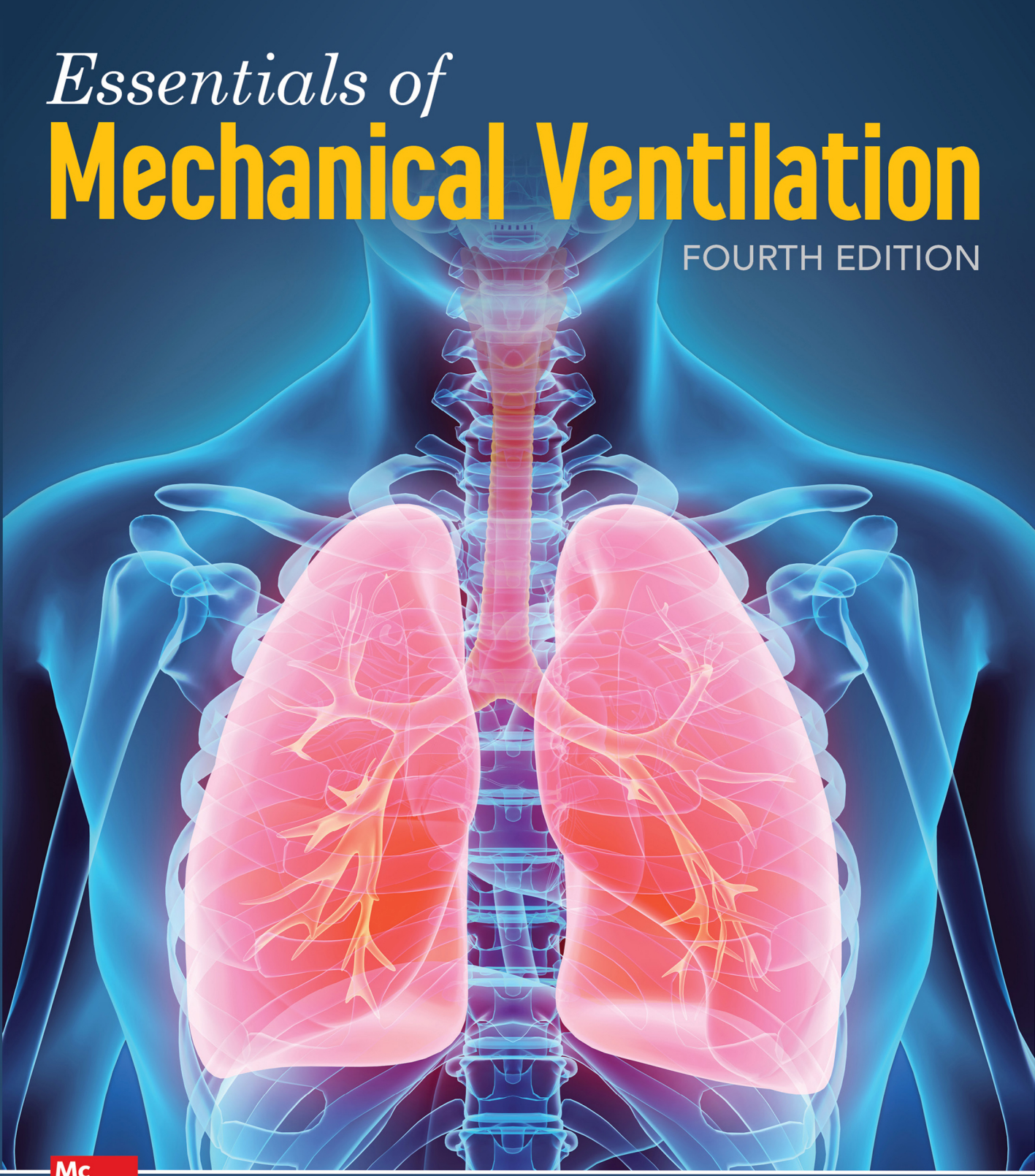
Essentials of Mechanical Ventilation

FOURTH EDITION



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Essentials of Mechanical Ventilation

Fourth Edition

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Dedication

For my wife, Susan; my daughters, Terri and Lauren; their spouses, Rob and Matt; and my grandchildren, Max, Abby, and Caris—who make every day enjoyable.
D.R.H.

For Cristina, the love of my life, and my children Robert, Julia, Katie, and Callie, who make it all worthwhile.
R.M.K.

