EIGHTH EDITION

# PILBEAM'S MECHANICAL VENTILATION

PHYSIOLOGICAL AND CLINICAL APPLICATIONS

J.M. CAIRO



Evolve\*

Student Resources on Evolve
Access Code Inside

# **Evolve®**

YOU'VE JUST PURCHASED

MORE THAN A TEXTBOOK!

Enhance your learning with Evolve Student Resources.

These online study tools and exercises can help deepen your understanding of textbook content so you can be more prepared for class, perform better on exams, and succeed in your course.



Activate the complete learning experience that comes with each NEW textbook purchase by registering with your scratch-off access code at

# http://evolve.elsevier.com/Cairo/Pilbeams/ventilation/

If your school uses its own Learning Management System, your resources may be delivered on that platform. Consult with your instructor.

If you rented or purchased a used book and the scratch-off code at right has already been revealed, the code may have been used and cannot be re-used for registration. To purchase a new code to access these valuable study resources, simply follow the link above.

Place Sticker Here

## **REGISTER TODAY!**



You can now purchase Elsevier products on Evolve!

Go to evolve.elsevier.com/shop to search and browse for products.



This page intentionally left blank

# **EIGHTH EDITION**

# PILBEAM'S MECHANICAL VENTILATION

PHYSIOLOGICAL AND CLINICAL APPLICATIONS

### J.M. Cairo, PhD, RRT, FAARC

Dean Emeritus and Professor Louisiana State University Health Sciences Center New Orleans, Louisiana



Elsevier 3251 Riverport Lane St. Louis, Missouri 63043

PILBEAM'S MECHANICAL VENTILATION: PHYSIOLOGICAL AND CLINICAL APPLICATIONS, EIGHTH EDITION Copyright © 2024 by Elsevier, Inc. All rights reserved.

No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system, without permission in writing from the publisher. Details on how to seek permission, further information about the Publisher's permissions policies and our arrangements with organizations such as the Copyright Clearance Center and the Copyright Licensing Agency, can be found at our website: www.elsevier.com/permissions.

This book and the individual contributions contained in it are protected under copyright by the Publisher (other than as may be noted herein).

### Notices

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds or experiments described herein. Because of rapid advances in the medical sciences, in particular, independent verification of diagnoses and drug dosages should be made. To the fullest extent of the law, no responsibility is assumed by Elsevier, authors, editors or contributors for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions, or ideas contained in the material herein.

Previous editions copyrighted © 2020, 2016, 2012, 2006, 1998, 1992 and 1986.

Content Strategist: Melissa Rawe

Content Development Specialist: Kristen Helm Publishing Services Manager: Deepthi Unni Project Manager: Sindhuraj Thulasingam

Design Direction: Ryan Cook

Printed in India

Last digit is the print number: 9 8 7 6 5 4 3 2 1



ISBN: 978-0-323-87164-8



# **Contributors**

**Robert M. DiBlasi, RRT-NPS, FAARC** Seattle Children's Hospital Seattle, Washington

**Terry L. Forrette, MHS, RRT, FAARC**Adjunct Associate Professor of Cardiopulmonary Science, LSU Health Sciences Center,
New Orleans, Louisiana

### **Ancillary Contributor**

**Sandra T. Hinski, MS, RRT-NPS**Faculty, Respiratory Care Division,
Gateway Community College,
Phoenix, Arizona

# **Preface**

he goal of *Pilbeam's Mechanical Ventilation* text has always been to provide clinicians with a strong physiological foundation for making informed decisions when managing patients receiving mechanical ventilation. The subject matter covered is derived from current evidence-based practices and is written in a manner that allows this text to serve as a resource for both students and practicing clinicians. As with previous editions, the eighth edition of *Pilbeam's Mechanical Ventilation* is presented in a concise manner that explains patient-ventilator interactions using a stepwise approach. Beginning with fundamental concepts and expanding to the more advanced topics, the text guides readers through a series of essential concepts and ideas, building on the information as the reader progresses through the text.

