EMERGENCY MEDICINE

Oxygen Therapy

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Abstract

The primary goal of oxygen therapy is to correct alveolar and/or tissue hypoxia. Therefore, any disorder causing hypoxia is a potential indication for oxygen administration. But the tissue oxygen delivery depends upon an adequate function of cardiovascular (cardiac output and flow), haematological (Hb and its affinity for oxygen) and the respiratory (arterial oxygen pressure) systems. Therefore, tissue hypoxia is not relieved by oxygen therapy alone – functioning of all the three organ systems also needs to be improved.

Oxygen therapy should be administered according to guidelines. Proper monitoring of oxygen therapy is recommended to ensure adequate oxygenation and to save precious oxygen from wastage. The use of pulse oximeter is a simple, quick, non-invasive, and reliable method to assess it.

Introduction

Many biochemical reactions in the body depend on oxygen utilisation. Supply of oxygen to the tissues depends on many factors like ventilation, diffusion across alveolar-capillary membrane, haemoglobin, cardiac output, and tissue perfusion. Oxygen therapy is required for respiratory failure in many conditions like severe asthma, chronic bronchitis, pneumonia, and myocardial infarction, etc. Through this article, we have made an attempt to review the physiological basis of hypoxia, basis of oxygen therapy, its indications, administration devices, and its hazards.

Respiratory system

The respiratory system is concerned with the delivery of an adequate amount of oxygen to and elimination of a corresponding amount of carbon dioxide from the cells of the body and maintenance of normal acid-base balance in the body. Proper supply of oxygen and elimination of carbon dioxide from various tissues of the body depends on the optimal functioning of various parts of the respiratory system like chest wall and respiratory muscles, airways and lungs, CNS (including medullary respiratory centres), spinal cord, CVS, and endocrine system. A disorder in any portion of these systems can lead to respiratory failure.

Respiratory failure

During respiratory failure, there is an inability to keep the arterial blood gases at normal level, while breathing air at rest at sea level, and the partial pressure of oxygen is usually below 60 mmHg with or without partial pressure of carbon dioxide above 49 mmHg in arterial blood.

Types

Respiratory failure may be acute or chronic. Acute respiratory failure develops suddenly or slowly if lungs are already diseased; while chronic respiratory failure develops slowly due to underlying lung disease. Respiratory failure may occur even if lungs are normal as in diseases of nervous system, chest wall, or upper airways. Inadequate gas exchange is associated with hypoxaemia with or without hypercarbia (Type-1 respiratory failure or lung failure), while inadequate ventilation leads to hypoxaemia with hypercarbia (Type-2 or ventilatory failure). Type-1 respiratory failure occurs when there is disease of peripheral gas exchanging parts of body and type-2 in COPD, bronchial asthma, neuromuscular disease, and chest wall disorders.

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Different causes of acute respiratory failure are  

### A. Defective ventilation
- Respiratory centre depression
- Drugs such as narcotics, anaesthetics, and sedatives
- Cerebral infarction
- Cerebral trauma
- Neuromuscular disorders
  - Myasthenia gravis
  - Guillain-Barre syndrome
  - Brain or spinal injuries
  - Polio, porphyria, botulism.
- Airways obstruction
  - Chronic obstructive pulmonary disease
  - Acute severe asthma
- Restrictive defects
  - Interstitial lung disease
  - Kyphoscoliosis, ankylosing spondylitis
  - Bilateral diaphragmatic palsy
  - Severe obesity

### B. Impaired diffusion and gas exchange
- Pulmonary oedema
- Acute respiratory distress syndrome
- Pulmonary thromboembolism
- Pulmonary fibrosis

### C. Ventilation-perfusion abnormalities
- Chronic obstructive pulmonary disease
- Pulmonary fibrosis
- Acute respiratory distress syndrome
- Pulmonary thromboembolism

### Management
The aims of therapy in respiratory failure are to achieve and maintain adequate gas exchange and reversal of the precipitating process that led to the failure. In type-1 respiratory failure, high concentration of oxygen is given to correct hypoxaemia. At the outset it should be determined whether the hypoxaemia can be relieved by oxygen therapy alone or it needs oxygen and ventilatory intervention. The decision is made on the presence or absence of hypercapnia and of lung disease. Patients with ARDS do not improve with simple oxygen therapy and they need mechanical ventilation (Positive end expiratory pressure - PEEP). In type-2 respiratory failure with previous normal lungs, there is inadequate alveolar ventilation and in these patients ventilatory assistance is needed. In patients with previous lung disease as in acute exacerbation of COPD, controlled oxygen therapy is needed. Mechanical ventilation should be avoided in patients with COPD as the weaning from the ventilator is very difficult.

