can be identified by rapid improvement of oxygenation with low levels of supplemental oxygen.

The four additional causes of hypoxaemia include: (1) low inspired oxygen fraction (FIO_2); (2) low atmospheric pressure (e.g. at high altitudes); (3) diffusion limitation (e.g. interstitial lung disease, *Pneumocystis jiroveci* pneumonia); and (4) pulmonary or extra-pulmonary right-to-left shunt; in contrast to alveolar hypoventilation, right-to-left shunt is minimally improved by even high levels of supplemental oxygen.

One additional cause of hypercapnia is excessive metabolic production of carbon dioxide in the setting of fever, sepsis, thyrotoxicosis or hyperthermia.

Step 2: Treatment

The second step of the ARF management cycle involves the treatment of the underlying cause. Respiratory support options include conventional oxygen therapy, high flow nasal cannula, continuous positive airway pressure, non-invasive ventilation, invasive mechanical ventilation and extracorporeal membrane oxygenation [Tables 1 and 2].[2-4] All of these options may be supplemented by prone positioning,[5,6] which alleviates compression of dorsal lung regions and improves lung aeration and gas exchange. Pneumothorax with continued air leak is worsened by positive pressure, and should be treated with chest tube insertion before instituting either non-invasive or invasive ventilation. Respiratory support may be escalated depending on the severity of hypoxaemia, the need for positive pressure to reverse atelectasis, the need for assisted ventilation and the need for airway protection [Table 3].

Table 2. PEEP-FIO_2 table

FIO_2 (%)	PEEP (cmH_2O)
25-30	5
35-40	5-8
45-50	8-10
55-60	10
65-70	10-14
75-80	14
85-90	14-18
95-100	18-24

FIO_2: inspired oxygen fraction, PEEP: positive end-expiratory pressure

Table 3. Problem-oriented selection of respiratory support.

Physiological problem	Concurrent conditions	Options for respiratory support
Hypoxaemia	Pneumonia	COT, HFNC, NIV and IMV
	Acute respiratory distress syndrome (P/F ratio ≥150)	COT, HFNC, NIV and IMV
	Acute respiratory distress syndrome (P/F ratio <150)	HFNC and IMV
	Acute cardiogenic pulmonary oedema	COT, CPAP, NIV and IMV
	Severe asthma	COT and IMV
	Multiple organ failure	IMV
	Inability to protect the airway e.g., coma, bulbar and dysfunction	IMV
	Hemodynamic instability	IMV
	Severe refractory hypoxaemia (P/F ratio <80)	ECMO
Hypercapnia	Chronic obstructive pulmonary disease	NIV and IMV
	Severe chest wall restriction e.g., due to kyphoscoliosis or obesity	NIV and IMV
	Neuromuscular weakness without bulbar dysfunction	NIV and IMV
	Inability to protect the airway e.g., coma, bulbar and dysfunction	IMV
	Haemodynamic instability	IMV

COT: conventional oxygen therapy, CPAP: continuous positive airway pressure, ECMO: extracorporeal membrane oxygenation, HFNC: high flow nasal cannula, IMV: invasive mechanical ventilation, NIV: non-invasive ventilation, P/F ratio: ratio of arterial oxygen partial pressure to inspired oxygen fraction

In general, with positive pressure ventilation, increasing FIO_2 and PEEP correct hypoxaemia; increasing ventilation by either increasing tidal volume (via direct control of volume or via increased inspiratory pressure above PEEP) or increasing respiratory rate correct hypercapnia. For patients with ARDS, lung protection strategies comprising low tidal volume ventilation (targeting 4–8 ml/kg predicted body weight) and a plateau pressure of <30 cmH_2O are linked to improved survival.[7] To facilitate low tidal volume ventilation, $PaCO_2$ may be allowed to rise with pH falling as low as 7.15 (i.e. permissive hypercapnia).[8] To mitigate recurrent patient-ventilator asynchrony in ARDS, besides ventilator optimisation, adequate analgesia and sedation, and neuromuscular paralysis may be required.

