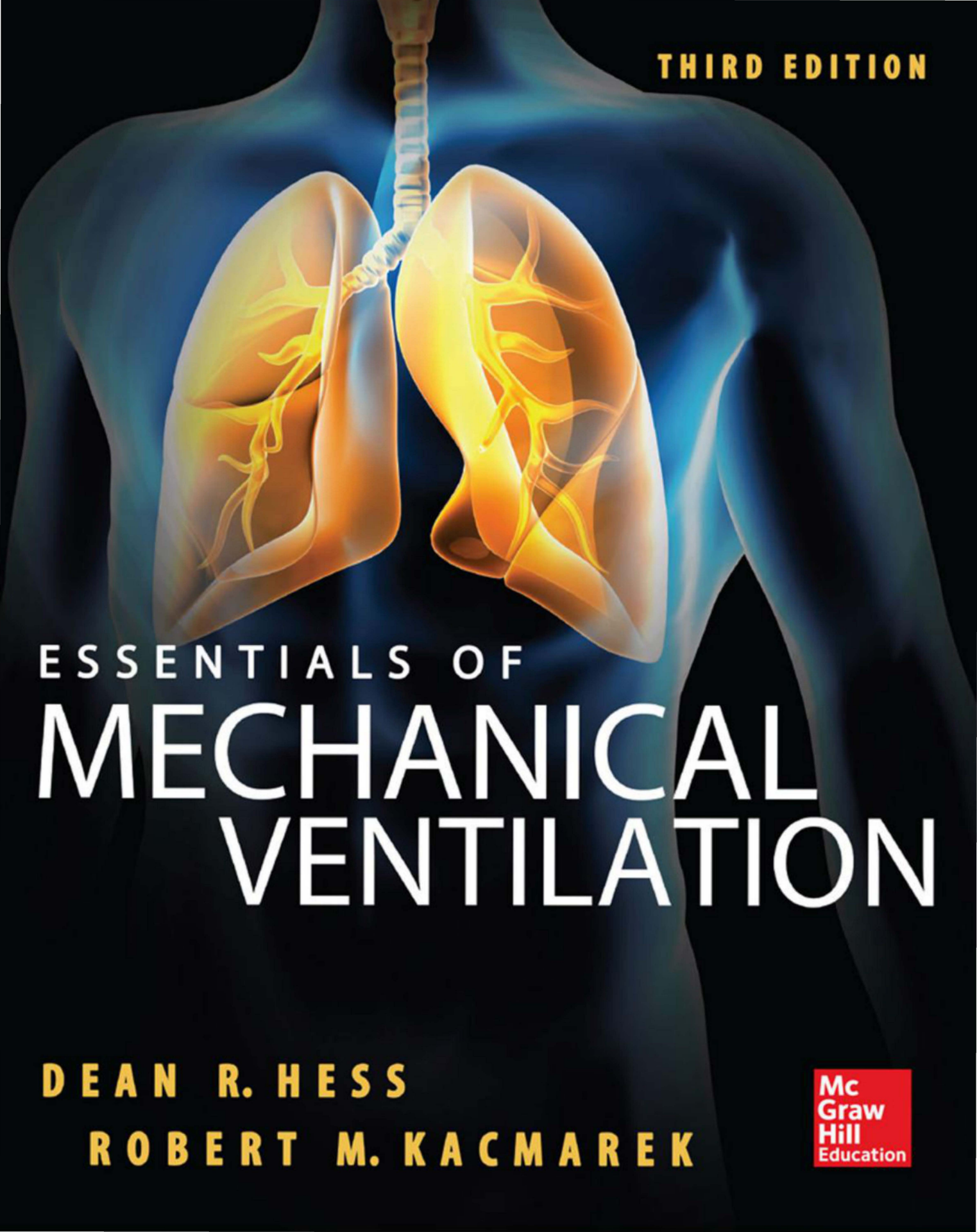
THIRD EDITION
ESSENTIALS OF
MECHANICAL
VENTILATION
DEAN R. HESS
ROBERT M. KACMAREK
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Essentials of Mechanical Ventilation

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Essentials of Mechanical Ventilation

Third Edition

DEAN R. HESS, PhD, RRT
Associate Professor of Anaesthesia
Harvard Medical School
Assistant Director of Respiratory Care Services
Massachusetts General Hospital
Boston, Massachusetts

ROBERT M. KACMAREK, PhD, RRT
Professor of Anaesthesia
Harvard Medical School
Director of Respiratory Care Services
Massachusetts General Hospital
Boston, Massachusetts

New York Chicago San Francisco Athens London Madrid
Mexico City Milan New Delhi Singapore Sydney Toronto

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Dedication

For Susan, Terri, Rob, Max, Abby, Lauren, and Matt—who make every day enjoyable.
D.R.H.