Contents

Preface

Mechanical ventilation is an integral part of the care of many critically ill patients. It is also provided at sites outside the ICU and outside the hospital, including long-term acute care hospitals and the home. A thorough understanding of the essentials of mechanical ventilation is requisite for respiratory therapists and critical care physicians. A general knowledge of the principles of mechanical ventilation is also required of critical care nurses, mid-level providers, hospitalists, and primary care physicians whose patients occasionally require ventilatory support.

This book is intended to be a practical guide to adult mechanical ventilation. We have written this book from our perspective of nearly 100 years of experience as clinicians, educators, researchers, and authors. We have made every attempt to keep the topics current and with a distinctly clinical focus. We have reviewed every word and updated the content as necessary. We have added new content such as mechanical ventilation of the obese patient and advanced monitoring procedures. Concepts such as driving pressure are included. We have checked the content against recently published clinical practice guidelines. As in the previous editions, we have kept the chapters short, focused, and practical.

Like previous editions, the book is divided into four parts. Part 1, *Principles of Mechanical Ventilation*, describes basic principles of mechanical ventilation and then continues with issues such as indications for mechanical ventilation, appropriate physiologic goals, and liberation from mechanical ventilation. Part 2, *Ventilator Management*, gives practical advice for ventilating patients with a variety of diseases. Part 3, *Monitoring During Mechanical Ventilation*, discusses blood gases, hemodynamics, mechanics, and waveforms. In the final part, *Topics Related to Mechanical Ventilation*, we discuss issues such as airway management, aerosol delivery, and extracorporeal life support.

This is a book about mechanical ventilation and not mechanical ventilators per se. We do not describe the operation of any specific ventilator (although we do discuss some modes specific to some ventilator types). We have tried to keep the material in this book generic and it is, by and large, applicable to any adult mechanical ventilator. We do not cover issues related to pediatric and neonatal mechanical ventilation. Because these topics are adequately covered in pediatric and neonatal respiratory care books, we have limited the focus of this book to adult mechanical ventilation. Although we provide a short list of suggested readings at the end of each chapter, we have specifically tried to make this a practical book and not an extensive reference book.

This book is written for all clinicians caring for mechanically ventilated patients. We believe that it is unique and hope you will enjoy reading it as much as we have enjoyed writing it.

Dean R. Hess, PhD, RRT
Robert M. Kacmarek, PhD, RRT

Abbreviations

A/C	Assist/control
AG	Anion gap
APRV	Airway pressure release ventilation
ARDS	Acute respiratory distress syndrome
ARDSnet	ARDS Network
AVAPS	Average volume assured pressure support
BAL	Bronchoalveolar lavage
BE	Base excess
BEE	Basal energy expenditure
BSA	Body surface area
CCI	Chronic critical illness
Cao_2	Oxygen content of arterial blood
$Cc'o_2$	Pulmonary capillary oxygen content
CDC	Centers for Disease Control and Prevention
CI	Cardiac index
C_L	Lung compliance
Cl^-	Chloride ion
CMV	Continuous mandatory ventilation
CO	Carbon monoxide
Co_2	Oxygen content of the blood
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CPP	Cerebral perfusion pressure
CPR	Cardiopulmonary resuscitation
CSV	Continuous spontaneous ventilation
CT	Computed tomography
$C\bar{v}o_2$	Mixed venous oxygen content
CVP	Central venous pressure
C_W	Chest wall compliance
Do_2	Oxygen delivery
EAdi	Electrical activity of the diaphragm
ECLS	Extracorporeal life support
ECMO	Extracorporeal membrane oxygenation
EELV	End-expiratory lung volume
EPAP	Expiratory positive airway pressure
f_b	Frequency of breathing; respiratory rate
f_c	Heart rate
Fio_2	Fraction of inspired oxygen
FRC	Functional residual capacity
Hb	Hemoglobin
HbCO	Carboxyhemoglobin
HCO_3^-	Bicarbonate concentration
HFJV	High-frequency jet ventilation
HFOV	High-frequency oscillatory ventilation
HFPPV	High-frequency positive pressure ventilation