It is apparent to critical care clinicians that implementing effective interprofessional care plans is required to achieve successful outcomes. Respiratory therapists are recognized as an integral part of effective interprofessional critical care teams. Their expertise in the areas of mechanical ventilation and respiratory care modalities is particularly valuable considering the pace at which technological advances are occurring in critical care medicine.

The application of mechanical ventilation principles to patient care is one of the most sophisticated respiratory care applications used in critical care medicine, making frequent reviewing helpful, if not necessary. *Pilbeam's Mechanical Ventilation* can be useful to all critical care practitioners, including practicing respiratory therapists, critical care residents and physicians, physician assistants, and critical care nurse practitioners.

### **ORGANIZATION**

This edition, like previous editions, is organized into a logical sequence of chapters and sections that build on each other as a reader moves through the book. The initial sections focus on core knowledge and skills needed to apply and initiate mechanical ventilation, whereas the middle and final sections cover specifics of mechanical ventilation patient care techniques, including bedside pulmonary diagnostic testing, hemodynamic testing, pharmacology of patients receiving ventilation, and a concise discussion of ventilator-associated pneumonia, as well as neonatal and pediatric mechanical ventilatory techniques and long-term applications of mechanical ventilation. The inclusion of some helpful appendixes further assists the reader in the comprehension of complex material, and an easy-access Glossary defines key terms covered in the chapters.

### **FEATURES**

The valuable learning aids that accompany this text are designed to make it an engaging tool for both educators and students. With clearly defined resources in the beginning of each chapter, students can prepare for the material covered in each chapter through the use of Chapter Outlines, Key Terms, and Learning Objectives.

Along with the abundant use of images and information tables, each chapter contains:

- Case Studies: Concise patient vignettes that list pertinent assessment data and pose critical thinking questions to readers to test their understanding of content learned. Answers can be found in Appendix A.
- Critical Care Concepts: Short questions to engage the readers in applying their knowledge of difficult concepts.
- Clinical Scenarios: More comprehensive patient scenarios covering patient presentation, assessment data, and treatment therapies. These scenarios are intended for classroom or group discussion.
- Key Points: Highlight important information as key concepts are discussed.
- Each chapter concludes with:
  - A bulleted Chapter Summary for ease of reviewing chapter content
  - Chapter Review Questions (with answers in Appendix A)
  - A comprehensive list of References at the end of each chapter for those students who want to learn more about specific topics covered in the text

Finally, several appendixes are included to provide additional resources for readers. These include a Review of Abnormal Physiological Processes, which covers mismatching of pulmonary perfusion and ventilation, mechanical dead space, and hypoxia. A special appendix on Graphic Exercises gives students extra practice in understanding the interrelationship of flow, volume, and pressure in mechanically ventilated patients. Answer Keys to Case Studies and Critical Care Concepts featured throughout the text and the end-of-chapter Review Questions can help the student track progress in comprehension of the content.

This edition of *Pilbeam's Mechanical Ventilation* has been updated to reflect commonly used equipment and techniques to ensure it is in step with the current modes of therapy. Case Studies, Clinical Scenarios, and Critical Care Concepts are presented throughout the text to emphasize this new information.

### **LEARNING AIDS**

### Workbook

The workbook for *Pilbeam's Mechanical Ventilation* is an easy-to-use guide designed to help the student focus on the most important information presented in the text. The workbook features clinical exercises directly tied to the learning objectives that appear in the beginning of each chapter. Providing the reinforcement and practice that students need, the workbook features exercises such as key term crossword puzzles, critical thinking questions, case studies, waveform analysis, and National Board for Respiratory Care (NBRC)-style multiple-choice questions.