For my children Robert, Julia, Katie, and Callie, who make it all worthwhile.
R.M.K.

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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 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 over 75 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. As in the previous editions, we have kept the chapters short, focused, and practical.

There have been many advances in the practice of mechanical ventilation over the past 10 years. Hence, much of the book is rewritten. 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 weaning 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, extracorporeal life support, and miscellaneous ventilatory techniques.

This is a book about mechanical ventilation and not mechanical ventilators. 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 covered 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 decided to limit the focus of this book to adult mechanical ventilation. Although we provide a short bibliography 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

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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
IBW	Ideal body weight (sometimes called predicted body weight)
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	Joules
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}
Pa_{O_2}/P_{AO_2}	Ratio of arterial PO_2 to alveolar P_{O_2}
Pa_{O_2}/F_{IO_2}	Ratio of arterial P_{O_2} to F_{IO_2}
$P(A\text{-}a)O_2$	Difference between alveolar P_{O_2} and arterial P_{O_2}
Pa_{CO_2}	Partial pressure of carbon dioxide in arterial blood
$\bar{P}alv$	Mean alveolar pressure
Palv	Alveolar pressure
Pa_{O_2}	Partial pressure of oxygen in arterial blood
P_{AO_2}	Alveolar P_{O_2}
PAP	Pulmonary artery pressure
PAV	Proportional-assist ventilation
$\bar{P}aw$	Mean airway pressure
Pb	Barometric pressure
Pb_{O_2}	Brain P_{O_2}
PC-CMV	Continuous mandatory ventilation with pressure control
PC-IMV	Pressure-controlled intermittent mandatory ventilation
PCIRV	Pressure-controlled inverse ration ventilation
P_{CO_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}
P_{H_2O}	Water vapor pressure
PEEP	Positive end-expiratory pressure

PEG	Percutaneous endoscopic gastrostomy
Peso	Esophageal pressure
$P_{ET}CO_2$	End-tidal P_{CO_2}
$P_{exh}CO_2$	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	Arterial pulse pressure variation
PRVC	Pressure-regulated volume control
PSV	Pressure support ventilation
$P_{tc}CO_2$	Transcutaneous P_{CO_2}
$P_{tc}O_2$	Transcutaneous P_{O_2}
$P\bar{v}CO_2$	Mixed venous P_{CO_2}
Pvent	Pressure-generated by the ventilator
PVI	Plethysmographic variability index
$P\bar{v}O_2$	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
Spco	Carbon monoxide measured by pulse oximetry
SpHb	Hemoglobin measured by pulse oximetry
SpMet	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

Objectives

1. List the factors affecting mean airway pressure during 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 the pulmonary, cardiac, renal, hepatic, gastric, and neuromuscular function.
5. Discuss the effects of positive pressure ventilation on nutrition, the airway, and sleep.
6. Describe methods that can be used to minimize the harmful effects of positive pressure ventilation.

Introduction

Ventilators used in 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 an individual patient, this demands application of strategies that maximize the potential benefit of mechanical ventilation while minimizing the potential for harm. Due to 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.

Mean Airway Pressure

During normal spontaneous breathing, intrathoracic pressure is negative throughout the ventilatory cycle. Intrapleural pressure varies from about –5 cm H_2O during exhalation to –8 cm H_2O during inhalation. Alveolar pressure fluctuates from +1 cm H_2O during exhalation to –1 cm H_2O during inhalation. The decrease in intrapleural pressure during inhalation facilitates lung inflation and venous return. Transpulmonary pressure is the difference between proximal airway pressure and intrapleural pressure. The greatest static transpulmonary pressure that can be generated normally during spontaneous inspiration is less than 35 cm H_2O.

Intrathoracic pressure fluctuations during positive pressure ventilation are opposite to those that occur during spontaneous breathing. During positive pressure ventilation, the mean intrathoracic pressure is usually positive. Intrathoracic pressure increases during inhalation and decreases during exhalation. Thus, venous return is greatest during exhalation and it may be decreased if expiratory time is too short or mean alveolar pressure is too high.

