Medical High Dependency Unit series, Article 3: Respiratory Support in the MHDU

CJ Wright, RM Allan & SA Gillon

Abstract

Acute respiratory failure is a life threatening condition encountered by Acute Physicians; additional non-invasive support can be provided within the medical high dependency unit (MHDU). Acute Physicians should strive to be experts in the investigation, management and support of patients with acute severe respiratory failure. This article outlines key management principles in these areas and explores common pitfalls.

Keywords

Severe respiratory failure, oxygen targets, high flow nasal cannula, non-invasive ventilation

Key points

- When managing the patient with respiratory failure consideration must be given to the underlying diagnosis to allow targeted management to be delivered
- High flow nasal cannula appear to have an outcome benefit in the management of hypoxaemic respiratory failure
- Non-invasive ventilation (NIV) is a standard of care for hypercapnoeic acute exacerbations of chronic obstructive pulmonary disease (AECOPD), it also has a role in the management of hypercapnoea in obesity, neuromuscular disease, and chest wall disease
- Besides acute cardiogenic pulmonary oedema (CPO), there is little evidence to support the use of continuous positive airways pressure (CPAP) devices in medical patients acutely
- A conservative fluid strategy should be adopted in patients with moderate to severe respiratory failure in the absence of shock. In shocked patients, conservative fluid management should be considered after the initial resuscitative phase
- Hyperoxia is associated with adverse outcomes and, in general, should be avoided

Introduction

Patients with acute or acutely decompensated respiratory failure account for a significant proportion of admissions to the Medical High Dependency Unit (MHDU). Many patients will have reversible pathology, although it should be noted that acute respiratory failure does carry a significant burden of both mortality and morbidity.[1] Some patients with moderate/severe or complex disease may require support beyond that available in the medical ward and may require management in the MHDU. In addition, patients with tracheostomy tubes are at high risk of device related and iatrogenic harm and as such require management in a specialist area – in many hospitals this will be within the MHDU. Respiratory monitoring and support typically available in the MHDU is listed in Box 1.

Box 1. Respiratory monitoring and support in MHDU

- Continuous oxygen saturation monitoring
- Arterial line monitoring – allows frequent drawing and analysis of arterial blood gases
- Enhanced nursing ratios – both for multi system monitoring and facilitating regular supportive respiratory care e.g. suction to aid clearance of respiratory secretions, position changes
- High flow nasal cannula – capable of delivering gas flows of up to 60 litres/min and fraction of inspired oxygen (FiO2) approaching 1.0
- Non-invasive ventilation
- Continuous positive airways pressure
- Tracheostomy care and management

This review article addresses several key areas in the management of respiratory failure in the MHDU, including pathophysiology, definitions, approach to respiratory failure of unclear aetiology, high flow nasal cannula, non-invasive ventilation, continuous positive airways pressure, fluid balance in respiratory failure and steroids. Tracheostomy management will be discussed in the next article in the series.

Oxygenation and Hypoxaemia

Respiratory failure is defined by the presence of hypoxaemia (low arterial oxygen tension) with or without hypercapnoea.[2] Hypoxaemia results from pathology in any step(s) of the process of transfer of atmospheric oxygen to the left side of the heart. The tension of oxygen decreases sequentially at every step as it transfers from the atmosphere to the left side of the heart (Box 2). This process is known as the "oxygen

Christopher Wright biography
Chris Wright is a consultant physician in critical care who completed acute medicine training in the West of Scotland and intensive care medicine training in London. His interests include MHDU, severe respiratory failure, ECMO, and global intensive care.

Russell Allan biography
Russell Allan is a consultant physician in critical care who completed both acute medicine and intensive care medicine training in the West of Scotland. His interests include MHDU, quality improvement, and simulation.

Stuart Gillon biography
Stuart Gillon is a consultant in critical care who has trained in Glasgow, London and Melbourne. His interests are critical care echocardiography and ECMO.

