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Oxygen Therapy

New Approach to Wellness Across Specialities

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Foreword
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Contents

CHAPTER

15

Oxygen Therapy in Children

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INTRODUCTION

Oxygen is one of the most important therapeutic agents used in the clinical management of pediatric patients suffering from cardiopulmonary diseases. Despite its importance in pediatric acute severe illnesses, oxygen therapy remains an inaccessible luxury for a large proportion of severely ill children admitted to hospitals in developing countries.[1] The gap between knowledge and practice of oxygen therapy is a common phenomenon and there is wide variation in approaches to oxygen therapy within pediatric clinical practice. Oxygen therapy within pediatric clinical practice is predominantly determined by institutional or individual practice or preference and arises from a lack of understanding of the relative merits and demerits of the different techniques of oxygen monitoring in children who are different from adults. This comprehensive review briefly summarizes the current and best available evidence regarding the oxygen therapy in pediatric clinical practice, and for the benefit of the reader, is systematically organized into the following sections:

- Historical aspects of oxygen therapy in pediatric clinical practice
- Hypoxia and assessment of inadequacy of oxygen delivery
- Physical and physiologic effects of oxygen therapy
- Indications of oxygen therapy
- Oxygen delivery devices
- High-flow oxygen versus low-flow oxygen in hospitalized pediatric patients
- Monitoring and maintenance of oxygen therapy
- Contraindications of oxygen therapy
- Oxygen toxicity
- Complications of oxygen therapy
- Weaning and discontinuation of oxygen therapy
- Oxygen therapy in children with coronavirus disease 2019 (COVID-19)
- Clinical pearls in oxygen therapy in pediatric clinical practice

HISTORICAL ASPECTS OF OXYGEN THERAPY IN PEDIATRICS

Oxygen was first discovered by Joseph Priestley in 1775, later named as oxygen by Antoine Lavoisier in 1778.[2] Oxygen therapy has its origins in the Pneumatic Institution founded in Bristol (UK) in 1779 by Thomas Beddoes, the aim of which was to explore the potential efficacy of this gas for the treatment of diseases.[3] The pediatric use of inhaled oxygen was first initiated by Julius Hess in 1934.[4] Later on, by 1940, oxygen therapy for treatment of cyanosis, apnea, and periodic breathing in the newborn became a standard of care.

HYPOXIA AND ASSESSMENT OF INADEQUACY OF OXYGEN DELIVERY

Continuous and adequate supply of oxygen to the organs and tissues are required for normal physiological function for adequate metabolism of carbohydrates and the production of adenosine

triphosphate (ATP). Inadequate oxygen delivery to tissues leads to hypoxia. Tissue hypoxia in children causes a myriad of undesirable problems, such as localized vasodilation, pulmonary vasoconstriction, metabolic acidosis, tissue necrosis, increased risk of kernicterus, and impairment of surfactant production, especially in the newborn.[5] Persistent tissue hypoxia can ultimately lead to serious and permanent brain injury and even death.[6] The oxygen carrying capacity of blood is determined by the concentration of hemoglobin present in the blood; its oxygen saturation (SpO_2) and diffusion to the organs and tissue and is largely affected by the rate of blood flow; and the efficiency with which oxygen is unloaded from hemoglobin to the tissues. Conditions such as pulmonary disease, hypoventilation, uneven matching (mismatch) of ventilation to perfusion, diffusion defects, intrapulmonary shunts or "right to left" cardiac shunts, or reduced oxygen carrying capacity due to anemia or abnormal blood hemoglobin can all lead to hypoxia.