Many of the beneficial and adverse effects associated with mechanical ventilation are related to mean airway pressure. Mean airway pressure is the average pressure applied to the airway during the ventilatory cycle. It is related to both the amount and duration of pressure applied during the inspiratory phase (peak inspiratory pressure, inspiratory pressure waveform, and inspiratory time) and the expiratory phase (positive end-expiratory pressure [PEEP] and respiratory rate).

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. Total shunt is the sum of the capillary and anatomic shunt.

Positive pressure ventilation usually decreases shunt and improves arterial oxygenation. An inspiratory pressure that exceeds the alveolar opening pressure expands a collapsed alveolus, and an expiratory pressure greater than alveolar closing pressure prevents its collapse. By maintaining alveolar recruitment with an adequate expiratory pressure setting, arterial oxygenation is improved. 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, decreased flow through the lungs, and worsening hypoxemia. Thus, mean airway pressure should be kept as low as possible if an anatomic right-to-left shunt is present.

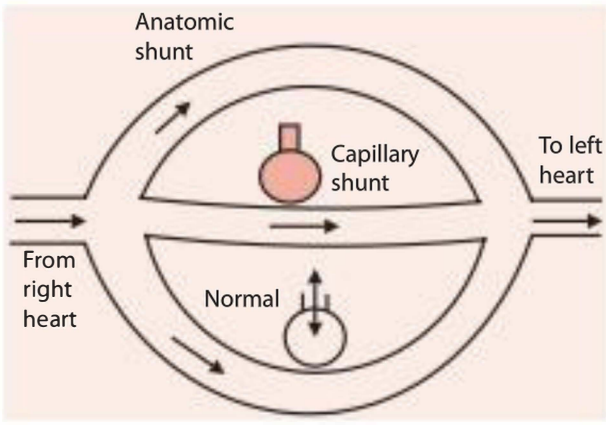

Figure 1-1 Schematic illustration of anatomic shunt and capillary shunt.

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

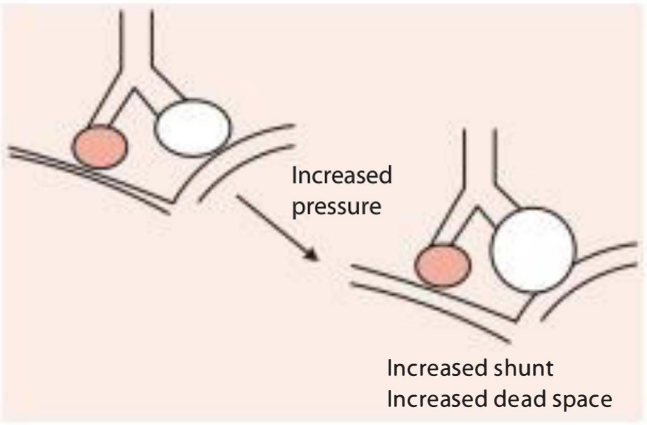

Figure 1-2 Alveolar overdistention, resulting in redistribution of pulmonary blood flow to unventilated units and an increased shunt.

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 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 respiratory frequency (f_b):

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

Ventilation can be either dead space ventilation ($\dot{V}_D$) or alveolar ventilation ($\dot{V}_A$). Minute ventilation is the sum of dead space ventilation 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 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 the $\dot{V}_E$. Mechanical dead space refers to the rebreathed volume of the ventilator circuit and acts as an extension of the anatomic dead space. Due to the

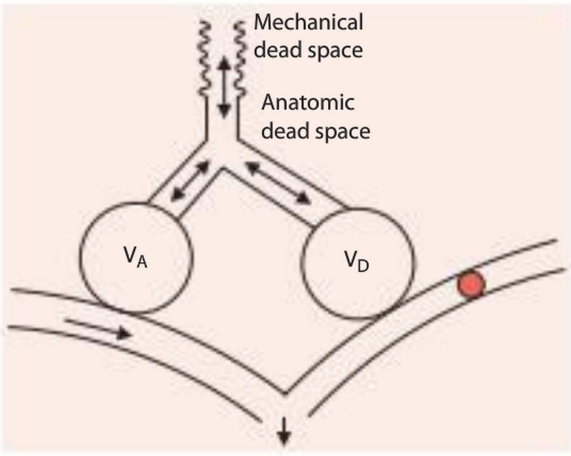

Figure 1-3 Schematic illustration of mechanical dead space, anatomic dead space, and alveolar dead space.

fixed anatomic dead space, a low tidal volume increases the dead space fraction and decreases alveolar ventilation. An increased dead space fraction will require a greater minute ventilation to maintain alveolar ventilation (and $PaCO_2$).

