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ADMINISTRATION AND MONITORING OF OXYGEN THERAPY

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INTRODUCTION

Medical oxygen is a common drug, in both the (pre-)hospital and home setting. Like any other therapeutic agent, it has specific indications for its use, appropriate dosage, method of administration and potential side effects. Given correctly, oxygen can be extremely beneficial to the patient; incorrect use may result in a variety of complications and even death.

Oxygen therapy is usually defined as the administration of oxygen at concentrations greater than those found in ambient air. The major indication for prescribing oxygen therapy and the only one that is evidence based, is hypoxaemia (arterial oxygen tension (P_{a,O_2}) <8.0 kPa or 60 mmHg). Oxygen is prescribed to increase oxygen tension and to decrease the work of breathing necessary to preserve a given P_{a,O_2} . There is no evidence of benefit from giving oxygen to patients who are not hypoxaemic, yet there is evidence that oxygen does not lessen breathlessness compared with air in non-hypoxaemic patients [1, 2]. The only

exceptions in this respect are carbon monoxide poisoning (high-dose oxygen decreases the half-life of carboxyhaemoglobin three-fold) and a moderate-sized pneumothorax that does not require drainage (high-dose oxygen can increase the reabsorption of air from a pneumothorax up to four-fold) [3].

Irrespective of the indication and setting of oxygen therapy, for oxygen to be administered, one always needs an oxygen source and a delivery system. For any individual patient, the appropriate device should be used to provide oxygen therapy in a given setting. A further requirement for those who prescribe oxygen therapy is to closely monitor the patient and to keep them within their personal target saturation range, while also assessing potential side effects in patients at risk of hypercapnic respiratory failure [4].

OXYGEN SOURCES

Most hospitals have piped oxygen systems between the wall oxygen

outlet and large liquid oxygen reservoirs outside the hospital. Measures should be taken and attention should be paid to rule out the risk of connecting the oxygen tubing to an incorrect (air) wall socket. Oxygen flow meters, which may provide a flow up to $15 \text{ L}\cdot\text{min}^{-1}$, should thus only be connected to the oxygen wall outlet. Most flow meters use a floating ball to indicate the flow-rate. The centre of the ball should correspond with the correct flow-rate marking [4]. In order to connect flow meters and regulators to the patient delivery device, oxygen tubing is needed. For emergency use, hospitals and the majority of medical centres and practices should have a supply of oxygen cylinders with a high-flow regulator capable of delivering flows $>6 \text{ L}\cdot\text{min}^{-1}$. These are also used by fast-response units, ambulances and rescue teams in pre-hospital care.

In the home setting, the oxygen concentrator is the most cost-effective and, therefore, the most frequently used stationary oxygen source [5]. An oxygen concentrator is an electrical device which ►

absorbs nitrogen and carbon dioxide from the air, so that only oxygen is passed to the patient. Hence, the device has to stay in a clean, dry and well-ventilated space. Because of the risk of a mechanical defect or power failure, an oxygen cylinder is always provided. Oxygen concentrators need regular servicing for proper functioning. In case of malfunctioning or with a flow-rate $>3 \text{ L}\cdot\text{min}^{-1}$, the oxygen concentration may drop. Most modern oxygen concentrators are equipped with an oxygen concentration alarm for this reason. Recently, portable concentrators and concentrators, which are able to refill small cylinders, have become available.

Due to the high cost, high weight and small capacity of oxygen cylinders, they have been largely replaced by the oxygen concentrator as a stationary oxygen source. Oxygen cylinders are, however, still distributed for both stationary and ambulatory use. The volume of oxygen cylinders may vary from 2 L (400 gas-litres at 200 bar) to 50 L. The 2 L cylinders (weight 4.75 kg) are frequently used for ambulation and may be carried in a special bag or a backpack. Smaller (0.8 L) or lightweight oxygen cylinders not only have a lower weight but also a smaller capacity. If combined with an oxygen conserving device, such as a pulse device, patients may, nevertheless, use these cylinders for the same amount of time as with a heavier 2 L cylinder. For ambulatory and active patients who want to leave their home regularly and are able to do so, the liquid oxygen system remains, however, the ultimate oxygen source. The stationary reservoir has a volume of 20–40 L, which is equivalent to 17,000–35,000 gas-litres (1 L of liquid oxygen (-183°C) evaporates at 15°C to 850 L of gaseous oxygen). The portable container has a volume of 0.5–2.0 L (425–1721 gas-litres), a full weight of 2.2–6.0 kg and can easily and safely be filled by the patient from the larger reservoir. Drawbacks of the liquid

