# Respiratory failure and non-invasive respiratory support during the COVID-19 pandemic: an update for re-deployed hospital doctors and Primary Care Physicians

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The case in this article is fictitious and therefore no consent was needed.

Box 1: What you need to know

- The majority of patients who become seriously unwell with COVID-19 do so primarily due to acute type 1 respiratory failure and benefit from early recognition of respiratory deterioration and appropriate escalation in respiratory support
- Respiratory deterioration can be identified by an increasing respiratory rate, an inability to talk in full sentences, the use of accessory muscles of breathing, decreasing oxygen levels and an increased requirement for supplementary oxygen.
- All patients with COVID-19 admitted to hospital and many of those in the community should have a ceiling of care established at the earliest opportunity.

Box 2: Education into practice

- What is the difference between T1RF and T2RF and how does this influence decisions regarding respiratory support?
- Do you know the difference between CPAP, EPAP, PEEP and IPAP?
- Would you feel comfortable explaining CPAP or non-invasive ventilation (NIV) to a patient/family member?

Box 3: How patients were involved in the creation of this article

A patient recovering from COVID-19 who was treated with different forms of non-invasive respiratory support during his admission to hospital was interviewed about the devices used, their application by staff and his experience of them.

Box 4: Sources and selection criteria

This article is based upon personal practice, clinical experience, papers available via PubMed and National guidelines and statements.

## Introduction

The first half of 2020 has seen a tremendous re-organisation of healthcare services in response to COVID-19, including staff re-deployment to acute specialities.[1] Continued preparedness for potential future re-deployment remains essential given the risk of further spikes in COVID-19 prevalence as society attempts to return to some form of normality.

The majority of people who become seriously unwell with COVID-19 have an acute respiratory illness,[2,3,4] of which approximately 14%[2] will require non-invasive

respiratory support. In addition to shifting into acute care settings in the short-term, primary care clinicians will also be caring for patients (or their loved ones) recovering from potentially traumatic experiences of respiratory illness. This article updates primary care and non-respiratory/intensivist specialist hospital doctors on the recognition and management of acute respiratory failure and will also support GPs in the subsequent outpatient care of patients during their recovery.

## Defining and classifying respiratory failure

The key purpose of the respiratory system is to move oxygen from the external environment into the bloodstream, and to remove the carbon dioxide generated by metabolism from the body. This requires adequate *ventilation* (i.e. the movement of gas into and out of the lungs) and *gas exchange* (i.e. the movement of oxygen and carbon dioxide between the lung and the blood). Respiratory failure refers to the situation in which one or both of these processes fails. This may lead to low oxygen levels (hypoxia) with or without high carbon dioxide levels (hypercapnia). Both hypoxia and hypercapnia act as stimuli to breathe, with hypercapnia the more powerful in most situations. They lead to breathlessness that manifests in patients as a variety of symptoms, including air hunger, chest tightness or an inability to breathe deeply enough (Box 5)

**Type 1 respiratory failure** (T1RF) is primarily a problem of gas exchange resulting in hypoxia *without* hypercapnia. **Type 2 respiratory failure** (T2RF), in contrast, occurs when there is reduced movement of air in and out of the lungs (hypoventilation) with or without interrupted gas transfer leading to hypercapnia and associated secondary hypoxia (Table 1). Important to understanding the differences between T1RF and T2RF is to remember that oxygen levels are very sensitive to problems with either gas exchange or ventilation. In contrast, owing to its much greater solubility in blood than oxygen, carbon dioxide levels are not as severely affected by problems with gas exchange, but are equally sensitive to problems with ventilation.

