Long term conditions

Supplementary Nasal Cannula Provision in Hypoxaemia Refractory to Non-Rebreathe Mask Treatment: A Case Study.

Adam Nolan[1], Ethan Singer[2]

[1] Physiotherapy, Glenfield Hospital, [2] Physiotherapy, Royal Brompton Hospital

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INTRODUCTION

Hypoxaemia refers to a low level of oxygen in the blood and may occur in response to diffusion impairment, shunting, deadspace and alveolar hypoventilation.[1] Pulse oximetry can measure oxygen saturations (that is oxygen which is bound to the red blood cell) (SpO2).[2] When SpO2 is low (<94% or <88% in patients as risk of hypercapnic respiratory failure) supplemental oxygen can be used to treat hypoxaemia.[3]

Oxygen therapy on the 'ward' (level one care) is often limited to fifteen litres per minute (L/Min) via a non-rebreathe mask (NRB). In other clinical areas, hypoxaemia may be treated with the provision of high flow nasal oxygen (HFNO) (which is *not* limited to 15L/Min and can provide up to 100% fraction of inspired oxygen (FiO2)). In some cases, continuous positive airways pressure (CPAP) and invasive mechanical ventilation (MV) may be used.

As noted during the COVID-19 pandemic, the availability of oxygen has previously been scarse.

High flow oxygen therapy is not always available. This may be of detriment to the patient as profound hypoxaemia may not be responsive to 15L/Min NRB therapy in isolation. Indeed this was the situation we faced in the treatment of our patient which we have recounted here. In this case study we will discuss how the time from hypoxaemia to 'normoxaemia' can be reduced using a combination therapy: a 15L/Min NRB and 4L/Min nasal cannulae.

There has been some research into the combined use of nasal cannulae and NRB therapy however this has been confined to patients with COVID-19.[4,5] To date, there appears to limited data on the effect of this treatment in a non-COVID-19 population.

PRESENTING CASE

An 82-year-old male was admitted to Glenfield Hospital. Thoracic surgery was subsequently undertaken to remove the left lower lobe of the lung in view of potential lung malignancy. Further bronchoscopy and mini-tracheostomy insertion were required as the patient's sputum burden was not wholly amenable to chest physiotherapy, this was secondary to altered anatomy post procedure. Weeks after the procedure (while still an inpatient), the patient developed dyspnoea and hypoxaemia (likely secondary to mucus plugging compounded by altered anatomy post-surgery). Oxygen saturations (SpO2) were 66% on 4L/Min nasal cannula. This was immediately treated with a 15L/Min NRB; however, the patient's oxygen saturations failed to exceed 77%. Consequently, nasal cannula 4L/Min was applied beneath the 15L/Min NRB and SpO2 reached 88%. Once the SpO2 was stable at 88%, help was sought to supervise the patient on a one-to-one basis while a HFNO was sourced and set-up. The patient was commenced on HFNO therapy with an FiO2 of 1.0 and approximately 60L/Min flow rate. The patient's SpO2 at this time was 91%. An urgent chest x-way was requested which showed complete collapse of the left lung with an apical space. The patient was soon listed for a repeat bronchoscopy in the afternoon.

Variable Performance Devices	Fixed Performance Devices
Nasal Cannula	Venturi Mask
Simple Oxygen Facemask	Cold Humidified Oxygen (utilising Bernoulli Principle)
Non-Rebreathe Mask	

Figure 1. Examples of Variable and Fixed Performance Devices.

DISCUSSION

In the example mentioned above, it is likely many factors contributed to this patient's hypoxaemia. Firstly, the gas exchange membrane would have been reduced on account of the left lung collapse, this would have necessitated shunting. Alveolar hypoventilation is likely to have occurred also.

Oxygen therapy is broadly classified into 'variable performance devices' and 'fixed performance devices' wherein the former is posited to provide a variable fraction of inspired oxygen (FiO2) and the latter is posited to provide a fixed and constant FiO2. Examples of these devices are included in Figure 1.

Although this is a commonly cited classification, it may resemble a false dichotomy as all devices may become variable performance devices if the patient's inspiratory flow demand (IFD) greatly exceeds the flow rate of oxygen. This