oxygen system are its high cost, dependency of the portable container on the large reservoir for refilling and the requirement for pressure-relief venting. The effect of the latter is that oxygen is consumed even if the patient does not use it [5].

OXYGEN DELIVERY DEVICES

During oxygen therapy, oxygen is delivered by either fixed- or variable-performance systems.

Fixed-performance devices

Fixed-performance systems deliver a predictable and consistent inspiratory oxygen fraction (F_{IO_2}) independent of fluctuations in the patient's breathing pattern. These are functionally systems with a large capacity, providing gas flows that exceed peak inspiratory flow demand. Examples of fixed-performance devices are the non-rebreathing mask, the partial rebreathing mask and the Venturi mask.

The non-rebreathing mask has an oxygen reservoir (balloon) with an inspiratory valve, which blocks exhaled air from flowing into the oxygen reservoir. A second valve in the reservoir opens with a negative pressure of 1–2 cmH_2O so that the reservoir is filled with oxygen. In addition, the mask has two unidirectional valves on each side of the mask that permit venting of expired air while preventing inspiration of room air. With a sufficient oxygen flow-rate to sustain the reservoir bag volume ($10\text{--}15 \text{ L}\cdot\text{min}^{-1}$) and proper application of this device a mean F_{IO_2} of 0.80–0.95 is attainable. These masks are most suitable for emergency use [4].

The partial rebreathing mask provides a high F_{IO_2} while conserving the oxygen requirement with approximately 30%. This makes this mask suitable for transport situations with limited

availability of oxygen. The oxygen flow-rate is regulated to permit the initial one-third of the expired tidal volume (anatomic dead space) to fill the reservoir, thus preventing the entry of gas containing carbon dioxide, which instead is exhaled through side ports in the mask. With correct application of this mask a F_{iO_2} of 0.70–0.85 may be reached [4].

The Venturi mask is an oxygen-powered air-entrainment mask, which is designed to provide a high gas flow at a known F_{iO_2} . Oxygen is delivered to a jet-mixing device, which increases gas velocity. The resulting entrainment of ambient air dilutes the stream of pure oxygen. The higher the oxygen flow-rate, the more air is entrained. As a result, Venturi masks deliver the same oxygen concentration regardless of the oxygen flow-rate, as long as this stays above a minimum, which is written on each device. The high gas flow promotes a consistent F_{iO_2} despite fluctuations in the patient's breathing pattern. Because the high gas flow usually exceeds the patient's inspiratory flow-rate, valves or a reservoir are not needed to prevent rebreathing. However, in patients with a respiratory rate of >30 breaths·min⁻¹, the flow-rate of the Venturi mask should be set above the minimum, as these patients may have an inspiratory flow-rate exceeding the gas flow delivered by the device [4]. If the flow-rate is not increased above the minimum level, ambient air may be inspired by the patient and, consequently, the F_{iO_2} may drop. Each mask is accompanied by a number of colour-coded jets that produce a known F_{iO_2} at a given oxygen flow-rate. If placed accurately on the patient's face, Venturi masks can deliver oxygen with a F_{iO_2} of 0.24, 0.28, 0.35, 0.40 or 0.60. Most Venturi masks also have an adaptor that permits aerosol therapy at a lower F_{iO_2} than is possible with conventional oxygen-powered wall-mounted nebulisers [4].