Step 3: Review

The third step of the ARF management cycle involves review and monitoring of the patient's comfort level (e.g. presence of accessory muscle use, fatigue and diaphoresis), pulse oximetry and pH. Patients with ARF requiring high FIO_2 (>50%) or respiratory support beyond conventional oxygen therapy should be monitored in an intermediate or intensive care unit. Patient discomfort is non-specific but an important warning sign for persistent hypoxaemia, hypercapnia, inadequate respiratory support or patient-ventilator asynchrony. Target SpO_2 should be 94%–98% for most acutely ill patients, and 88%–92% for patients at risk of hypercapnic respiratory failure.[9,10]

Hyperoxaemia (over-oxygenation) can increase hypercapnia via loss of hypoxic vasoconstriction (thereby worsening ventilation-perfusion mismatch), the Haldane effect (oxygen displacing carbon dioxide from haemoglobin) and blunting of the hypoxic drive. Hyperoxaemia can also theoretically promote free radical formation and organ dysfunction, and is associated with mortality.[10] When patients are on supplemental oxygen, hyperoxaemia can be avoided by targeting SpO_2 ≤98%. Unless a patient is fully alert and comfortable, arterial blood gas analysis to check for pH and hypercapnia should be done at least once when managing a patient with ARF.

KEY CLINICAL TOOL 1: PULSE OXIMETRY

Pulse oximetry allows non-invasive and continuous monitoring of blood oxygen, and devices are widely available for hospital, clinic and home usage. Spectrophotometric probes placed over the fingers, toes, earlobes or forehead skin allow detection of pulsatile flow and calculation of the ratio between oxyhaemoglobin and total (oxygenated and deoxygenated) haemoglobin. Accurate detection of pulsatile flow can be verified by hearing the periodic beeping of the pulse oximeter or by observing the plethysmographic waveform. Further confirmation can be done by checking whether the digital heart rate derived from the detected pulsatile signal matches the heart rate measured using another method.

Oxyhaemoglobin saturation measured by pulse oximetry (SpO_2) closely matches that measured by arterial blood gas analysis (SaO_2) when SaO_2 is above 90%, though overestimation of SaO_2 by SpO_2 measurement can occur when SaO_2 is below 90%.[11] However, limitations of pulse oximetry can lead to inability to obtain readings (when the pulsatile flow is poor or absent) or to a discordance between SpO_2 and SaO_2 [Table 4].[12] Patients can look well in the presence of low SpO_2 if SpO_2 is substantially less than SaO_2.[13] Conversely, patients can look ill despite SpO_2 exceeding 90%, if SpO_2 is substantially more than SaO_2. For instance, Black patients tend to have overestimation of SpO_2, which is termed racial bias of pulse oximetry.[14] Assuming correct sensor application, if pulse oximetry readings cannot be reliably obtained or if SaO_2-SpO_2

Table 4. Limitations of pulse oximetry.

Limitations	Causes
Inability to detect SpO_2	Incorrect probe application Motion artefact (excessive motion) Nail polish Artificial nails Vasoconstriction Hypotension Hypothermia
$SpO_2 < SaO_2$	All causes for inability to detect SpO_2 Prominent venous pulsation detected by pulse oximeter (e.g. from venous congestion or arteriovenous shunting) Severe anaemia (Hb <5 g/dL) Methaemoglobin (SpO_2 trends towards 85%) Sulfhaemoglobin (SpO_2 trends towards 85%) Methylene blue
$SpO_2 > SaO_2$	Dark skin pigmentation High levels of glycated haemoglobin Carboxyhaemoglobin Methaemoglobin (SpO_2 trends towards 85%) Sulfhaemoglobin (SpO_2 trends towards 85%)

Hb: haemoglobin, SaO_2: oxygen saturation as measured by arterial blood gas, SpO_2: oxygen saturation as measured by pulse oximetry

discordance is suspected, arterial blood gas analysis would then be required.

