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PERIOPERATIVE CRITICAL CARE



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CHAPTER

2

Oxygen Therapy in the Perioperative Period

Bindiya Salunke, Madhusudan Jaju, Amol Kothekar

INTRODUCTION

Oxygen is essential element required for aerobic metabolism for the synthesis of adenosine triphosphate (ATP) within mitochondria and for survival. ATPs are the primary energy source for all active metabolic processes. Concentration of oxygen in the air at or near sea level (21% or partial pressure of 160 mm Hg) is more than sufficient for healthy individuals with intact respiratory drive. Oxygen supplementation is often required in perioperative period due to wide range of pathophysiological effects of anesthesia and surgery on respiratory function and lung physiology. Hence, understanding the respiratory physiology in relation to oxygen and pathophysiology of conditions needing oxygen supplementation is vital for management of perioperative patient. Oxygen is considered as a lifesaving drug in various emergencies; however, there is a growing evidence of toxicities associated with excess oxygen use. Hence, it is equally important to avoid its use in the unindicated cases.

PHYSIOLOGY

Atmospheric oxygen enters the body by the process known as respiration, the process of carrying oxygen to the tissues, which is known as transportation. Oxygen utilization is the process of oxidation of glucose into ATP, water and carbon dioxide. The processes of respiration and transportation are described in brief.

Respiration

It deals with the transport of atmospheric oxygen from the alveoli and subsequently into the blood. Alveolar gas equation explains the physiology of the former component of transport of atmospheric oxygen to the alveoli. Alveolar arterial gradient (A–a gradient) deals with the later component of transport of alveolar oxygen into the arterial blood.

Alveolar Gas Equation

The simplified alveolar gas equation is described here:[1]

$$PAO_2 = (P_{atm} - PH_2O)\ FiO_2 - PaCO_2/R$$

where,

PAO_2 = partial pressures of O_2 in the alveolar gas

P_{atm} = the atmospheric pressure (At sea level average value = 760 mm Hg)

PH_2O = partial pressure of water (Average value = 45 mm Hg)

FiO_2 = the fraction of inspired oxygen (Average value = 0.21)

$PACO_2$ = partial pressures of CO_2 in the alveolar gas (average value = 40–45 mm Hg)

R = the respiratory exchange ratio VCO_2/VO_2, in which VCO_2 is the rate of CO_2 elimination from the lungs and VO_2 is the rate of O_2 uptake from the alveolar gas (average value = 0.8–0.82)

Putting all these values in the equation, alveolar oxygen pressure is calculated below:

$$PAO_2 = (760 - 47)\ 0.21 - 40/0.8 = 149 - 40/0.8$$
$$= 149 - 50 = 99 \text{ mm Hg (approximately)}$$

Clinical utility of alveolar gas equation: Alveolar gas equation highlights three key determinants of alveolar oxygen pressure, viz., atmospheric pressure, fraction of inspired oxygen (FiO_2), and partial pressure of carbon dioxide in alveoli (blood).

Decrease in the partial pressures of O_2 in the alveolar gas (PAO_2) at high altitude is due to drop in atmospheric pressure. Decrease in PAO_2 can also occur when there is drop in FiO_2. The older anesthesia machines without 'link 25' safety mechanism had potential risk for delivering hypoxic mixture by possible decrease in FiO_2 when incorrect flow settings of oxygen and nitrous oxide were used.

Alveolar gas equation beautifully explains decrease in PAO_2 in conditions which increase partial pressure of carbon dioxide (PCO_2), e.g., respiratory depression. For each 8 mm Hg increase in PCO_2, there is 10 mm Hg drop in PAO_2. When PCO_2 increases from 40 to 80 mm Hg, PAO_2 drops by 50 to the value of 49 mm Hg.

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PAO_2 (at PCO_2 of 40) = (760 – 47) 0.21 – 40/0.8 = 149 – 40/0.8
= 149 – 50 = 99 mm Hg
PAO_2 (at PCO_2 of 48) = (760 – 47) 0.21 – 48/0.8 = 149 – 48/0.8
= 149 – 60 = 89 mm Hg]
PAO_2 (at PCO_2 of 80) = (760 – 47) 0.21 – 80/0.8 = 149 – 80/0.8
= 149 – 100 = 49 mm Hg

This is one of the common causes of perioperative hypoxemia. The physiological treatment of it is normalization of PCO_2 rather than supplemental oxygen. Alveolar gas equation is essential for calculation of alveolar–arterial or A–a gradient which can be used for identification of cause of hypoxemia (see later).

Alveolar–arterial Gradient (A–a Gradient)

Oxygen diffuses from the alveoli, against a pressure gradient into the mixed venous blood of the alveolar capillaries. Hence, partial pressure of oxygen in arterial blood (PaO_2) will always be lesser than the alveolar partial pressure of oxygen (PAO_2).

Oxygen diffuses from alveoli to the capillary blood through a membrane known as alveolar capillary membrane. As oxygen diffuses across a pressure gradient, the alveolar PAO_2 (upstream) will always be higher than arterial (downstream) PaO_2 **(Fig. 1)**.

Alveolar–arterial gradient (A–a gradient) is the difference between the partial pressure of oxygen in alveoli (A) and the artery (a), and can be calculated by the following formula:

$$\text{A–a gradient} = PAO_2 - PaO_2$$

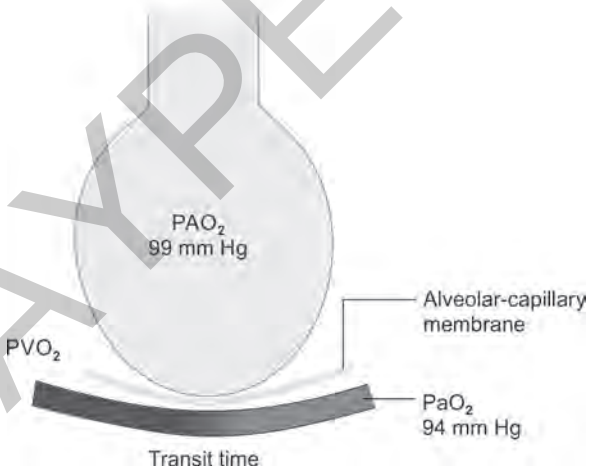


Fig. 1: Schematic diagram showing anatomy of alveolar unit. Oxygen gradient between alveolar and arterial blood is known as alveolar–arterial gradient (A–a gradient).