HFV	High-frequency ventilation
HME	Heat and moisture exchanger
Hz	Hertz
I:E	Inspiratory time to expiratory time ratio
ICP	Intracranial pressure
ICU	Intensive care unit
IMV	Intermittent mandatory ventilation
iNO	Inhaled nitric oxide
IPAP	Inspiratory positive airway pressure
ISB	Isothermal saturation boundary
IVAC	Infection-related ventilator-associated condition
j	Joule
LV	Left ventricle
LVSWI	Left ventricular stroke work index
MAP	Mean arterial pressure
MDI	Metered-dose inhaler
MIC	Maximum insufflation capacity
MIE	Mechanical insufflation–exsufflator
MMV	Mandatory minute ventilation
MODS	Multiple organ dysfunction syndrome
MPAP	Mean pulmonary artery pressure
NO	Nitric oxide
Na^+	Sodium
NAVA	Neurally adjusted ventilatory assist
NIV	Noninvasive ventilation
NPE	Neurogenic pulmonary edema
OI	Oxygenation index
ΔPaw	Change in airway pressure
ΔP_L	Transpulmonary pressure
ΔPOP	Plethysmographic waveform amplitude
ΔPpl	Change in pleural pressure
$P(a\text{-}et)CO_2$	Difference between arterial and end-tidal P_{CO_2}
$P(A\text{-}a)O_2$	Difference between alveolar Po and arterial P_{O_2}
$PaCO_2$	Partial pressure of carbon dioxide in arterial blood
$\bar{P}alv$	Mean alveolar pressure
Palv	Alveolar pressure
PaO_2	Partial pressure of oxygen in arterial blood
PAO_2	Alveolar P_{O_2}
PaO_2/PAO_2	Ratio of arterial PO_2 to alveolar P_{O_2}
PaO_2/FIO_2	Ratio of arterial P_{O_2} to F_{IO_2}
PAP	Pulmonary artery pressure
PAV	Proportional-assist ventilation
$\bar{P}aw$	Mean airway pressure
Pb	Barometric pressure
PbO_2	Brain P_{O_2}
PBW	Predicted body weight
PC-CMV	Continuous mandatory ventilation with pressure control
PC-IMV	Pressure-controlled intermittent mandatory ventilation
PCIRV	Pressure-controlled inverse ration ventilation
PCO_2	Partial pressure of carbon dioxide
PCV	Pressure-controlled ventilation
PCWP	Pulmonary capillary wedge pressure
Pdi	Transdiaphragmatic pressure
$P\bar{E}CO_2$	Mixed exhaled P_{CO_2}
PH_2O	Water vapor pressure
PEEP	Positive end-expiratory pressure

PEG	Percutaneous endoscopic gastrostomy
Peso	Esophageal pressure
P_{ETCO_2}	End-tidal P_{CO_2}
P_{EXHCO_2}	Measured mixed exhaled P_{CO_2} including gas compressed in the ventilator circuit
pH	Negative log of the hydrogen ion concentration
PI	Plethysmographic perfusion index
PI_{max}	Maximum inspiratory pressure
PI_{min}	Minimal value of the plethysmographic perfusion index
PIP	Peak inspiratory pressure
Pmus	Pressure generated by the respiratory muscles
PMV	Prolonged mechanical ventilation
P_{O_2}	Partial pressure of oxygen
Pplat	Plateau pressure
PPV	Pulse pressure variation
PRVC	Pressure-regulated volume control
PSV	Pressure support ventilation
P_{TCCO_2}	Transcutaneous P_{CO_2}
P_{TCO_2}	Transcutaneous P_{O_2}
$P_{\bar{v}O_2}$	Mixed venous P_{CO_2}
Pvent	Pressure-generated by the ventilator
PVI	Plethysmographic variability index
P_{v-O_2}	Mixed venous P_{O_2}
PVR	Pulmonary vascular resistance
$\dot{Q}_C$	Cardiac output
$\dot{Q}_S/\dot{Q}_T$	Pulmonary shunt
R	Respiratory quotient
R_E	Expiratory resistance
REE	Resting energy expenditure
REM	Rapid eye movement
R_I	Inspiratory resistance
RSBI	Rapid shallow breathing index
RVSWI	Right ventricular stroke work index
Sa_{O_2}	Hemoglobin oxygen saturation of arterial blood
SBT	Spontaneous breathing trial
Scv_{O_2}	Central venous oxygen saturation
SID	Strong ion difference
SIMV	Synchronized intermittent mandatory ventilation
Sjv_{O_2}	Jugular venous oxygen saturation
Sp_{CO}	Carbon monoxide measured by pulse oximetry
Sp_{Hb}	Hemoglobin measured by pulse oximetry
Sp_{Met}	Methemoglobin measured by pulse oximetry
Sp_{O_2}	Hemoglobin oxygen saturation measured by pulse oximetry
SVI	Stroke volume index
$S\bar{v}_{O_2}$	Mixed venous oxygen saturation
SVR	Systemic vascular resistance
SVRI	Systemic vascular resistance index
T_E	Expiratory time
T_I	Inspiratory time
T_T	Total cycle time
UUN	Urine urea nitrogen
$\dot{V}$	Flow
$\dot{V}_A$	Alveolar ventilation
$\dot{V}/\dot{Q}$	Ratio of ventilation to blood flow