Coronavirus disease 2019 (COVID-19) is caused by a novel coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The virus enters the body via the respiratory tract, and in severe cases causes widespread inflammation in the lungs. Approximately 14% of all people with COVID-19 develop severe respiratory disease with T1RF and a further 5% will become critically unwell,[2] many of whom will require treatment in intensive care.[5-7]

In this article, we focus on patients with COVID-19 who do not require intubation and invasive ventilation, but require non-invasive respiratory support, usually due to severe hypoxaemia that is refractory to simple oxygen therapy. In our experience, which is in keeping with reports from other authors [8,9], the clinical picture in this group of patients is unusual. For example, despite extensive inflammatory changes in the lungs, which may be evident radiologically, *ventilation* appears to be less severely impaired than *gas exchange*. Indeed, patients often have no difficulty moving large volumes of gas into and of the lungs, hence the lack of hypercapnia/T2RF in most

patients and the observation that dyspnoea may be absent or disproportionately mild, relative to the severity of disease.[4,9]

The pathophysiology remains uncertain at the time of writing, and the clinical course is highly variable. However, the above findings have led some authors to suggest that there are different stages of severe COVID-19. In the early phase, hypoxia may be driven primarily by damage or dysregulation to the pulmonary blood vessels,[8-11] leading to impaired transfer of oxygen from the lungs to the bloodstream. If the patient continues to deteriorate, however, the clinical picture may become more typical of acute respiratory distress syndrome (ARDS) due to other aetiology, with diffuse alveolar damage and inflammatory oedema that necessitates intubation and mechanical ventiation.[8,10,12]

## Recognising and assessing respiratory distress

Clinical indicators of respiratory failure include an increasing respiratory rate (RR), decreasing oxygen levels or increasing supplementary oxygen requirement. Patients may also describe chest tightness, an inability to breathe deeply and may look and/or feel increasingly anxious which exacerbates breathlessness due to an increase in respiratory rate to the point of inefficiency alongside psychological effects including fear. These symptoms warrant prompt clinical review.

Oxygen levels are assessed by peripheral oxygen saturation ($SpO_2$) and/or arterial/capillary oxygen partial pressure ($Pa/cO_2$), with $SpO_2$ favoured during COVID-19 [13] due to its ease and speed of monitoring, unless there is concern of hypercapnia. The normal range for $SpO_2$ is 94-98%, although clinicians have been advised to target 92-96% during the COVID-19 pandemic. This will aid conservation of hospital oxygen supplies with evidence from clinical trials demonstrating that these lower oxygen target ranges are safe.[14] This range is sufficiently high to help alleviate dyspnoea and maintain tissue oxygenation, whilst avoiding the potentially harmful effects of hyperoxia. The normal range for $PaO_2$ is 12.6-14 kPa.[15]

Key features of respiratory distress are summarised in Box 5.[16]

Box 5: Recognising respiratory distress/failure

Symptoms:

- Breathlessness
- Chest discomfort
- Anxiety/agitation

Signs:

- Tachypnoea (early)
- Inability to speak complete sentences and gasping
- Use of accessory muscles of breathing including leaning forward in the ‘tripod position’, contraction of neck and abdominal muscles and retraction of lower ribs during inspiration.
- Purse lipped breathing
- Nasal flaring
- Tracheal tug

- Low oxygen saturations
- Cyanosis (late)
- Drowsiness (late)

As noted above, breathlessness may not be as prevalent or severe as expected for a given level of hypoxia in COVID-19. [4,9] Therefore extra vigilance is required as decreasing $SpO_2$ and an increased oxygen requirement may be the only marker of deterioration and a need for escalation of treatment.