(PAO_2: partial pressures of oxygen in alveolar gas; PaO_2: partial pressure of oxygen in arterial blood; PVO_2: mixed venous partial pressures of oxygen)

Table 1: Alveolar–arterial gradient (A–a gradient) and efficacy of 100% oxygen to correct PaO_2 in various clinical conditions.[2]

Disease state	*A-a gradient*	*Correctable with 100% oxygen*	*Causes*
Low inspired pO_2	Normal	Yes	High altitude
Hypoventilation	Normal	Yes	CNS depression, neuromuscular disease, skeletal abnormalities of the chest wall, opiate and other CNS depressant use
Diffusion disorder	Elevated	Yes	Interstitial lung disease
V/Q mismatch	Elevated	Yes	• *Increased V/Q*: Chronic bronchitis, mucus plugging, pulmonary edema • *Decreased V/Q*: Pulmonary embolism, emphysema
Shunting	Elevated	No	• *Alveolar*: Pneumonia, severe pulmonary edema, atelectasis, ARDS • *Cardiac*: Eisenmenger syndrome, cyanotic heart disease • *Vascular*: Pulmonary AV shunt

(ARDS: acute respiratory distress syndrome; AV shunt: arteriolar venous shunt; CNS: central nervous system; V/Q: ratio between ventilation and perfusion of lungs; pO_2: partial pressure of oxygen; PaO_2: partial pressure of arterial oxygen)

A young nonsmoker adult has A–a gradient of 5–10 mm Hg when breathing room air. This gradient can go up to 30 at 100% FiO_2.

Clinical utility of alveolar–arterial gradient: Calculation of A–a gradient can help in diagnosis of underlying mechanism of decrease in PaO_2 (hypoxia). **Table 1** explains the A–a gradient in various disease states. It also differentiates clinical conditions based on the efficacy of 100% oxygen to correct PaO_2.

Oxygen Transportation

Oxygen Contents of Blood

The major component of oxygen binds with hemoglobin and minor component is dissolved in the plasma. Oxygen content of the blood can be measured by the formula given below:

Arterial oxygen content = bound oxygen + dissolved oxygen

$$CaO_2 = (SaO_2 \times Hb \times \text{Oxygen combining capacity of hemoglobin}) + (\text{oxygen solubility} \times PaO_2)$$
$$= (SaO_2 \times Hb \times 1.31 \times 0.01) + (0.0225 \times PaO_2)$$

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where,

- 1.31 = Hüfner's constant
- Hb = the amount of hemoglobin (g/dL)
- SaO_2 = the arterial hemoglobin saturation in percent
- 0.0225 = the solubility coefficient of oxygen at body temperature
- PaO_2 = the partial pressure of oxygen in arterial blood in kilopascals (kPa).

At Hb of 15 g/dL, saturation of 100% and PaO_2 of 99 mm Hg (13.19 kPa), oxygen content in the blood is 19.94 mL (almost 20 mL) for each 100 mL or 200 mL for each 1,000 mL or one liter, out of which 19.65 mL is carried by hemoglobin and 0.29 mL is dissolved.

Oxygen Delivery and Extraction

At cardiac output of 5 L/min, 1,000 mL of oxygen (SpO_2 100%) is delivered to the tissues out of which 250 mL is utilized and 750 mL (SvO_2 or mixed venous saturation of 75%) returns to the heart. Oxygen consumption is roughly 250 mL/min. Factors affecting oxygen consumption are listed in **Table 2**.

At higher altitude, inspired oxygen drops due to drop in atmospheric pressure leading to proportionate drop in PAO_2. Body compensates by CO_2 washout to reduce PCO_2 (see alveolar gas equation) and by increasing hemoglobin to increase oxygen content of the blood.

Physiologic Determinants of Oxygenation

Shunt fraction and Functional Residual Capacity play major role in the physiology of oxygenation.

Shunt Fraction

Pulmonary shunt is an area of the lung which gets perfusion in the absence of ventilation. The cardiac output reaching shunt area does not take part in oxygenation. Pulmonary shunt can be quantified in percentage, known as shunt fraction. The normal range of shunt fraction for an adult is around 4–10%. In pathological conditions, the shunt fraction is significantly greater and even breathing 100% oxygen does not fully oxygenate the blood. Classic example is acute respiratory distress syndrome (ARDS), application of positive end-expiratory pressure (PEEP) "recruits" the collapsed alveoli reducing shunt fraction and hence improving oxygenation.[3]

Table 2: Factors affecting oxygen consumption (VO_2).

Increased VO_2	*Decreased VO_2*
Exercise and physiotherapy	*General anesthesia*: Sedation, muscle relaxants, analgesics
Trauma (Surgery)	Antipyretics
Shivering and agitation	Mechanical ventilation
Inflammation, sepsis, pyrexia	Shock state

Functional Residual Capacity (FRC)

The amount of air left in the lung at the end of normal expiration is called functional residual capacity (FRC). Two most important functions of FRC are:

1. It helps in prevention of alveolar collapse and in healthy lungs; most of the alveoli are open at FRC. In diseased lungs (pulmonary edema, ARDS, etc.) there is collapse of significant number of alveoli at FRC causing hypoxemia due to shunt. Addition of PEEP (either invasive or noninvasive) increases FRC which helps in opening of these alveoli, reduces shunt and improves oxygenation. PEEP may also improve lung compliance and reducing work of breathing as it is easy to inflate already opened alveoli than to reopen closed alveoli.
2. It represents the oxygen store available for gas exchange during the apnea. Preoxygenation or denitrogenation of FRC replaces air or nitrogen for oxygen. Adequate preoxygenation helps in providing safe apnea time for airway procedures like intubation or bronchoscopy.