### Hypoxia and hypoxaemia
Hypoxia is lack of oxygen at the tissue level while hypoxaemia implies a low arterial oxygen tension below the normal expected value (85-100 mmHg).

### Hypoxaemia due to pulmonary disorders:
Presentation of acute respiratory failure is sudden in a previously healthy person while it is gradual in a patient with pre-existing chronic respiratory disease. There are signs and symptoms of hypoxaemia along with those of the underlying and/or complicating disease. The general features attributed to hypoxaemia are restlessness, palpitation, sweating, altered consciousness, headache, confusion, and cyanosis. Blood pressure may initially rise but it falls as the severity of hypoxaemia worsens. Hypercapnia accompanies hypoxaemia whenever there is hypoventilation, such as conditions listed at A in Table.

During acute respiratory failure we require higher concentration of oxygen initially to correct hypoxaemia so as to prevent organ damage. Not only the correction of PaO\textsubscript{2} but also oxygen delivery to the tissue and its proper utilisation are equally important. Diagnosis is made from the total clinical presentation of the disease and the relevant investigations. Acute respiratory failure is established by blood gas and pH determinations.

### ARDS
In such cases to correct hypoxaemia, ventilator controlled administration of oxygen often with PEEP.
(positive end expiratory pressure) is required. The desirable PaO₂ of about 60 mmHg with lowest possible FiO₂ is achieved with PEEP of about 10-15 cmH₂O. After the initial 24 hours, FiO₂ should not exceed 60% (to reduce the risk of O₂ toxicity).

Acute severe bronchial asthma
Patients with acute severe asthma or status asthmaticus have severe airways obstruction and inflammation. They are generally hypoxaemic. Hypoxaemia is corrected by giving oxygen via nasal cannula or face mask at a flow rate of 4-6 L/min to achieve FiO₂ of 35-40%. Flow rate may be adjusted to maintain PaO₂ of about 80 mmHg or more. The risk of hypercarbia and CO₂ narcosis is more in COPD rather than acute severe asthma and in such cases assisted ventilation is required. Administration of sedatives and tranquilizers must be avoided. Sedatives may precipitate the CO₂ retention not only in patients with COPD but also in asthma.

Severe pneumonia
In severe acute viral or bacterial pneumonias, there may be hypoxaemia and respiratory failure. Oxygen is given at a flow rate of 4-6 L/min to achieve PaO₂ above 60 mmHg. Bronchial hygiene and treatment with antibiotics and other drugs is meanwhile continued.

Interstitial lung disease
Patients may have respiratory failure due to fulminant onset or because of intercurrent infection. The lungs are stiff and compliance is low. As these patients need oxygen for prolonged periods, one should wean oxygen to FiO₂ of about 40% as early as possible. Some patients may become dyspnoeic even after mild exertion and such cases benefit from oxygen administration before and after physical activity.

Pulmonary thromboembolism
Hypoxaemia in the presence of pulmonary thromboembolism is common but not essential. Oxygen is required when there is breathlessness and hypoxaemia which depends on the amount of pulmonary circulation occluded.

Pulmonary infarction is prevented by alveolar oxygen and systemic bronchial vascular anastomosis which can be enriched with oxygen therapy.

Spontaneous pneumothorax and pneumomediastinum
Hypoxaemia may be precipitated by these in a patient having pre-existing lung disease. 100% oxygen may be given for a short period as it causes denitrofication of pleural and mediastinal air which is then ultimately absorbed in the circulation. Such a modality should not be tried beyond 12-16 hours to avoid oxygen toxicity.

Indications for oxygen therapy
Oxygen can be given in high or low concentration in all the conditions associated with hypoxaemia. In conditions like COPD in which there is a risk for hypercarbia, low concentration should be used.