VAC	Ventilator-associated condition
VAE	Ventilator-associated event
VAP	Ventilator-associated pneumonia
VC	Vital capacity
$\dot{V}CO_2$	Carbon dioxide production
$\dot{V}_D$	Dead space ventilation
$\dot{V}_E$	Minute ventilation
$\dot{V}_I$	Inspiratory flow
VCV	Volume-controlled ventilation
VC-CMV	Continuous mandatory ventilation with volume control
VC-IMV	Volume-controlled intermittent mandatory ventilation
V_D/V_T	Dead space-to-tidal volume ratio
VILI	Ventilator-induced lung injury
$\dot{V}O_2$	Oxygen consumption
VS	Volume support
V_T	Tidal volume
W	Work
τ	Time constant

Part 1
Principles of Mechanical Ventilation

Chapter 1
Physiologic Effects of Mechanical Ventilation

- **Introduction**
- **Intrathoracic Pressure Changes**
- **Pulmonary Effects**
  - Shunt
  - Ventilation
  - Atelectasis
  - Barotrauma
  - Ventilator-Induced Lung Injury
  - Pneumonia
  - Hyperventilation and Hypoventilation
  - Oxygen Toxicity
- **Cardiac Effects**
- **Renal Effects**
- **Gastrointestinal Effects**
- **Nutrition Effects**
- **Sedation and Delirium**
- **Neuromuscular Effects**
- **Airway Effects**
- **Sleep Effects**
- **Patient-Ventilator Asynchrony**
- **Mechanical Malfunctions**
- **Points to Remember**
- **Additional Reading**

Objectives

1. Compare intrathoracic pressure during spontaneous breathing and positive-pressure ventilation.
2. Describe the effects of positive-pressure ventilation on shunt and dead space.
3. Discuss the roles of alveolar overdistention and opening/closing on ventilator-induced lung injury.
4. Discuss the physiologic effects of positive-pressure ventilation on pulmonary, cardiac, renal, gastrointestinal, and neuromuscular function.
5. Describe the effects of sedation and delirium in the mechanically ventilated patient.
6. Discuss the effects of positive-pressure ventilation on nutrition, the upper airway, and sleep.
7. Describe methods that can be used to minimize the harmful effects of positive-pressure ventilation.

Introduction

Ventilators for adult acute care use positive pressure applied to the airway opening to inflate the lungs. Although positive pressure is responsible for the beneficial effects of mechanical ventilation, it is also responsible for many potentially deleterious side effects. Application of mechanical ventilation requires an understanding of both its beneficial and adverse effects. In the care of a patient, this demands application of strat egies that maximize the potential benefit of mechanical ventilation while minimizing the potential for harm. Because of the homeostatic interactions between the lungs and other body systems, mechanical ventilation can affect nearly every organ system of the body. This chapter provides an overview of the beneficial and adverse physiologic effects of mechanical ventilation.

Intrathoracic Pressure Changes

During normal spontaneous breathing, intrathoracic pressure is negative throughout the ventilatory cycle. Intrapleural pressure varies from about −5 cm H_2O during exha lation to −8 cm H_2O during inhalation. Alveolar pressure fluctuates from +1 cm H_2 during exhalation to −1 cm H_2O during inhalation. The decrease in intrapleural pres sure during inhalation facilitates lung inflation and venous return.

Transpulmonary pressure is the difference between proximal airway pressure and intrapleural pressure. The greatest transpulmonary pressure that can be generated nor mally during spontaneous inspiration is about 30 cm H_2O. Transalveolar pressure, also called alveolar stress, should be limited to 20 cm H_2O during positive-pressure ventilation.

Intrathoracic pressure fluctuations during positive-pressure ventilation are oppo site to those that occur during spontaneous breathing. During positive-pressure

ventilation, intrathoracic pressure is usually positive. Intrathoracic pressure increases during inhalation and decreases during exhalation.

Pulmonary Effects

Shunt

Shunt is perfusion (blood flow) without ventilation (Figure 1-1). Pulmonary shunt occurs when blood flows from the right heart to the left heart without participating in gas exchange. The result of shunt is hypoxemia. Shunt can be either capillary shunt or anatomic shunt. Capillary shunt results when blood flows past unventilated alveoli. Examples of capillary shunt are atelectasis, pneumonia, pulmonary edema, and acute respiratory distress syndrome (ARDS). Anatomic shunt occurs when blood flows from the right heart to the left heart and completely bypasses the lungs. Normal anatomical shunt occurs due to the Thebesian veins and the bronchial circulation. Abnormal anatomic shunt occurs with congenital cardiac defects and with a patent foramen ovale. Total shunt is the sum of the capillary and anatomic shunt.