Christopher J Wright
MBChB, MRCP, FFICM

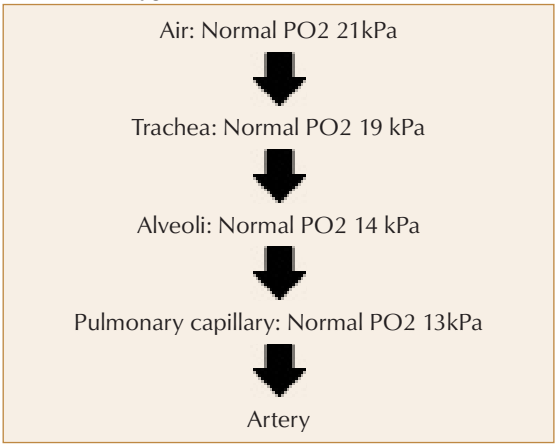
Russell M Allan
MBChB, MRCP, FFICM

Stuart A Gillon
MBChB, MRCP, FFICM

Consultants in Critical Care, Queen Elizabeth University Hospital, Glasgow

Correspondence
Christopher Wright, Department of Critical Care, 1st Floor, Queen Elizabeth University Hospital, 1345 Govan Road, Glasgow, G51 4TF
Email: christopherwright3@nhs.net

Box 2. The oxygen cascade



cascade" and it provides a systematic framework for determination of pathology underpinning respiratory failure. The oxygen cascade should be considered in conjunction with the mechanisms of hypoxaemia and these are described in Box 3.

Ventilation and hypercapnoea

Ventilation is the movement of gas in and out of the lungs facilitating gas exchange and subsequent oxygenation and clearance of CO_2. Tidal volume (Vt) describes the volume of gas inhaled/exhaled in a normal breath. Minute ventilation (MV) is the product of Vt x Frequency (Breaths/minute). CO_2 is significantly more soluble than O_2, and $PaCO_2$ is directly related to the MV. In normal physiology central chemoreceptors in the medulla detect changes in pH related to $PaCO_2$ and adjust MV accordingly. In states of significant hypoxaemia PaO_2 may also influence MV. Not all of the Vt is involved in gas exchange – the portion where no gas exchange occurs is known as dead space. The conducting airways are described as the anatomical dead space. Alveolar dead space describes alveoli that are ventilated but not perfused – in normal physiology this is minimal, however it may become clinically significant in situations such as pulmonary embolism or low cardiac output states. Physiological dead space describes both anatomical and alveolar dead space.[2]

In many cases of respiratory failure, rapid shallow breathing occurs with a resultant decrease in inspiratory time, this results in lower Vt and tachypnoea with subsequent increased load on the respiratory muscles – as muscle fatigue occurs the MV falls and hypercapnoea and acidosis occurs.[4]

Oxygen administration associated hypercapnoea and the hypoxic drive

It is widely believed that in some patients with chronic hypoxaemia the primary determinant of MV switches from $PaCO_2$ to PaO_2 – this is known as the hypoxic drive. It is believed that oxygen administration resulting in higher PaO_2 may decrease this drive and subsequently cause hypercapnoea.[2] While this may be the case in some patients, it is more likely that there are other mechanisms at play in oxygen administration associated hypercapnoea.

Box 3. Mechanisms of hypoxaemia[2,3]

Global alveolar hypoventilation

- can occur in a variety of conditions including COPD, obesity hypoventilation, neuromuscular hypoventilation, drug toxicity eg opiates.

V/Q mismatch and shunt

- V/Q varies throughout the lung in normal physiology, however, overall it is 1 and almost all blood entering the left side of the heart is oxygenated. Reduction in ventilation of a given perfused lung unit results in a reduction in V/Q – blood passing through this unit "shunts" gas exchange and reaches the left side of the heart poorly oxygenated. At low shunt fractions (<30%) administration of supplemental oxygen results in an improvement in arterial oxygen tension. In "true shunt" blood passes though the lungs with no gas exchange, this may be caused by intrapulmonary causes (eg dense consolidation, ARDS, pleural effusion, pneumothorax) or intracardiac causes (eg septal defect resulting in right to left intracardiac flow of blood). "True shunt" is not correctable with administration of supplemental oxygen.

Diffusion problems

- can occur due to increased thickness of alveolar membrane (eg pulmonary oedema, pulmonary fibrosis), reduced alveolar surface area (destructive processes such as emphysema), reduction in capillary transit time (hyperdynamic states), or reduced pulmonary capillary blood volume (hypovolaemia).