Time required for the desaturation after apnea depends on FRC and oxygen consumption and hence varies from individual to individual. Larger FRC and reduced oxygen consumption provide longer apnea time.

HYPOXIA VS. HYPOXEMIA

Hypoxemia is defined as a decrease in the partial pressure of oxygen in the blood. Hypoxia is defined by reduced level of tissue oxygenation.

Types of Hypoxia

Following types of hypoxia are described:

1. *Hypoxic hypoxia*:[4] This is further divided into:
   a. Diminished oxygen content
   b. Obstruction of the airway
   c. Abnormal lung conditions
   d. Pulmonary shunt or extrapulmonary right to left shunt
2. *Anemic hypoxia*: Due to reduced hemoglobin
3. *Stagnant hypoxia*: due to reduced cardiac output or reduced regional blood flow
4. *Histotoxic hypoxia*: (e.g., cyanide poisoning)
5. *Cytopathic hypoxia*: (e.g., secondary to sepsis and inflammation)[5]

Pathophysiology and Management of Hypoxia and Hypoxemia in Perioperative Period

Hypoxemia during perioperative period can occur anytime; however, it is more common during induction or emergence

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of anesthesia. Common causes of hypoxemia during perioperative period are:

- *Obstruction of the airway:* Both mechanical (laryngospasm, bronchospasm, complete ventilation failure) and functional obstruction (respiratory depression, residual paralysis) of the airway.
- *Shunt*: Atelectasis, one lung ventilation. Inhalation anesthetics can increase severity of hypoxemia due to shunt by inhibiting hypoxic pulmonary vasoconstriction.
- Anemic hypoxia due to sudden massive blood loss.

Management of Hypoxia in Perioperative Period

Prevention: Prevention of hypoxia is of prime importance. Perioperative physician should anticipate, prevent and treat all possible causes of hypoxia.

Preoperative measures: Preoperative aerobic exercise and inspiratory muscle training, especially for patients with high risk of postoperative pulmonary complications.[6] This has been proven to reduce postoperative atelectasis and pneumonia. This can be achieved by:

- Incentive spirometry 10 times per hour.
- Deep breathing exercises.

Intraoperative measures:

- *Preoxygenation*: It increases the duration of safe apnea till definitive airway is achieved. The primary goal of preoxygenation is to denitrogenate FRC of the lungs and to achieve end-tidal oxygen concentration (ETO_2) of greater than 90%.[7] Following techniques can be utilized during preoxygenation.
  - Oxygen delivery 100% FiO_2 via anesthesia circuit with well-fitted mask. This can be achieved by either 3 minutes tidal volume breathing or eight vital capacity breaths.[8]
    - High-flow nasal cannula (HFNC) is another option and it continues to provide apneic oxygenation during the process of intubation.
    - If patient cannot achieve saturation greater than 93–95% before intubation with high FiO_2 alone, noninvasive positive pressure ventilation should be applied. Continuous positive airway pressure (CPAP) or PEEP will help decrease the degree of shunts and will also prevent absorption atelectasis caused due to high FiO_2.[9]
  - Position of patient
    - Supine position increases the risk of atelectasis of the dependent lung. Patients who are at high risk of hypoxemia (critically ill patients, patients having morbid obesity or abdominal distension) should be preferably preoxygenated in a propped up position. If the patient is immobilized (e.g., spine injury) then reverse Trendelenburg position is ideal. Head elevated position additionally provides benefit of better laryngeal view on direct laryngoscopy.[10]
- *Bag-mask ventilation*: Periods of apneas should be avoided during induction by ventilating the patient with bag-mask ventilation. This will benefit in several ways like:
  - Increases oxygenation through distension of alveoli and decreases shunting
  - Minimizes accumulation of carbon dioxide by actively washing it out which will otherwise cause acidosis and cerebral vasodilatation.

  Modified rapid sequence intubation allowed gentle mask ventilation in patients who are considered as full stomach. Care must be taken to prevent risk of regurgitation and aspiration by limiting inspiratory pressure below 25 cm of H_2O.[11,12]
- *Apneic oxygenation:* It extends the duration of safe apnea.[13] It can be provided either by a nasal cannula set at 15 L/min. Benefit of short-term apneic oxygenation during intubation procedure outweighs the risk of delivery of such high flows of dry and cold oxygen via nasal cannula both in patients with high risk for hypoxemia and the patient having difficult intubation. Alternatively, a HFNC with 100% FiO_2 and flow rate up to 70 L/min can be safely used.

  Apneic oxygenation will work best with patent upper airway. Ideal position during apneic ventilation should aim at maximizing upper airway patency, and this is achieved by head elevated position and chin lift-jaw thrust position.[14]
- *Prevention of atelectasis:* Atelectasis can be prevented by the use of PEEP unless contraindicated. Recruitment maneuvers can be used for reversal of atelectasis during anesthesia.[15] It is prudent to avoid higher FiO_2 to reduce and prevent recurrence of atelectasis.[16]

Postoperative measures to prevent hypoxia[17]

- Continuation of incentive spirometry as advised preoperatively
- Deep breathing and cough every 2 hourly
- Early mobilization of patients
- Head end of bed elevated >30 degrees
- Use of bronchodilators for patients with chronic obstructive pulmonary disease (COPD) and asthma[18]
- Good postoperative analgesia
- Utilization of regional blocks (epidural or facial plane blocks) for analgesia as much as possible
- Opioid-free analgesia postoperatively as much as possible.

