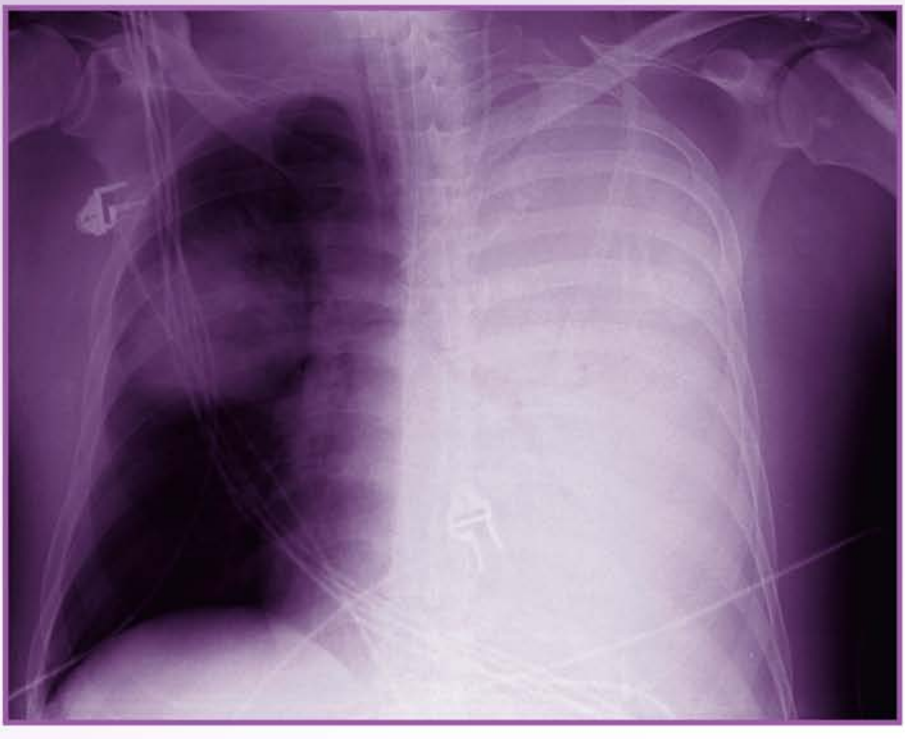
Lung Biology in Health and Disease Volume 213
Executive Editor: Claude Lenfant

Practical Pulmonary and Critical Care Medicine

Respiratory Failure



edited by
Zab Mosenifar
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Practical Pulmonary and Critical Care Medicine

LUNG BIOLOGY IN HEALTH AND DISEASE

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Practical Pulmonary and Critical Care Medicine

Respiratory Failure

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Introduction

Surely, all of us would certainly concur with the first sentence of the Preface prepared by Drs. Mosenifar and Soo Hoo: "Over the past decade, the pace of progress in medicine has been astounding. New developments in diagnosis, management, and therapeutics occur at a breathtaking rate." On the other hand, one could argue with the time frame, that is, why only the past decade? This is a significant point, especially when it comes to pulmonary and critical care. Pulmonary medicine started to blossom many, many decades ago but especially during the last 30 years or so. Critical care started its course, to what it is today, with the development of the oxygen electrode (and later oximetry) and with the recognition of the value of blood pH measurements in the late 1950s. At the same time, the use of ventilators and respiratory assist devices became more common.

However, in reality it matters little when progress began. Today, what counts is that new developments are being introduced at such a fast pace that progress may exceed the ability to fully transfer and utilize all this knowledge in the practice of medicine. Sure enough, tertiary hospitals have the facilities and work force to adjust to and quickly adapt the changes as they occur. On the other hand, hospitals which are community based and distant from academic centers may find

it more difficult to take full advantage of the newer approaches for the management of pulmonary patients and/or critical care situations.

What could be considered a dual standard between tertiary and community hospitals is well recognized today, and has raised concerns of policy makers, politicians, and medical leaders. The question is frequently raised about how fast, and how well, research advances are translated into the practice of medicine, including pulmonary and critical care medicine.

This monograph, *Practical Pulmonary and Critical Care Medicine: Respiratory Failure* and its companion volume titled *Practical Pulmonary and Critical Care Medicine: Disease Management* represent important steps to minimize translation difficulties. Indeed, they both present a very practical approach to pulmonary and critical care medicine. The first volume addresses, and very carefully describes, the "tools" to provide the most optimal care and management of respiratory failure. The second volume examines a number of situations—both pulmonary and non-pulmonary—that require the use of these tools. Together, these volumes will not only enrich the knowledge of the readers, but also will provide a wealth of practical information that has the potential to positively impact on the care of their patients.

The editors, Dr. Zab Mosenifar and Dr. Guy W. Soo Hoo, have assembled contributors with outstanding expertise and a wealth of practical experience, coming from institutions/environments with large patient populations, and a wide variety of cases and medical situations. They share their very practical and real experiences throughout the volumes.

As the Executive Editor of the series of monographs Lung Biology in Health and Diseases, I am proud to present these two volumes and to express my most sincere thanks to the editors and the authors.

Claude Lenfant, MD
Gaithersburg, Maryland, U.S.A.

Preface

Over the past decade, the pace of progress in medicine has been astounding. New developments in diagnosis, management, and therapeutics occur at a breathtaking rate. A new disease emerges and, aided by the ease of transcontinental travel, threatens to become the next pandemic. And just as quickly, the causative agent is identified with diagnostics and therapeutics soon to follow. Old diseases have their mysteries unraveled, and targeted therapeutics provide hope where there was once despair. Some conditions come under control in less than a generation's span, while others plod along inexorably to the end, with little available to alter their course.

Just as the conditions of disease have changed, so have the conduits for information. The speed and availability of electronic databases now allow access to vast warehouses of information at the click of a mouse or flick of a stylus. Where once the resident or houseofficer carried a worn copy of a spiral bound manual, handheld computers are now the essential accessories. The grand old textbooks have followed suit, available in versions abbreviated to fit file-size limits or available in their own electronic internet-based versions. This electronic world not only allows but mandates frequent content changes. Information can be updated daily and even more frequently if necessary. The online resource is now predominant in an arena that was once the domain of the print journal.

Whereas attendance at national or international meetings once offered the latest developments, this information can now be accessed from a remote site and disseminated at near-instantaneous speed, certainly before one can return home.

Why then, in this information rich era, would there be a need for this book? First of all, even though there is instant access and availability to volumes of information, there remains a dearth of practical information. No one functions in the vacuum of cyberspace or isolation of an information warehouse. Everyone faces the limitations of available technology, restricted formularies, time pressures, and treatment preferences. In addition to the wealth of knowledge, one requires the wisdom of experience and expediency of practical management. The best technology or therapeutic is only as effective as the treatment that can be instituted by the lone practitioner. Treatment available only to the few or the very specialized usually has no role in general management.

