Part II: Respiratory Failure

Chapter 18: Oxygen Therapy

T E Oh

Oxygen is required in aerobic metabolic pathways to produce biological energy from food fuels. With inadequate oxygenation, anaerobic metabolism leads to decreased biological energy and harmful lactic and metabolic acidosis. Oxygen therapy is thus indicated whenever tissue oxygenation is impaired, in order to allow essential metabolic reactions to occur, and to prevent complications attributed to hypoxaemia.

The common clinical indications are:

1. Acute respiratory failure:

(a) without CO_2 retention, i.e. asthma, pneumonia, pulmonary oedema, and pulmonary embolism;

(b) with CO_2 retention, i.e. chronic bronchitis, chest injuries, unconscious drug overdose, postoperative hypoxaemia, and neuromuscular disease.

2. Acute myocardial infarction.

3. Cardiac failure.

4. Shock of any cause.

5. Increased metabolic demands, i.e. burns, multiple injuries, and severe infections.

6. Postoperative states.

7. Carbon monoxide poisoning.

Arterial Oxygen Tension (PaO_2)

Tissue oxygenation depends upon oxygen delivery and PaO_2. It is difficult to suggest a "safe" PaO_2 value at which hypoxia poses few significant problems. Each situation needs to be judged by the requirements for oxygen and the availability of oxygen. In general, additional inspired oxygen is required when PaO_2 is 60 mmHg (8.0 kPa) or less, and profound hypoxaemia is present and death is imminent, when PaO_2 is less than 30 mmHg (4.0 kPa). The clinical significance of some common PaO_2 and saturation values are listed (Table 1).

Oxygen Dissociation Curve

Tissue oxygenation depends partly on the Hb oxygen saturation and the shift of the oxygen dissociation curve. A shift of the curve to the right (Fig 1) favours Hb unloading of oxygen, and thus oxygen delivery to the tissues. Conversely, a shift to the left increases the

affinity of Hb for oxygen with reduced tissue oxygenation. The factors influencing the position of the curve are shown in Table 2.

Oxygen Flux and Requirements

Oxygen delivery to the cells is represented by the oxygen cascade (Table 3). The supply of oxygen is dependent upon the haemoglobin (Hb), oxygen saturation % (SaO_2) and cardiac output (Q). "Oxygen flux" denotes the total amount of oxygen delivered to the body per minute and is given by the equation:

Oxygen flux = 1.34 x Hb in g/dL x (SaO_2/100) x (Q in mL/min)/100 = 1000 mL/min

(1.34 = oxygen-carrying capacity of Hb in mL/g Hb.) The amount of oxygen carried dissolved in blood is negligible.

Hence the oxygen supply for a normal adult is approximately 1000 mL/min. However, not all of this amount is available for cellular utilization. Oxygen diffuses from tissue capillaries to mitochondria in cells. Mean tissue PO_2 varies from organ to organ and is higher near capillaries, especially near the arteriole end. Although mitochondria in tissue cells may operate at a PaO_2 of 8-40 mmHg (1.06-5.32 kPa), diffusion requires a capillary-tissue cell gradient. Thus tissue extraction of oxygen from blood is generally limited, and mitochondrial function jeopardized, at a PaO_2 of less than 300 mmHg (4.0 kPa) or a saturation of 30%. The available oxygen/min is therefore less than the supply (by about 250-300 mL/min) and is approximately 700 mL in a normal man.

Normal oxygen consumption at rest is about 200-250 mL/min. The oxygen reserve (availability minus consumption) in a normal man at rest is thus about 450-500 mL/min. Some factors in the sick person increase oxygen consumption greatly, i.e. fever, sepsis, shivering, restlessness and hypercatabolism. When other associated factors concomitantly reduce oxygen supply and availability, then the oxygen reserve may be reduced to critical levels and place the patient in jeopardy. A concept of minimum or "critical" oxygen flux of 400 mL/min is useful, below which tissue hypoxia may occur. Thus the use of additional oxygen to relieve hypoxaemia must be considered with measures to:

1. Reduce oxygen requirements (i.e. by cooling, paralysis, and controlled ventilation).

2. Increase oxygen availability (by correcting anaemia, low cardiac output, and adverse factors which shift the dissociation curve to the left).

Methods of Oxygen Therapy

The basic requirements of methods or devices for use in oxygen therapy are:

1. Control of fractional inspired oxygen concentration (FIO_2).
2. Prevention of excessive CO_2 accumulation.
3. Minimal resistance to breathing.
4. Efficient and economical use of oxygen.
5. Acceptance by patients.