Reduced fraction of inspired oxygen

- clinically relevant examples include administration of hypoxic gas mixtures (eg in failure of the medical oxygen supply or erroneous connection to medical air) and changes in atmospheric pressure at altitude (eg in aeromedical transfers)

Definitions and severity of respiratory failure

Type 1 respiratory failure describes hypoxaemia with PaO2 <8 kPa, Type 2 respiratory failure describes hypercapnoea with PaCO2 >6.0 kPa.[3] Beyond describing the presence or absence of hypercapnoea, this classification has little clinical value as most causes of respiratory failure can lead to both Type 1 and Type

2 respiratory failure. It also takes no account of the oxygen concentration of the inspired gas.

The PaO2:FiO2 (PF) ratio quantifies the severity of hypoxaemic respiratory failure in critically ill patients. It is a component of the widely used Berlin definition of the Acute Respiratory Distress Syndrome (ARDS).[1] The definition of ARDS is a PF <40kPa in the presence of positive end expiratory pressure (PEEP) of 5cmH20, which develops within 7 days of onset of symptoms/insult, is associated with bilateral infiltrates on chest imaging, and isn't fully explained by cardiogenic pulmonary oedema or fluid overload.[1] It is subdivided into mild (PF 26.6-40), moderate (PF 13.3-26.6), and severe (PF<13.3).[1] The moderate to severe categories apply to invasively ventilated patients only, however it is argued that the advent of high flow nasal cannula (HFNC) opens the definition to non-invasively ventilated patients.[5] In the ARDS population mortality increases as the PF ratio decreases.[1]

Diagnostic pitfalls and respiratory failure of uncertain aetiology

The underlying cause of respiratory failure is often clearly apparent, however, in some patients the aetiology will be unclear. In clinical practice these patients are often labelled as having ARDS – however, it is important to remember that ARDS is a clinical syndrome and requires an underlying aetiology to be sought in order to direct specific management. Diagnostic pitfall also lies with the patient with bilateral consolidative changes on plain chest x ray failing to improve with several days of antibiotic therapy and with no positive microbiology – this should serve as a cognitive stop point to explore alternative diagnoses.

Given the potential pitfalls in diagnosis, some critical care experts advocate a standardised investigatory approach to patients with severe respiratory failure in the ICU including wide ranging infective and autoimmune screens, detailed imaging, and echocardiography.[6] While this would not be appropriate in all patients with respiratory failure in the MHDU, we would certainly consider such an approach in patients with unclear aetiology, on high levels of respiratory support in the MHDU, particularly if not responding to initial treatment. One such standardised approach is described in Box 4.

Box 4. Standardised approach to patient with severe respiratory failure of unclear aetiology[6]

Standard laboratory investigations
- Severe community acquired pneumonia screen: sputum for micro, gargle swab for viral PCR, consider testing for PCP, urine for legionella and pneumococcal antigen
- Viral blood testing: HIV, CMV, HSV
- Autoimmune and vasculitis screen: ANCA (MPO/PR3), RhF, ANA, complement, anti-GBM, AMA, immunoglobulins, anti-dsDNA
- Urine protein:creatinine ratio

Imaging*
- CT chest and CTPA
- Consider CT of Abdo/Pelvis also in case of intra-abdominal pathology driving respiratory failure
- Echo

Bronchoalveolar lavage*
- Bacterial and fungal cultures
- Respiratory standard viral PCR, PCP, CMV/HSV
- TB screening and culture

* Imaging and lavage may not always be necessary and benefits need to be weighed against risks, which include risk of transportation for CT scanning.

Oxygenation targets in MHDU

The British Thoracic Society (BTS) guidance on the administration of oxygen mandates the use of targeted oxygen saturation (SaO_2) ranges against which the administration of oxygen can be titrated.[3] The BTS guidance warns against hyperoxia and recommends an SaO_2 target of 94-98% in the general acute hospital population, with an amended target of 88-92% in patients with COPD and those with risk factors for hypercapnoea (e.g. morbid obesity, neuromuscular disease, cystic fibrosis, bronchiectasis with fixed air flow obstruction, and chest wall disease).