*Case scenario 1: A 50 year-old mathematician has been self-isolating for 8 days with fever and a cough. He was brought to the Emergency Department by ambulance as he has become increasingly breathless over the last 2 days.*

*He can complete sentences but has an elevated RR of 24 breaths per minute (normal range 12-20). $SpO_2$ was 72% breathing room air when the paramedics arrived, and 90% on 4 litres per minute (L/min) oxygen via nasal cannula. Cardiovascular observations are normal and he is apyrexial.*

Box 6: Approach to assessing and managing respiratory failure

- Focused history and examination alongside commencing supplementary oxygen if required to achieve target $SpO_2$
- Commence treatment for underlying cause if possible
- Escalate supplementary oxygen as required to achieve target $SpO_2$ and consider arterial or capillary blood gas analysis (ABG or CBG) to differentiate T1RF from T2RF
- If required commence non-invasive respiratory support (CPAP or NIV)

## Management of respiratory failure

The tenets of managing respiratory failure are to maintain adequate oxygen levels, avoid hypercapnia, treat the underlying cause where possible (e.g. antibiotics for bacterial pneumonia) and reduce the risk of potential complications (e.g. venothromboembolism prophylaxis), whilst optimising hydration, nutrition and patient comfort. Additionally in COVID-19 management, there needs to be enhanced focus on infection prevention and control, location of patient care, prompt decisions regarding ceilings of care and recruitment into research trials. [17, 18]

Table 3 lists terminology used in non-invasive respiratory support.

*Case scenario (continued): You increase supplementary oxygen delivery to your patient by applying a facemask delivering 8 L/min of oxygen. His $SpO_2$ improves to 96% and RR falls to 18/min.*

*Later that day his nurse alerts you that his RR has increased to 26/min, he is using accessory muscles of breathing and $SpO_2$ is 90% despite switching to a reservoir mask delivering 15 L/min oxygen. The patient is commenced on Continuous Positive Airway Pressure (CPAP) and the intensive care team are informed.*

*Type 1 respiratory failure*

The aim of supportive treatment in T1RF is to improve oxygenation.

1. The first intervention is to increase the fraction of inspired oxygen ($FiO_2$). The choice of delivery device (Table 2) is determined by the oxygen requirement and patient comfort. Patients should receive the lowest FiO2 required to maintain target $SpO_2$.

2. Applying Positive End Expiratory Pressure (PEEP) to the airway during expiration can further improve oxygenation by splinting open the alveoli and smaller airways that may otherwise close during expiration in respiratory infection. This reduces de-oxygenated blood flowing through areas of poorly ventilated lung (intrapulmonary shunting) and effectively increases the surface area available for gas exchange.[19,20] PEEP may be indicated alongside increasing FiO2 when target oxygenation has not been achieved.

The most effective means of achieving PEEP in non-intubated patients is with continuous positive airway pressure (CPAP). This is different to Non-invasive ventilation (NIV) (see Table 3). It involves wearing a snug fitting mask through which pressurised air is applied. It is most commonly used to splint open the *upper* airway in obstructive sleep apnoea; in T1RF it is splinting open the *lower* airways in order to increase gas exchange.

Respiratory support will progress from the most simple, best-tolerated devices using the least oxygen to achieve target $SpO_2$ – typically, in sequence: nasal cannula, oxygen mask and then CPAP or high flow nasal oxygen (HFNO). A venturi mask may be used instead of nasal cannula or oxygen mask in patients who are susceptible to T2RF (Table 1), to deliver a precise $FiO_2$.

Box 7: Contraindications to CPAP

- Agitated or uncooperative patient
- Reduced conscious level with inability to protect airway
- Facial burns or trauma
- Persistent vomiting
- Unstable cardiorespiratory status or respiratory arrest
- Copious respiratory secretions
- Non-drained pneumothorax

CPAP is recommended when treating COVID-19, [13] with suggested pressure ranges from 8 – 14 cm $H_2O$, and a growing evidence base to support these recommendations. [21]

A further therapy to improve oxygenation advocated in patients with COVID-19 is awake proning. [22] Proning is an established therapy for refractory T1RF in intubated patients. [23,24] It improves oxygenation by minimising ventilation/perfusion mismatching in the lung. Proning has been increasingly used in awake patients with COVID-19 [22,25,26] although high quality evidence regarding its efficacy in awake patients is still awaited.