In acute lung conditions (without underlying chronic lung disease) like pulmonary embolism, pneumonia, tension pneumothorax, acute severe asthma, pulmonary oedema, or myocardial infarction, a higher concentration of oxygen can be given. Similarly in fibrosing alveolitis, there is no retention of CO₂, so high concentration can be given as in these conditions there is no danger of induction of hypoventilation. Maintaining PaO₂ above 60 mmHg gives O₂ saturation of 90%. During acute exacerbation of COPD, chemoreceptor drive for ventilation is eliminated which leads to reduced alveolar ventilation. Hypoxaemia should be reduced immediately by giving oxygen generally in a concentration of 24% to improve oxygenation without losing the respiratory stimulant effect.

Goals of oxygen therapy
The goal is to relieve hypoxaemia by increasing alveolar tension, to reduce the work of breathing, and to decrease the work of myocardium. Oxygen
should be used like a drug in various conditions and its dose should be individualised. Arterial blood gases should be measured repeatedly in patients with acute respiratory failure on oxygen therapy. The goal is to maintain PaO₂ above 60 mmHg. Oxygen should be given in low dose continuously since small increase in FiO₂ causes increase in PaO₂ as most patients of COPD lie on the steep part of oxy-haemoglobin curve. O₂ can be calculated by the formula 20+4xO₂ flow (L/min). 

Monitoring oxygen therapy

Oxygen therapy should be given continuously and should not be stopped abruptly until the patient has recovered, since sudden discontinuation can wash-out small body stores of oxygen resulting in fall of alveolar oxygen tension. The dose of oxygen should be calculated carefully. Partial pressure of oxygen can be measured in the arterial blood. Complete saturation of haemoglobin in arterial blood should not be attempted. Arterial PO₂ of 60 mmHg can provide 90% saturation of arterial blood, but if acidosis is present, PaO₂ more than 80 mmHg is required. In a patient with respiratory failure, anaemia should be corrected for proper oxygen transport to the tissue. A small increment in arterial oxygen tension results in a significant rise in the saturation of haemoglobin. Under normal situations, no additional benefit is secured by raising PaO₂ level to greater than 60 to 80 mmHg. An increase of 1% oxygen concentration elevates oxygen tension by 7 mmHg. It is necessary to maintain normal haemoglobin level in the presence of respiratory disease as proper oxygen transport to the tissues is to be maintained.

Measurement of arterial blood gases repeatedly is difficult so a simple and non-invasive technique like pulse oximeter may be used to assess oxygen therapy.

When to stop oxygen therapy

Weaning should be considered when the patient becomes comfortable, his underlying disease is stabilised, his BP, pulse rate, respiratory rate, skin colour, and oxymetry are within normal range. Weaning can be gradually attempted by discontinuing oxygen or lowering its concentration for a fixed period for e.g., 30 min. and re-evaluating the clinical parameters and SpO₂ periodically. Patients with chronic respiratory disease may require oxygen at lower concentrations for prolonged periods. Prospects
of long term domiciliary oxygen need to be discussed with the patient after the acute need is over.

Dangers of oxygen therapy

There are three types of risks associated with oxygen use.

1. Physical risks

Oxygen being combustible, fire hazard and tank explosion is always there. This is more with high concentration of oxygen, use of pressure chambers, and in smokers. Catheters and masks can cause injury to the nose and mouth. Dry and non-humidified gas can cause dryness and crusting.

2. Functional risks

Patients who have lost sensitivity to CO₂ and are upon the hypoxic drive are in danger of ventilatory depression as seen in patients of COPD. Hypoventilation can lead to hypercapnia and CO₂ narcosis although the risk is small with low flow oxygen therapy. Arterial pH may be a better guide than PaCO₂ for monitoring oxygen therapy. As long as pH does not suggest acidosis, long term oxygen therapy can benefit the patients with CO₂ retention.

3. Cytotoxic damage

COPD patients on long term oxygen therapy, on autopsy, show proliferative and fibrotic changes in their lungs. In acute conditions, most of the structural damage occurs from high FiO₂ as the oxygen can lead to the release of various reactive species which attack the DNA, lipids, and SH-containing proteins.