The terms "hypoxia" and "hypoxemia" are not synonymous. Hypoxemia is defined as a decrease in the partial pressure of oxygen in the blood whereas hypoxia is defined as reduced level of tissue oxygenation, which can be due to either defective delivery or defective utilization of oxygen by the tissues. Hypoxemia and hypoxia do not always coexist. Patients can develop hypoxemia without hypoxia if there is a compensatory increase in hemoglobin level and cardiac output (CO). Similarly, there can be hypoxia without hypoxemia. To identify and assess a child's need for oxygen therapy, several physical signs and laboratory values can be assessed. Physical signs, such as cyanosis, confusion, tachycardia, retractions, nasal flaring, and expiratory grunting (infants) can be indications that the child needs oxygen therapy.[6]

A simple noninvasive device, pulse oximetry is now most commonly used to identify hypoxemia in the clinical setting. In the child with a normal pH, PCO_2, temperature and diphosphoglycerate, and noninvasive SpO_2 value of approximately 90% and 95% corresponds to arterial pressures (PaO_2) of 60 and 80 mm Hg, respectively.[7] Pulse oximetry has its limitations and is known to be inaccurate in carbon monoxide poisoning and other conditions where the oxyhemoglobin dissociation curve shifts either to the left (increased affinity for O_2) or the right (decreased affinity for O_2). In an anemic child, SpO_2 might be in the normal range despite a reduced PaO_2. Many pulse oximeters are affected by movement artifact and impaired peripheral perfusion.[8,9] Arterial blood gas (ABG) analysis is an invasive, but a very accurate method used to measure partial pressure of oxygen (PaO_2) for detecting hypoxemia in children.

PHYSICAL AND PHYSIOLOGIC EFFECTS OF OXYGEN THERAPY IN CHILDREN

Individual responses to oxygen therapy in the pediatric age group vary greatly, depending on the particular cause of hypoxia and the degree of impairment. Hypoxia caused by hypoventilation and ventilation/perfusion anomalies associated with pulmonary disease is most responsive to oxygen therapy,[7] on the other hand, oxygen supplementation in hypoxia caused by cardiac shunts, shock, or hemoglobin deficiency/dysfunction might lead to only small increases in tissue oxygenation, though even this small increment may prevent life-threatening hypoxia in the child.[10] Medical oxygen, when administered to the patient is a dry gas and to preserve ciliary function and minimize atelectasis and tracheitis, it needs to be humidified before providing it to children.

INDICATIONS OF OXYGEN THERAPY IN CHILDREN

The therapeutic indication for providing oxygen in children is similar to adults, i.e., when the PaO_2 falls to below 60 mm Hg. However, PaO_2 alone is not sufficient to determine oxygen delivery to tissues. The rational for oxygen therapy is to prevent or correct cellular hypoxia, caused by hypoxemia (low PaO_2), and thus prevent potentially irreversible damage to vital organs. Simply stated, oxygen therapy is a modality to provide oxygen according to target saturation rates (as per the age of the child and nature or pattern

of the disease) in order to achieve normal or near normal SpO_2 levels for ill children by increasing the concentration of inhaled oxygen. The World Health Organization (WHO) recommends oxygen administration in children if SpO_2 level <90% with signs of respiratory distress.[11] The WHO Emergency Triage Assessment and Treatment (ETAT) guidelines recommend a target SpO_2 of 94% for children with emergency signs for severe disease [severe pneumonia, septic shock, severe anemia, central nervous system (CNS) infection, or heart failure] where oxygen delivery from the lungs to body tissues is impaired, or where vital organs may be susceptible to low oxygen levels.[12,13] Common indications for oxygen therapy in pediatric clinical practice are enumerated in **Box 1**.

Oxygen therapy in children must be provided at accurate and safe levels with the lowest possible fractional concentration of inspired oxygen (FiO_2) by careful selection of the oxygen delivery device. The target range of SpO_2 is different for different age groups of children (preterm newborn, newborn, infant, children, and adolescent), disease pattern (acute or chronic), and/or type of disease.

BOX 1: Common indications for oxygen therapy in pediatric clinical practice.