Variable-performance devices

Variable-performance systems are patient dependent and have a small capacity or no capacity at all. The intratracheal F_{iO_2} delivered depends on the dead space of the device (100–300 mL in masks), the size of the anatomic reservoir (nasopharynx volume), the inspiratory peak flow, respiratory rate, length of the expiratory pause and the oxygen flow-rate. Accordingly, the oxygen flow-rate must be adjusted on the basis of oximetry measurements and, when necessary, blood gas measurements. Mouth breathing results in either the same or a higher F_{iO_2} , especially when the respiratory rate is increased [6]. Examples of commonly used variable performance systems are the simple face mask and nasal cannula.

The simple face mask, which is sometimes referred to as an, medium concentration (MC) mask or Hudson mask, covers the nose and mouth. It does not contain valves or a reservoir. With an oxygen flow-rate of 5–15 L·min⁻¹, a simple face mask may deliver a F_{iO_2} of 0.40–0.60 [4]. In children, a F_{iO_2} of 0.80 may be reached. However, different brands of this mask can ►



Figure 1. Oxygen delivery devices. From left to right: nasal cannulae, nasal catheter with foam manchet, Venturi mask with two jets and simple face mask.

deliver a different FiO_2 at a given oxygen flow-rate. In order to prevent rebreathing of carbon dioxide, the oxygen flow-rate should exceed the minute ventilation of the patient. In clinical practice, an oxygen flow-rate of $5 \text{ L}\cdot\text{min}^{-1}$ has shown to be sufficient [7]. Given the risk of carbon dioxide retention, this mask is unsuitable for patients with hypercapnic respiratory failure. With regard to the maximum FiO_2 or the consistency of the FiO_2 , the simple mask does not perform better than the other variable performance systems. □

Nasal cannulae are a cheap, convenient and effective means of delivering a FiO_2 of 0.21–0.50 if the nose is not blocked or severely congested [8]. As a rule of thumb, the FiO_2 increases with 0.03–0.04 for each extra $\text{L}\cdot\text{min}^{-1}$ of oxygen. Flow-rates $>6 \text{ L}\cdot\text{min}^{-1}$ do not further increase the FiO_2 [8]. With regard to the consistency of the FiO_2 , nasal cannulae seem to perform as well as a Venturi mask. Moreover, nasal cannulae are more likely to be left in position by the patient and less likely to fall off, they do not cause claustrophobia and do not have a risk of rebreathing carbon dioxide. In addition, patients may eat and speak while using oxygen therapy. If kept on for several hours, some patients may experience discomfort and nasal dryness at flows $>4 \text{ L}\cdot\text{min}^{-1}$. Patient preference seems, nonetheless, strongly in favour of nasal cannulae compared with masks [9].

MONITORING OF OXYGEN THERAPY

Patients with hypoxaemia should be closely monitored until their clinical situation becomes stabilised. As oxygen therapy is directed against hypoxaemia, it is also mandatory to monitor the effect of this therapy on oxygenation by checking if the oxygen saturation is kept within the target range [4]. By monitoring oxygen saturation, one

can judge if the oxygen delivery device and/or oxygen flow-rate need to be adjusted. In patients at risk for hypercapnic respiratory failure, monitoring of oxygenation is not enough. In these patients, ventilation needs to be monitored as well, by serial blood-gas measurements. To monitor oxygen therapy pulse oximetry, transcutaneous or end-tidal carbon dioxide monitoring and arterial, arteriolised or venous blood gases may be used.

One of the easiest, most reliable and cost-effective measurements to clinically assess hypoxaemia is pulse oximetry. Hence, in all breathless or acutely ill patients, oxygen saturation should be checked with pulse oximetry [4]. Pulse oximetry measures haemoglobin oxygen saturation by detecting the absorption of light at two wavelengths, which correspond to the absorption peaks of oxygenated and deoxygenated haemoglobin. As methaemoglobin and carboxyhaemoglobin have similar light absorption characteristics to oxyhaemoglobin, patients with methaemoglobinaemia or carbon monoxide poisoning may have a greatly reduced total arterial blood oxygen content despite normal oxygen saturation levels measured by pulse oximetry [10]. In most subjects, modern oximeters reflect the arterial oxygen saturation accurately to within 1–2% of directly measured arterial oxygen saturation at saturations above about 70% [11]. It has been estimated that an oxygen saturation of $\geq 92\%$, measured by pulse oximetry, has a sensitivity of 100% and specificity of 86% for excluding hypoxaemia, defined as an arterial oxygen tension $<60 \text{ mmHg}$ (8.0 kPa) [12]. Although the accuracy of pulse oximetry is diminished in patients with poor peripheral perfusion, many types of oximeter may remain accurate as long as the oximeter is able to obtain a reading [13]. It is, therefore, important to ensure that the best available signal for the