### **FOR EDUCATORS**

Educators using the Evolve website for *Pilbeam's Mechanical Ventilation* have access to an array of resources designed to work in coordination with the text and aid in teaching this topic. Educators may use the Evolve resources to plan class time and lessons, supplement class lectures, or create and develop student exams. These Evolve resources offer:

- More than 800 NBRC-style multiple-choice test questions in ExamView
- PowerPoint Presentation with more than 650 slides featuring key information and helpful images
- An Image Collection of the figures appearing in the book

Jim Cairo New Orleans, Louisiana

# **Acknowledgments**

number of dedicated individuals should be recognized for their contributions to this project. I wish to offer my sincere gratitude to Sue Pilbeam for her support over the years. I also want to thank Terry Forrette, MHS, RRT, FAARC, for authoring the chapter on Ventilator Graphics; Rob DiBlasi, RRT-NPS, FAARC, for authoring the chapter on Neonatal and Pediatric Ventilation; and Theresa Gramlich, MS, RRT, and Paul Barraza, RCP, RRT, for their contributions in earlier editions of this text. I also thank Sandra Hinski, MS, RRT-NPS, for authoring the ancillaries that accompany this text and Amanda Dexter, MS, RRT, and Gary Milne, BS, RRT, for their suggestions related to ventilator graphics. As in previous editions, I want to express my sincere appreciation to all of the respiratory therapy practitioners and educators who provided valuable suggestions and comments

during the course of writing and editing the eighth edition of *Pilbeam's Mechanical Ventilation*.

I would like to offer special thanks for the guidance provided by the staff of Elsevier throughout this project, particularly Content Strategist, Melissa Rawe; Director of Content Development, Ellen Wurm-Cutter; Content Development Specialist, Kristen Helm; Project Manager, Sindhuraj Thulasingam; and Publishing Services Manager, Deepthi Unni. Their dedication to this project has been consistently helpful, and I feel fortunate to have had the opportunity to work with such a professional group.

I particularly wish to thank my wife, Rhonda for always providing love and support for me and all of our family. Her gift of unconditional love and encouragement inspires me every day.

# **Contents**

### 1 Basic Terms and Concepts of Mechanical Ventilation, 1 Physiological Terms and Concepts Related to Mechanical Ventilation, 2 Normal Mechanics of Spontaneous Ventilation, 2 Ventilation and Respiration, 2 Gas Flow and Pressure Gradients Durina Ventilation, 2 Units of Pressure, 3 Definitions of Pressures and Gradients in the Lungs, 3 Lung Characteristics, 4 Compliance, 5 Resistance, 6 Time Constants, 8 Types of Ventilators and Terms Used in Mechanical Ventilation, 10 Types of Mechanical Ventilation, 10 Negative Pressure Ventilation, 10 Positive Pressure Ventilation, 10 High-Frequency Ventilation, 11 Definition of Pressures in Positive Pressure Ventilation, 11 Baseline Pressure, 12 Peak Pressure, 13 Plateau Pressure, 13 Pressure at the End of Exhalation, 13 Summary, 13 References, 16 **How Ventilators Work, 17** Historical Perspective on Ventilator Classification, 17 Internal Function, 18 Power Source or Input Power, 18 Electrically Powered Ventilators, 18 Pneumatically Powered Ventilators, 18 Positive and Negative Pressure Ventilators, 19 Control Systems and Circuits, 19 Open-Loop and Closed-Loop Systems to Control Ventilator Function, 19 Control Panel (User Interface), 20 Pneumatic Circuit, 21 Power Transmission and Conversion System, 23 Compressors (Blowers), 23 Volume Displacement Designs, 24 Volume Flow-Control Valves, 26

### How a Breath Is Delivered, 29

Summary, 27

References, 28

Basic Model of Ventilation in the Lung During Inspiration, 30 Factors Controlled and Measured During Inspiration, 31 Pressure-Controlled Breathing, 32