Acute hypoxemia:
- Acute respiratory infections
- Shock
- Sepsis
- Asthma
- Meningitis
- Cardiac failure

Abnormalities in quality or type of hemoglobin:
- Acute blood loss
- Carbon monoxide poisoning
- Trauma

Others:
- Pneumothorax
- Perioperative emergencies
- Postoperative state resulting in hypoxemia

In hospitalized children, oxygen should be administered and weaned to achieve target SpO_2 level between 94 and 99%.[14] Most of the critically ill newborn babies require oxygen. The ideal SpO_2 target for extremely low birth weight (LBW) infants remains unknown and is likely to be patient-specific and dependent on various factors, including gestational age, chronologic age, underlying disease, and transfusion status. Current delivery room resuscitation guidelines recommend the use of room air for term newborns and preterm newborns of ≥35 weeks' gestation and FiO_2 of 0.21–0.3 for preterm infants of <35 weeks' gestation. Most appropriate evidence-based strategies advocate targeting SpO_2 for newborns to 91–95% with close monitoring.[4,15] A target saturation of 94% during the resuscitation phase will compensate for the potential of reduced oxygen delivery, as well as compensate for the error of the test inherent with the use of some pulse oximeters.[12]

OXYGEN DELIVERY DEVICES IN PEDIATRIC CLINICAL PRACTICE

Methods of oxygen delivery in children can be noninvasive (through a face mask, head box, incubator or tent, or holding tubing close to an infant's face), semi-invasive (insertion of prongs or catheters into the upper airway), or invasive (ventilators). Oxygen delivery systems are categorized as low-flow (variable performance) systems or high-flow (fixed performance) systems. With low-flow systems, 100% oxygen mixes with room air during inspiration and the room air is entrained, making the proportion (percentage) of delivered oxygen variable. High-flow devices provide a high flow of premixed gas such that the child is not required to inhale room air and the proportion of oxygen delivered is consistent. The methods used for administration of oxygen to children should be safe, simple, and effective. It is also important to select an oxygen delivery system that suits the child's age, size, needs, clinical condition, and therapeutic goals. Different devices and methods for oxygen therapy in pediatric clinical practice are summarized in **Table 1**.

TABLE 1: Oxygen delivery devices and interfaces used in pediatric clinical practice.

Oxygen delivery device	Type of flow	Flow rate	FiO_2 achieved	Advantages	Disadvantages
Nasal cannula	Low flow	• 0.5–1 L/min for neonates • 1–2 L/min for infants • 1–4 L/min in preschool children • 6 L/min in school children	0.24 at 1 L/min to 0.44 at 6 L/min	• Most commonly used device when FiO_2 required is between 22 and 60% • Allows the child to eat, talk, and cough without interruption • Humidification is not required at low-flow rates	• FiO_2 inconsistent • May cause drying of nasal mucous membranes at high flow rates
Simple face mask	Low flow	6 L/min	Up to 0.6 L/min	• Useful for acute situations and short-term use only (set at 6–10 L/min) • Snugly fits on patient's face without much discomfort • Increases the size of the oxygen reservoir beyond the limit of anatomic reservoir	• Inconsistent FiO_2 delivery • Minimum of 6 L/min to be maintained for avoiding hypercarbia • Mask must be removed for eating and drinking
Nasopharyngeal catheters	Low flow	• 0.5 L/min for neonates • 1 L/min for infants	Higher positive end-expiratory pressure (PEEP) is achieved	• Most economical of all the methods • Better oxygenation with moderate positive distending pressure (PDP) is achieved with a lower oxygen flow than with nasal prongs	• Requires close supervision • Prone to blockage • If displaced downward into the esophagus causes gagging, vomiting, and gastric distention
Head boxes, incubators, and tents	Low flow	7–10 L/min	>0.5 L/min	• Actual FiO_2 can be determined precisely with an oxygen analyzer • No increased risk of airway obstruction or gastric distention • Humidification is not necessary	• Requires high oxygen flows to achieve adequate concentrations of oxygen and to avoid carbon dioxide accumulations • Interferes with feeding
Nonrebreather mask (NRBM)	Low flow	10–15 L/min	0.8–0.95 L/min	• Works as a high flow system as it provides high FiO_2 • Good for short-term oxygen therapy	• Inconsistent FiO_2 delivery • Can cause hypercarbia