For some patients, CPAP with or without awake proning may be used as a holding measure while awaiting intubation. The intensive care team should be involved in this situation**.** HFNO is a theoretical alternative to CPAP, combining increased FiO2 with up to 5 cm $H_2O$ PEEP [27] but NHS England has advised against its use when treating COVID-19 due to lack of efficacy, high oxygen consumption and risk of infection spread. [13] In some patients, CPAP is sufficient to support patients' breathing and avoid the risks of invasive ventilation altogether. [21]

*Type 2 respiratory failure*

In Type 2 respiratory failure, (Table 1) alveolar hypoventilation leads to hypercapnia with associated hypoxia. Management aims to reduce carbon dioxide whilst maintaining acceptable oxygenation, target $SpO_2$ 88-92%. This ensures safe levels of oxygen whilst avoiding the risk of worsening hypercapnia. [15] Reduction in carbon dioxide is achieved by increasing minute ventilation; the product of respiratory rate and tidal volume (MV = RR x $V_T$). This is achieved by:

1. Increasing the RR. This is difficult to achieve in an awake, unwell patient, who is already tachypnoeic. Beyond a certain level, further increases in respiratory rate do not clear more carbon dioxide, since breathing becomes inefficient due to increased dead-space ventilation (i.e. ventilation of parts of the airway where no gas exchange occurs).

2. Increasing the volume of air moved with each breath (tidal volume, $V_T$) reduces alveolar carbon dioxide levels. As carbon dioxide readily diffuses from the pulmonary capillaries into the alveolar space the blood level is closely affected by changes in alveolar ventilation. This is the basis for NIV which delivers positive airway pressure that varies during the breathing cycle, providing an elevated baseline or end-expiratory airway pressure (PEEP/EPAP), and a separate, elevated inspiratory pressure (IPAP), which is provided when the patient starts breathing in. (Table 3).

Pressure settings vary depending upon the patient's underlying condition.[28] For example, high pressure will be needed in COPD (e.g. IPAP 20; EPAP 4 cm $H_2O$) and obesity (e.g. IPAP 20; EPAP 8 cm $H_2O$) due to airway resistance and poor chest wall compliance, respectively. By contrast, in neuromuscular disease respiratory system compliance is usually normal so lower pressures (e.g. IPAP 12; EPAP 4 cm $H_2O$) are required. Pressures can be titrated based on arterial/capillary carbon dioxide partial pressure (Pa/c$CO_2$). Increasing PEEP/EPAP to improve oxygenation must be matched by an equal increase in IPAP to maintain the same pressure support (i.e. maintain the same effective tidal volume and carbon dioxide control).

In COVID-19, T2RF is uncommon. [17] However, patients vulnerable to hypoventilation including those with COPD or obesity (Table 1) as well as patients with very severe disease and/or fatigue may be at higher risk of developing T2RF. In those with T2RF due to severe COVID-19 and/or fatigue, NIV should only be used as a bridge to urgent intubation, or as the ceiling of care.

Other considerations

When using CPAP or NIV in COVID-19, a non-vented mask with a bacterial/viral filter between the patient and the expiratory port is required to reduce environmental contamination. [13]

Whilst it is beyond the scope of this article to discuss the haematological abnormalities associated with COVID-19, there is evidence that it induces a prothrombotic state that increases the risk of VTE. [29,30] Therefore clinicians must have a high index of suspicion for pulmonary embolism that will further impact the degree of respiratory failure.

## Ceiling of care, intubation and invasive ventilation

The ceiling of care is the maximal level of care that is appropriate for a given patient, considering a combination of factors including their (or their advocates) wishes, underlying co-morbidities and frailty. Guidance exists from professional bodies to support this decision making. [31] Best practice is for individuals, their family members and their GP or secondary care team to discuss and jointly agree a ceiling of care prior to becoming acutely unwell or being unable to express their wishes. All patients with COVID-19 in hospital and many of those in the community should have a ceiling of care established at the earliest opportunity.[32,33] This may be limited to a defined level of the respiratory support in the home or ward environment depending on the outcome of the discussions detailed above.

