

Oxygen Therapy - An Overview

Maj Saleem Ahmed MBBS, FCPS

Classified Anaesthesiologist
CMH Peshawar

Oxygen is a normal constituent of air, which is required for aerobic metabolism within the cell. In the absence of O_2 , anaerobic metabolism takes place, which causes acidosis and electrolyte imbalance, hence jeopardizing the life of the cell.

Oxygen is carried from the atmosphere to the cell in two forms in the blood. One is combined with hemoglobin and the other is in the dissolved form. The oxygen is off loaded at the tissue level and the blood then returns to the heart and lungs for reoxygenation.

The oxygen utilization takes place at the mitochondria. When the PO_2 drops, then anaerobic metabolism starts. This is the situation, which is called "hypoxia" for which we administer O_2 .

There are four types of hypoxia, namely

- a. Hypoxic hypoxia
- b. Anaemic hypoxia
- c. Apnoeic hypoxia
- d. Histotoxic hypoxia

Description of types of hypoxia is beyond the scope of this article.

At this point if we examine oxygen hemoglobin curve, it will be evident that the curve is steep below 60 mm Hg PO_2 . This means that the oxygen reserve is low and O_2 off-load is difficult. It hampers O_2 supply to the cells. So whenever PO_2 goes below 60 mmHg the cells will suffer hypoxia. In such a situation oxygen therapy is indicated.

INDICATIONS:

The common clinical indications are

1. Acute Respiratory Failure:
 - a: without CO_2 retention, e.g. asthma, pneumonia, pulmonary edema and pulmonary embolism.
 - b: with CO_2 retention, e.g. ch bronchitis, chest injuries, drug overdose, post operative hypoxaemia and neuromuscular disease.
2. Acute myocardial infarction.
3. Cardiac failure.
4. Shock due to any cause.

5. Increased metabolic demands e.g. Burns, multiple injuries and severe infections.
6. Post operative state.
7. Carbon monoxide poisoning.

METHODS OF OXYGEN THERAPY:

The basic requirements of a method or device 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.

CLASSIFICATION OF APPARATUS AND DEVICES.

A. Fixed Performance System.

1. High flow venturi-type masks (FiO_2 is independent of patient factors)

Oxygen flow rate is set at 6-8 L/min. and with air entrainment total flow rate becomes 40-60 L/min. These mask may not deliver the intended FiO_2 if severe dyspnoea is present.

2. Low – flow Spontaneous Breathing Circuits. These include anaesthetic circuits and circuits to deliver continuous positive airway pressure (CPAP) or 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 catheters at Low flow rates (less than 2L/min)

2. Small Capacity System.

- a) Nasal catheters at high flows.
- b) Simple semirigid plastic masks e.g. M.C., Edinburgh, Harrio, Hudson).

- c) Tracheostomy masks.
- d) T-piece circuit
- e) Face tent

3. Large Capacity System

- a. Soft plastic masks e.g. Pneumask, Polymask, Oxyaire.
- b. Oxygen headbox, tents, cots and incubators.

PAEDIATRIC OXYGEN THERAPY

- a. Oxygen Head box or
- b. Incubators.
- c. Oxygen Cot / tent.

Following table shows the efficiency of the described systems.

Apparatus / Device	O ₂ Flow L/min	% Conc. Achieved
Nasal Catheters	2-6	25-40
Semirigid masks eg (MC, Edin burgh, Hudson, Harris)	4-15	35-70
Venturi-Type mask e.g ventimask	6 -12	25-60
Soft Plastic masks. eg. Pnenmask, Polymask, OXgaire	4-15	40-80
Ventilators	varying	21-100
Anaesthetic circuits	varying	21-100
Plastic head hood	4-8	30-50
Oxygen Tents and Cot	7-10	60-80
Incubators.	3-8	upto 40.

HAZARDS OF OXYGEN THERAPY

1. Carbon Dioxide Narcosis.

This happens in patients who are suffering from COAD and are maintaining ventilation because of hypoxic drive. When higher concentration of O₂ is given the hypoxic drive goes away and the patient starts hypoventilating ultimately going into CO₂ narcosis.

2. Oxygen Toxicity

- a. Neurological Effects (Paul Best effects)
- b. Lung Toxicity
- c. Bronchopulmonary dysplasia
- d. Retrolental Fibroplasia

CORRECT CLINICAL USE OF OXYGEN .

Oxygen is a drug and has to be used correctly. Oxygen is given usually as a temporary measure to relieve hypoxaemia, but in no way replaces the definitive treatment of the underlying cause. Oxygen therapy must be assessed by frequent measurements of ABGs and alveolar – arterial oxygen gradient. It must be

continuous and not intermittent.

Oxygen administered for both adults and children over 50% should be given for as short a period as possible. But when profound hypoxaemia is life threatening, higher even 100% concentration is never withheld.

1. Mild hypoxaemia

Nasal catheters at 2-3 L/min or a simple mask at 4L/min are suitable.

2. Moderate to sever Hypoxaemia without CO₂ retention.

PaO₂ is approximately 50-60 mmHg.

Simple mask with flow rate of 4-15 L/min.

Extremely dyspnoeic patient with large PIFR requires highest possible flows.

3. Hypoxaemia with CO₂ retention.

Controlled O₂ therapy with a venturi type mask 24% concentration is started and ABGs are measured every 30 min to 60 min. PaCO₂ is < 75 mm Hg and rise is less than 10 mmHg, then the O₂ is increased to 25%. If hypoxaemia persists then increase FiO₂ gradually to 40 to 50%.

4. Profound hypoxaemia.

PaO₂ is less than 50 mmHg. Mechanical ventilation is indicated.

CPAP by mask may be tried.

If high FiO₂ is required to maintain O₂ saturation in the blood then PEEP may have to be used to help reduce FiO₂.

BIBLIOGRAPHY

1. Nunn JF: Oxygen. Applied Respiratory Physiology 3rd Ed. Butterworths, London, 1987
2. Shaprio BA, Harrison R.A, Kacmarek RM, Cane RD: Evaluating Bronchial Hygiene Therapy Clinical Application of Respiratory Care 3rd Ed. Year Book Medical Publishers, Chicago, 1985.
3. Basic Pathology: Robbins and Kumar 5th Ed. W.B. Saunders.
4. Harrison's Principal of internal Medicine 12th Ed. McGraw hill, International Book Company.
5. National conference on Oxygen Therapy: Chest 82:234, 1984
6. Anaesthesia Ronald D. Miller, 3rd Ed Churchill Livingstone.
7. Davidson's Principles and Practice of Medicine. Christopher R.W. Edwards 16th Ed. Churchill Livingstone.
8. Clinical Medicine; Parveen and Michael Clark, 3rd Ed. Bailliere Tindall.
9. Winter PM, smith G. The toxicity of oxygen . Anaesthesiology 37: 210, 1972.
10. Nash G, Blennerhasset JB, Pontoppidan H: Pulmonary lesions associated with oxygen therapy and artificial ventilation. N.Engl. J. Med 276: 368, 1967.
11. Freeman B, Crapo J: Hyperoxia increases O₂ radical production in rate lungs and lung mitochondria J Biol chem 256:10986,1981.