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Oxygen delivery device	Type of flow	Flow rate	FiO_2 achieved	Advantages	Disadvantages
Venturi masks	High flow	• 2–4 L/min = 24% O_2 • 12–15 L/min = 60% O_2	Fixed FiO_2	Guarantees delivery of a fixed and consistent FiO_2	• Can cause hypercarbia • Humidification can alter oxygen concentration
High-flow nasal cannula (HFNC)	High flow	• 1–2 L/min • 4 L/min in infants up to 40 L/min or more in adolescents	Adjustable 21–100%	• Used when a higher FiO_2 with some end-expiratory pressure is required and ventilation is adequate • Delivers a mixture of air and oxygen • More comfortable • Incidental delivery of PEEP	• Not suitable in upper airway abnormalities that may make HFNC ineffective or potentially dangerous such as severe hypoxia, hemodynamic instability, facial bone or skull base trauma, and pneumothorax • Should be applied carefully in patients with a decreased level of consciousness, congenital heart disease, acute asthma, or chronic respiratory failure

HIGH-FLOW OXYGEN VERSUS LOW-FLOW OXYGEN IN HOSPITALIZED PEDIATRIC PATIENTS

The use of high-flow nasal cannula (HFNC) oxygen delivery systems to provide respiratory support to children has increased over the past decade. HFNC is an oxygen supply system capable of delivering humidified and heated oxygen at a flow rate of up to 60 L/min. This delivery of oxygen at an adjustable FiO_2 (between 21 and 100%), heated (34°C–37°C) with nearly 100% relative humidity can avoid mucosal injury, patient discomfort from cold, dry air, and encourage the clearance of secretions and reduce bronchoconstriction. The basic principle of HFNC is to set a higher oxygen flow than the inspiratory demand flow according to the clinical situation.[16] HFNC systems also provide washout of the nasopharyngeal dead space, decrease inspiratory resistance, improve airways conductance and pulmonary compliance, as well as decrease metabolic work by providing heated and humidified gas.[16] HNFC can be initiated as the first-line therapy for all age groups of children with various etiologies of acute respiratory distress. HFNC in neonates and preterm babies is noninferior to continuous positive airway pressure (CPAP) in preventing intubation and invasive ventilation; however, HNFC should only be used for moderate-to-severe bronchiolitis in the pediatric ward and intensive care unit (PICU) for the best results.[17] Humidification of oxygen, either heated or unheated is recommended and used while providing high-flow oxygen therapy. Many HFNC systems are designed with in-built warming and humidification systems that provide oxygen at appropriately humidification and as close to normal body temperature which is nonirritating to the mucosa of the upper airways, thereby increasing patient comfort.

MONITORING AND MAINTENANCE OF OXYGEN THERAPY IN PEDIATRIC CLINICAL PRACTICE

Children who are on oxygen therapy should be regularly monitored by regular pulse oximetry to determine whether they still need oxygen and whether those who are already on oxygen have developed worsening of respiratory distress. It is unlikely that a child who has normal SpO_2 while breathing room air has impaired ventilation; however, once oxygen is administered, SpO_2 can be maintained at normal levels despite severe hypercapnea. As pulse oximetry cannot indicate the adequacy of ventilation, children receiving oxygen therapy must also be assessed clinically for respiratory effort, respiratory rate and the level of consciousness to check for the adequacy of ventilation. A child with inadequate ventilation will have slow or shallow breathing and will be lethargic. Any concern about the adequacy of ventilation should prompt efforts to ensure that the airway is clear and protected; and the child is positioned to facilitate chest expansion (e.g., sitting in a semirecumbent position at 20–30°, head-up to reduce diaphragmatic splinting).