Volume-Controlled Breathing, 32 Flow-Controlled Breathing, 32 Time-Controlled Breathing, 32 Overview of Inspiratory Waveform Control, 32 Phases of a Breath and Phase Variables, 33 Beginning of Inspiration: The Trigger Variable, 34 The Limit Variable Durina Inspiration, 35 Termination of the Inspiratory Phase: The Cycling Mechanism (Cycle Variable), 38 Expiratory Phase: The Baseline Variable, 40 Types of Breaths, 43 Summary, 43 References, 45

### **Establishing the Need for Mechanical** Ventilation, 46

Acute Respiratory Failure, 47 Recognizing the Patient in Respiratory Distress, 47 Definition of Respiratory Failure, 47 Recognizing Hypoxemia and Hypercapnia, 48 Patient History and Diagnosis, 48 Central Nervous System Disorders, 48 Neuromuscular Disorders, 50 Increased Work of Breathing, 50 Physiological Measurements in Acute Respiratory Failure, 51 Bedside Measurements of Ventilatory Mechanics, 51 Failure of Ventilation and Increased Dead Space, 53 Failure of Oxygenation, 54 Overview of Criteria for Mechanical Ventilation, 54 Possible Alternatives to Invasive Ventilation, 55 Noninvasive Positive Pressure Ventilation, 55 Intubation Without Ventilation, 56 Ethical Considerations, 56 Summary, 58 References, 60

### Selecting the Ventilator and the Mode, 62

Noninvasive and Invasive Positive Pressure Ventilation: Selecting the Patient Interface, 63 Noninvasive Positive Pressure Ventilation, 63 Invasive Positive Pressure Ventilation, 64 Full and Partial Ventilatory Support, 64 Breath Delivery and Modes of Ventilation, 64 Type of Breath Delivery, 64 Targeting Volume as the Control Variable, 65 Targeting Pressure as the Control Variable, 65 Timing of Breath Delivery, 67 Modes of Ventilation, 67 History of Intermittent Positive Pressure Breathing and Intermittent Positive Pressure Ventilation: Understanding the Terminology, 67