Positive-pressure ventilation usually decreases shunt and improves arterial oxygenation. However, if positive-pressure ventilation produces overdistention of some lung units, this may result in redistribution of pulmonary blood flow to unventilated regions (Figure 1-2). In this case, positive-pressure ventilation paradoxically results in hypoxemia.

Although positive-pressure ventilation may improve capillary shunt, it may worsen anatomic shunt. An increase in alveolar pressure may increase pulmonary vascular resistance, which could result in increased flow through the anatomic shunt (eg, patent foramen ovale), decreased flow through the lungs, and worsening hypoxemia. Thus, alveolar pressure should be kept as low as possible if an anatomic right-to-left shunt is present.

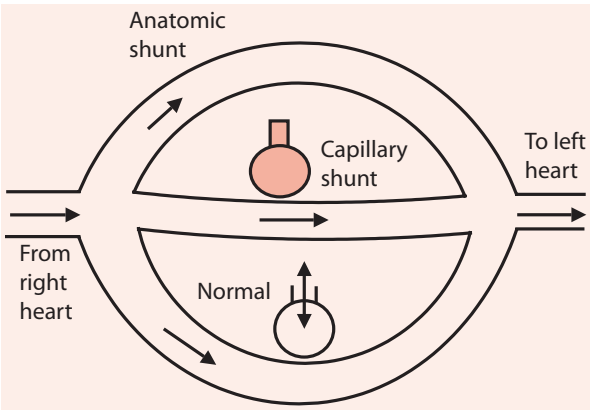

Figure 1-1 Schematic illustration of anatomic shunt and capillary shunt.

A relative shunt effect can occur with poor distribution of ventilation, such as might result from airway disease. With poor distribution of ventilation, some alveoli are underventilated relative to perfusion (shunt-like effect and low ventilation-perfusion ratio), whereas other alveoli are overventilated (dead space effect and high ventilation-perfusion ratio). Positive-pressure ventilation may improve the distribution of ventilation, particularly by improving the ventilation of previously underventilated areas of the lungs.

Ventilation

Ventilation is the movement of gas into and out of the lungs. Tidal volume (V_T) is the amount of gas inhaled or exhaled with a single breath,

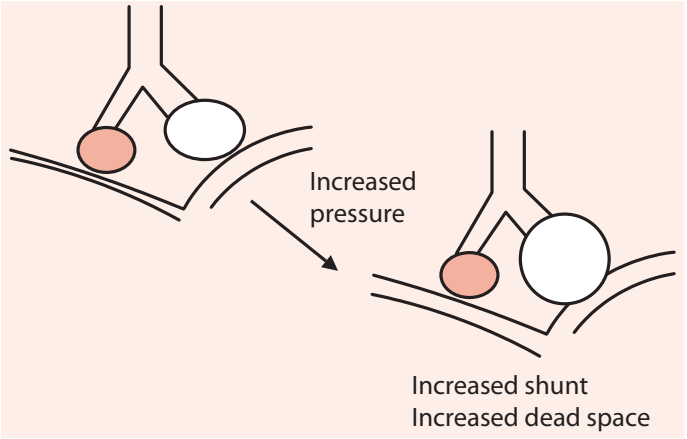

Figure 1-2 Alveolar overdistention, resulting in redistribution of pulmonary blood flow to unventilated units and an increased shunt.

and minute ventilation ($\dot{V}_E$) is the volume of gas breathed in 1 minute. Minute ventilation is the product of tidal volume (V_T) and breathing frequency (f_b):

$$\dot{V}_E = V_T \times f_b$$

Ventilation is either dead space ventilation ($\dot{V}_D$) or alveolar ventilation ($\dot{V}_A$). Minute ventilation is the sum of dead space and alveolar ventilation:

$$\dot{V}_E = \dot{V}_D + \dot{V}_A$$

Alveolar ventilation participates in gas exchange (Figure 1-3), whereas dead space ventilation does not. In other words, dead space is ventilation without perfusion. Anatomic dead space is the volume of the conducting airways of the lungs and is about 150 mL in normal adults. Alveolar dead space refers to alveoli that are ventilated but not perfused, and it is increased by any condition that decreases pulmonary blood flow. Total physiologic dead space fraction (V_D/V_T) is normally about one-third of $\dot{V}_E$. Mechanical dead space refers to the rebreathed volume of the ventilator circuit and acts as an extension of the anatomic dead space. Because of the fixed anatomic dead space, a low tidal volume increases the dead space fraction and decreases alveolar ventilation. An increased V_D/V requires a greater $\dot{V}_E$ to maintain $\dot{V}_A$ (and $Paco_2$).