The methods available to raise PaO_2 are shown (Table 4) and classified below. Oxygen head hoods, tents and incubators are used in paediatrics but are impractical in adults. Anaesthetic circuits and resuscitator bags are used to preoxygenate patients prior to endotracheal intubation. Oxygen administration is thus achieved largely by facemasks or nasal catheters.

Classification of Apparatus and Devices

It is important to know if the FIO_2 delivered by the apparatus will vary with the patient's ventilation. Apart low-flow spontaneous breathing circuits, some manual resuscitator bags, and ventilators, no device will deliver 100% oxygen, unless the oxygen is supplied at a rate greater than peak inspiratory flow rate (PIFR). PIFR in adults is about 25-35 L/min at rest, increasing markedly to over 60 L/min in dyspnoeic states. The apparatus and devices for oxygen therapy are classified below.

A. Fixed Performance Systems (FIO_2 is independent of patient factors.)

1. *High-flow Venturi-type Masks*

Oxygen flow entrains air by the venturi principle to delivery a fixed FIO_2 which remains accurate. Ventimasks (Vickers) come in separate oxygen concentrations, i.e. 24%, 28%, 35%, 40%. Other newer masks, i.e. Hudson, McGraw, Medi-shield, and Inspiron, use a facemask with a short "elephant trunk" hose attached to an interchangeable entrainment disc to allow a range of concentrations. The oxygen flow rate is set at 6-8 L/min depending on the FIO_2 chosen, entraining room air to give a resultant total flow rate of 40-60 L/min. Since room air is entrained, the use of a humidifier is not essential. The high-flow system also eliminates rebreathing and the need for a tight fit to the face. However, these masks may not deliver the intended FIO_2 if severe dyspnoea is present. The large PIFR in such patients may exhaust the reservoir in the newer, smaller volume masks, leading to the FIO_2 being lower and fluctuant. This is overcome by increasing the oxygen flow rate to 12-14 L/min (to give total inspired gas flows over 60 L/min).

2. *Low-flow Spontaneous Breathing Circuits*

These include anaesthetic circuits and circuits to deliver continuous positive airway pressure (CPAP) or spontaneous PEEP. These circuits incorporate a reservoir bag to deliver an FIO_2 set by the fresh gas mixture via an endotracheal tube or airtight mask.

B. Variable Performance Systems (FIO_2 depends upon oxygen flow, device factors and patient factors)

1. *No Capacity System*

Nasal catheter at low flow rates (less than 2 L/min). Hence there is insufficient oxygen storage in the airway during the expiratory pause to significantly affect the next inspiration. FIO_2 then depends upon the added oxygen flow rate and the peak inspiratory flow rate. In order to maintain the sam FIO_2, the added oxygen flow rate will need to be altered with each change in peak inspiratory flow.

2. *Small Capacity System*

(a) *Nasal catheters at high flows*

Significant oxygen storage occurs during the expiratory pause and varies with the length of the pause. Together with the breath-to-breath variation of PIFR, FIO_2 thus varies with ventilation. Moreover, major differences in alveolar oxygenation occur between mouth breathers and nose breathers, with a lower alveolar oxygen percentage found with the former group. The high flow rates may cause discomfort and drying of nasal mucosa. Nasal catheters are, however, cheap and easy to use, and the patient is able to eat or drink with them in situ. CO_2 rebreathing does not occur.

(b) *Simple, semi-rigid plastic masks* (i.e. MC, Edinburgh, Harris, Hudson)

Since some CO_2 rebreathing occurs, especially at low flows, the oxygen flow rate should be set at 4 L/min or greater. FIO_2 varies with patient ventilation and the oxygen flow rate (Table 5). A maximum concentration of only 60-70% oxygen is achieved by these masks. The tables of FIO_2 for these simple masks should only be used as guidelines. Large discrepancies between the delivered FIO_2 and that received by the patient (i.e. intratracheal FIO_2) occur with increasing rate and depth of breathing (i.e. increased PIFR). The standard oxygen flowmeter has a maximum flow rate of only 15 L/min and may not deliver adequate inspired oxygen. Special high-flow flowmeters or twin linked flowmeters are used in such a patient.

(c) *Tracheostomy masks*

These are small, plastic masks placed over the tracheostomy tube or stoma. The patient will inspire less oxygen than delivered, as dilution by room air occurs. Otherwise, they perform similarly to simple face masks.