Filling this void is the guiding premise of this book. Even if mutations threaten to render current antimicrobials ineffective or new disease entities emerge, there remains a need for comprehensive and effective supportive care. This care is best provided not by the disease specialist, but by physicians who carry a broader perspective while also maintaining focus on the most pressing problems. In the new lexicon of medicine, this is the hospitalist or intensivist. Because many of the most immediate life-threatening conditions involve the respiratory tract, management often falls under the domain of pulmonary and critical care, either as the pulmonologist and/or intensivist. The need for coordinated comprehensive care is further highlighted by increasingly vulnerable patients as a result of increased longevity, treatment modalities that strip a patient's immune system, and emerging disease entities. The modern pulmonary and critical care physician not only has to deal with the many complexities of illness, but also must choose the best available approach to facilitate recovery.

This has required the intensivist to assume many roles in patient management, but none more important than as the functional equivalent of a chief executive with oversight over total management. This has required familiarity with areas where they are not generally considered expert, and reliance on a multi-disciplinary approach to care. These sections in the text are authored by experts in the area who provide a broad overview, but highlight those issues most important in managing critically ill patients. This brings the intensivist's view to the specialist's world.

As every bibliophile knows, the advantage of a textbook often lies in the additional or complementary information that is often encountered through perusal of its pages. Electronic sources are often unforgiving in their searches, limiting access to pre-determined and pre-defined categories. With a book, one can often identify the needed content within its covers even if one is not quite certain of the initial focus of inquiry. The topic may not be in the first section, but is invariably covered in a subsequent area.

Practical Pulmonary and Critical Care is divided into two volumes based partly on space limitations and partly by design. This first volume focuses

on immediate management and diagnosis. For the practicing physician, this is often referred to in billing as the first hour or more of critical care management. This often involves the patient with respiratory failure, one of the most common entry conditions into a hospital or critical care unit. These patients often present in extreme distress and the underlying diagnosis may not be immediately apparent or may require further investigation. Other patients are intubated as part of support measures while therapy is directed at other organs.

This volume includes chapters and strategies on immediate management of these patients. This includes an extensive treatise on oxygen strategies, helium–oxygen, and non-invasive ventilation. If the patient fails these conservative measures, the focus shifts to intubation and ventilator management. The alphabet soup of ventilator modes and waveform interpretation is clarified with a section that allows comparisons between multiple vendors. These patients demand close monitoring, and a chapter is dedicated to the nuances of monitoring their response to therapy as well as mechanical ventilation. Other sections deal with the delicate task of discontinuing mechanical ventilation and patients who require long-term ventilatory support.

These patients often have multi-organ system dysfunction. In addition to ventilatory support, they often require other invasive procedures, either therapeutically or to provide or guide therapeutics. A comprehensive section is dedicated to the most commonly performed procedures, with an abundance of detailed figures and tables. This includes coverage of procedures once reserved for surgical colleagues, such as tube thoracostomy or tracheostomy. Equally important are imaging studies that help diagnose and monitor a patient's response to therapy. The section on radiology covers the gamut with respect to patient management, including the use of the radiograph for confirmation of location of invasive devices, diagnosis, and illustrative cases. The radiographs are all derived from recent cases and reproduce well, providing important detail useful in management. Each topic is covered with a focus on details that facilitate its implementation and potential pitfalls, as well as a practical perspective on its role in overall management.

It is this perspective that not only defines this book, but hopefully lends an enduring quality to its content. There have been very few scientific developments that have completely altered the management of patients. Changes are often incremental and incorporated over several years of practical experience, although medications may be the one exception. The experience of time allows techniques to undergo further refinement. Practical aspects of management are highlighted with detailed descriptions of procedures, protocols, or guidelines. There is often much to gain from historical perspective, and this information is judiciously included as well as that which is evidence based. Therefore, even though technology, medications, and formularies have changed over the past decade, patients with acute respiratory failure still have the same basic requirements in their management. Ventilators must provide the best support while inflicting the least amount of ventilator-associated injury. And once the road to recovery is

reached, assisted ventilation should be removed as soon as possible. These basic tenets will not change in the foreseeable future, only the details in implementation. Although a paper-based textbook may not have the allure of electronic media, it can provide a roadmap and framework for efficient and pragmatic care. Once in place, additional information can only enhance the work and final product. We share a common goal to enhance the recovery of patients from a critical illness. We hope this textbook can contribute to that end result.

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1

Oxygen Therapy and Airway Management

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I. Introduction

Oxygen administration and airway management are two of the fundamental aspects of management in a patient with acute respiratory failure. Proper application of technical aspects of oxygen therapy and airway management can be life saving. Despite the importance of these therapies and their frequent use in the acute care setting, their nuances are often under-appreciated.

II. Oxygen Therapy

Oxygen (O_2) is an elemental gas that is necessary for life in aerobic organisms. In the absence of O_2 (hypoxia), cellular respiration ceases and irreversible cellular injury and death occur within minutes. At normal atmospheric pressure and temperature, O_2 exists as an odorless, tasteless, and colorless gas. It represents one-fifth of the earth's atmosphere by volume (20.96%).

A. Medical Oxygen

Medical grade O_2 is manufactured by fractional distillation of liquefied air (1,2). It is stored as a liquid to reduce the size of the storage container (1 L of liquid O_2 produces 860 L of gaseous O_2). The liquid O_2, stored outside the hospital in a cryogenic container, is converted to gaseous O_2 and delivered to the patient

care areas via a bulk gas delivery system. By convention, O_2 is supplied at a pressure of 50 lbs/in^2. Oxygen can also be delivered from a medical gas cylinder at a pressure of 2000 lbs/in^2 when full. Cylinders are frequently used to supply O_2 during transport of patients to remote locations of the hospital, such as diagnostic areas. Cylinders are identified by letter codes to indicate size and gas capacity. The cylinders most frequently used in the hospital are small E-cylinders. They are typically green in color, and a label affixed to the side of the cylinder indicates the cylinder contents. When cylinders are used, it is important to calculate the duration of flow to avoid inadvertent loss of O_2 supply (Table 1).

B. Indications for Oxygen Therapy

The most important indication for O_2 therapy is to treat hypoxemia. The alveolar gas equation illustrates how increasing the inspired O_2 fraction (F_IO_2) increases the alveolar PO_2 (P_AO_2) and subsequently the arterial PO_2 (P_aO_2):

$$P_AO_2 = F_IO_2 \times \text{EBP} - 1.25 \times P_aCO_2$$

where EBP is the barometric pressure corrected for water vapor pressure. The effect of increasing F_IO_2 on the P_aO_2 is a function of the physiologic cause of hypoxemia. In cases of shunt ($V/Q = 0$), supplemental O_2 therapy has little effect on P_aO_2. If the cause of hypoxemia is low V/Q or diffusion defect, supplemental O_2 therapy will effectively increase the P_aO_2.