The critical care literature supports the BTS guidance, in particular the avoidance of hyperoxia. Hyperoxia in the first 24 hours after ICU admission is associated with excess mortality,[7] and is associated with excess morality in the post cardiac arrest population.[8] Furthermore, the administration of supplemental oxygen to patients suffering from an acute myocardial infarction in the absence of significant hypoxaemia has recently been shown to lack benefit in a large high quality multicentre randomised controlled trial (RCT).[9]

The recent Italian single centre RCT OXYGEN-ICU study[10] compared a conservative SaO2 target of 94-98% against a liberal hyperoxic target of 97-100% in adult patients admitted to the ICU with projected length of stay of >72 hours. The study reported a significant mortality reduction in the conservative group, however recruitment ended early due to an earthquake in the region and it was subsequently statistically underpowered. The multi-centre RCT ICU-ROX is currently underway in Australia and New Zealand and this will hopefully provide high quality evidence regarding the use of conservative versus liberal oxygen therapy in critically ill patients.[11]

Modalities of advanced non-invasive respiratory support frequently available in MHDU

The MHDU can provide advanced non-invasive respiratory support for patients with reversible pathology failing on standard management. Common modalities include high flow nasal cannula (HFNC), non-invasive ventilation (NIV), and continuous positive airways pressure (CPAP).

High flow nasal cannula

Delivery of oxygen via high flow nasal cannula (HFNC) has been increasingly utilised over the last decade. This supportive therapy is known by a number of different names – HFNC, high flow nasal oxygen, and heated and humidified HFNC.[5] It provides oxygen at flow rates of up to 60 litres/min and a FiO2 of approaching 1.0 via wide bore nasal cannula using a heated and humidified circuit. The primary role of HFNC in the MHDU is to provide support to patients with acute hypoxaemic respiratory failure who are failing to respond to conventional oxygen delivery systems. It has a number of theoretical benefits over conventional therapy, which are described in Box 5.

Despite the exponential rise and increasing volume of experience in the use of HFNC in patients with moderate to severe respiratory failure, high quality evidence directing and supporting its provision is limited.

The FLORALI study[12] is the largest high quality multicentre RCT of the use of HFNC in acute hypoxaemic respiratory failure. FLORALI compared standard oxygen therapy, non-invasive ventilation, and HFNC in the management of patients with hypoxaemic respiratory failure. The majority of patients recruited had community acquired pneumonia as their primary diagnosis and there was a significant reduction in mortality in the HFNC group at 90 days. Subsequent systematic reviews[13,14] have failed to show reduction in requirement for invasive ventilation nor outcome benefit with HFNC use but have shown that HFNC are well tolerated and viewed as comfortable by patients.[14]

There is some evidence that patients who require intubation after >48 hours of HFNC therapy have worse ICU outcomes when compared to those who require intubation within 48 hours of initiation of HFNC.[15] Prediction of deterioration on HFNC can be difficult – in our experience patients on high flows and high FiO2 can appear relatively well compensated while using HFNC, this can mask impending, precipitous deterioration. In light of this, we would urge caution in the use of HFNC with high FiO_2 outside of the critical care setting in patients who remain for active treatment and escalation. Some markers of likelihood of deterioration on HFNC include deteriorating PF ratio despite HFNC use and the co-existence of additional non-respiratory organ failures.[5]

Box 5. Theoretical benefits of heated and humidified HFNC[5]

Heat/humidification
- Enhances mucociliary function
- Protects against dried secretions
- Ameliorates tracheal/large airway inflammation in post extubation patients

High flow rates
- Better matched to peak inspiratory flow rates of the hypoxaemic patient
- CO2 clearance via washout of deadspace
- Provides a degree of positive end expiratory pressure (PEEP).

Comfort
- Well tolerated when compared with NIV/CPAP
- Able to eat and communicate while using HFNC

From a holistic perspective, the development of HFNC has had a number of advantages, primarily the maintenance of communication and oral nutrition that the device facilitates. Additionally, HFNC is a useful ceiling of active respiratory support in some patients in whom invasive ventilation within the ICU is deemed not appropriate.