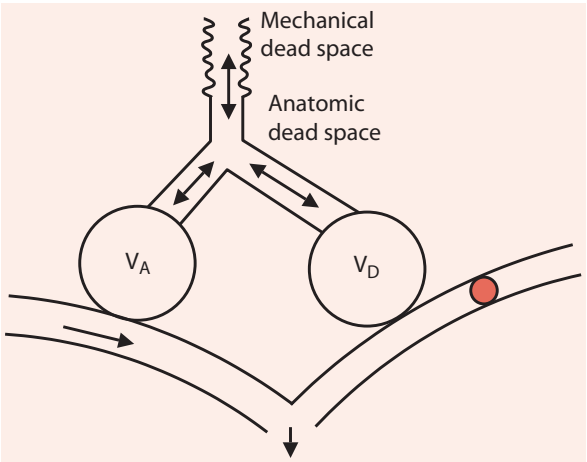

Figure 1-3 Schematic illustration of mechanical dead space, anatomic dead space, and alveolar dead space.

Because mechanical ventilators provide a tidal volume

and respiratory rate, any desired level of ventilation can be provided. The level of ventilation required depends on the desired $Paco_2$, $\dot{V}_A$, and tissue CO_2 production ($\dot{V}$). This is illustrated by the following relationships (note that the factor 0.863 is not used if the measurements are made at the same conditions and using the same units):

$$Paco_2 \propto \dot{V}co_2/\dot{V}_A$$

and

$$Paco_2 = (\dot{V}co_2 \times 0.863)/(\dot{V}_E \times [1 - V_D/V_T])$$

A higher $\dot{V}_E$ will be required to maintain $Paco_2$ if $\dot{V}co_2$ is increased, such as occurs with fever and sepsis. If dead space is increased, a higher $\dot{V}_E$ is required to maintain the same level of $\dot{V}_E$ and $Paco_2$. If this level of ventilation is undesirable due to its injurious effects on the lungs and hemodynamics, $Paco_2$ can be allowed to increase (permissive hypercapnia). Mechanical ventilation can produce overdistention of normal alveoli, resulting in alveolar dead space. Mechanical ventilation can also distend airways, increasing anatomic dead space.

Atelectasis

Atelectasis is a common complication of mechanical ventilation. This can be the result of preferential ventilation of nondependent lung zones with passive ventilation, the weight of the lungs causing compression of dependent regions, or airway obstruction. Breathing 100% oxygen may produce absorption atelectasis, and it should be avoided if possible. Use of positive end-expiratory pressure (PEEP) to maintain lung volume is effective in preventing atelectasis.

Barotrauma

Barotrauma is alveolar rupture due to overdistention. Barotrauma can lead to pulmonary interstitial emphysema, pneumomediastinum, pneumopericardium, subcutaneous emphysema, and pneumothorax. Pneumothorax is of greatest clinical concern, because it can progress rapidly to life-threatening tension pneumothorax. Pneumomediastinum and subcutaneous emphysema rarely have major clinical consequences.

Ventilator-Induced Lung Injury

Alveolar overdistention causes acute lung injury and is determined by the difference between intra-alveolar pressure and the intrapleural pressure. Peak alveolar pressure (end-inspiratory plateau pressure) should be as low as possible. Many authorities have suggested that plateau pressure (Pplat) should not exceed 30 cm H_2O. But in the presence of normal chest wall mechanics, Pplat should be kept less than 28 cm H O to avoid injurious stress and strain on the lungs. Alveolar distention is also affected by intrapleural pressure. Thus, a stiff chest wall may be protective against alveolar overdistention. Overdistention is minimized by limiting tidal volume to 4 to 8 mL/kg predicted body weight, driving pressure (Pplat – PEEP) to less than 15 cm H_2O, and alveolar distending pressure (stress) to less than 20 cm H_2O.

Ventilator-induced lung injury can also result from cyclical alveolar collapse during exhalation and reopening during subsequent inhalation. This injury is ameliorated by the application of PEEP to avoid alveolar derecruitment. Ventilating the lungs in a manner that promotes alveolar overdistention and derecruitment increases inflammation in the lungs (biotrauma). Inflammatory mediators may translocate into the pulmonary circulation, resulting in systemic inflammation.