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Treatment of Established Hypoxia

Supplementation of oxygen is the first line of treatment of hypoxia. Supplemental oxygen can be delivered to patient at normal atmospheric pressure and is referred as normobaric oxygen therapy or at higher than atmospheric pressure is known as hyperbaric oxygen therapy. Hyperbaric oxygen therapy is beyond the purview of this chapter.

Depending on the dependency of patients upon the oxygen delivery device to correct any oxygen delivery deficiency, they can be classified as:[19]

- *Low dependency*: When supplemental oxygen alone is effective to correct hypoxia in spontaneously breathing patient, e.g., nasal prongs, standard facemask.
- *Medium dependency*: When spontaneously breathing patient requires additional respiratory support along with supplemental oxygen to correct hypoxia, e.g., CPAP mask, HFNC, etc.
- *High dependency*: When patient requires supplemental oxygen and full respiratory support to maintain oxygenation, e.g. Noninvasive positive-pressure ventilation (NIPPV), Intermittent positive pressure ventilation (IPPV).

Continuous positive airway pressure, NIPPV, and IPPV are beyond the purview of this chapter. Here we are discussing low-dependency devices and HFNC.

Low-dependency devices can be further classified as variable performance devices or fixed performance devices depending on if fixed amount of oxygen is delivered to patient during both the phases of respiration or its variable depending on patient's respiratory rate, tidal volume or flow rate.

Depending on reservoir capacity, low-dependency oxygen delivery devices can be classified as:

- No capacity oxygen delivery devices, e.g., nasal cannulae
- Low-capacity oxygen delivery devices (Capacity < 100 mL), e.g., pediatric facemask, tracheostomy mask
- Medium-capacity oxygen delivery devices (Capacity = 100–250 mL), e.g., standard facemask
- High-capacity oxygen delivery devices (Capacity = 250–1,500 mL), e.g., rebreathing mask, nonrebreathing mask (NRBM)
- Very high-capacity oxygen delivery devices (Capacity > 1,500 mL), e.g., incubator, head tent.

Commonly available devices: Commonly available oxygen devices in day-to-day use are explained in detail here:

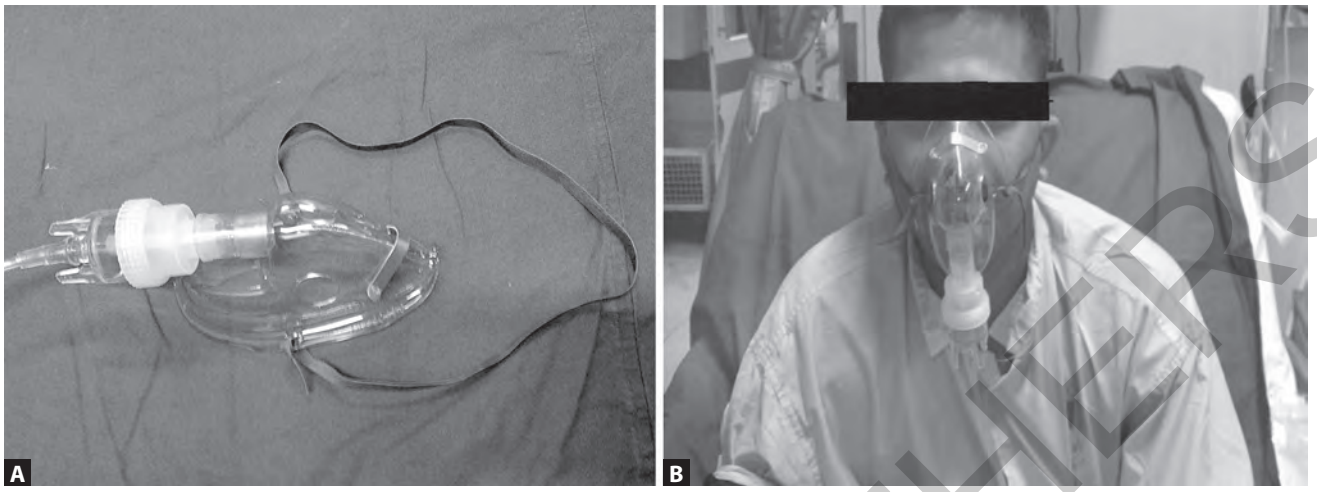
- *Nasal cannulae* ***(Fig. 2)****:* These deliver unhumidified oxygen to the nasopharynx. Its advantages being less claustrophobic and allows easy accessibility to mouth for uninterrupted talking, and eating. But its major drawback being unpredictability of FiO_2 and oxygen delivery >2 L/min can be uncomfortable and can cause drying of nasal mucosa.



Fig. 2: Patient receiving oxygen via nasal prongs.

- *Facemask* ***(Figs. 3A and B)****:* Here oxygen flow of 2–5 L/min should be used and rebreathing is seen commonly if oxygen flow rate is kept <2 L/min or if patient has very high minute ventilation.
- *Nonrebreathing masks* ***(Fig. 4)****:* They have reservoir bag connected to the facemask via a flap valve which prevents rebreathing. Oxygen supply enters the reservoir bag first and then enters the facemask. Reservoir bag provides extra oxygen to match the patient's peak inspiratory flow rate (PIFR) which is higher than the oxygen supply flow rate. Presence of two one-way flap valve on the mask prevents entrainment of room air during inspiration and allows expiratory air to vent out to atmosphere. FiO_2 up to 75–90% can be achieved at oxygen flow rate of 12–15 L/min.[19] NRBM should be used with caution, they provide higher FIO_2 than the face mask and may mask severe respiratory failure. It should not be encouraged to use NRBM except for short-term use or use in patients with do-not-intubate (DNI) orders. If the patient's FiO_2 is in this range, need of ventilation should be assessed.
- *Partial rebreathing masks* ***(Fig. 5)****:* They are similar to the NRBM except for the absence of flap valve to the oxygen reservoir. These masks are supposed to allow rebreathing of the heated and humidified dead space gas while preventing rebreathing of alveolar gases. Dead space gas does not contain expired CO_2 and has oxygen concentration similar to fresh gas. Mapleson A breathing system (Magill circuit) also utilizes similar advantage of dead space rebreathing.