When initiating therapy with HFNC, we currently favour the FLORALI approach, with a flow rate of 50l/min and FiO2 of 1.0, adjusted to maintain SaO2 >92%. The HFNC dose finding trial FRONTIERS[16] will hopefully provide comprehensive guidance to the initiation and titration of HFNC. Weaning of HFNC is again an area lacking in high quality evidence. At present we favour weaning FiO2 first against set SaO2 target; when the FiO2 has weaned to <= 0.4 then flow rate should be weaned; when flow is at <=30l/min we would advocate a trial of liberation from HFNC onto a standard oxygen delivery system.

Non-invasive ventilation (NIV)

NIV equipment consists of an interface (face mask, nasal mask, or hood) with expiratory outlet, tubing (usually single limb), ventilator, oxygen supply (either supplemental via interface or blended by ventilator). NIV is also known by multiple other, largely proprietary, acronyms – these include BiPAP and NIPPY. The ventilator works by delivering both patient triggered assisted breaths and mandatory back up breaths, this subsequently increases Vt and MV and reduces respiratory muscle work load and respiratory distress with subsequent improvements in gas exchange and metabolic status. The inspiratory pressure delivered by the ventilator is known as

Box 6. BTS NIV Guidance: Indications, contraindications, adverse factors (note gas parameters in kPa)[20]

Indications

COPD after initial medical therapy
- pH<7.35, pCO2>6.5, RR>23

Neuromuscular disease with resp illness
- pH<7.35, pCO2>6.5
- RR>20 if usual vital capacity (CV) < 1 litre even if pCO2<6.5

Obesity
- pH<7.35, pCO2>6.5, RR>23
- Somnolence with daytime pCO2>6.0

Contraindications

Absolute
- Fixed upper airway obstruction, severe facial deformity/facial burns

Relative/Adverse features (if using NIV in presence of these should be within critical care area)
- pH<7.15, GCS<8, confusion/agitation, cognitive impairment, requirement for sedation, pH<7.25 with additional adverse factor

Indications for ICU referral
- Peri-arrest
- Failure tolerate NIV or need for IV sedation
- Failure to improve pCO2
- Unable to maintain SaO2 85-88% on NIV
- Predicted difficult airway

Box 7. BTS NIV Guidance: Settings and initiation[20]

COPD
- Pressure settings: 15/3 initially, titrated over 30 mins to maximum of 30/8, titrate against clinical response including RR and chest wall movement
- Back up rate: 16-20
- I:E ratio: 1:2 to 1:3
- Inspiratory time: 0.8-1.2

Obesity
- Pressure settings: as per COPD guidance initially, may need higher maximum pressures – only after expert review
- Back up rate: 16-20
- I:E ratio: 1:1
- Inspiratory time: 1.2-1.5

Chest wall disease
- Pressure settings: as per COPD guidance
- Back up rate: 16-20
- I:E ratio: 1:1
- Inspiratory time: 1.2-1.5

Neuromuscular disease
- Pressure settings: initially 10/3 or IPAP 5 above usual setting if on home NIV
- Back up rate: 16-20
- I:E ratio: 1:1
- Inspiratory time: 1.2-1.5

inspiratory positive airway pressure (IPAP) or pressure support (PS), while the expiratory pressure is known as expiratory positive airway pressure (EPAP).

The use of NIV for hypercapnoeic respiratory failure secondary to acute exacerbation of COPD (AECOPD) failing to respond to initial medical management is now widespread. This follows major landmark trials in the 1990s which showed a reduction in mortality with the use of NIV when compared to standard management.[17,18] Although some institutions provide all NIV within a critical care area, there is evidence to support its use in specialist areas outside of critical care for AECOPD as long as appropriate nurse training and monitoring is in place.[19]

There are also a number of additional conditions beyond AECOPD in which the use of NIV is now recommended for the management of hypercapnoeic exacerbations.[20] These include obesity hypoventilation syndromes (OHS), neuromuscular disease (NMD), and chest wall disease (CWD). There is some evidence that NIV provides a more rapid improvement in respiratory distress and metabolic disturbance in patients with acute cardiogenic pulmonary oedema (CPO) when compared to conventional therapy, however its effect on mortality is less clear.[21] NIV has previously been advocated for use in moderate to severe respiratory failure in immunocompromised patients.[22] However recent evidence suggests that it does not improve outcomes[23] and may actually increase mortality when compared to standard O2 therapy and HFNC[24] – this may be due to the emergence of HFNC. The use of NIV in patients with hypercapnoea due to a primary diagnosis of asthma or pneumonia is not currently supported – indeed the presence of a respiratory acidosis in such patients should prompt ICU referral.[20]