Spontaneously breathing patients with acute respiratory failure may have a high respiratory drive and breathe with large tidal volumes. This has the potential to generate injurious transpulmonary pressure swings. This is of particular concern in patients with lung injury and is more likely with pressure-targeted modes of ventilation. Spontaneous breathing can also result in the movement of gas from one region of the lungs to another, without a significant change in overall tidal volume. This phenomenon, called pendelluft, can result in tidal recruitment and local overdistention of dependent lung regions, as well as deflation/reinflation of corresponding nondependent regions. Pendelluft can occur during spontaneous breathing with either volume-control or pressure-control ventilation. Sedation and, in some cases, paralysis might be necessary to prevent patient self-inflicted lung injury.

An important characteristic of the lungs of mechanically ventilated patients is heterogeneity. That is, some lung units are normal, some are prone to overdistention, some are prone to collapse, some are consolidated, and some are fluid filled. Alveolar wall stress is magnified when a collapsed alveolus is adjacent to one that is open (stress raiser). Recruitment of collapsed alveoli thus improves homogeneity within the lungs and decreases the potential of injury because it reduces opening/closing injury and the effects of stress raisers. If the collapsed alveolus cannot be recruited, however, a high recruiting pressure in the open alveolus will increase the potential for injury due to stress raisers. Thus, setting the ventilator is often a compromise between maximum recruitment and overdistention.

Pneumonia

Ventilator-associated pneumonia (VAP) can occur during mechanical ventilation. This is more common during invasive ventilation than with noninvasive ventilation. VAP most often results from aspiration of oropharyngeal secretions around the cuff of the endotracheal tube. A number of prevention strategies can be bundled to reduce the risk of VAP.

Hyperventilation and Hypoventilation

Hyperventilation lowers $Paco_2$ and increases arterial pH. This should be avoided because of the injurious effects of alveolar overdistention and an alkalotic pH. Respiratory alkalosis causes hypokalemia, decreased ionized calcium, decreased cerebral blood flow, and increased affinity of hemoglobin for oxygen (left shift of the oxyhemoglobin dissociation curve). Relative hyperventilation can occur when mechanical ventilation is provided for patients with chronic compensated respiratory acidosis. If a normal Paco is established in such patients, the result is an elevated pH. Because severe hypercapnia

appears to be independently associated with higher mortality in patients with ARDS, it should be avoided unless the alternative is an injurious ventilatory pattern.

Oxygen Toxicity

A high inspired oxygen concentration is considered toxic. What is less clear is the level of oxygen that is toxic. Oxygen toxicity is probably related to F_{IO_2} as well as the amount of time that the elevated F_{IO_2} is breathed. Although the clinical evidence is weak, it is commonly recommended that an F_{IO_2} greater than 0.6 be avoided, particularly if breathed for a period more than 48 hours. Whether permissive hypoxemia should be tolerated to avoid oxygen toxicity is an area of controversy.

High F_{IO_2} can result in a higher than normal P_{aO_2}. This may produce an elevation in P_{aCO_2} due to the Haldane effect (ie, unloading CO_2 from hemoglobin), due to improving blood flow to low-ventilation lung units (ie, relaxing hypoxic pulmonary vasoconstriction), and due to suppression of ventilation (less likely). However, this is usually not an issue during mechanical ventilation because ventilation can be controlled. A high P_{aO_2} can produce retinopathy of prematurity in neonates, but this is not known to occur in adults.

Poorer outcomes have been reported with excessive oxygen administration for critically ill patients. A reasonable target S_{pO_2} during mechanical ventilation is 88% to 95%, which corresponds to a P_{aO_2} of 55 to 80 mm Hg. There are, however, several exceptions such as carbon monoxide poisoning and absorption of free air such as pneumocephalus.

Cardiac Effects

With spontaneous breathing, venous return to the right atrium is greatest during inhalation, when the intrathoracic pressure is lowest. During positive-pressure ventilation, venous return is greatest during exhalation and it may be decreased if expiratory time is too short or mean alveolar pressure is too high. Increased intrathoracic pressure decreases venous return and right heart filling. This effect is greatest with high alveolar pressure, high lung compliance, low chest wall compliance, and low circulating blood volume. Hypotension results when left heart filling and cardiac output are reduced. In the presence of left heart failure, the increase in right atrial pressure and subsequent reduction in venous return might assist the failing heart. Positive-pressure ventilation decreases left ventricular afterload as well as preload, both of which might be beneficial in the presence of left heart failure.