individual patient is obtained. Accuracy of pulse oximeters may also be influenced by skin pigmentation, particularly in dark skinned persons at saturation levels $<80\text{--}85\%$; motion of the patient's hand; and malpositioning of the sensor [14]. In general, finger and earlobe measurements are more accurate than toe measurements [15]. Finger probes may be more accurate than ear probes but only on fingers without nail varnish or acrylic finger nails. Pulse oximetry will be normal in a patient with normal oxygen tension but abnormal blood pH or carbon dioxide tension (PCO_2) or with a low blood oxygen content due to anaemia. Blood gases and full blood count tests are therefore required as early as possible in all situations where these measurements may affect patient outcomes.

During oxygen therapy, monitoring of ventilation is particularly useful in patients with a risk of hypercapnic respiratory failure. The latter can be detected reliably by pulse oximetry only when patients breathe room air [16, 17]. Thus, to determine the adequacy of ventilation during oxygen therapy the arterial carbon dioxide tension (Pa,CO_2) has to be measured. This may be accomplished by transcutaneous or end-tidal carbon dioxide monitors, or by blood gas measurements.

Transcutaneous PCO_2 (P_{tc},CO_2) measurement makes use of the fact that carbon dioxide diffuses through body tissue and skin. The first commercially available P_{tc},CO_2 monitors, which were introduced in 1980, used a locally heated electrochemical sensor that was applied to the skin surface. This technique provides a continuous noninvasive estimation of the Pa,CO_2 and is nowadays used routinely in clinical practice. At 42°C , the P_{tc},CO_2 correlates well with the corresponding Pa,CO_2 , with the P_{tc},CO_2 being $\sim 5 \text{ mmHg}$ higher, due to the elevated temperature and increased skin ►

metabolism [18, 19]. In order to obtain a significant correlation between transcutaneous oxygen tension ($P_{tc}O_2$) and $P_{a}O_2$ a sensor temperature of approximately 44°C is needed. Sensors for $P_{tc}CO_2$ analysis are available as a single PCO_2 sensor, as a combined PCO_2 /oxygen tension (PO_2) sensor and, more recently, as a combined PCO_2 /arterial oxygen saturation measured by pulse oximetry (SpO_2) sensor [20]. Continuous development of this technique during the last twenty years has made $P_{tc}CO_2$ monitors easier and more reliable for use in clinical practice. The present $P_{tc}CO_2$ sensors still need to be regularly re-membraned and calibrated, however. Preliminary results, obtained with a new technique, show that long-term stable and calibration-free CO_2 monitoring is possible [21].

End-tidal carbon dioxide tension ($P_{ET}CO_2$) monitoring is the noninvasive measurement, by infrared spectroscopy or colorimetric detection, of the partial pressure or maximum concentration of CO_2 at the very end of expiration. Following the first clinical studies in the 1970s, $P_{ET}CO_2$ monitoring has been widely established in the hospital and prehospital environments. $P_{ET}CO_2$, being a reflection of circulation, ventilation and metabolism, is normally 2–5 mmHg lower than $P_{a}CO_2$ [22]. Any condition causing ventilation/perfusion mismatch will cause the net $P_{ET}CO_2$ to underestimate the $P_{a}CO_2$ [23]. Hence, in patients with chronic obstructive pulmonary disease (COPD) the $P_{ET}CO_2$ correlates poorly with $P_{a}CO_2$. A further disadvantage of this technology is the need for calibration with a previously analysed gas mixture. Also, high concentrations of oxygen can cause infrared capnometry to provide erroneously elevated PCO_2 readings [24]. Finally, with a continuous (oro-)nasal oxygen flow, it is impossible to measure $P_{ET}CO_2$ reliably. Given all these drawbacks,

$P_{ET}CO_2$ monitoring is unsuitable for the monitoring of ventilation during oxygen therapy.