CONTRAINDICATIONS OF OXYGEN THERAPY IN PEDIATRIC CLINICAL PRACTICE

There are no absolute contraindications to oxygen therapy in pediatric clinical practice if indications are judged to be present clinically. Supplemental O_2 should be administered with caution in children presenting with paraquat poisoning and with acid inhalation. The administration of oxygen can lead to pulmonary vasodilation and subsequent system ischemia in some congenital heart defects because of an unbalanced circulation. In children with chronic carbon dioxide retention, oxygen administration may cause further increases in carbon dioxide and respiratory acidosis.[18] Children with chronic neuromuscular disorders, chest wall deformities, cystic fibrosis, morbid obesity, and chronic lung disease of prematurity are at higher risk.

OXYGEN TOXICITY

There is a Janus-faced dimension to oxygen therapy in pediatric clinical practice in that in addition to being essential, it is toxic under certain conditions. Oxygen toxicity in children can be divided into two groups; one in which the child is exposed to very high concentrations of oxygen for a short duration, and the second where the child is exposed to lower concentrations of oxygen but for a longer duration, which can result in acute and chronic oxygen toxicity, respectively.

Acute oxygen toxicity manifests generally with CNS effects, while chronic oxygen toxicity has mainly pulmonary effects. Severe cases of oxygen toxicity can lead to cell damage and even death. It has been shown that exposure to high concentrations of oxygen first damages the capillary endothelium, followed by interstitial edema (0–12 hours), worsening pulmonary compliance and vital capacity (12–30 hours), followed by thickening of the alveolar-capillary membrane (30–72 hours).[19,20] If the process continues, type 1 alveolar cells are destroyed, and type II cells proliferate.[21] An exudative phase follows, resulting in a low ventilation/perfusion ratio, physiologic shunting, and worsening hypoxemia. $FiO_2 > 0.50$ presents a significant risk of absorption atelectasis.[21] The risk of absorption atelectasis may be greatest in children breathing at low tidal volumes.[7] Unrestricted or unmonitored administration of oxygen to preterm infants is associated with a disastrous increase in the incidence of retinopathy of prematurity (ROP), chronic lung disease, and necrotizing enterocolitis. The onset and rate of progression of oxygen toxicity in children is influenced by a variety of conditions, procedures, and drugs.

Oxygen toxicity is managed by reducing the exposure to increased oxygen levels. The lowest possible concentration of oxygen that alleviates tissue hypoxia is optimal in most decompensated neonates. Treatment for oxygen toxicity is purely symptomatic; therefore, it is imperative to monitor for early recognition of toxicity. Abrupt or sudden discontinuation of oxygen at the onset of toxicity may at time aggravate symptoms. Damage due to oxygen-induced pulmonary toxicity is reversible in most children. Infants who have survived

bronchopulmonary dysplasia will ultimately recover near-normal lung function, since the lungs continue to grow during the first 5 –7 years of life.

COMPLICATIONS OF OXYGEN THERAPY IN PEDIATRIC CLINICAL PRACTICE

CO_2 narcosis: This occurs in children who have chronic respiratory obstruction or insufficiency which results in hypercapnea (i.e., raised $PaCO_2$). In these children, the respiratory center relies on hypoxemia as the stimulus to maintain adequate ventilation. If these children are given oxygen with a high FiO_2, it can blunt their respiratory drive, causing respiratory depression and further rise in $PaCO_2$.[18] Therefore, one must be cautious while using high FiO_2 in the presence of reduced minute ventilation.[19]

Pulmonary atelectasis: High concentrations of oxygen (FiO_2 > 60%) may damage the alveolar membrane when inhaled for >48 hours resulting in pathological lung changes.[19]

Retinopathy of prematurity: Neonates exposed to high levels of oxygen are at risk for developing ROP, as oxygen promotes neovascularization of the retina and can cause vision loss or blindness. Mild ROP in infants frequently reverses without intervention and the eyesight may become normal in later years.[22,23]