The BTS have published extensive guidance on the use of NIV in acute hypercapnoeic respiratory failure.[20] Facemask is the preferred interface, and pressure support/controlled ventilation is recommended over volume assist/controlled modes. Conventional starting settings have been IPAP of 12cmH20 and EPAP of 4cmH20 titrated to a maximal IPAP of 20, however, current guidance suggests that those IPAP settings are well below those often required.[20] Patient selection and subsequent monitoring is key as there is some evidence that patients who fail on NIV and require invasive mechanical ventilation have an excess mortality when compared to a matched control population.[25] The BTS guidance for patient selection and treatment settings is summarised in Boxes 6 and 7.

Box 8: Strategies to consider if failing to improve on NIV

For persisting hypercapnoea with acidosis
- Can FiO2 be reduced?
- Is MV adequate?
  - ◊ Ensure asynchrony adequately addressed (as above)
  - ◊ Can tidal volume be increased via increasing delta pressure (IPAP – EPAP) by increasing IPAP further (beware this can reduce tolerance, cause non-voluntary glottis narrowing or increase leak)
  - ◊ Is the patient's spontaneous respiratory rate high enough?
  - ◊ Minimise mask leak
  - ◊ Is re-breathing of exhaled gas occurring (ensure expiratory valve adequately functioning – this may be aided by increased EPAP)

For hypoxaemia
- Increase FiO2
- Lung recruitment via increasing EPAP

Failure to improve on NIV or patient discomfort on NIV should prompt an overall review of the patient's condition and a search for any technical factors which may be having an adverse effect. Patient-ventilator asynchrony is a common cause of patient discomfort and failure to improve while on NIV – findings may include failure to deliver a breath despite patient effort, delivery of a breath with no patient effort, or continued delivery of ventilator breath while the patient is exhaling. In these circumstances it is important to ensure correct mask fit (without excess air leak), and appropriate ventilator trigger.[20] Continued delivery of a ventilator breath during patient exhalation may be due to an excessively long inspiratory time or high IPAP on the ventilator – these parameters should be reviewed and reduced if necessary.[20] Following the identification and resolution of any asynchrony, if improvement in gas exchange is not present or adequate then further review of therapy should be initiated, this is illustrated in Box 8.

Haemodynamic instability can occur during NIV therapy manifested mainly by hypotension. This is often due to decreased venous return in response to the increase in intrathoracic pressure associated with the application of positive pressure ventilation; it usually resolves with a fluid bolus. Hypotension, particularly during high pressure NIV, should also prompt examination to look for features of pneumothorax.

NIV should be used for most of the first 24 hours of therapy as tolerated; breaks for meals and communication are encouraged. Over the next 48-72 hours breaks off NIV should be extended during the day with overnight NIV continued, once NIV has been off for a full day it may be advisable to give a final night of support prior to a trial of full liberation from the ventilator.[20]

Patients should have escalation decisions discussed (with patient if possible) and documented early on during their NIV treatment.[20] Patients failing to improve despite NIV and maximal medical management should be referred to ICU for consideration of invasive mechanical ventilation if deemed appropriate for consideration of escalation. There has been a degree of therapeutic nihilism with regard to the provision of invasive ventilation for patients with AECOPD, however, a large UK prospective cohort study has brought this approach into question.[26] The study revealed that 62% of patients admitted to ICU with AECOPD survived to 180 days. Of the survivors who responded to a questionnaire, 73% stated that their overall quality of life was similar or better than it was pre-admission, and 96% stated they would undergo the same treatment again.[26]

It should be noted that application of NIV for some patients can be an uncomfortable and unpleasant experience, and in those who fail to improve and are on a trajectory towards death and in whom escalation to ICU is deemed not appropriate, its discontinuation is appropriate to allow the focus to shift to palliation.