Treating the Cause of Respiratory Failure, 110 Continuous Mandatory Ventilation, 68 Volume-Controlled Continuous Mandatory Ventilation, 70 Selecting the Appropriate Ventilator, 110 Pressure-Controlled Continuous Mandatory Ventilation, 70 Evaluation of Ventilator Performance, 111 Intermittent Mandatory Ventilation, 72 Initial Ventilator Settings for Specific Patient Situations, 111 Spontaneous Modes, 73 Chronic Obstructive Pulmonary Disease, 111 Bilevel Positive Airway Pressure, 75 Guidelines for Patients With Chronic Obstructive Additional Modes of Ventilation, 76 Pulmonary Disease, 111 Pressure Auamentation, 76 Asthma, 113 Pressure-Regulated Volume Control, 77 Guidelines for Patients With Asthma, 114 Volume Support Ventilation, 77 Neuromuscular Disorders, 114 Mandatory Minute Ventilation, 78 Guidelines for Patients With Neuromuscular Disorders, 115 Adaptive Support Ventilation, 78 Closed Head Injury, 115 Airway Pressure Release Ventilation, 79 Guidelines for Patients With a Closed Head Injury, 116 Proportional Assist Ventilation, 79 Acute Respiratory Distress Syndrome, 117 Neurally Adjusted Ventilatory Assist, 80 Guidelines for Patients With ARDS, 117 Summary, 80 Acute Cardiogenic Pulmonary Edema and Congestive References, 83 Heart Failure, 119 Guidelines for Patients With Congestive Heart Failure, 119 Initial Ventilator Settings, 85 Summary, 120 **Determining Initial Ventilator Settings During** References, 122 Volume-Controlled Ventilation, 86 Initial Patient Assessment, 124 Initial Settings During Volume-Controlled Ventilation, 86 Setting Minute Ventilation, 86 Documentation of the Patient-Ventilator System, 125 Tidal Volume and Rate, 88 The First 30 Minutes, 128 Relationship of Tidal Volume, Flow, Total Cycle Time, and Mode, 128 Inspiratory-to-Expiratory Ratio, 91 Sensitivity, 129 Inspiratory Flow and Flow Patterns, 92 Tidal Volume, Rate, and Minute Ventilation, 129 Setting the Minute Ventilation: Special Considerations, 95 Correcting Tubing Compliance, 130 Inspiratory Pause During Volume Ventilation, 95 Alveolar Ventilation, 130 Determining Initial Ventilator Settings During Pressure Monitoring Airway Pressures, 130 Ventilation, 96 Peak Inspiratory Pressure, 131 Setting Baseline Pressure: Physiological Positive Plateau Pressure, 131 End-Expiratory Pressure, 96 Set Pressure, 131 Determining Tidal Volume Delivery in Pressure Transairway Pressure: PIP Minus Pplat, 131 Ventilation, 97 Mean Airway Pressure, 131 Initial Settings for Pressure Support Ventilation, 98 End-Expiratory Pressure, 131 Initial Settings for Pressure Control Ventilation, 98 Driving Pressure, 132 Initial Settings for Bilevel Positive Airway Pressure Pressure Limit, 133 Ventilation, 99 Checking the Circuit: Checking for Leaks, 133 Initial Settings for Pressure Ventilation Modes Vital Signs, Blood Pressure, and Physical Examination of With Volume Taraetina, 99 the Chest, 134 Initial Settings of Pressure-Regulated Volume Control, 99 Heart Rate, 134 Initial Settings of Volume Support, 100 Temperature, 135 Summary, 100 Systemic Arterial Blood Pressure, 135 References, 102 Central Venous Pressure, 135 Pulmonary Artery Pressure, 135 Final Considerations in Ventilator Setup, 103 Physical Examination of the Chest, 135 Selection of Additional Parameters and Final Ventilator Management of Endotracheal Tube and Tracheostomy Setup, 103 Tube Cuffs, 136 Selection of Fractional Concentration of Inspired Cuff Pressure Measurement, 136 Oxygen, 103 High Cuff Pressure, 138 Sensitivity Setting, 104 Nonexistent or Low Cuff Pressure, 138 Humidification, 106 Cut in the Pilot Tube, 139 Alarms, 107 Tube and Mouth Care, 140 Action During Ventilator Alarm Situations, 109 Monitoring Compliance and Airway Resistance, 140 Periodic Hyperinflation or Sighing, 109 Static Compliance, 140 Final Considerations in Ventilator Equipment Setup, 110 Dynamic Characteristic (Dynamic Compliance), 140 Preparing the Patient, 110 Airway Resistance, 141 Establishing an Interface, 110 Bedside Measurement of Pressure-Volume Curves, 142 Manual Ventilation, 110 Comment Section of the Ventilator Flow Sheet, 144 Cardiovascular Stabilization, 110 Summary, 144 Ventilator Needs, 110 References, 146

9	Ven	tilator	Grap	hics,	147
---	-----	---------	------	-------	-----

Relationship of Flow, Pressure, Volume, and Time, 148 A Closer Look at Scalars, Curves, and Loops, 148

Scalars, 148

Comparison of Pressure-Controlled Ventilation and Volume-Controlled Ventilation. 150

Determining the Mode of Ventilation, 150

Components of the Pressure-Volume Loop, 150

Components of the Flow-Volume Loop, 151

Summary: Normal Scalars, Loops, and Curves, 151

Using Graphics to Monitor Pulmonary Mechanics, 154 Assessing Patient-Ventilator Asynchrony, 156

Advanced Applications, 158

Auto-PEEP and Air Trapping, 158

Titrating PEEP, 159

APRV Settings, 159

Integrated Ventilator and Esophageal Graphics, 159 Assessing Overdistention During Pressure-Controlled