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Figs. 3A and B: (A) Facemask; (B) Patient receiving oxygen via facemask.



Fig. 4: Nonrebreathing mask.



Fig. 5: Partial rebreathing mask.

- *Venturi mask **(Figs. 6A and B)**:* This is an example of fixed performance device. It works on Bernoulli effect and Venturi principle. Here, mixing of room air and oxygen occurs due to delivery of oxygen at high velocity through a small orifice and shear forces. There is entrainment of room air but at fixed rate. Mask adapters are of different sizes and color-coded, which help us to adjust the FiO_2. To ensure target FiO_2 and adequate CO_2 clearance, oxygen flow rate should exceed patient's minute ventilation.[20] These masks can provide FiO_2 from 0.24 to 0.60% **(Table 3)**.

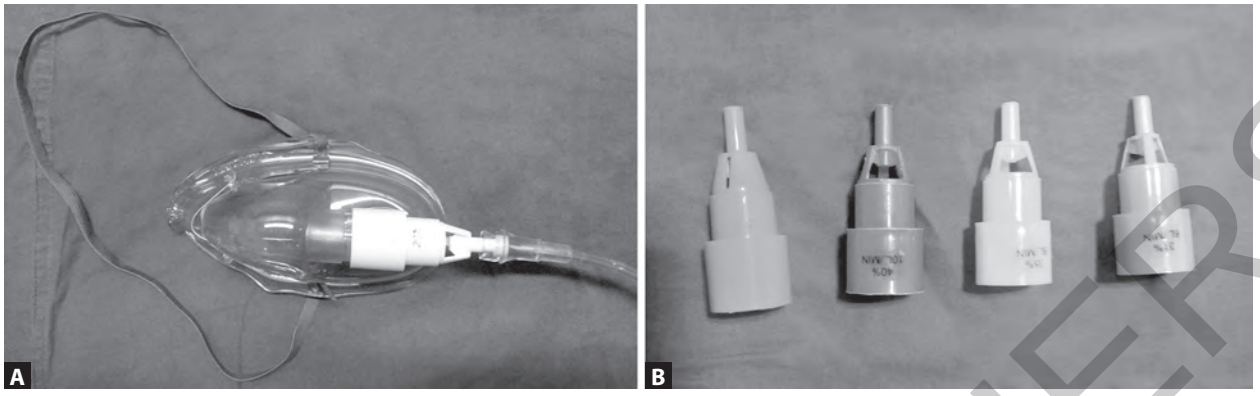
HIGH-FLOW NASAL CANNULA (FIG. 7)

Conventional oxygen delivery systems typically deliver variable and unreliable FiO_2 due to low flows (e.g., nasal cannula or facemask) leading to difficult in its monitoring and titration. High-flow systems like Venturi masks and NRBM can provide more or less fixed FiO_2, however, with delivery of cold and dry gases. Oxygen given by high-flow systems for prolonged periods is poorly tolerated by patients due to inadequate warming and humidification of inspired gas.

High-flow nasal cannula is an innovative oxygen delivery system which delivers high flows of heated and humidified gases which offer advantage of both fixed FiO_2 and better patient acceptance. HFNC provides heated and humidified oxygen at high flows to the patient. It can deliver FiO_2 of 0.21–1.00% at flow rates of up to 60 L/min. The flow rate and FiO_2 can be independently titrated based on the patient's flow and FiO_2 requirements.

High-flow nasal cannula may also be used in safe environment with less monitoring availability in stable cooperative patients who do not have severe hypoxemia.[21]

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Figs. 6A and B: (A) Venturi mask; (B) Venturi connecters for different FiO_2.

Table 3: Venturi devices with color coding, oxygen flow rate, air entrainment and total flow to patient.[19]

Color of device	*FiO_2 (Fraction of inspired oxygen)*	*Oxygen flow rate (L/min)*	*Air entrainment (L/min)*	*Total flow (L/min)*
Blue	0.24	2	51	53
White	0.28	4	41	45
Orange	0.31	6	41	47
Yellow	0.35	8	37	45
Red	0.40	10	32	42
Green	0.60	15	15	30



Fig. 7: Patient receiving oxygen via high-flow nasal cannula.

Physiology and Mechanics of High-flow Nasal Cannula

Washout of Nasopharyngeal Dead Space

High flows delivered by HFNC tend to washout dead space from the upper airway;[22] this improves the efficiency of ventilation as the proportion of alveolar to minute ventilation increases. HFNC in healthy volunteers lead to approximate 20% fall in minute ventilation due to a decrease in tidal volume without any change in respiratory rate during sleep.[23] This effect is probably due to lesser requirements of minute ventilation due to washout of nasopharyngeal dead space. In patients with PaO_2:FiO_2 ratio < 300 mm Hg; use of HFNC at 40 L/min lead to decrease in minute ventilation due to decreased respiratory rate and constant tidal volume without any change in arterial CO_2 levels.[24]

Positive End-expiratory Pressure

High-flow nasal cannula offers some resistance to expiratory flow leading to modest PEEP. The PEEP[25] effect is almost twice high when the mouth is closed compared to when mouth is open. At 50 L/min of inspiratory flow, PEEP of 3.31 ± 1.05 cm H_2O is generated with closed mouth and 1.73 ± 0.82 H_2O with open mouth.

Improvement of Mucociliary Clearance and Patient Comfort[26]

Warm and humidified gases prevent dryness of the upper airways, enhance the mucociliary clearance, and are generally well accepted by the patients.

Fixed Fraction of Inspired Oxygen

Higher flows close to the patients PIFR reduces room air dilution of oxygen providing fixed FiO_2. This may reduce the transient hypoxemic episodes.