Intubation and invasive ventilation are indicated when deterioration occurs despite the measures above, in a patient suitable for full support. In those where invasive ventilation has been deemed inappropriate, CPAP or NIV could be used as the ceiling of care. Prior to commencing these therapies the ceiling of care should be documented.

## A patient's experience

*The patient improved with CPAP, avoiding invasive ventilation.*

During recovery he provided insightful feedback: Nasal cannula and the reservoir mask were tolerable as he was able to communicate, eat and drink. In contrast, CPAP was "claustrophobic" and "suffocating". He was scared of vomiting and being unable to remove the mask, whilst "simple communication with staff became a task" which made him angry. Better explanation of the rationale behind treatment would have helped his tolerance.

Such insights are valuable and can inform our decision-making regarding treatments, particularly in those where CPAP or NIV may be of marginal benefit.

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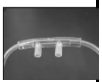
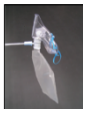
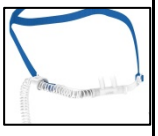
**Table 1 – Differentiating Type 1 and Type 2 Respiratory Failure**

| | Oxygen (arterial $PO_2$) | Carbon dioxide (arterial $PCO_2$) | Minute ventilation (see explanation in T2RF paragraph) | Pathophysiology | |
|---|---|---|---|---|---|
| Type 1 | Low | Normal or low (<6 kPa) | Normally high, as the patient will typically hyperventilate in response to the hypoxia. | Caused by a problem with transfer of gas from the lung into the blood.<br><br>May be due to a problem with diffusion of gas from alveolus into blood, but more often due to poor matching of gas and blood flow ('VQ matching') across the lung.<br><br>Due primarily to its low blood solubility, both these problems have a much greater impact on oxygen than carbon dioxide. The $PCO_2$ is therefore normal (or in fact usually low, due to hyperventilation and increased 'blowing off' of carbon dioxide from the lung). | **Pneumonia**, in sputum.<br><br>**Heart failure**, i impairing gas e<br><br>**Asthma**, in whi and mucus plu asthma, typica<br><br>**Pulmonary em** regions of lung<br><br>**High altitude p** uneven pulmo into the alveol |
| Type 2 | Low | Elevated (>6 kPa) | Low | Primarily due to a failure of ventilation i.e. a failure to move enough gas into and out of the lungs, leading to reduced oxygen intake and reduced removal of $CO_2$ from the lungs.<br><br>The impairment of ventilation may be due to reduced respiratory drive, increased airway resistance, reduced compliance (increased 'stiffness') of the respiratory system, or to respiratory muscle weakness.<br><br>In type 2 respiratory failure underlying gas exchange may be normal when there is respiratory muscle weakness with underlying healthy lungs, or impaired when there is lung disease with a 'mixed' T1 and 2 RF picture e.g. severe COPD. | **Opiate toxicity** in the central r<br><br>**Iatrogenic ove** abolished in a (raised base ex vasoconstrictio<br><br>**Neuromuscula** dystrophy. Oft<br><br>**Reduced chest**<br><br>**Increased airw**<br><br>**Severe impairr** both poor gas |

**Table 2 – Oxygen delivery devices**

| | Nasal cannula | Simple face mask | Venturi Mask | Reservoir mask | Nasal high flow oxygen | CPAP (Continuous Positive Airway Pressure) |
|---|---|---|---|---|---|---|
| Image |  |  |  |  |  |  |
| Indication | Mild-moderate hypoxia (T1RF) | Moderate-severe hypoxia (T1RF) | Hypoxia with chronic hypercapnia | Severe hypoxia | Very severe hypoxia | Very severe hypoxia |