Ventilation, 160

Inspiratory Rise Time Control: Sloping or Ramping, 161

Flow Cycling During Pressure Support Ventilation, 161

Summary, 162

Bibliography, 165

### 10 Assessment of Respiratory Function, 166

Noninvasive Measurements of Blood Gases, 167

Pulse Oximetry, 167

Capnography (Capnometry), 170

Technical Considerations, 170

Physiological Considerations, 171

Clinical Applications, 172

Volumetric Capnometry, 175

Exhaled Nitric Oxide Monitoring, 177

Transcutaneous Monitoring, 178

Transcutaneous PO2, 178

Transcutaneous PCO<sub>2</sub>, 179

Technical Considerations, 179

Indirect Calorimetry and Metabolic Measurements, 180

Overview of Indirect Calorimetry, 180

Assessment of Respiratory System Mechanics, 183

Measurements, 183

Airway Pressure Measurements, 183

Flow Measurements, 184

Clinical Applications, 185

Summary, 188

References, 190

### 11 Hemodynamic Monitoring, 192

Review of Cardiovascular Principles, 193

Factors Influencing Cardiac Output, 193

Obtaining Hemodynamic Measurements, 195

Hemodynamic Monitoring Systems, 195

Systemic Artery Catheterization, 196

Central Venous Lines, 197

Pulmonary Artery Catheterization, 197

Interpretation of Hemodynamic Profiles, 200

Heart Rate, 200

Systemic Arterial Pressure, 200

Right Atrial and Pulmonary Artery Pressures, 201

Cardiac Output, 204

Mixed Venous Oxygen Saturation, 205

Oxygen Delivery, 205

Shunt Fraction, 205

Vascular Resistance, 206

Ejection Fraction, 206

Cardiac Work, 206

Clinical Applications, 207

Summary, 210

References, 211

### 12 Methods to Improve Ventilation in Patient-Ventilator Management, 213

Correcting Ventilation Abnormalities, 214

Common Methods of Changing Ventilation Based on

P<sub>a</sub>CO<sub>2</sub> and pH, 214

Respiratory Acidosis: Volume and Pressure Ventilation Changes, 214

Respiratory Alkalosis: VC-CMV and PC-CMV Changes, 216

Metabolic Acidosis and Alkalosis, 217

Metabolic Acidosis, 217

Metabolic Alkalosis, 218

Mixed Acid-Base Disturbances, 218

Increased Physiological Dead Space, 218

Increased Metabolism and Increased Carbon

Dioxide Production, 219

Intentional latrogenic Hyperventilation, 219

Permissive Hypercapnia, 220

Procedures for Managing Permissive Hypercapnia, 220

Contraindications of Permissive Hypercapnia, 220

Airway Clearance During Mechanical Ventilation, 221

Secretion Clearance From an Artificial Airway, 221

Hazards and Complications of Suctioning, 223

Closed-Suction Catheters (Inline Suction Catheters), 223

Aspiration of Subglottic Secretions, 224

Normal Saline Instillation, 225

Assessment After Suctioning, 225

Administering Aerosols to Ventilated Patients, 226

Types of Aerosol-Generating Devices, 228

Ventilator-Related Factors, 228

Patient-Related Factors, 229

Circuit-Related Factors, 229

Use of Pressurized Metered-Dose Inhaler During

Mechanical Ventilation, 229

Use of Small-Volume Nebulizers During Mechanical

Ventilation, 230

Technical Problems Associated With Continuous

Nebulization Using an External Gas Source, 230

Nebulization Provided by the Ventilator, 231

Use of Nebulizers During Noninvasive Positive Pressure Ventilation, 231

Patient Response to Bronchodilator Therapy, 232

Postural Drainage and Chest Percussion, 232

Flexible Fiberoptic Bronchoscopy, 233

Additional Patient Management Techniques and Therapies