Table 1 Duration of Flow (in Hours) from an E-Cylinder as a Function of O_2 Flow and the Pressure of Gas Remaining in the Cylinder

Cylinder pressure (lbs/in^2)	Flow (L/min)														
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
500	2.3	1.2	0.8	0.6	0.5	0.4	0.3	0.3	0.3						
600	2.8	1.4	0.9	0.7	0.6	0.5	0.4	0.4	0.3	0.3	0.3				
700	3.3	1.6	1.1	0.8	0.7	0.5	0.5	0.4	0.4	0.3	0.3	0.3	0.3		
800	3.7	1.9	1.2	0.9	0.7	0.6	0.5	0.5	0.4	0.4	0.3	0.3	0.3	0.3	
900	4.2	2.1	1.4	1.1	0.8	0.7	0.6	0.5	0.5	0.4	0.4	0.4	0.3	0.3	0.3
1000	4.7	2.3	1.6	1.2	0.9	0.8	0.7	0.6	0.5	0.5	0.4	0.4	0.4	0.3	0.3
1100	5.1	2.6	1.7	1.3	1.0	0.9	0.7	0.6	0.6	0.5	0.5	0.4	0.4	0.4	0.3
1200	5.6	2.8	1.9	1.4	1.1	0.9	0.8	0.7	0.6	0.6	0.5	0.5	0.4	0.4	0.4
1300	6.1	3.0	2.0	1.5	1.2	1.0	0.9	0.8	0.7	0.6	0.6	0.5	0.5	0.4	0.4
1400	6.5	3.3	2.2	1.6	1.3	1.1	0.9	0.8	0.7	0.7	0.6	0.5	0.5	0.5	0.4
1500	7.0	3.5	2.3	1.8	1.4	1.2	1.0	0.9	0.8	0.7	0.6	0.6	0.5	0.5	0.5
1600	7.5	3.7	2.5	1.9	1.5	1.2	1.1	0.9	0.8	0.7	0.7	0.6	0.6	0.5	0.5
1700	7.9	4.0	2.6	2.0	1.6	1.3	1.1	1.0	0.9	0.8	0.7	0.7	0.6	0.6	0.5
1800	8.4	4.2	2.8	2.1	1.7	1.4	1.2	1.1	0.9	0.8	0.8	0.7	0.6	0.6	0.6
1900	8.9	4.4	3.0	2.2	1.8	1.5	1.3	1.1	1.0	0.9	0.8	0.7	0.7	0.6	0.6
2000	9.3	4.7	3.1	2.3	1.9	1.6	1.3	1.2	1.0	0.9	0.8	0.8	0.7	0.7	0.6

An unusual, but occasionally effective, use of O_2 therapy is the re-absorption of air leaks, such as a small pneumothorax, subcutaneous emphysema, or pneumocephalous (3,4). If the patient is breathing room air, the composition of the leaked gas is presumably 21% O_2 and 78% nitrogen. Breathing 100% O_2 clears nitrogen from the blood, creates a diffusion gradient for nitrogen to diffuse from the extra-vascular space, and thus effectively reduces the volume of the leaked gas. In a theoretical assessment of normobaric O_2 therapy to treat pneumocephalus, Dexter and Reasoner (3) reported that modestly increasing the F_IO_2 from 0.21 to 0.4 decreased the total air absorption time by 67%, whereas increasing the F_IO_2 from 0.8 to 1.0 decreased the total air absorption time by only an additional 3%.

Another important use of O_2 is treatment of carbon monoxide poisoning. The half-life of carboxyhemoglobin is about five hours breathing 21% O_2 at ambient pressure, a little more than one hour breathing 100% O_2 at ambient pressure, and $<$30 min breathing 100% O_2 at 3 atm of pressure. Weaver et al. (5) conducted a double-blind, randomized trial to evaluate the effect of hyperbaric-oxygen treatment on cognitive sequelae that commonly occur following carbon monoxide poisoning. Patients with symptomatic acute carbon monoxide poisoning were assigned to three hyperbaric oxygen treatments or one normobaric-oxygen treatment plus two sessions of exposure to normobaric room air. Cognitive sequelae at six weeks were less frequent in the hyperbaric-oxygen group (25%) than in the normobaric-oxygen group (46%).

Anaerobic infections and necrotizing fasciitis (6) may also be responsive to O_2 therapy. However, the treatment is usually with hyperbaric oxygen, rather than normobaric oxygen, and this application of hyperbaric O_2 therapy is controversial.

C. Monitoring Oxygen Therapy

Oxygen should not be administered without an objective assessment of its effect. Because the most frequent indication for O_2 therapy is to treat hypoxemia, the clinical effect of O_2 administration is usually monitored with pulse oximetry or arterial blood gas analysis. Most commonly, pulse oximetry is used and a target O_2 saturation (S_pO_2) of $>$92% is acceptable. If the O_2 therapy is assessed by arterial blood gas analysis, a $P_aO_2 > 60$ mm Hg is usually acceptable.

S_pO_2 or P_aO_2 should always be assessed relative to the amount of inspired O_2. In the case of low-flow O_2 delivery systems (e.g., nasal cannula), the arterial O_2 level is assessed relative to the administered O_2 flow. In the case of high-flow O_2 delivery systems and during mechanical ventilation, the arterial O_2 level is assessed relative to the F_IO_2.

Oxygen analyzers use one of several methods to measure O_2 concentration(2). Polarographic and galvanic cell analyzers use an electrochemical principle to measure changes in PO_2, which is then converted to a display of $\%O_2$. Paramagnetic analyzers use the Pauling principle, which is based on the fact that

O_2 is a paramagnetic gas. A wheatstone bridge O_2 analyzer uses the principle of thermoconductivity. In the zirconium analyzer, an electric potential is developed across heated zirconium oxide that is proportional to the PO_2. Zirconium analyzers are very precise and used in applications such as indirect calorimetry.