Positive-pressure ventilation may increase pulmonary vascular resistance. The increase in alveolar pressure, particularly with PEEP, has a constricting effect on the pulmonary vasculature. The increase in pulmonary vascular resistance decreases left ventricular filling and cardiac output. Increased right ventricular afterload can result in right ventricular hypertrophy, with ventricular septal shift and compromise of left ventricular function. Increased pulmonary vascular resistance with PEEP produces a West Zone 1 effect, which increases dead space, and thus results in less alveolar ventilation and a higher P_{aCO_2}.

Renal Effects

Urine output can decrease secondary to mechanical ventilation. This is partially related to decreased renal perfusion due to decreased cardiac output and may also be related to elevations in plasma antidiuretic hormone and reductions in atrial natriuretic pep tide that occur with mechanical ventilation. Fluid overload frequently occurs during mechanical ventilation, due to decreased urine output, excessive intravenous fluid administration, and elimination of insensible water loss from the respiratory tract due to humidification of the inspired gas.

Gastrointestinal Effects

Invasively or noninvasively ventilated patients may develop gastric distention (meteorism). Stress ulcers and gastrointestinal bleeding can occur in mechanically ventilated patients, and stress ulcer prophylaxis should be provided. Gastric and splanchnic perfusion is usually maintained provided that cardiac output is not impaired.

Nutrition Effects

Appropriate nutritional support is problematic in mechanically ventilated patients. Underfeeding can result in respiratory muscle catabolism and increases the risk of pneumonia and pulmonary edema. Overfeeding increases metabolic rate and thus increases the required minute ventilation. Overfeeding with carbohydrates increases $\dot{V}CO_2$, further increasing the ventilation requirement.

Sedation and Delirium

Most critically ill mechanically ventilated patients have pain. Assessment of pain and provision of adequate analgesia is essential, but continuous deep sedation should be avoided to the extent possible. Intravenous opioids are recommended for pain control. Minimizing the depth and duration of sedation is an important practice in the care of mechanically ventilated patients. This can be achieved by practices such as proto cols to minimize sedation or daily spontaneous awakening trials. Non-benzodiazepine sedatives such as propofol or dexmedetomidine are preferred. An appropriate seda tion target is a Riker Sedation-Agitation Scale (SAS) score of 3 to 4, or a Richmond Agitation-Sedation Scale (RASS) score of −2 to 0.

Delirium may affect as many as 80% of mechanically ventilated critically ill patients, resulting in increased mortality and hospital length of stay. The Confusion Assessment Method for the ICU (CAM-ICU) and the Intensive Care Delirium Screening Checklist (ICDSC) are the most valid and reliable delirium monitoring tools for adult critically ill patients. Early mobilization of adult ICU patients is recommended to reduce the incidence and duration of delirium.

The mnemonic ABCDEF has been proposed to remind clinicians of important steps of care in mechanically ventilated patients:

A: Assess, prevent, and manage pain
B: Both spontaneous awakening trials and spontaneous breathing trials
C: Choice of analgesia and sedation
D: Delirium; assess, prevent, manage
E: Early mobility and exercise
F: Family engagement and empowerment

Neuromuscular Effects

Mechanically ventilated patients are at increased risk of critical illness weakness (polyneuropathy and polymyopathy). Survivors of ARDS have a reduced 6-minute walk distance 1 year after discharge. Controlled mechanical ventilation is associated with adverse effects on diaphragm (and other respiratory muscles) structure and function, known as ventilator-induced diaphragmatic dysfunction. On the other extreme, excessive respiratory muscle activity can result in muscle fatigue. Thus, an appropriate balance between respiratory muscle activity and support from the ventilator is important. Mobilization of mechanically ventilated patients is used increasingly to address generalized weakness in this patient population.

Airway Effects

Critically ill patients are usually mechanically ventilated through an endotracheal or tracheostomy tube. This puts these patients at risk for complications of artificial airways such as laryngeal edema, tracheal mucosal trauma, contamination of the lower respiratory tract, sinusitis, loss of the humidifying function of the upper airway, and communication problems. These complications can be avoided through appropriate use of noninvasive ventilation.

Sleep Effects

Mechanical ventilation can have positive and negative effects on sleep. For patients receiving prolonged mechanical ventilation, ventilation at night may improve sleep quality. However, during sleep, the apneic threshold for $Paco_2$ increases, and lowering $Paco_2$ level below this threshold due to excessive ventilation may rapidly lead to central apneas and periodic breathing. Thus, use of modes with backup ventilation (eg, continuous mandatory ventilation rather than pressure support ventilation) is preferable. Proportional modes (eg, neurally adjusted ventilatory assist and proportional assist ventilation) may improve asynchrony during sleep, but their effect on sleep quality has not been conclusively demonstrated. Noninvasive ventilation improves sleep in