Because mechanical ventilators provide a tidal volume and respiratory rate, any desired level of ventilation can be provided. The level of ventilation required depends upon the desired $PaCO_2$, alveolar ventilation, and tissue CO_2 production ($\dot{V}CO_2$). 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 $\dot{V}_A$ 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 should be avoided if possible. Use of 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 (Figure 1-4). 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. Alveolar distention is determined by the difference between intra-alveolar pressure and the intrapleural pressure. The peak alveolar pressure (end-inspiratory plateau pressure) should ideally be as low as possible and less than 30 cm H_2O. 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 (eg, 4-8 mL/kg ideal body weight) and alveolar distending pressure (< 25 cm H_2O). Ventilator-induced lung injury can also

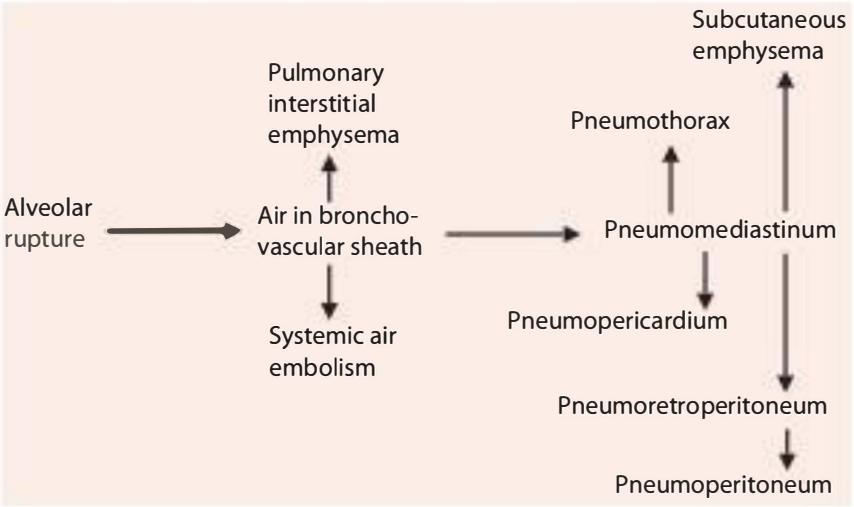

Figure 1-4 Barotrauma-related injuries that can occur as the result of alveolar rupture.

result from cyclical alveolar collapse during exhalation and re-opening 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. An important characteristic of the lungs of mechanically ventilated patients is heterogeneity; that is, some lung units are prone to overdistention and others are prone to collapse.

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, 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 $Paco_2$ is established in such patients, the result is an elevated pH. Hypercapnia during mechanical ventilation may be less injurious than the traumatic effects of high levels of ventilation to normalize the $Paco_2$. A modest elevation of $Paco_2$ (50-70 mm Hg) may not be injurious and a pH as low as 7.20 is well tolerated by most patients.

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 FIO_2 as well as the amount of time that the elevated FIO_2 is breathed. Although the clinical evidence is weak, it is commonly recommended that an FIO_2 greater than 0.6 be avoided, particularly if breathed for a period more than 48 hours. High FIO_2 levels can result in a higher than normal PaO_2. A high PaO_2 may produce an elevation in $PaCO_2$ 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 PaO_2 can produce retinopathy of prematurity in neonates, but this is not known to occur in adults.

Cardiac Effects

Positive pressure ventilation can decrease cardiac output, resulting in hypotension and potential tissue hypoxia. This effect is greatest with high mean airway pressure, high lung compliance, and low circulating blood volume. Increased intrathoracic pressure decreases venous return and right heart filling, which may reduce cardiac output. 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.

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 $PaCO_2$.

The adverse cardiac effects of positive pressure ventilation are ameliorated by lower mean airway pressure. When high mean airway pressure is necessary, circulatory volume loading and administration of vasopressors may be necessary to maintain cardiac output and arterial blood pressure.