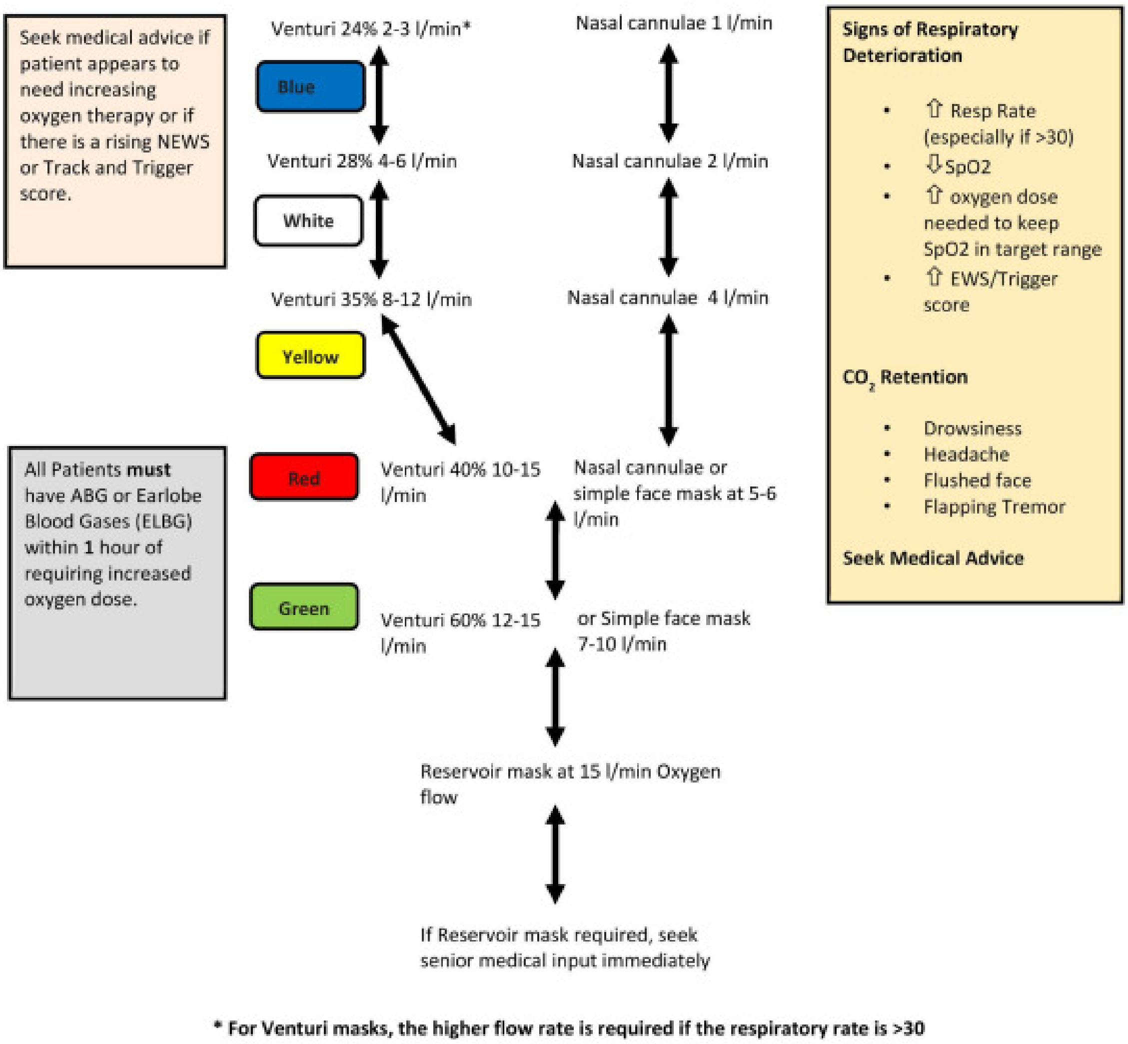

Figure 2. Illustration of Compatible Flow Rates for Venturi Valves.[3]

has been echoed by O'Driscoll et al.[3] who notes that although a Venturi valve may have a *suggested* flow rate (for instance 35% Venturi valve requires a suggested 8L/Min), clinicians can use up to 12L/Min provided the respiratory rate exceeds thirty. This has been illustrated in Figure 2.

At rest, a one's inspiratory flow demand (IFD) is roughly 30L/Min.[6] This means that on 15L/Min a patient is still diluting the FiO2 with 15L/Min of room air). As such, the FiO2 the patient receives (even with for instance a Venturi mask running at 15L/Min with a 60% valve) is not going to be exactly 60%.

Furthermore, during exercise, one's IFD can reach roughly 100L/Min.[6] Exercise mimics respiratory distress on account of the increased respiratory rate and tidal volume. If we apply this axiom to the example mentioned above, our gentleman may have been receiving only 15L/Min of FiO2 1.0 and 85L/Min of room air thus owing to his poor response to supplemental oxygen.

As described above, the provision of 4L/Min nasal cannula appeared to attenuate the hypoxaemia. This was the only treatment provided as examination findings were unremarkable; chest radiography had not yet been undertaken.

An additional 4L/Min of FiO2 1.0 may have been sufficient to meet the patient's IFD. There is research support for this idea from Kumar et al.[4] in the instance of COVID-19 induced hypoxaemia.[4] However, Kumar et al.[4] used 6L/Min and not 4L/Min which may suggest this method is only efficacious with higher flow rates.

Conversely, we concede that with the aforementioned IFD in mind, it is unlikely that an additional 4L/Min would be sufficient to meet a patient's IFD during respiratory distress. In this instance we would have to assume that 19L/Min of FiO2 1.0 was sufficient to treat the profound hypoxaemia. Unfortunately, in the absence of any real-time measurement of IFD, we are limited in our reasoning for this improvement.

CONCLUSION

In conclusion, supplemental nasal cannula use in hypoxaemia refractory to 15L/Min NRB therapy may be a useful as a 'holding therapy' while alternative forms of oxygen therapy are sourced. The mechanisms of additional nasal cannula therapy are unknown but may be related to the optimisation of IFD.

SUMMARY

Although there have been improvements in technology which have permitted the delivery of advanced respiratory support, this is often a timely endeavor. This case study has demonstrated a single method which can maintain a patient using novel application of existing older technology in order to address hypoxaemia, maintain safety and prevent deterioration while other treatments are set up.

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