(d) *T-piece circuit*

A T-piece is a simple, large bore, non-rebreathing circuit attached to an endotracheal or tracheostomy tube. Humidified oxygen is delivered through one limb of the T, and expired gas leaves via the other limb. The T-piece can be a fixed performance device if the fresh gas flow rate and the circuit volume are sufficient to provide for the patient's PIFR.

(e) *Face tent*

This is a large, semi-rigid plastic half-mask which wraps around the chin and cheeks. The oxygen mixture is delivered from the bottom of the mask, and gases are exhaled through the open, upper part. It is used to provide added humidification from a heated humidifier. Otherwise it has no advantages over the simple face mask.

3. *Large Capacity System*

Significant oxygen and CO_2 storage (i.e. rebreathing) occurs in these devices.

(a) *Soft plastic masks,* i.e. Pneumask, Polymask, Oxyaire. These masks have an added reservoir bag and thus a large effective dead space. FIO_2s greater than semi-rigid masks are possible, but considerable CO_2 rebreathing occurs if the oxygen supply fails or is reduced. They are potentially dangerous in patients without cardiopulmonary reserve and should be used with high oxygen flow rates. Rebreathing can be eliminated and delivered FIO_2 increased further if unidirectional valves are added, but asphyxia may occur in the unconscious patient if a valve become faulty.

(b) *Oxygen headbox, tents, cots and incubators* (see below).

Paediatric Oxygen Therapy

The PIFR of children, because of their smaller size, approximate more closely with the flow rate of oxygen delivery devices. Hence, higher FIO_2s are achieved. However, it is difficult to retain nasal catheters and masks on (especially restless) children. A single nasal catheter, placed at the level of the uvula and taped to the face, is well tolerated and is useful in infants and small children.

Oxygen Headbox or Hood

Oxygen is delivered into a box encasing the child's head and neck. The FIO_2 depends on the fresh gas flow, size of box, leak around the neck, head position, and how often the box is removed. It is a useful method in infants and small children, but high flow rates should be supplied and monitoring of oxygen concentration near the face is essential.

Incubator

Incubators provide oxygen as well as a neutral thermal environment. Patient access and recovery of oxygen concentration after opening the incubator are problems. The use of a headbox inside an incubator is common to give a more stable oxygen environment.

Oxygen Cot/Tent

Oxygen cots or tents may be used to nurse large children. Access, long recovery time for oxygen concentration and the difficulty to achievge high FIO_2s are problem.

Hazards of Oxygen Therapy

1. CO_2 Narcosis

When high oxygen concentrations are administered to patients with ventilatory failure who are dependent on a hypoxic drive, severe respiratory depression may occur, with loss of consciousness and, ultimately, death. This is seen particularly in patients with acute exacerbation of chronic bronchitis. Controlled oxygen therapy is needed in such cases. When patients are seen with CO_2 narcosis due to oxygen, the oxygen should not be withdrawn suddenly as a dangerous fall in PaO_2 will result. Such patients should be encouraged to breathe or, if unconscious, should be immediately ventilated.

2. Oxygen Toxicity

(a) *Neurological Effects (Paul Bert effects)*

Idiopathic epilepsy occurs with exposure to oxygen at more than 3 atmospheres absolute.

(b) *Lung Toxicity*

Pulmonary toxicity following exposure to high inspired concentrations of oxygen is a recognized clinical problem, but knowledge of the disorder remains limited. There is no specific pathological lesion, but progressive decrease in lung compliance occurs associated with the development of haemorrhagic interstitial and intra-alveolar oedema, and ultimately, fibrosis. The exact mechanism of the toxic effects of oxygen on the lung remains unknown, but it is generally thought that oxygen has a direct effect on lung tissue, affecting capillary endothelium before alveolar epithelium. A biochemical pathogenesis of oxygen-free radicals and hydrogen peroxide causing lung tissue injury has been suggested. Additional indirect factors that have been suggested include increased sympathetic activity, reduced surfactant activity, and absorption collapse. Differentiation of oxygen toxicity from other conditions of lung damage (i.e. ARDS) is extremely difficult, and the damage may be a common response to different types of injury.

It is generally agreed that oxygen pulmonary toxicity is dependent upon the duration of exposure and the concentration. However, precise details about "safe" periods of exposure and "safe" concentrations are unknown. Individual susceptibility to oxygen damage varies both within and among species. In patients with irreversible brain damage, ventilation with 100% oxygen produced changes in arterial oxygenation at 40 hours, and changes were reversible at this stage if the oxygen level was reduced to under 50%.