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 peptide 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.

Gastric Effects

Patients being mechanically ventilated may develop gastric distention (meteorism). Stress ulcer formation and gastrointestinal bleeding can also occur in mechanically ventilated patients, and stress ulcer prophylaxis should be provided.

Nutritional 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.

Neurologic Effects

In patients with head injury, positive pressure ventilation might increase intracranial pressure. This is related to a decrease in venous return, which increases intracranial blood volume and pressure. If high mean airway pressure is used, cerebral perfusion can also be compromised due to arterial hypotension.

Delirium is common in mechanically ventilated patients. The mnemonic ABCDE has been proposed to remind clinicians of important steps of care in mechanically ventilated patients (Awakening and Breathing, Choice of sedative and analgesic, Delirium monitoring, and Early mobilization). Such an evidence-based protocol may improve patient outcome, including mortality. Benzodiazepine-based sedation may increase the risk of delirium, when compared with agents such as dexmedetomidine.

Neuromuscular Effects

Mechanically ventilated patients are at increased risk of critical illness and weakness (polyneuropathy and polymyopathy). If the respiratory muscles are not used during mechanical ventilation (ie, paralysis), ventilator-induced diaphragm dysfunction can occur. 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.

Hepatosplanchnic Effects

PEEP can reduce portal blood flow. However, the clinical importance of the effects of positive pressure ventilation on hepatosplanchnic perfusion is unclear.

Airway Effects

Critically ill patients are usually mechanically ventilated through an endotracheal or tracheostomy tube. This puts these patients at risk for all of the 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.

Sleep Effects

Mechanically ventilated patients may not have normal sleep patterns. Sleep deprivation can produce delirium, patient-ventilator asynchrony, and sedation-induced ventilator dependency.

Patient-Ventilator Asynchrony

Lack of synchrony between the breathing efforts of the patient and the ventilator may be due to poor trigger sensitivity, auto-PEEP, incorrect inspiratory flow or time setting, inappropriate tidal volume, or inappropriate mode. Asynchrony can also be caused by nonventilator issues such as pain, anxiety, and acidosis.

Mechanical Malfunctions

A variety of mechanical complications can occur during mechanical ventilation. These include accidental disconnection, leaks in the ventilator circuit, loss of electrical power, and loss of gas pressure. The mechanical ventilator system should be monitored frequently to prevent mechanical malfunctions.

Points to Remember

- Many of the beneficial and adverse effects of mechanical ventilation are related to mean airway pressure.
- Positive pressure ventilation usually improves arterial P_{O_2} and P_{CO_2}, but may increase shunt and dead space under some conditions.

- Atelectasis, barotrauma, acute lung injury, pneumonia, hypoventilation or hyperventilation, and oxygen toxicity are pulmonary complications of positive pressure ventilation.
- Positive pressure ventilation can produce adverse cardiac, renal, nutritional, neurologic, hepatic, and airway effects.
- An ABCDE approach (Awakening and Breathing, Choice of sedative and analgesic, Delirium monitoring, and Early mobilization) may improve outcomes of mechanically ventilated patients.
- Asynchrony commonly occurs and should be corrected by use of appropriate ventilator settings and by addressing nonventilator issues leading to asynchrony.

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Chapter 2
Physiologic Goals of Mechanical Ventilation

- **Introduction**
- **Tidal Volume and Alveolar Distending Pressure**
  Tidal Volume
  Alveolar Distending Pressure
  Positive End-Expiratory Pressure
- **Permissive Hypercapnia**
- **Oxygen Toxicity**
- **Gas Exchange Targets**
  Oxygenation
  Ventilation
  Acid-Base Balance
- **Patient-Ventilator Synchrony**
- **Points to Remember**
- **Additional Reading**

Objectives

1. Discuss the pressure and volume targets to be used when ventilating patients.
2. Define permissive hypercapnia, discuss when it should be employed, and discuss problems with its use.
3. Discuss concerns regarding the use of high oxygen concentrations in critically ill patients.
4. List the gas exchange and acid-base targets for critically ill patients.
5. Discuss concerns regarding patient-ventilator synchrony.