Reducing Work of Breathing[22]

High-flow nasal cannula may help in reducing work of breathing by many mechanisms. It reduces inspiratory flow resistance. The PEEP effect may help in improving oxygenation and increasing compliance. Active heating

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and humidification reduces the metabolic demand of gas conditioning.

Indications of High-flow Nasal Cannula

Indications of HFNC are expanding; typical indications are listed below:[27]

- Postoperative respiratory failure
- Prevention or treatment of extubation failure
- Preoxygenation and apneic oxygen during intubation and airway procedures
- Comfort care in palliative patients

Postoperative Respiratory Failure

High-flow nasal cannula has been used both for prevention and treatment of postoperative extubation failure and respiratory failure. BiPOP[28] study has compared continuous HFNC (50 L/min with FiO_2 50%) versus BIPAP (pressure support 8 cm H_2O; PEEP 4 cm H_2O; FiO_2 50% at least 4 hours per day) in 830 postoperative cardiothoracic surgery patients who were either high risk for respiratory failure due to pre-existing risk factors or had failed spontaneous breathing trial or extubation. Both groups had similar treatment failure and mortality rates. BiPAP group had higher incidence of skin breakdown.

In a multicenter randomized controlled trial (OPERA study) comparing HFNC versus standard oxygen therapy via nasal prongs or facemask, in adults undergoing major abdominal surgery and having moderate-to-high risk of postoperative pulmonary complications, HFNC was not associated with improved pulmonary outcomes.[29]

Prevention or Treatment of Extubation Failure

High-flow nasal cannula is an attractive oxygenation strategy for reducing the risk of reintubation in the ICU. In patients with PaO_2/FiO_2 ratio less than or equal to 300,[30] HFNC compared with Venturi mask provides better oxygenation, better patient comfort, fewer episodes of desaturation and most importantly five times lower reintubation and ventilatory support. Benefits of HFNC for prevention of extubation failure are well demonstrated. In patients who are at low risk for reintubation, HFNC for 24 hours, compared to the conventional oxygen therapy[31] reduces postextubation respiratory failure and reintubation within 72 hours. In patients who are at high risk for reintubation, HFNC is as good as NIV for preventing both reintubation and postextubation respiratory failure. As HFNC is generally better tolerated than NIV, its role in prevention of postextubation failure looks promising. Patients having hypercapnia during the spontaneous[31] breathing trial were excluded in the study; hence, the result cannot be applied to these patients. HFNC has been studied for weaning and extubation in both, in patients with high risk and low risk for extubation. In high-risk and nonhypercapnic patients, HFNC is independently associated with lowering postextubation failure.[32]

Preoxygenation and Apneic Oxygen during Intubation and Airway Procedures

High-flow nasal cannula can be used for both preoxygenation and apneic oxygenation **(Fig. 8)** during intubation or other airway procedures. Advantage of HFNC over conventional preoxygenation is that there is no need to engage an operator to achieve a tight mask seal. Another important advantage is the same setup can be continued during intubation for apneic oxygen. Disadvantage is difficulty in monitoring endpoint of preoxygenation. Conventionally,[33] ETO_2 of 90% is considered as endpoint of maximal preoxygenation. With HFNC, capnography cannot be used due to dilution of excelled gases with the high flows. Transnasal humidified rapid insufflation ventilatory exchange (THRIVE)[34] technique has used HFNC for 3 minutes for preoxygenation. Initial flow rate was 30 L/min which was increased to 70 over the first minute. Apneic oxygen using THRIVE has shown to significantly increase safe apnea time for intubation.[35]

Comfort Care in Palliative Patients

High-flow nasal cannula can be used for comfort care of palliative patients having respiratory distress. Its detailed applications are beyond the purview of this chapter.

Success of HFNC does not necessarily mean end of NIV era. Combining use of high-flow nasal oxygen with noninvasive ventilation may improve gas exchange and reduce the work of breathing. HFNC-NIV combination in patients who are high risk for extubation failure is associated with decreased reintubation rates compared to HFNC alone.[36]



Fig. 8: High-flow nasal cannula (HFNC) during apneic ventilation.

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Physiology of FiO_2 Delivery

For a device with a reservoir bag [NRBM, partial rebreathing mask (PRBM)], keeping the flow close to minute ventilation and ensuring proper functioning of the flap valves prevents drop in FiO_2 due to entrainment of air close to patient. For high-flow devices like Venturi, which do not have flap valve or a reservoir bag, ensuring the delivered flow close to the patient's PIFR prevents drop in FiO_2 due to entrainment of air close to patient. Venturi masks, especialy the one with high FiO_2 have total flow rates significantly lower than average PIFR of 60 L/min (*see* **Table 3**) and hence may deliver FiO_2 5–10% lower than the actual setting.[19] For low-flow devices like face mask or nasal prongs, we can never achieve fixed FiO_2. HFNC ensures fixed FiO_2 by providing high flow rate close to patient's PIFR. They have additional benefit of washing out of nasopharyngeal dead space which increased alveolar ventilation without increasing total minute ventilation. **Table 4** shows the comparison of various oxygen devices described in the chapter.

SPECIAL SITUATIONS

- *Obesity:*[37] Obese patients are at more risk of hypoxia during induction, intubation and also after extubation due to following reasons:
  - Reduced FRC below closing capacity
  - Significant atelectasis and shunting in dependent lung regions

Table 4: Comparison of various oxygen devices.