No damage was seen in volunteers breathing 50% for long periods, and no clinically relevant signs of toxicity appeared when 100% oxygen was used for less than 15-20 hours. Even when using high FIO_2s pulmonary toxicity does not always occur. The alveolar oxygen content or the FIO_2 appears to be more important, although the arterial tension (PaO_2) may modify oxygen tolerance. Damage in healthy lungs can occur, but whether the response is similar in lungs with pre-existing disease remains unclear. Patients with pulmonary injury may, paradoxically, be more resistant to oxygen toxicity. The converse may, however, be true. In general, clinical signs of toxicity (i.e. dyspnoea, substernal pain, deteriorating gas exchange and X-ray changes) are not normally detected with using oxygen less than 50% or 100% for short periods less than 24 hours.

(c) *Bronchopulmonary dysplasia,* a paediatric chronic lung disease originating in the neonatal period has similar abnormalities. This is seen when the immature lung is ventilated with high FIO_2s. Barotrauma is thought to be the major predisposing factor, but oxygen may accelerate the pathological process.

(d) *Retrolental Fibroplasia*

Blindness is caused by changes in retinal vessels. This occurs in premature babies under 1200 g weight (about 28 weeks) exposed to high oxygen concentrations, and relates to PaO_2 and retinal immaturity. Oxygen appears to stimulate immature vessels to vasoconstrict and obliterate, resulting in neovascularization, with haemorrhage, fibrosis, retinal detachment and blindness. PaO_2 should be kept between 50-80 mmHg (6.6-10.6 kPa).

Correct Clinical Use of Oxygen

Oxygen is a drug and has to be used correctly. It is given usually as a temporary measure to relieve hypoxaemia, but in no wat replaces the definitive treatment of the underlying cause. Oxygen therapy must be assessed by frequent measurements of blood gases and the alveolar-arterial oxygen gradient. PaO_2 must always be related to FIO_2 and the ventilation pattern. It is meaningless to quote the PaO_2 with no reference to the concentration or flow rate of oxygen. Finally, oxygen therapy must be *continuous not intermittent,* which may be harmful (with profound hypoxaemia).

In practice, oxygen administration for both adults and children over 50% should be given for as short a period as possible (except retrolental fibroplasia situations above), although when profound hypoxaemia is life-threatening (even 100%) concentrations should never be withheld. Conversely, restricting oxygen (using low concentrations) in hypoxic patients who are not oxygen sensitive is ignorant, stupid and dangerous. This is commonly seen with hypoxic patients who have raised $PaCO_2$, even though the hypercarbia is due to central depression from other causes (i.e. head injury or drug overdose) or neuromuscular failure (i.e. flail chest), and despite the hypoxia being profound. Other examples of this unfortunately common "asphyxia therapy" of rationing oxygen in hypoxia, is acute severe asthma (especially when the patient is hypocarbic), shock and sepsis. Hence, oxygen should never be restricted to hypoxic patients, except for those are sensitive (below).

1. Mild Hypoxaemia

Nasal catheters at 2-3 L/min or a simple mask at 4 L/min are suitable. If an oral (Guedel) airway is used, the nasal catheters must be placed inside the airway instead of the nares.

2. Moderate to Severe Hypoxaemia without CO_2 Retention

Example: when PaO_2 is approximately 50-60 mmHg (6.7-8.0 kPa). A simple mask is used with a flow rate of 4-15 L/min according to the PaO_2 and patient requirements. Extremely dyspnoeic patients with large PIFR will require oxygen delivered as high a flow as possible.

3. Hypoxaemia with CO_2 Retention

Controlled oxygen therapy with a venturi-type mask is used. A concentration of 24% is started, and blood gases are measured after 30-60 minutes. If the $PaCO_2$ is then below 75 mmHg (10 kPa), and if the rise in $PaCO_2$ is less than 10 mmHg (1.3 kPa), then the oxygen

is increased to 28%. Since these patients lie on the steep part of the oxygen dissociation curve, a small rise in PaO_2 will result in a relatively large increase in oxygen available to tissues. The FIO_2 may be increased further in the same way if hypoxaemia persists.

Nasal catheters at low flows may be used but are not ideal. At higher flows, controlled oxygen therapy cannot be achieved with these catheters.

4. Profound Hypoxaemia

Example: when PaO_2 is less than 50 mmHg (6.7 kPa). Mechanical ventilatory support is indicated. Continuous positive airway pressure (CPAP) by mask may be tried initially to avoid intubation. Positive end expiratory pressure (PEEP) may be used, to help reduce the FIO_2 being given.