Introduction

Many clinical management decisions are designed to return abnormal physiologic function to normal or to return abnormal laboratory data to normal. However, during mechanical ventilation, it may not be prudent to target normal blood gas values irrespective of the tidal volume (V_T) delivered, pressure applied, or FIO_2 used. The inappropriate use of the ventilator may cause lung injury, activate inflammatory mediators, and potentially cause or extend multisystem organ failure. Of particular concern are patients with acute respiratory distress syndrome (ARDS), asthma, or chronic obstructive pulmonary disease (COPD), whose lungs have abnormal mechanics. Regardless of the pathophysiology requiring ventilatory support, the primary goals of mechanical ventilation should be to (1) cause no additional injury, avoiding ventilator-induced lung injury by minimizing lung stress and strain, (2) maintain gas exchange and acid-base balance at a level appropriate for the specific patient, accepting hypercapnia and hypoxemia where indicated, and (3) ensure patient-ventilator synchrony, selecting the mode and ventilator settings that best match the patient's respiratory drive while ensuring lung protection.

Tidal Volume and Alveolar Distending Pressure

Tidal Volume

In the past, approaches to mechanical ventilation suggested V_T of 10 to 15 mL/kg of ideal body weight (IBW). We now know that these V_T are excessive for any patient who requires mechanical ventilation. A V_T of greater than 10 mL/kg IBW should be avoided in all acutely ill patients regardless of their lung mechanics. Since it is impossible to clinically detect localized overdistention, an acceptable V_T in a given patient must be judged relative to alveolar distending pressure.

Alveolar Distending Pressure

Alveolar distending pressure is assessed by measuring end-inspiratory plateau pressure (Pplat), which reflects mean peak alveolar pressure. To measure Pplat, a 0.5- to 2-second end-inspiratory breath-hold is applied. Pplat should be limited to 30 cm H_2O if chest

wall compliance is normal. This is generally achieved by using a V_T of 4 to 8 mL/kg IBW for patients with ARDS and a V_T no greater than 10 mL/kg IBW for any patient requiring acute mechanical ventilation. Exceeding this Pplat target should be avoided in the absence of a stiff chest wall.

Positive End-Expiratory Pressure

The recommended level of positive end-expiratory pressure (PEEP) is 8 to 15 cm H_2O for mild ARDS and 10 to 20 cm H_2O for moderate to severe ARDS, which is needed to maintain lung recruitment. If PEEP is set at 10 to 20 cm H_2O and Pplat is limited to 30 cm H_2O, then only 10 to 20 cm H_2O ventilating pressure (driving pressure) is available. This may result in a V_T of only 4 to 6 mL/kg IBW. In this case, minute ventilation is adjusted by increasing the respiratory rate, provided that air trapping does not occur.

For patients with flow limitation (eg, COPD) and auto-PEEP, applied PEEP may be useful to improve the ability of the patient to trigger the ventilator. For most other patients, a PEEP of 5 cm H_2O is reasonable to maintain functional residual capacity and prevent atelectasis. This level of PEEP will usually have no adverse affects. However, PEEP levels as low as 0 may be necessary for patients who are hemodynamically unstable, or who have a large bronchopleural fistula.

Permissive Hypercapnia

Permissive hypercapnia is the deliberate limitation of ventilator support to avoid alveolar overdistension, allowing $Paco_2$ levels greater than normal (50-100 mm Hg). Allowing the $Paco_2$ to rise to these levels should be considered when the only alternative is a potentially dangerous increase in alveolar distending pressure or significant levels of auto-PEEP. The potential adverse effects of an elevated $Paco_2$ are listed in Table 2-1. Most of the more important clinical problems occur at $Paco_2$ levels above 150 mm Hg. However, even small increases in $Paco_2$ increase cerebral blood flow and permissive hypercapnia is generally contraindicated when intracranial pressure is increased (eg, acute head injury). Elevated $Paco_2$ also stimulates ventilation and may contribute to asynchrony, but patients are usually sedated when permissive hypercapnia is used.