KEY CLINICAL TOOL 2: ARTERIAL BLOOD GAS

Blood gas analysis can be performed using point-of-care devices or in central laboratories. Venous blood gas-derived values of pH and serum bicarbonate are approximately that of arterial blood gas, though venous oxygen and carbon dioxide levels deviate from arterial levels. As such, even though both arterial blood and venous blood can be analysed, only the former can be used to determine hypoxaemia and hypercapnia. After considering hypoxaemia, the acid-base status should be determined from pH, $PaCO_2$ and serum bicarbonate [Table 5].[15]

The severity of hypoxaemic respiratory failure is often determined by taking the ratio of the partial pressure of arterial oxygen (PaO_2) to FIO_2 (i.e. the P/F ratio). For instance, a patient who has a PaO_2 of 80 mmHg while receiving FIO_2 of 50% via a Venturi mask has a P/F ratio of 160. Generally, a P/F ratio 200–299 indicates mild hypoxaemic, a P/F ratio 100–199 indicates moderate hypoxaemic, while a P/F ratio of <100 indicates severe hypoxaemic. P/F ratio can guide therapy: consider the prone position in early ARDS when P/F ratio is <150,[6] and consider ECMO for severe refractory hypoxaemia when P/F ratio is <80.[16] Additionally, monitoring the P/F ratio allows tracking of worsening (decreasing P/F ratio) or improving (increasing P/F ratio) oxygenation status. A change in the P/F ratio can reflect either a change in a patient's underlying disease or the net effect of positive pressure ventilation on the cardiorespiratory system. With regards to the latter point, increasing PEEP may have opposing effects on oxygenation by reversing lung atelectasis and decreasing cardiac output in a hypovolaemic patient.

Table 5. Arterial blood gas interpretation.

pH	Primary problem	Expected compensation	Interpretation
<7.35	Hypercapnia ($PaCO_2$ >45 mmHg)	$[HCO_3]$ increases by 1 mmol/L for every 10 mmHg increase in $PaCO_2$ above 40 mmHg	Acute respiratory acidosis
<7.35	Hypercapnia ($PaCO_2$ >45 mmHg)	$[HCO_3]$ increases by 3.5 mmol/L for every 10 mmHg increase in $PaCO_2$ above 40 mmHg	Chronic respiratory acidosis
<7.35	Metabolic acidosis (HCO_3 <22 mmol/L)	$PaCO_2$ (mmHg) = 8 + (1.5 × [HCO_3 in mmol/L]) ± 2	Metabolic acidosis
>7.45	Hypocapnia ($PaCO_2$ <35 mmHg)	$[HCO_3]$ decreases by 2 mmol/L for every 10 mmHg decrease in $PaCO_2$ below 40 mmHg	Acute respiratory alkalosis
>7.45	Hypocapnia ($PaCO_2$ <35 mmHg)	$[HCO_3]$ decreases by 5 mmol/L for every 10 mmHg decrease in $PaCO_2$ below 40 mmHg	Chronic respiratory alkalosis
>7.45	Metabolic alkalosis (HCO_3 >26 mmol/L)	$PaCO_2$ (mmHg) = 21 + (0.7 × [HCO_3 in mmol/L]) ± 2	Metabolic alkalosis

Note: If more than one acid-base disorder is present, the observed compensation would deviate from the expected compensation. HCO_3: serum bicarbonate, $PaCO_2$: partial pressure of arterial carbon dioxide