Type	*Flow rate (in L/min)*	*FiO_2 (in %)*	*Indication*	*Advantage*	*Disadvantage*	*Caution*
Nasal cannula	1–2 L/min	Variable	Minimal oxygen supplementation	• Possible to give low flow rates like 1–2 L/min without rebreathing • Better patient acceptance as patient can talk, eat and drink • No claustrophobia	• No reservoir hence entrainment of room air • FiO_2 is unpredictable • Oxygen flow > 2 L/min causes drying of nasal mucosa	• Not for moderate and severe respiratory failure • Not for mouth breathers
Facemask	2–5 L/min	Variable	Moderate oxygen supplementation	Can be used for nebulized drug delivery	Rebreathing if oxygen flow rate is kept 2 L/min or if patient has very high minute ventilation	Lower flows can cause rebreathing
NRBM	12–15 L/min	75–90%	High oxygen supplementation	• Higher FiO_2 • Do not need electric supply • Good system for short term use, e.g., transport, remote location like MRI suite	• Cold dry gases • FiO_2 can vary	• Can mask severe respiratory failure • Patients who require such high FiO_2, generally need additional therapy like HFNC, NIV or IMV
PRBM	≥8 L/min	60–80%	Moderate-high oxygen supplementation	Partial humidification and heating duet to rebreathing of anatomical dead space	Rebreathing of alveolar gas if flow rate is not adequate	• Can mask severe respiratory failure. • See NRBM
Venturi mask	• Flow meter setting 2–15 L/min • Total flow delivered to the patient 30–53 L/min	24–60%		• Gives precise oxygen concentration • Can be used in COPD patients requiring oxygen supplementation as FiO_2 does not exceed	• Skin irritation • Uncomfortable for patient due to cold dry gases	Delivered FiO_2 is lower than the set FiO_2 (Especially for high FiO_2 setting) since total flow rates are significantly lower than average PIFR of 60 L/min
HFNC	Up to 70 L/min	21–100%	Moderate respiratory failure	• Fixed FiO_2 • Heated humidified • Patient comfort	• Requires electric power source and distilled water supply • Difficult to use during transport • Not MRI compatible	Cautious to use in patients with altered consciousness and excessive secretion (risk of aspiration), and hemodynamic instability

(COPD: chronic obstructive pulmonary disease; FiO_2: fraction of inspired oxygen; HFNC: high-flow nasal cannula; IMV: invasive mechanical ventilation; NIV: noninvasive ventilation; NRBM: nonrebreathing mask; PIFR: peak inspiratory flow rate; PRBM: partial rebreathing mask)

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- Increased resting metabolic rate and increased oxygen consumption

Following precautions can help in avoiding hypoxia in these patients:

- Induction of anesthesia
  - Preoxygenation in head up or ramped position
  - Adequate preoxygenation ensuring ETO_2 > 80–90%
  - Use of NIV during preoxygenation especially in critically ill patients
  - Avoiding apnea during induction
- Intubation
  - Apneic oxygenation during laryngoscopy
- Extubation
  - Head up position
  - Chest physiotherapy and intensive spirometry
  - Oxygen administration
  - Use of CPAP/NIV if patient was using one preoperatively or whose hypoxia postoperatively is not responding to chest physiotherapy

- Effect of regional blocks of respiratory function
  - *Interscalene block*: Phrenic nerve block is commonly associated with this block and is usually well tolerated unless patient has compromised respiratory system (COPD, morbid obesity). That is why, it is recommended to avoid this block in patients with contralateral respiratory pathology.
  - *Epidural analgesia*: It is boon for major thoracic and abdominal surgeries. It resulted in pain-free ventilation and increased abdominal ventilation and ability to cough in the postoperative period.[38] Even high thoracic epidural anesthesia (sensory blockade from C4 to T8) was not detected by patients as respiratory distress, even when it showed no change in FRC and reduced both forced expiratory volume in one second (FEV1) and vital capacity by 8–10%.[39]
- *Flail chest:* It generally occurs following trauma. It is characterized by either double fractures of three or more contiguous ribs or combined sternal and rib fractures resulting in an unstable segment of the chest wall, which moves paradoxically with respiration. It results in reduction in effective ventilation leading to hypoventilation.[40]

CURRENT EVIDENCE FOR USE OF SUPPLEMENTAL OXYGEN THERAPY IN PERIOPERATIVE AND CRITICALLY ILL PATIENTS

Oxygen is a drug and should be used only in indicated cases at a particular dose (flow rate of FiO_2) and duration. Evidence suggest indiscriminate use of oxygen leading to hyperoxia causes increased production of reactive oxygen species (ROS), which promote a deleterious inflammatory response leading to secondary tissue damage and/or apoptosis. Oxygen has direct lung toxicity causing atelectasis and tracheobronchitis. The studies looking at effect of various oxygenation strategies in critically ill patients are listed in **Table 5**.

Guidelines for Use of Supplemental Oxygen Therapy

Following recommendations are suggested by British Thoracic Society Guideline for oxygen use in adult in 2017:

Recommendation of use of oxygen in perioperative care:[41]

- Target saturation recommended for most of the patients is 94–98%, except for those at risk of hypercapnic respiratory failure, where recommendation is 88–92%.
- Pulse oximetry monitoring is recommended in all postoperative patients.
- For patients using the *patient-controlled analgesia* (PCA), oxygen saturation should be monitored every two hourly with oximetry and if needed oxygen should be administered to keep patient within recommended saturation range.

Weaning and discontinuation of oxygen therapy[41]

- If patient is clinically stable and maintaining oxygen saturation above or near upper limit of recommended target saturation for around 4–8 hours, then oxygen concentration should be steadily lowered and eventually weaned off.
- Before cessation of oxygen therapy, patients should be stepped down to lowest oxygen concentration, i.e., 2 L/min via nasal cannula and 1 L/min for patients at risk of hypercapnic respiratory failure.
- Oxygen therapy should be stopped when the patient is clinically stable on low-concentration oxygen with the oxygen saturation within the recommended range on two consecutive observations.
- After stopping oxygen therapy, oxygen saturation on air should be monitored for 5 minutes and again at 1 hour, if it remains in the desired range.
- After 1 hour, if oxygen saturation remains within target range, then patient can be safely discontinued from oxygen therapy. However, if saturation drops below the patient's target range after stopping oxygen therapy, we should restart oxygen therapy to maintain the patient in the target range and the patient should be clinically re-evaluated to establish the cause for deterioration.