Long term oxygen therapy

Long term domiciliary oxygen therapy was used initially for patients with COPD and chronic respiratory insufficiency but now its use has also been extended to patients with breathing disorders during sleep or exercise.

Selection of patients

Following guidelines are used to select patients for instituting the treatment.

1. A definite documented diagnosis responsible for chronic hypoxaemia.
2. An optimal medical treatment should be in effect.
3. Patient in a stable condition.
4. Oxygen administration should have been shown to improve hypoxaemia and provide clinical benefit in such patients.

The following specific indices are used while prescribing long term oxygen therapy:

1) At rest in non-recumbent position, PaO₂ ≤ 55 mmHg.
2) Patient with PaO₂ more than 55 mmHg if:
   a) While on optimal medical treatment, shows features of hypoxic organ dysfunction like secondary pulmonary hypertension, cor-pulmonale, polycythaemia, or CNS dysfunction.
   b) Patient shows fall in PaO₂ below 55 mmHg during sleep with disturbed sleep pattern, cardiac arrhythmias, or pulmonary hypertension. These patients are benefitted by nocturnal oxygen therapy.
   c) There is fall in PaO₂ during exercise and oxygen therapy improves exercise tolerance.

Benefits

Long term oxygen therapy benefits patients with COPD and other chronic pulmonary diseases with hypoxaemia as it increases their survival and quality of life. Patients of interstitial lung disease become comfortable and there occurs improvement in pulmonary hypertension and right heart failure.

Oxygen dosage

COPD patients are given oxygen at the rate of 1-
2 L/min. Some of the patients with other chronic respiratory diseases may require higher flow rates. PaO$_2$ should be maintained at 60 mmHg or so. During sleep or exercise or other activities, flow rate may be increased by 1-2 L/min.

Home oxygen

The aim of oxygen therapy at home is to make the patient active and encourage exercise and other activities outside the home. Patients of COPD with hypoxaemia at rest, having arterial PaO$_2$ below 55 mmHg or patients with cor pulmonale or secondary polycythaemia having PaO$_2$ between 55-59 mmHg in a stable clinical state need home oxygen.

Two types of oxygen systems are available for use at home:

a) Stationary (Compressed high pressure gas cylinders or O$_2$ concentrators):
   
   These are useful for bedridden patients. They are of low cost. They separate oxygen from nitrogen by adsorption principle of a molecular sieve. They are not portable and create a lot of noise. They need backup of tank system if there is electricity failure.

b) Portable system (Transfilling gaseous or liquid system):
   
   They are useful for ambulatory patients including those who have to remain away from house for work. They are light weight and can provide oxygen at the rate of 2 L/min for about 6-9 hours. Oxygen is filled from a stationary source. They are costly. Oxyspec (single prong nasal cannula), oxymizer and oxymizer pendant conserver cannula (their reservoir system stores oxygen during exhalation), electronic demand oxygen delivery system (Pulser supplying short bolus of enriched oxygen early in inspiration on triggering of inspiratory sensor by negative nasal pressure), and small polythene transtracheal catheter (for transtracheal oxygen administration) are thought to be useful to improve efficiency of oxygen delivery, to reduce cost of oxygen delivery, and to improve cosmetic appearance.

Perspectives of domiciliary oxygen use in India

As the facilities and expertise for domiciliary oxygen therapy are available mainly in cities, use is restricted mainly to cities. Although this sector is not organised, more and more people are using this facility and with increased compliance. At present, there is no organised supply of oxygen and the cost is high. Supply is difficult in rural areas. Also, the patients have to be selected carefully taking into account their education, income, and social status.

Various studies in India show that although the treatment is still irregular and inadequate, patients show definite improvement in well-being and prolongation of life span. Limited sources of supply, high costs, difficulty in procurement of oxygen, lack of medical expertise, no clear-cut policy on reimbursement to employees, all come in the way of its frequent use. Let us hope these difficulties are resolved in due course of time.

Conclusion

With these basic guidelines for oxygen administration, one can easily deliver a fairly consistent concentration. The hazards of oxygen toxicity must be kept in mind but hypoxia must not be left untreated in view of toxicity since hypoxia is common and the damage it causes is rapid and severe in comparison to oxygen toxicity which is uncommon and even pulmonary injury caused by it is relatively slow in development.

References