Continuous positive airways pressure (CPAP)

CPAP delivers continuous application of positive airways pressure usually in the range of 5-15cmH20, it is usually used in combination with supplemental oxygen. Commonly used interfaces include tight fitting facemask or hood. Most ICU and NIV ventilators have a CPAP mode, it can also be delivered using a specialised circuit containing a Bousiggnac valve, tight fitting mask, oxygen tubing, and a high flow oxygen flow meter. CPAP has some potential respiratory and haemodynamic advantages when compared to standard oxygen therapy. From a respiratory perspective CPAP may decrease respiratory muscle work load and splint open and recruit previously de-recruited alveoli, from a cardiovascular perspective CPAP decreases pre-load via an increase in intrathoracic pressure and decreases after load via a decrease in transmural pressure.[27]

CPAP may improve physiological parameters in patients with acute cardiogenic pulmonary oedema,[27] however effect on overall outcome is unclear.[21,28] There is no evidence to support the use of CPAP as a measure of intubation avoidance in medical patients with acute hypoxaemic respiratory failure out with the setting of cardiogenic pulmonary oedema, in fact it may be associated with an increased rate of adverse events.[29]

In light of the above evidence and the emergence of HFNC, we have observed a decline in the use of traditional face mask CPAP within the MHDU setting.

Fluid balance in acute respiratory failure

Patients with multi-system critical illness often require volume resuscitation, particularly within the first 24 hours of their presentation. However, tissue fluid oedema can develop rapidly in the context of capillary leak and can be deleterious in the critically ill patient.[30] There is some evidence that a persistently positive fluid balance may result in adverse outcomes.

A large multicentre RCT performed by the ARDSNET clinical trials group compared a liberal fluid strategy to a conservative strategy in mechanically ventilated patients with ARDS.[31] The mean fluid balance at 7 days in the liberal group was positive 6992mls compared to negative 136mls in the conservative group. There was no difference in 60 day mortality, however, the conservative group had a shorter duration of both ICU stay and mechanical ventilation. There was no increase in incidence of shock or requirement for renal replacement therapy in the conservative group.

In the absence of shock and pre-renal AKI we advocate the use of a conservative fluid strategy in patients with moderate to severe respiratory failure in the MHDU. In those patients presenting with both shock and respiratory failure, we would advocate consideration of a conservative fluid strategy after the first 24 hours.

Steroids in acute respiratory failure

While steroids are a standard of care in many acute respiratory conditions (eg AECOPD, acute asthma exacerbations, pulmonary vasculitis, cryptogenic organising pneumonia, some cases of interstitial lung disease), there are some conditions in which their use is more controversial.

RCTs looking at steroids in the setting of mechanically ventilated patients with ARDS have produced mixed results.[32,33] There is not enough evidence to support their routine use in the MHDU population with severe respiratory failure at present, although failure to improve with initial therapy should prompt a review of the diagnosis and consideration of a potentially steroid responsive condition.

Steroids have also been proposed as an adjunctive treatment in the management of severe community acquired pneumonia (CAP). A European multicentre RCT compared methylprednisolone 0.5mg/kg twice daily for 5 days to placebo in patients with severe CAP, there was no mortality reduction, however, steroids were associated with a reduction in treatment failure.[34] While this is not enough to mandate the widespread use of steroids in severe CAP, they should be considered in patients presenting with particularly severe disease on significant amounts of non-invasive respiratory support.

Conclusion

The assessment and management of the patient with acute respiratory failure is complex. An accurate diagnosis is necessary to direct specific patient centred management. Failure to improve with initial therapy should prompt a review of the diagnosis, a standardised approach may be considered in such cases and where initial diagnosis is unclear.

Supportive therapies such as HFNC and NIV allow time for patient specific management to work, however, patient selection and regular review and monitoring is key to avoid iatrogenic harms and subsequent adverse patient outcomes. CPAP should be avoided other than in cases of acute cardiogenic pulmonary oedema failing to respond to initial therapy. Hyperoxia should be avoided, and therapy should be titrated to set oxygenation targets. A conservative fluid strategy should be considered.

"Escalation may not be appropriate for some patients - in this group if it is clear that the patient is dying, cessation of non-invasive advanced respiratory support should be discussed with a view to providing palliation."

Above all, it should be remembered that advanced non-invasive respiratory therapies are supportive, and without appropriate diagnosis and targeted management they are of limited or no benefit.

Declaration of competing interests

Nothing to declare.

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