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Table 5: Effect of various oxygen strategies on outcome in critically ill patients.

Name of trial or author *Year*	*Study type*	*Patient population*	*Intervention*	*Comparison*	*Endpoints*	*Outcome*	*Conclusion*
AVOID[42] 2015	• RCT • 9 metropolitan hospitals • Melbourne	441 patients with ST-elevation myocardial infarction	• No oxygen therapy • (No O_2, unless O_2 saturation falls below 94%)	• Routine oxygen therapy • (Prehospital supplemental O_2 at 8 L/min and in hospital as per hospital protocol)	• *Primary endpoint*: myocardial infarct size as assessed by cardiac enzymes, troponin I, and creatine kinase	• Statistically significant reduction in creatine kinase • Non statistically significant decrease in infarct size	• No significant benefit of routine oxygen therapy for reducing myocardial infarct size, improving patient hemodynamic, or alleviating symptoms • Possible harm with routine oxygen therapy due to larger myocardial infarct size at 6 months and also higher frequency of myocardial infarction recurrence
DETO2X-AMI[43] Published in 2017	• RCT (multicenter) • 35 Swedish hospitals	6,629 patients with suspected myocardial infarction and O_2 saturation ≥90%	Ambient room air	Supplemental oxygen (6 L/min for 6–12 hours)	• Primary endpoint: All-cause mortality at 1 year • Secondary endpoint: All-cause mortality within 30 days after randomization, and rehospitalization with myocardial infarction or heart failure	No significant difference	No benefit of supplemental oxygen in suspected myocardial infarction patients with O_2 saturation ≥ 90%
SO2S trial[44] 2017	• RCT • 136 participating centers in the United Kingdom	8,003 nonhypoxic patients with acute stroke	• Continuous oxygen • Nocturnal oxygen	*Control group*: Oxygen only if clinically indicated	The primary outcome: Death or disability at 3 months using the modified Rankin Scale score	No statistically significant difference in primary outcome in primary and subgroup analysis	The prophylactic use of low-dose oxygen supplementation in nonhypoxic patients with acute stroke did not reduce death or disability at 3 months
Oxygen-ICU[45] 2016	RCT (single center) Modena University Hospital, Italy	• Adult patients admitted to ICU with an expected length of stay of 72 hours or longer	Conservative oxygen therapy (Target SpO_2 = 94–98%, PaO_2 = 70–100 mm Hg)	Conventional oxygen therapy (Target SpO_2 = 97–100%, PaO_2 up to 150 mmHg, FiO_2 ≥0.4	• *Primary outcome*: ICU mortality. • *Secondary outcome*: occurrence of new organ failure and infection 48 hours or more after ICU admission	• Conservative oxygen therapy lead to statistically significant 43% relative risk (RR) reduction in mortality	Striking mortality and organ failure benefit in critically ill patients given a conservative oxygen regimen *Contd...*

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Contd...

Name of trial or author *Year*	*Study type*	*Patient population*	*Intervention*	*Comparison*	*Endpoints*	*Outcome*	*Conclusion*
		• Originally planned sample size 660 patients (unplanned early termination of the trial due to difficulty in patient enrolment) • Final sample size 480 patients				• New organ failure and infection were also less common in conservative oxygen therapy group	
HYPRESS[46] 2017	• Two-by-two factorial, multicenter, randomized, clinical trial • 22 centers in France	• 442 patients • Septic shock • (stopped prematurely for safety reasons)	FiO_2 of 1.0 for first 24 hours (hyperoxia)	FiO_2 set to target an arterial oxygen saturation of 88–95% (normoxia) during the first 24 hours	*The primary endpoint*: Mortality at day 28	The trial was stopped prematurely due to observation of significant increase in 28-day mortality and doubled incidence of ICU acquired weakness and atelectasis in hyperoxia group	In patients with septic shock, setting FiO_2 to 1.0 to induce arterial hyperoxia might increase the risk of mortality
ICU-ROX[47] 2019	• RCT • 21 ICUs in Australia and New Zealand	1,000 patients	• Conservative oxygen group • SPO_2 90–96% • Decreasing FIO_2 if SPO_2 97% or above	• Usual oxygen therapy • Maintaining SPO_2 above 90% with no upper limit	*The primary outcome*: The number of ventilator-free days from randomization to day 28	No difference in primary endpoint	Conservative oxygen therapy did not reduce number of ventilator-free days
Jonge et al.[48] 2019	Meta-analysis of 17 RCT comparing the effect of high (80%) vs. standard (30–35%) FiO_2 on the incidence of SSI		Surgical patients receiving 80% FiO_2 intraoperatively	Surgical patients receiving 30–35% FiO_2 intraoperatively	Incidence of surgical site infection (SSI)	No overall reduction in SSI with high FiO_2. Significant reduction in SSI in intubated patients {RR: 0.80 [95% confidence interval (CI): 0.64–0.99]}	No definite benefits with SSI with high FiO_2 in adult surgical patients undergoing general anesthesia
IOTA[49] 2018	• Systemic review and meta-analysis • 25 RCTs comparing liberal and conservative oxygen therapy in acutely ill adults (aged ≥18 years)	16,037 patients	Liberal oxygen group (median FiO_2 of 0.52 for a median duration of 8 hours)	Conservative oxygen group (median FiO_2 0.21)	*Outcomes*: Mortality and morbidity (disability, risk of any hospital-acquired infection, and hospital length of stay)	• Increased mortality – In -hospital (RR 1·21, 95% CI 1·03-1·43), – At 30 days (RR 1·14, 95% CI 1·01-1·29), – At longest reported follow-up (RR 1·10, 95% CI 1·00-1·20) • Morbidity outcomes were similar between groups	In a heterogeneous group of ICU patients hyperoxia increased mortality without improving other patient-important outcomes

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