WEANING AND DISCONTINUATION OF OXYGEN THERAPY IN PEDIATRIC CLINICAL PRACTICE

A weaning trial in children on oxygen therapy should be best done in the morning, when there are likely to be adequate staff to observe the child throughout the day. If weaning is initiated and/or supplemental oxygen is discontinued in the late evening or night, then oxygen desaturation that sometimes occurs during sleep might increase the risk for hypoxemia, which may go unrecognized during the night. For children on oxygen therapy who are in a stable condition (stable vital signs, with no respiratory distress, feeding adequately and normal level of consciousness, and with SpO_2 > 90%), oxygen should be interrupted once a day for 10–15 minutes and the children carefully examined for changes in clinical signs and SpO_2 to assess whether supplemental O_2 still required.[11] Children should receive supplemental oxygen until their SpO_2 on room air is 90%. If the SpO_2 is 90% after a trial on room air, they should remain off oxygen, and their SpO_2 should be rechecked after 1 hour, as late desaturation can sometimes occur. Continuous pulse oximetry monitoring needs to be done for at least 30 minutes postcessation of oxygen therapy.[11]

OXYGEN THERAPY IN CHILDREN WITH CORONAVIRUS DISEASE 2019

The child with COVID-19 infection should receive oxygen supplementation to achieve a target SpO_2 ≥ 94% during resuscitation if the child has emergency signs (obstructed or absent breathing, severe respiratory distress, central cyanosis, shock, coma, or convulsions), and once the child is stable, the target should be set at SpO_2 > 90%.[12]

The preferred mode of oxygen delivery in children with COVID-19 infection is nasal prongs or nasal cannula for infants and children <5 years of age. It is recommended that only those children having respiratory distress should receive oxygen when their SpO_2 < 90%.[24] Initially, oxygen should be administered by nasal prongs at a standard flow rate (0.5–1 L/min for neonates, 1–2 L/min for infants, 2–4 L/min for older children, and 4–6 L/min for adolescents) or through an appropriately sized face mask (>4 L/min) to reach an SpO_2 of ≥94%. If severe hypoxemia persists despite maximal flow rates, then oxygen should be given by CPAP or by a face mask with a reservoir bag. HHHFNC (heated humidified high-flow nasal cannula) or HFNC can be tried in children with mild acute respiratory distress syndrome (ARDS) without evidence of hemodynamic instability, altered mental status, or multiorgan failure though it is known to generate aerosol, thereby increasing the risk of transmission of COVID-19.[25] With all these noninvasive methods if the child's oxygenation status does not improve or

respiratory status deteriorates then he/she should be considered for invasive mechanical ventilation.

CONCLUSION

- Supplemental oxygen only relieves hypoxemia, it does not improve ventilation, or correct the underlying cause of respiratory failure.
- SpO_2 is a measure of oxygenation and not ventilation. Therefore, beware of oxygen therapy at high FiO_2 in the presence of reduced minute ventilation.
- Many children in the recovery phase of an acute respiratory illness are characterized by ventilation/perfusion mismatch and can be managed by maintaining their SpO_2 % within the low 90s range as long as they are clinically improving, feeding well, and do not have obvious respiratory distress.
- Normal SpO_2 values may be found despite rising blood carbon dioxide levels (hypercapnia).
- Oxygen therapy at high FiO_2 has the potential to mask signs and symptoms of hypercapnia.
- Therapeutic procedures and handling may increase the child's oxygen consumption and lead to worsening hypoxemia; therefore, it is mandatory to closely monitor and assess children on oxygen therapy at regular intervals.
- Children with cyanotic congenital heart disease normally have SpO_2 between 60 and 90% at room air. Strategies to increase $SpO_2 > 90\%$ with supplemental oxygen are not recommended except in emergency situations due to the risk of causing ventilation/perfusion mismatch by increasing circulation to the pulmonary system while adversely decreasing systemic circulation.
- Unless clinically contraindicated, an attempt to wean the child off oxygen therapy and/or ventilation should be attempted at least once per shift.
- Although a pediatrician may consider specific methods and devices for oxygen administration and initially select the most appropriate one at the onset of therapy, it is important to understand that with therapy individual patients change, and their medical conditions evolve too. Therefore, constant re-evaluation of the child on oxygen therapy is critical to ensure the necessity for continued oxygen administration as well as decide which method or device is the best possible for each child from time to time.