D. Physiologic Complications of Oxygen Therapy

Worsening hypercapnia with O_2 administration in patients with chronic obstructive pulmonary disease (COPD) has been of concern for many years. This has resulted in conservative use of O_2 in these patients and has likely produced unnecessary hypoxemia in some patients. In patients with stable COPD, continuous O_2 administration has been shown to be life-prolonging (7). Oxygen should never be withheld from a hypoxemic patient in acute respiratory failure. Moreover, the risk may be relatively low (8), and the mechanism for hypercarbia when O_2 is administered to the patient with COPD is controversial. When hypercarbia occurs with O_2 administration, this may be due to the Haldane effect (9), an increase in alveolar dead space due to the effects of O_2 on pulmonary blood flow and $\dot{V}/\dot{Q}$ mismatch, or suppression of the respiratory drive. Aubier et al. (10) reported that an increased P_aCO_2 following O_2 administration in patients with COPD occurred with no change in minute ventilation. Crossley et al. (11) reported that an increased F_IO_2 in mechanically ventilated patients with COPD (P_aO_2 increase to about 200 mm Hg) did not change P_aCO_2, respiratory drive, or dead space. Gomersall et al. (12) reported no significant difference in requirement for mechanical ventilation or mortality for patients with COPD in acute respiratory failure in whom O_2 was administered with a goal $P_aO_2 > 50$ or >70 mm Hg. However, Robinson et al. (13) reported a reduction in minute ventilation and an increase in alveolar dead space in patients with COPD who had worsening hypercapnia with O_2 administration. These results, as well as those by Dick et al. (14) and Dunn et al. (15), suggest that a decrease in ventilatory drive may contribute to the hypercapnia that occurs with O_2 administration in some patients with COPD.

Concern for hypercapnia is also reported in patients with asthma exacerbation. Chien et al. (16) reported an increase in P_aCO_2 in patients with acute asthma who received an F_IO_2 of 1.0, but the increase in P_aCO_2 was modest (maximum of 10 mm Hg) and the clinical significance of this is unclear.

Absorption atelectasis has been demonstrated in patients breathing 100% O_2 (17). Absorption of gas behind occluded airways is more likely to produce atelectasis if the alveolar gas composition is 100% O_2 rather than room air. If the alveolar gas is room air, uptake of O_2 results in an alveolar volume change of 20%, delaying the onset of atelectasis. Although this effect has been demonstrated primarily during anesthesia, it is likely that it might also occur during acute respiratory failure. In mechanically ventilated patients, this complication may be avoided by the use of positive end-expiratory pressure (PEEP).

Experimental evidence implicates the formation of reactive O_2 species (superoxide anion, hydroxyl radical, and hydrogen peroxide) in the pathogenesis of pulmonary O_2. The clinical importance of this, however, has been debated for many years (18,19). It is common teaching that maintaining the $F_IO_2 < 0.6$ avoids O_2 toxicity, although there are few data to support this. Early human reports of "respirator lung" showed septal edema, endothelial cell damage, hyaline membrane deposition, and interstitial fibrosis. However, these findings may have been the result of high ventilating pressure rather than hyperoxia per se. Normal human volunteers exposed to hyperoxia develop chest discomfort, dyspnea, cough, headache, and paresthesia within 12 hours of exposure and these effects persist for several days. However, similar effects have not been shown to occur in patients with acute respiratory failure.

Patients treated with bleomycin have an increased risk for oxygen-induced lung toxicity (20). An F_IO_2 of 0.35 to 0.4 is sufficient to cause toxicity in this setting. Bleomycin may impair the lungs' antioxidant defenses, increasing the burden of reactive O_2 species produced in the lung by a high F_IO_2. Bleomycin O_2 toxicity may be steroid responsive.

Retinopathy of prematurity (21) and bronchopulmonary dysplasia (22) are complications that are associated with O_2 therapy in infants. Similar disorders are not known to occur in patients older than neonates.

E. Technical Hazards of Oxygen Therapy

An important physical characteristic of O_2 is its ability to support combustion. Accordingly, the potential fire hazard of O_2 should be appreciated when supplemental O_2 is administered. Improper handling and storage of high-pressure gas cylinders can result in rapid escape of gas, turning the cylinder into a dangerous projectile. Because O_2 is stored dry, it must be humidified when delivered at high concentrations. This is of particular concern when the upper airway is bypassed with an endotracheal tube (ETT) or tracheostomy tube. When low-flow O_2 is administered, the need for humidification is less, and humidifiers for this application have not been shown to be useful (23–25).

F. Oxygen Administration Devices

Oxygen therapy systems are generally categorized as either low- or high-flow devices (1,2). Low-flow devices deliver O_2 at flow rates insufficient to meet the inspiratory flow demand of the patient. The additional required flow is inhaled from the room air. The F_IO_2 from low-flow devices may vary from breath-to-breath, depending on the breathing pattern. High-flow devices provide flow sufficient to meet the patient's inspiratory flow demand and thus maintain a precise F_IO_2 that is unaffected by changes in breathing pattern. Relevant characteristics of O_2 delivery devices are shown in Table 2.

The nasal O_2 cannula (Fig. 1) is the most widely used O_2 delivery device. It consists of two short, soft, pliable plastic prongs, each about one-half inch in

Table 2 Oxygen Delivery Devices for Adult Applications

Device	Usual flow range	Approximate oxygen concentration	Comments
Nasal cannula	1–6 L/min	24–40%	F_IO_2 is reduced with nasal obstruction; can be less effective with mouth breathing; F_IO_2 varies with breathing pattern
Simple mask	5–10 L/min	30–60%	Flows <5 L/min result in rebreathing; F_IO_2 varies with breathing pattern
Non-rebreathing mask	Flow must be high enough to prevent full collapse of reservoir bag during inhalation; flows ≥ 12 L/min are often required	Theoretically, a non-rebreathing mask will deliver close to 100% O_2; in reality, however, it delivers concentrations of 60–80% because the mask does not fit tightly over the face	If S_pO_2 remains low despite use of a non-rebreathing mask, consider using a high-flow O_2 delivery device
Air-entrainment mask	Use at least the flow stamped on colored adapter	O_2 concentration is stamped on the colored adapter	When mask is removed, administer O_2 by nasal cannula to provide target S_pO_2
High-flow oxygen system	>30 L/min	24–100%, set by air and O_2 flow meters on blender	Gas should be humidified with high-flow system

length. The cannula is easily applied and well tolerated by most patients when used with flows of ≤ 6 L/min. It is held in place by an elastic band around the head or, more commonly, by looping the delivery tubing over the ears and holding it in place with an adjustable slide under the chin. Oxygen delivery by nasal cannula produces an F_IO_2 of approximately 0.24 at 1 L/min to about 0.40 at 6 L/min. The F_IO_2 from a nasal cannula is highly variable even if the O_2 flow remains constant (Table 3). A number of studies using various methodologies confirm that the F_IO_2 resulting from use of a nasal cannula is highly variable and dependent upon the O_2 flow, inspiratory flow, and minute ventilation (26–36). The amount of delivered O_2 is reduced if O_2 is breathed from only one of the prongs, as may occur if the prongs are displaced or there is unilateral nasal obstruction (37). There are

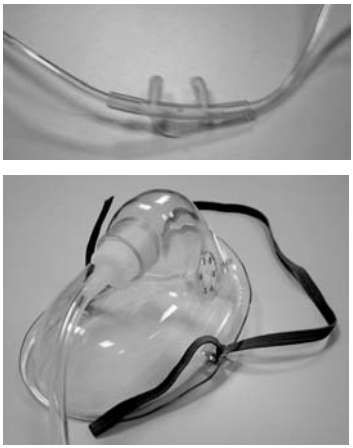

Figure 1 Nasal cannula and simple mask.

conflicting data regarding the effect of mouth breathing when a nasal cannula is used (38,39). Although mouth breathing may decrease the F_IO_2, O_2 therapy by nasal cannula can be effective even with mouth breathing.