Arterial blood gas measurements remain the “gold standard” to assess and monitor ventilation during oxygen therapy. A change in the oxygen dose may take 20 min for the $P_{a}O_2$ and arterial oxygen saturation (SaO_2) to equilibrate, but for the $P_{a}CO_2$, this may take 30–60 min [4, 25]. Arterialised capillary gases from the earlobe (but not from the finger) may provide a pH and PCO_2 , which are almost identical to the arterial values [26, 27]. The capillary PO_2 is, however, about 3.7–7.5 mmHg (0.5–1.0 kPa) lower than the arterial PO_2 , particularly at oxygen tensions >60–75 mmHg (8.0–10.0 kPa) [28]. Thus, most patients can be safely managed by earlobe blood gases and pulse oximetry, even in emergency settings. Capillary blood gases are, however, very vulnerable to mistakes in technique and should only be performed by trained personnel. In general, earlobe sampling is safer and less painful than an arterial puncture but due to the unknown accuracy of earlobe samples in shock or hypotension (systolic blood pressure <90 mmHg), it is recommended to use arterial blood gas analysis in these circumstances [4]. Arterial blood gases should also be used for accurate measurements of PO_2 , like in candidates for long-term oxygen therapy.

OXYGEN THERAPY IN THE HOSPITAL AND PREHOSPITAL SETTING

For all acutely ill patients not at risk of hypercapnic respiratory failure or receiving terminal palliative care, oxygen therapy in both the hospital and pre-hospital setting is aimed at achieving a (near-)normal oxygen saturation. Because oxygenation is reduced in the supine position, patients should be allowed to maintain the most erect position possible unless there are good

reasons for immobilisation [29]. Critically ill patients and patients having a saturation <85% should be treated immediately with high-dose oxygen therapy with a partial or non-rebreathing mask at 15 L·min⁻¹. Early intubation and ventilation may be needed. In seriously ill patients, medium-dose oxygen therapy should be started, preferably with nasal cannulae (2–6 L·min⁻¹) or a simple face mask (5–10 L·min⁻¹). Besides pulse, blood pressure, temperature and respiratory rate, oxygen saturation should be checked by pulse oximetry. The recommended target saturation range for these patients is 94–98% [4]. For patients aged >70 yrs, a slightly lower target saturation range may be acceptable, as many of these people have an oxygen saturation <94% when clinically stable [30]. In order to maintain the target saturation range, patients need to be monitored, oxygen delivery devices should be adjusted and oxygen flow-rates need to be titrated up or down. In all critically ill patients, in patients with an unreliable oximetry signal and in patients with unexpected signs or symptoms, blood gases must be checked. If these patients become hypercapnic or show other signs of respiratory deterioration (invasive) ventilation should be considered. Once patients have become clinically stable with acceptable oxygen saturation the oxygen flow-rate should be reduced. Humidification is only needed in case of an artificial airway, for patients who require high-flow oxygen therapy for >24 h and for patients who are troubled by dryness of their upper airways [4].

Pending urgent blood gas analysis, for most patients with (a significant likelihood of) COPD or other known risk factors for hypercapnic respiratory failure, like neuromuscular disorders, chest wall deformities or morbid obesity, a target saturation of 88–92% seems sufficient [4]. In these patients, low-dose oxygen therapy should be ►

commenced by means of a 28% Venturi mask at 4 L·min⁻¹ or a 24% Venturi mask at 2–4 L·min⁻¹. However, if their saturation stays below 88% nasal cannulae (2–6 L·min⁻¹) or a simple mask at 5 L·min⁻¹ should be tried. Preferably, treatment should be based on the results of blood gas measurements during previous acute exacerbations as hypercapnic respiratory failure can occur even at saturations <88% [31]. With a history of preceding respiratory failure, the lowest oxygen dose should be sought. If a patient is suspected of having respiratory acidosis due to excessive oxygen therapy, the latter should not be discontinued abruptly but, instead, should be diminished gradually depending on oxygen saturation and blood gas results. The sudden withdrawal of oxygen may lead to rebound hypoxaemia, which may be more dangerous than the hypercapnic respiratory failure itself [4]. Once these patients have stabilised, nasal cannulae may be used at flow-rates of 1–2 L·min⁻¹ to achieve the same target range as with a Venturi mask.