| | | | | | | |
|---|---|---|---|---|---|---|
| Oxygen flow rate | 1-6 L/min | 5-10 L/min | 2-15 L/min (varies according to desired $FiO_2$) | 15 L/min | Up to 70 L/min | 15 L/min |
| $FiO_2$ | ~24-50% | 40-60% | 24-60% | 60-90%, if bag inflated | Up to 100% | Up to 100% |
| Pros | Well tolerated<br><br>Widely available | Widely available<br><br>Simple to use | Widely available,<br><br>Simple to use.<br><br>Predictable $FiO_2$. | Widely available<br><br>Simple to use | Provides PEEP (~5 cm $H_2O$)[10] when breathing with mouth closed.<br>Warmed and humidified.<br>Nasal cannula allow patients to talk, eat and drink | Provides PEEP<br>Potentially avoid need for invasive ventilation<br>Lower oxygen use, compared with high flow nasal oxygen |
| Cons | Unpredictable $FiO_2$<br><br>Non-humidifed oxygen can cause dryness of the upper airway potentially causing crusting and epistaxis | Unpredictable $FiO_2$<br><br>Risk of rebreathing at low oxygen flows<br><br>Non-humidifed oxygen can cause dryness of the upper airway potentially causing crusting and epistaxis | May not necessarily increase alveolar ventilation sufficiently to lower $PCO_2$<br><br>Non-humidifed oxygen can cause dryness of the upper airway potentially causing crusting and epistaxis | $FiO_2$ unpredictable, risk of rebreathing at low oxygen flows<br><br>Non-humidifed oxygen can cause dryness of the upper airway potentially causing crusting and epistaxis | Aerosol generating procedure, requiring level 2 PPE/side room in COVID-19 pandemic<br><br>Very high oxygen flow rate means transfer of patients not possible<br><br>Widespread use during COVID-19 surge may lead hospital oxygen demand to exceed supply. [34] | Aerosol generating procedure, requiring level 2 PPE/side room in COVID-19 pandemic<br><br>Difficult to tolerate for some due to claustrophobia, inability to talk, eat and drink<br><br>Can cause pressure sores, for example, on the bridge of nose if mask is applied too tightly or breaks in treatment are not scheduled |

**Table 3 – Terminology of non-invasive respiratory support**

| Primary term | Synonyms used in intensive care | Meaning |
|---|---|---|

| | | |
|---|---|---|
| PEEP (Positive End-Expiratory Pressure)<br>*Or*<br>EPAP (Expiratory Positive Airway Pressure) | e-PEEP<br>Low PEEP<br>$P_{low}$ | This is the pressure set during expiration, the lowest pressure during the respiratory cycle. It is primarily used to splint open additional alveolar units and therefore improve oxygenation. |
| CPAP (Continuous Positive Airway Pressure) | e-PEEP<br>Low PEEP<br>$P_{low}$ | Like PEEP and EPAP, this is also the pressure set during expiration. It is used to splint open the lower airway (improve oxygenation) but is also the term for treatment when the aim is to splint open the upper airway, for example when treating obstructive sleep apnoea.<br>Unlike PEEP and EPAP, this terminology would not be used when giving NIV where the pressure delivered is variable, so the term “continuous” becomes inappropriate. |
| IPAP (Inspiratory Positive Airway Pressure) | $P_{high}$<br>Inspiratory Pressure<br>i-PEEP<br>High PEEP | This is the pressure set during inspiration, the highest pressure in the lung during the respiratory cycle. |
| PS (Pressure Support) | Driving pressure (ΔP)<br>Pressure Assist | PS =IPAP-EPAP<br>This controls the amount of ventilation as increasing the PS will increase the tidal volume in each breath. |
| NIV (Non-invasive Ventilation) | BiPAP<br>Bi level<br>NIPPV | Positive airway pressure that varies during the breathing cycle, to provide not only an elevated baseline airway pressure (PEEP/EPAP), but also an elevated inspiratory pressure (IPAP), which is triggered when the patient starts taking a breath in. |