Table 2-1 Physiologic Effects of Permissive Hypercapnia

- Shift of the oxyhemoglobin dissociation curve to the right
- Decreased alveolar Po_2
- Stimulation and depression of the cardiovascular system
- Central nervous system depression
- Increased ventilatory drive
- Pulmonary vasoconstriction (pulmonary hypertension)
- Systemic vasodilatation (systemic hypotension)
- Increased intracranial pressure
- Anesthesia ($Paco_2$ > 200 mm Hg)
- Decreased renal blood flow ($Paco_2$ > 150 mm Hg)
- Leakage of intracellular potassium ($Paco_2$ > 150 mm Hg)

Permissive hypercapnia may adversely affect oxygenation in some patients. Elevated $Paco_2$ and acidosis shift the oxyhemoglobin dissociation curve to right. This decreases the affinity of hemoglobin for oxygen, decreasing oxygen loading in the lungs but facilitating unloading of oxygen at the tissues. As illustrated by the alveolar gas equation, an increase in alveolar Pco_2 results in a decrease in alveolar Po_2. For each $Paco_2$ rise of 1 mm Hg, the Pao_2 decreases by about 1 mm Hg. When permissive hypercapnia is allowed, optimal efforts to maximize oxygenation should be used.

As illustrated in Figure 2-1, carbon dioxide stimulates or depresses some parts of the cardiovascular system, but opposite effects can occur via stimulation of the autonomic nervous system. It is thus difficult to predict the precise response of the cardiovascular system to permissive hypercapnia. An increase in Pco_2 might cause pulmonary hypertension and it might affect cardiac output. Rarely, pharmaceutical agents might need to be adjusted in the presence of permissive hypercapnia, but this is usually the result of acidosis and not the elevated Pco_2 per se.

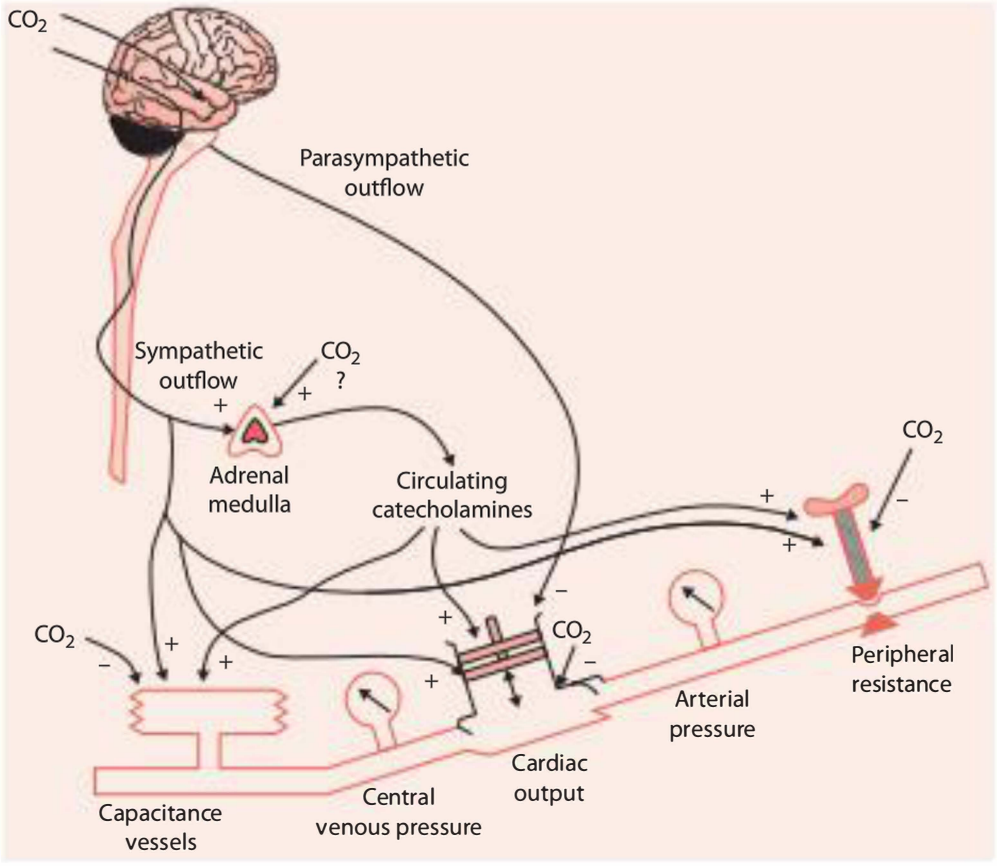

Figure 2-1 This diagram illustrates the complexity of the mechanisms by which carbon dioxide influences the circulatory system. See text for details. (Reproduced with permission from Nunn JF. Carbon dioxide. In: Nunn JF, ed. *Applied Respiratory Physiology*. 2nd ed. London, UK: Butterworth and Co.; 1977:334-374.)