The majority of O_2 is wasted when it is delivered by nasal cannula. Most of the O_2 flow during the expiratory phase is wasted. It is only during the initial period of inspiration that the supplemental flow of O_2 is needed. It is this first part of inspiration that is delivered to the alveoli and participates in gas exchange. The remainder of the inspired gas fills the anatomic dead space and is exhaled without contributing in gas exchange. Oxygen-conserving devices conserve O_2 by using a reservoir, which provides O_2 flow only during the inspiratory phase,

Table 3 Estimation of F_IO_2 from a Nasal Cannula

Cannula flow	2 L/min (33 mL/sec)
Tidal volume	500 mL
Anatomic reservoir	50 mL (nasal passages and nasopharynx)
Inspiratory time	1 sec
Volume of O_2 inspired	
33 mL	O_2 flow
50 mL	Anatomic reservoir
88 mL	Amount of O_2 in the inspired air; 417 mL × 0.21
Volume O_2 inspired 171 mL	

$F_IO_2 = 171$ mL $(O_2)/500$ mL (tidal volume) $= 0.34$. Similar calculations can be used to calculate the effects of changes in O_2 flow or inspiratory time.

Table 4 Oxygen Delivery Systems Designed to Conserve O_2 by Eliminating Waste During the Expiratory Phase

Reservoir O_2 cannula (*moustache and pendant types*): A small reservoir fills with O_2 (20 mL for the moustache style and 40 mL for the pendant) during exhalation. At the beginning of inhalation, a bolus of O_2 is drawn from the reservoir. This may allow a reduction in O_2 flow by 50–75%. Reservoir cannulae are not well accepted by patients due to their appearance.
Transtracheal O_2 catheter: This is a small-diameter catheter surgically inserted into the trachea. It is connected to a small flange and held in place by an adjustable chain. As O_2 is continuously delivered directly into the trachea, a reduction in the O_2 flow by about 50% is possible. Complications of the catheter placement include infection, bleeding, and subcutaneous emphysema. The catheter must be cleaned regularly to prevent mucus accumulation. Catheter obstruction is prevented by instilling saline and inserting a cleaning wire into the lumen of the catheter.
Demand oxygen conservers: These devices only deliver O_2 during the inspiratory phase. When the patient begins an inspiration, this creates a negative pressure in the supply tubing and causes a demand valve to open and supply a dose of O_2.

either by providing an O_2 bolus at the beginning of inspiration or by administering O_2 directly into the trachea (Table 4) (40,41). These devices are not commonly used in the acute care settings, but patients receiving chronic O_2 therapy may use one of these systems. Considerable variability in the performance of these devices has been reported (42).

The simple O_2 mask (Fig. 1) is used when a higher F_IO_2 is needed than can be attained with a nasal cannula or when a cannula is not appropriate (e.g., with nasal obstruction). The simple mask increases the F_IO_2 over that achieved by a nasal cannula because it adds a volume of 100 to 200 mL over the face, which serves as an O_2 reservoir. Additional air is inhaled through small holes in the mask. To avoid rebreathing, a minimum flow of 5 L/min must be used with O_2 delivery by face mask (43). The simple mask is a low-flow O_2 delivery device capable of providing an F_IO_2 of 0.3 to 0.6 at flows of 5 to 10 L/min (44). The F_IO_2 is dependent on the size of the mask and the patient's breathing pattern. Simple masks are subjectively less appealing than nasal cannulae and may cause claustrophobia, muffling of speech, and difficulty eating and drinking.

The non-rebreathing mask (Fig. 2) increases the F_IO_2 by adding a reservoir bag. It has a one-way valve between the bag and the mask and another one-way valve over one or both mask ports. All of the exhaled tidal volume is directed through the mask ports because of the valve between the mask and the bag. The bag fills with 100% O_2 during exhalation. During inhalation, the mask valves close and the bag valve on the reservoir opens. The O_2 flow must be high enough to prevent the bag from emptying during inhalation. Theoretically, the

Figure 2 Non-rebreathing mask.

non-rebreathing mask will deliver 100% O_2. However, these masks do not provide an airtight fit on the face, and the valves do not provide a perfect seal (45). At flows of 10 to 15 L/min, an F_IO_2 of 0.6 to 0.8 may be achieved. Moreover, the non-rebreathing masks have a valve over only one of the exhalation ports. This allows inhalation of room air if the O_2 supply flow becomes inadequate.

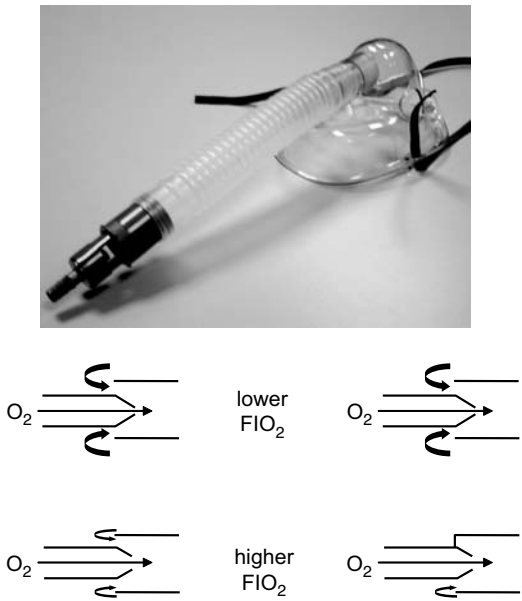

Figure 3 The air-entrainment mask and its principle of operation.