If these patients develop a critical illness, however, they should have the same initial target saturation as other critically ill patients. Depending on blood gas results, these patients may need controlled oxygen therapy or noninvasive mechanical ventilation. The target saturation also needs to be adjusted to 94–98% if these patients develop a serious illness, their P_{a,CO_2} is normal and there is no history of previous hypercapnic respiratory failure. In these circumstances, blood gases need to be repeated at least every 30–60 min [4].

Although they have become clinically stable, particularly patients with COPD may still be hypoxaemic shortly before their discharge from the hospital. To be discharged securely, these patients may need a prescription for short-term home oxygen therapy. However, most of these patients will become less hypoxaemic

during the first 3 months at home. Based on arterial blood gas results measured 6 and 12 weeks following discharge, oxygen therapy can be stopped in up to 45% of these patients [32, 33]. In patients who are still hypoxaemic at 12 weeks following discharge, short-term oxygen therapy may need to be changed into long-term home oxygen therapy.

OXYGEN THERAPY IN THE HOME SETTING

The main reason to prescribe oxygen at home is to alleviate hypoxaemia. The latter may exist permanently or only during sleep or exercise.

Long-term oxygen therapy (LTOT) is aimed at relieving daytime hypoxaemia and secondary consequences (pulmonary hypertension, cor pulmonale and hypoxia in the tissues). If prescribed for at least 15 h·day⁻¹, LTOT is the only therapy which is able to prolong survival in COPD patients with hypoxaemia [33]. LTOT should only be prescribed in patients who are clinically stable, who have stopped smoking and who have been treated optimally otherwise. As pulse oximetry and capillary blood gases are unreliable with respect to the P_{a,O_2} , at least two arterial blood gases are needed with an interval of at least 3 weeks to prescribe LTOT. The oxygen flow-rate should be titrated with pulse oximetry to reach a S_{a,O_2} 91–96% at all times [34]. Most patients are treated by an oxygen concentrator and nasal cannulae. For ambulation, small cylinders or liquid oxygen may be used.

In COPD patients, there is no proof that oxygen therapy for only nocturnal hypoxaemia improves survival, diminishes pulmonary hypertension or extends the moment that LTOT is needed [35]. Hence, oxygen therapy should be reserved for patients with nocturnal hypoxaemia and serious cardiac

complaints or clinical problems, like polycythaemia or heart failure, that respond well to the administration of oxygen.

By diminishing dynamic hyperinflation and ventilatory response, oxygen may reduce dyspnoea and improve, to some extent, the exercise tolerance and quality of life in COPD patients with exercise-induced hypoxaemia. It is, however, impossible to predict which patients will show a positive effect. Moreover, as the benefits often do not offset the ballast of the mobile oxygen source, many patients do not want to use this therapy in the long term [36].

SUMMARY

Irrespective of the setting, oxygen therapy is prescribed predominantly to treat hypoxaemia. In clinically unstable patients with a risk of hypercapnic respiratory failure, a target saturation range of 88–92% is usually acceptable, pending blood gas results. In order to avoid respiratory failure, controlled or low-flow oxygen therapy is frequently needed. However, when severely ill, these patients may need a medium- or even high-flow oxygen therapy to reach a higher target saturation range. In all other breathless or acutely ill patients aged <70 yrs, a target saturation range of 94–98% is recommended. Depending on their situation, these patients may need high-, medium- or low-dose oxygen therapy in order to achieve this. Pulse oximetry is the best method to check if the oxygen saturation is kept in the target range. Monitoring of oxygen saturation by pulse oximetry may require a change in delivery device and/or oxygen flow-rate. Patients with hypoxaemia should be closely observed until their clinical situation becomes stabilised. Although some patients may still need oxygen at home, 3 months following discharge, only a proportion of them satisfy the criteria for long-term oxygen therapy. ■

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