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"Oxygen Therapy: New Approach to Wellness across Specialities" is meant for a vast and diverse reader pool, ranging from general practitioners to specialists of Respiratory Medicine, Internal medicine, Critical Care and several other Broad and Super Medical and Surgical Specialties, as well as postgraduate trainees, and healthcare workers.

The book begins with the evolution of oxygen on earth, the isolation of oxygen in pure form in the medieval ages, and covers in broad perspective the evolution of oxygen therapy in medicine from the past to the present. In addition to this, the basic sciences and physiology of respiratory failure, gas exchange, oxygen transport delivery and utilization at cellular level are also covered. Chapters pertaining to oxygen sources, administration, devices, and clinical uses in different respiratory as well as nonrespiratory conditions, both acute and chronic in adults and children have been included, as are oxygenation during procedures such as ventilation and bronchoscopy. The oxygen crisis during coronavirus disease 2019 (COVID-19) has also been dealt with, with valuable lessons for the future pandemic preparedness, including oxygen audits for health services. The historical contributions of all pioneers whose efforts led to oxygen therapy and assisted ventilation achieving their current status in modern medicine have been commemorated by a photo gallery in the chapter on "The Past, Present and Future of Oxygen Therapy". All articles have been written and edited to follow a standard pattern. Each article includes an introduction and summary or keypoints, with references for those who wish to read further. To break the monotony of reading subjective articles and make it more lively and interesting for the reader, some articles have been written in a question-answer format and some real-life case reports that reflect commonly encountered clinical scenarios have also been included for the reader to develop a better grasp of the subject. The informative chapters contain high-quality graphics, pictures, tables, and charts to capture the reader's interest and make it more informative and interesting. We have attempted to provide a concise, yet comprehensive review of the historical and current perspectives, theory, practice, and clinical applications of oxygen therapy in a nutshell and I hope both teachers and students will appreciate our endeavor. While some overlap may exist between the content of different articles, I would like to clarify that this is with the intent to ensure each article remains complete in itself, mainly for the convenience of the reader. We hope that this book is effective in capturing the reader's attention and fulfills the purpose that it was written for, and its usefulness grows much worth beyond the time and effort which it took to write and create.

Nikhil Sarangdhar DNB is an esteemed academic and clinician, serves as an Assistant Professor in the Department of Pulmonary Medicine at DY Patil University School of Medicine in Nerul, Navi Mumbai, Maharashtra, India. With an impressive academic career, he has contributed extensively to the field of respiratory medicine. He currently holds the position of Editor for the Yearbook of Respiratory Medicine and the NCCP(I) Lung Bulletin (having completed five issues). He is also the Section Editor for Pulmonology in the MacMaster Textbook of Internal Medicine (Southeast Asia Edition). A prolific author, he has published 70 research articles and contributed 33 chapters to textbooks. He has been recognized for his leadership in the National College of Chest Physicians (India), where he has served as "Joint Secretary" from 2022 to 2024 and was re-elected for the 2024–2026 term. He has been the Organizing Secretary for NAPCON 2016 and 2020 and is the Convenor of the NCCP(I) All-India PG Quiz since 2019. In addition to his leadership roles, he has been instrumental in developing national guidelines for nebulization therapy, pneumococcal and influenza vaccination in adults, pleural effusion management, and treatment protocols for drug-resistant tuberculosis. His significant contributions to medical education and research have earned him numerous prestigious awards, including the Prof PS Shankar and Prof KC Mohanty Chest Oration (2020), the Sardari Lal Dr VK Arora TAI Oration (2024), and multiple Young Scientist Awards from esteemed organizations such as the Indian Chest Society and the Association of Physicians of India. He is highly regarded by peers and mentors, including Prof Emeritus Nadoja Dr PS Shankar, PadmaShri Prof Dr D Behera, and Prof Dr Parvaiz Koul, who have recognized his outstanding contributions to the medical community.

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