G. High-Flow Devices

An air-entrainment mask (Fig. 3) consists of a mask, a jet nozzle, and air-entrainment ports (46–49). Oxygen is delivered through the jet nozzle, which increases its velocity. The high-velocity O_2 entrains ambient air into the mask due to the viscous shearing forces between the gas traveling through the nozzle and the stagnant ambient air. The F_IO_2 depends on the nozzle size and the size of the entrainment ports. Commercially available systems use interchangeable jets or adjustable entrainment ports. Obstruction of the entrainment port or downstream obstruction decreases entrainment and increases F_IO_2. To deliver a fixed F_IO_2, the flow to the mask must exceed the peak inspiratory flow of the patient. This may be difficult to achieve with a higher F_IO_2 settings (Table 5).

A high-flow O_2 delivery system can be used to deliver a precise F_IO_2 and any concentration between 0.21 and 1.0. Such a system provides sufficient flow to meet the inspiratory demands of the patient (50). Because the total flow is set higher than the inspiratory flow of the patient (typically 30–60 L/min), the gas should be humidified. An air and an O_2 flow meter can be used to deliver a precise F_IO_2 (Fig. 4) and a variety of patient interfaces can be used (Fig. 5). The F_IO_2 can be calculated from the flow rates of air and O_2 (Table 6). A blender uses pressurized sources of air and O_2 to deliver a precise F_IO_2. Blenders are compact and convenient but more expensive than using two flow meters.

H. Heliox

Heliox is a gas mixture of helium and oxygen that is clinically useful in some circumstances due to its low density. Because helium does not support life, it must always be delivered in a gas mixture containing at least 20% oxygen. There is an increasing interest in its therapeutic use in patients with obstructive lung

Table 5 F_IO_2, Minimum Flow Requirements, Outputs, and Entrainment Ratios for an Air-Entrainment Mask

F_IO_2 setting	Minimum O_2 flow (L/min)	Entrainment ratio (O_2:Air)	Total flow (L/min)
0.24	4	1:25	104
0.28	4	1:10	44
0.31	6	1:7	48
0.35	8	1:5	48
0.40	8	1:3	32
0.50	12	1:1.7	32
0.60	12	1:1	24
0.70	12	1:0.6	19

Source: From Branson RD. The nuts and bolts of increasing arterial oxygenation: devices and techniques. Respir Care 1993; 38:672–686.

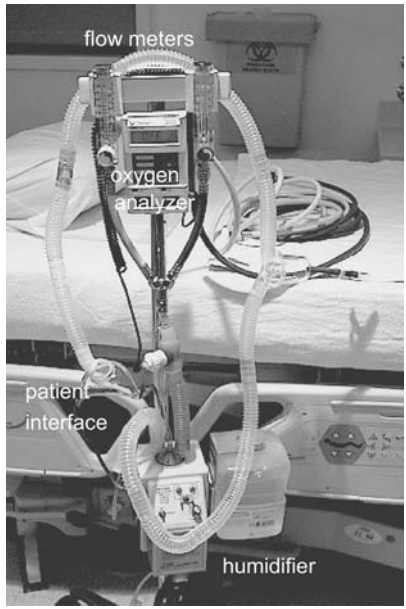

Figure 4 High-flow O_2 delivery system.

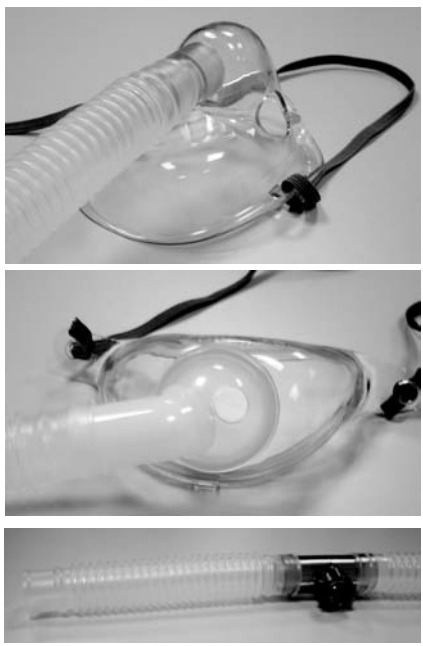

Figure 5 Patient interfaces used with high-flow O_2 delivery systems.

Table 6 Air:O_2 Ratios and Determination of Air and O_2 Flows

Air:O_2 = (1.0 − F_IO_2)/(F_IO_2 − 0.21)
If an F_IO_2 of 0.4 is desired:
Air:O_2 = (1.0 − 0.4):(0.4 − 0.21) = 0.6:0.2 = 3:1
If a total flow of 60 L/min is required, an air flow of 45 L/min and an O_2 flow of 15 L/min will produce a F_IO_2 of 0.4.
If an F_IO_2 of 0.6 is desired:
Air:O_2 = (1.0 − 0.6):(0.6 − 0.21) = 0.4:0.4 = 1:1
If a total flow of 60 L/min is required, an air flow of 30 L/min and an O_2 flow of 30 L/min will produce a F_IO_2 of 0.6.

diseases, and this has been the source of several reviews (51,52) and meta-analyses (53,54). Although the role of heliox has been reported beneficial in case series and anecdotal reports, current evidence is insufficient to allow a recommendation for the use of heliox as a standard therapy for any specific patient population.

One use of heliox is to reduce resistance with upper airway obstruction (55,56). An example is post-extubation stridor, where use of heliox has been reported to be beneficial (57). There is also interest in the use of heliox for acute asthma (58,59). In spontaneously breathing patients with asthma, heliox decreases P_aCO_2, increases peak flow, and decreases pulsus paradoxus. There may be benefit related to the combination of heliox with aerosol bronchodilator delivery in patients with acute asthma or COPD (60–63). When heliox (rather than air or oxygen) is used to power the nebulizer, the flow should be increased by about 50% to assure adequate output from the nebulizer (64). As demonstrated in several meta-analyses, however, the benefit of heliox in the management of patients with acute asthma has yet to be conclusively demonstrated (53,54). The role of heliox in the treatment of COPD is unclear (65–67). COPD is a disease of small airways—a region of the lungs in which flow is density independent. Heliox has been reported to decrease work of breathing in some, but not all, patients with COPD when evaluated just prior to extubation (68). Benefit has been reported for the use of heliox with non-invasive ventilation in patients with COPD (69–72), and methods to administer heliox with a BiPAP ventilator have been described (73).