TAKE HOME MESSAGES

- Acute hypoxaemic respiratory failure is an acute reduction of arterial oxygen partial pressure to <60 mmHg (equivalent to an arterial oxygen saturation <90%, and reflected by SpO_2 <90%. It may be accompanied by an increase in $PaCO_2$ to >45 mmHg and a decrease in blood pH to <7.35 (i.e. acidaemia), leading to acute hypercapnic respiratory failure.
- The management of ARF entails a three-step cyclical loop. The first step (Check) involves recognising possible ARF given an abnormal level of consciousness or an abnormal respiratory rate. Confirming hypoxaemic respiratory failure using pulse oximetry, ascertaining hypercapnia using arterial blood gas analysis and thoroughly evaluating ARF's causes must be done.
- The second step (Treatment) of the ARF management cycle involves treatment of the underlying cause. Respiratory support options include conventional oxygen therapy, high flow nasal cannula, continuous positive airway pressure, non-invasive ventilation, invasive mechanical ventilation and extracorporeal membrane oxygenation. All of these options may be supplemented by prone positioning.
- The third step (Review) involves review and monitoring of the patient's comfort level, pulse oximetry and pH. Patients

with ARF requiring high FIO_2 (>50%) or respiratory support beyond conventional oxygen therapy should be monitored in an intermediate or intensive care unit.

Closing Vignette

You recognised that the patient had acute hypoxaemic respiratory failure secondary to multi-lobar pneumonia. Arterial blood gas analysis demonstrated a normal pH and $PaCO_2$. You provided oxygen via a Venturi mask, adjusting the FIO_2 to 35% to keep SpO_2 between 94% and 98%. One hour later, the patient became more tachypneic, desaturated further, and required an FIO_2 of 50%. You transferred the patient to the intensive care unit for high flow nasal cannula, starting at an FIO_2 of 50% and a flow rate of 50 L/min, and achieving an SpO_2 of 94%. Fortunately, over the next 5 days, he improved on high flow oxygen, did not require intubation and was transferred to the general floor ward for rehabilitation.

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Conflicts of interest

See KC is the handling editor of the PACC series.

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See: Acute respiratory failure

SMC CATEGORY 3B CME PROGRAMME

Online Quiz: https://www.sma.org.sg/cme-programme

Deadline for submission: 6 pm, 03 February 2023

Question	True	False
1. An arterial oxygen partial pressure of 50 mmHg indicates hypoxaemic respiratory failure.		
2. An arterial carbon dioxide partial pressure of 30 mmHg indicates hypercapnia.		
3. Acute respiratory distress syndrome can be present in a patient with both pneumonia and cardiac failure.		
4. Acute respiratory distress syndrome can be diagnosed in a patient who is receiving oxygen via high flow nasal cannula.		
5. Acute respiratory failure requires arterial blood gas analysis for confirmation.		
6. Acute hypercapnic respiratory failure requires arterial blood gas analysis for confirmation.		
7. Ventilation-perfusion mismatch can cause hypoxaemia.		
8. Ventilation-perfusion mismatch can cause hypercapnia.		
9. Hypoxaemia caused by alveolar hypoventilation is generally difficult to correct with supplemental oxygen.		
10. Hypoxaemia caused by right-to-left shunt is generally difficult to correct with supplemental oxygen.		
11. Non-invasive ventilation is recommended for acute respiratory failure due to severe asthma.		
12. Prone positioning can be used with conventional oxygen therapy.		
13. Prone positioning can be used with continuous positive airway pressure.		
14. For patients with acute respiratory distress syndrome, the target tidal volume should be 8-10 ml/kg predicted body weight.		
15. For patients with acute respiratory distress syndrome, arterial carbon dioxide partial pressure may be allowed to rise with pH falling as low as 7.15.		
16. The target peripheral oxygen saturation should be 88%-92% for most acutely ill patients.		
17. Hyperoxaemia can worsen hypercapnia predominantly by blunting the hypoxic drive.		
18. Black skin pigmentation generally leads to an under-estimation of the arterial oxygen saturation from the measured peripheral oxygen saturation.		
19. Venous blood gas-derived values of pH and serum bicarbonate are approximately that of arterial blood gas.		
20. Ratio of arterial oxygen partial pressure to inspired oxygen fraction (P/F ratio) can change without a change in a patient's underlying disease.		