Care must be taken to administer heliox in a safe and effective manner. To avoid administration of a hypoxic gas mixture, it is recommended that 20% oxygen/80% helium is mixed with oxygen to provide the desired helium concentration and F_IO_2. If an F_IO_2 greater than 0.40 is required, the limited concentration of helium is unlikely to produce clinical benefit. For spontaneously breathing patients, heliox is administered by face mask with a reservoir bag (Fig. 6). A Y-piece attached to the mask allows concurrent delivery of aerosolized medications. Sufficient flow is required to minimize contamination of

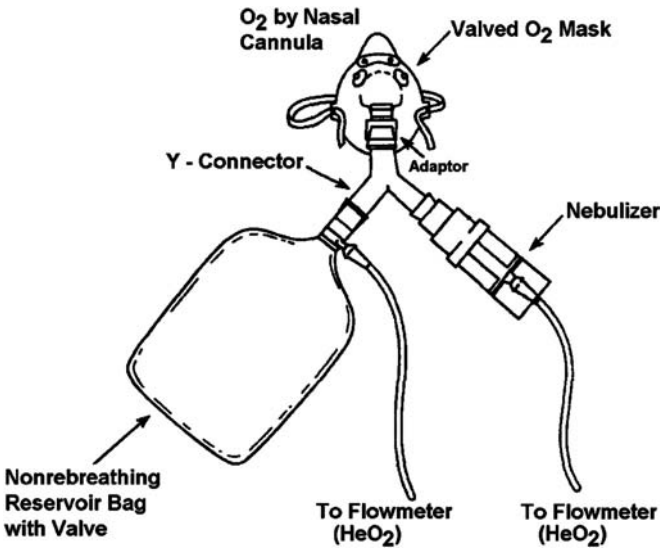

Figure 6 Delivery system for heliox administration for spontaneously breathing patients.

the heliox with ambient air. This is often at least 12 to 15 L/min and requires 3 to 6 H-size cylinders per day. When using an oxygen-calibrated flow meter for heliox therapy, it must be remembered that the flow of heliox (80% helium and 20% oxygen) will be 1.8 times greater than the indicated flow. Heliox administration during mechanical ventilation can be problematic (74–78). Ventilators are designed to deliver a mixture of air and oxygen. The density, viscosity, and thermal conductivity of helium affect the delivered tidal volume and the measurement of exhaled tidal volume. With some ventilators (e.g., Puritan–Bennett), no reliable tidal volume is delivered with heliox, whereas there may be a much higher delivered tidal volume than desired for other ventilators. One commercially available ventilator has been approved for use with heliox (VIASYS AVEA, Palm Springs, California, U.S.A.).

III. Airway Management

When oxygen delivery remains compromised without mechanical assistance, endotracheal intubation should be considered. The goal of this section is to describe the practical aspects of endotracheal intubation. Fundamental key points useful in enhancing a successful endotracheal intubation will be discussed.

A. Indications

Endotracheal intubation is indicated to protect the airway and reduce the risks of pulmonary aspiration. It also offers a pathway for patients who require prolonged positive pressure ventilation and frequent suctioning. In addition, the ETT can be used to administer emergency medications when intravenous access is not available. Most frequently, endotracheal intubation enables the patient to undergo surgical procedures and allow delivery of inhalational anesthetics. Outside the operation room, endotracheal intubation typically involves patients who are in respiratory failure, shock, or cardiopulmonary arrest (79). Despite the increasing use of non-invasive ventilation, most patients receive mechanical ventilation invasively via an ETT.

B. Nasotracheal vs. Orotracheal Intubation

The ETT can be placed either nasally or orally. Nasotracheal intubation is typically indicated for patients undergoing oral surgeries or when intubation through the mouth is unsuccessful. It is contraindicated in patients with basilar skull fracture, mid-facial trauma, coagulopathy, nasal polyps, and epistaxis. For long-term mechanical ventilation, orotracheal intubation is preferred because a nasotracheal tube may increase the work of breathing as well as the risk of sinusitis. Because the orotracheal route usually permits placement of a larger ETT, clearance of secretions may also be easier.

C. Airway Assessment

A thorough airway evaluation should be performed prior to intubation to assess the degree of difficulty in airway management. Careful review of the medical history and a detailed examination of the anatomic characteristics allow identification of potential difficult mask ventilation or tracheal intubation.

Anatomic features such as a short muscular neck, receding mandible, prominent upper incisors, small mouth with a high arched palate, and limited movement of the mandible suggest increased likelihood of potential airway problems (80).

D. Predicting Difficult Mask Ventilation

Langeron and colleagues (81) identified five independent predictors for difficult mask ventilation: age >55 years, body mass index $>26\ \mathrm{kg/m^2}$, lack of teeth, presence of a beard, and history of snoring. When two or more of the risk factors are present, the likelihood of difficulty with mask ventilation is high. Furthermore, difficult intubation is encountered more frequently when difficult ventilation is present.

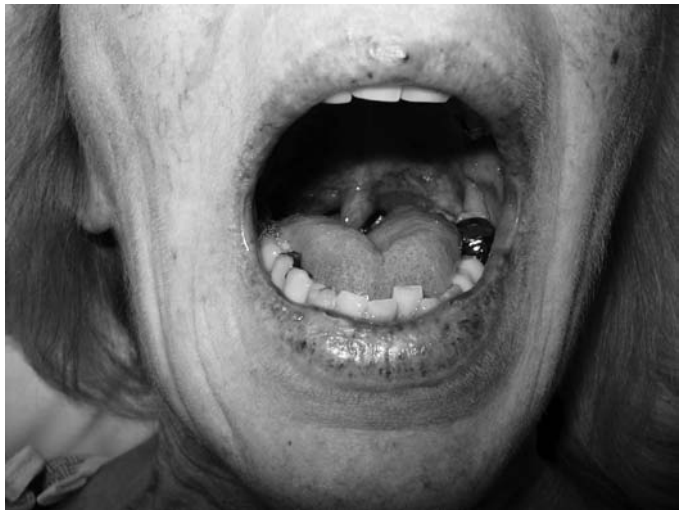

Figure 7 An example of the Mallampati class I airway.

E. Predicting Difficult Tracheal Intubation

The Mallampati classification (82) is widely used in predicting the ease of laryngoscopy. It compares tongue versus pharyngeal size. The observation is divided into four categories. The evaluation is performed while the patient is sitting with mouth open and tongue protruding. Visualization of the oropharyngeal structures is then noted (Fig. 7).

- Class I: the soft palate, uvula, and faucial pillars are all visible.
- Class II: the tonsillar pillars and the base of uvula are obscured by the base of the tongue.
- Class III: only the soft palate is visible.
- Class IV: soft palate is not visible.

When the pharyngeal class is Mallampati III or IV, a more difficult intubation is expected. Unfortunately, it is not easy to use the Mallampati classification for urgent or emergent intubation.

Simple measurement of the thyromental distance, which is the length between the prominence of the thyroid cartilage and the bony point of the chin, can be used to aid in predicting difficult intubation. When the thyromental distance is <7 cm and the Mallampati class is III or IV, difficult intubation can be anticipated (83). The performance of the Mallampati test and the measurement of the thyromental distance should identify most cases of difficult intubation and allow appropriate preparations.

F. Laryngeal Visualization by Direct Laryngoscopy

Laryngoscopic view of the glottic opening is also used to predict the ease of tracheal intubation (84). The extent of laryngeal exposure is categorized into grades I to IV. When the glottic opening is fully visualized (grade I view), endotracheal intubation is relatively easy. If only the posterior portion of the glottic opening is seen (grade II view), intubation is technically more difficult. When the laryngoscopic view is grade III (only the tip of epiglottis is visible) or grade IV (only the soft palate is seen), success of tracheal intubation by direct laryngoscopy is expected to be low.

G. Maneuvers to Improve Laryngoscopic Visualization

Better laryngeal visualization of the glottic opening facilitates endotracheal intubation. Several simple techniques have been advocated to improve laryngoscopic visualization of the glottis. The "BACK" maneuver (simple back-pressure on the thyroid or cricoid cartilage) displaces the larynx posteriorly and reduces the failure rate to visualize any part of the glottis from about 9.2% to 1.6% (85). The "BURP" maneuver (backward, upward, and rightward pressure of the larynx) has also been reported to improve laryngeal visualization (86). In a comparative study (87), both the BURP maneuver and the BACK maneuver were found to be effective. However, the BURP maneuver was shown to be superior. Neither maneuver was associated with significant complications.

The jaw thrust maneuver is a technique commonly used to relieve laryngeal obstruction caused by the base of the tongue. By grasping the angles of the jaw with one hand on each side, the mandible can be displaced forward to keep the pharyngeal airway patent (88). The mandibular advancement can also improve

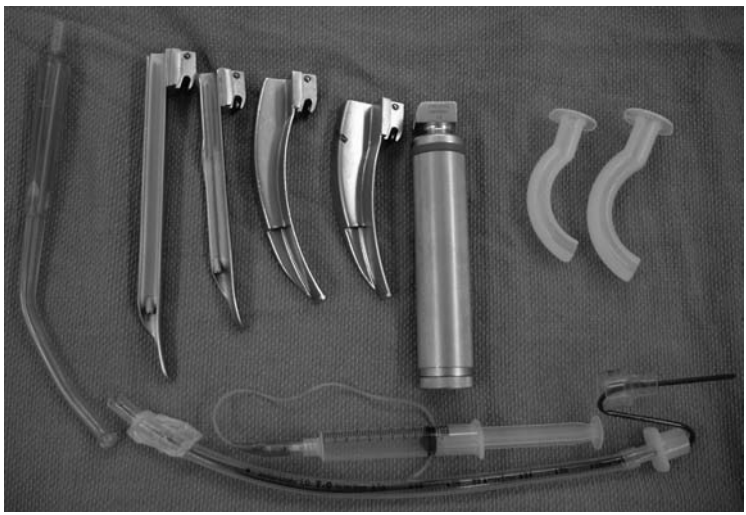

Figure 8 Equipment for endotracheal intubation.

Table 7 Medications Commonly Used to Facilitate Endotracheal Intubation

	Induction agent		Depolarizing muscle relaxant	Non-depolarizing muscle relaxant	
Medication	Propofol	Etomidate	Succinylcholine	Rocuronium	Vecuronium
Concentration	10 mg/mL	2 mg/mL	20 mg/mL	10 mg/mL	1 mg/mL
Dosage	1–2 mg/kg	0.2–0.6 mg/kg	1–2 mg/kg	0.6–1.2 mg/kg	0.08–0.1 mg/kg
Absolute contraindications	Hypersensitivity to propofol, soybean oil, egg, and glycerol	Known sensitivity to etomidate	Malignant hyperthermia susceptibility, myopathies, burns, increased intracranial pressure	Known hypersensitivity to rocuronium	Known hypersensitivity to vecuronium
Side effects/clinical considerations	Hypotension, apnea, pain at injection site	Myoclonus, adrenal suppression	Bradycardia, hyperkalemia, increased intracranial pressure	Increased sensitivity in patients with myasthenia gravis, Eaton–Lambert syndrome	Increased sensitivity in patients with myasthenia gravis, Eaton–Lambert syndrome

Source: From Donnelly AJ, Cunningham FE, Baughman VL. Anesthesiology and Critical Care Drug Handbook. Ohio: Lexi-Comp Inc., 2000.

laryngeal inlet view during nasal fiberoptic laryngoscopy (89). Tamura and colleagues (90) have also demonstrated that such a maneuver can improve laryngeal view during laryngoscopy performed by inexperienced physicians. When the mandibular advancement is combined with the BURP maneuver, further improvement of laryngeal visualization can be obtained.

H. Preparation for Orotracheal Intubation

Before attempting endotracheal intubation, preparation is needed to ensure that the equipment (Fig. 8) is working and medications (Table 7) are available.

1. *Laryngoscope*: a laryngoscope consists of a handle and a blade. The number on the blade reflects its length; therefore, an adult with a long neck may require a larger blade. The blade can be curved (MacIntosh), straight (Jackson–Wisconsin), or straight with a curved tip (Miller). A curved (MacIntosh) #3 blade is most commonly used for intubation in adults; however, the choice of the blade is entirely dependent on individual preference. A MacIntosh blade may provide more room to pass the ETT, while the straight blade gives better exposure of the glottic opening. Regardless, blades of different size and shape should be readily available. When checking the laryngoscope, it is important to ensure that there are batteries in the handle and the light in the blade illuminates well.
2. *ETT*: a proper-sized ETT should be determined prior to intubation, usually an internal diameter of 7.5 mm for adult males and 7.0 mm for adult females. If bronchoscopy is anticipated, endotracheal tubes with 8.0 mm or larger internal diameters are preferred, as the smaller ETTs may hinder or prevent the passage of the adult bronchoscope. When examining the ETT, the cuff should be gently inflated with a syringe and checked for leaks. A stylet can be inserted in the ETT to provide rigidity, which can allow the practitioner to direct the ETT with more control.
3. *Oral airway*: 8.0 and 9.0 mm oral airways are commonly used in adults to maintain a patent conduit above the laryngeal inlet, just cephalad to the epiglottis. Choosing the proper size oral airway is important as airway obstruction can occur if the inserted oral airway is too big or too small. When an ideal size oral airway is placed on the side of the patient's face, the proximal end of the oral airway should be at the lip and the distal tip of the oral airway at the angle of the jaw.
4. *Medications*: induction agents are commonly used to sedate or ensure unconsciousness in patients prior to direct laryngoscopy. Muscle relaxants, either depolarizing or non-depolarizing, are often utilized to facilitate orotracheal intubation. Vasoconstrictors, such as phenylephrine, and antihypertensive drugs, such as labetalol, should be readily available to maintain hemodynamic stability.