in Ventilated Patients, 235

Sputum and Upper Airway Infections, 235

Fluid Balance, 236

Psychological and Sleep Status, 236

Duration and Magnitude of Positive Pressures, 312

Patient Safety and Comfort, 237 Derecruitment Maneuver, 275 Patient Safety, 237 Summary of Recruitment Maneuvers in ARDS, 275 Patient Comfort, 237 Importance of Body Position During Positive Pressure Patient-Centered Mechanical Ventilation, 238 Ventilation, 275 Transport of Mechanically Ventilated Patients Within an Positioning in a Patient With ARDS: Prone Positioning, Acute Care Facility, 238 Summary, 239 Patient Position in Unilateral Lung Disease, 278 References, 241 Additional Patient Cases, 279 Summary, 280 13 Improving Oxygenation and Management of References, 282 Acute Respiratory Distress Syndrome, 244 Basics of Oxygenation Using F<sub>1</sub>O<sub>2</sub>, PEEP Studies, and Ventilator-Associated Pneumonia, 286 Pressure-Volume Curves for Establishing Optimal Epidemiology, 287 PEEP, 246 Causes and Risk Factors, 287 Basics of Oxygen Delivery to the Tissues, 246 Pathogenesis of Ventilator-Associated Pneumonia, 289 Introduction to Positive End-Expiratory Pressure and Diagnosis of Ventilator-Associated Pneumonia, 289 Continuous Positive Airway Pressure, 248 Clinical Diagnosis, 289 Pathophysiology of Atelectasis, 248 Bacteriological (Quantitative) Diagnosis, 291 Goals of PEEP and CPAP, 249 Treatment of Ventilator-Associated Pneumonia, 291 Terminology, 249 Strategies to Prevent Ventilator-Associated Technical Aspects of PEEP and CPAP Devices, 249 Pneumonia, 291 Application of CPAP and PEEP to the Patient's Airway, Nonpharmacological Interventions, 293 Pharmacological Interventions, 295 Circuitry for Spontaneous CPAP With Freestanding Summary, 296 Systems and Mechanical Ventilators, 250 References, 297 PEEP Ranges, 250 15 Sedatives, Analgesics, and Paralytics, 299 Minimum or Low PEEP, 250 Sedatives and Analgesics, 300 Therapeutic PEEP, 251 Monitoring the Need for Sedation and Analgesia, 300 Optimal PEEP, 251 Benzodiazepines, 301 Indications for PEEP and CPAP, 251 Neuroleptics, 302 Initiating PEEP Therapy, 251 Anesthetic Agents, 302 Selecting the Appropriate PEEP/CPAP Level Opioids, 303 (Optimal PEEP), 251 Application of PEEP Above 5 cm H<sub>2</sub>O, 251 Paralytics, 304 Monitoring Neuromuscular Blockade, 305 Optimal PEEP Study, 252 Depolarizing Agents, 305 Use of Pulmonary Vascular Pressure Monitoring with Nondepolarizing Agents, 305 PEEP, 257 Summary, 306 Contraindications and Physiological Effects of PEEP, 259 References, 307 Contraindications for PEEP, 259 Pulmonary Effects of PEEP, 259 **Extrapulmonary Effects of Mechanical** Transmission of Airway Pressure to Pleural Space, 260 Ventilation, 309 Uses of PEEP for Problems Other Than ARDS, 260 Effects of Positive Pressure Ventilation on the Heart and Weaning From PEEP, 261 Thoracic Vessels, 309 Acute Respiratory Distress Syndrome, 261 Adverse Cardiovascular Effects of Positive Pressure Pathophysiology, 263 Ventilation, 310 Changes in Computed Tomogram with ARDS, 263 The Thoracic Pump Mechanism During Normal ARDS as an Inflammatory Process, 265 Spontaneous Breathing and During Positive Pressure Two Categories of ARDS, 266 Ventilation, 310 ARDS: A Heterogeneous Disorder—Normal Lung versus Increased Pulmonary Vascular Resistance and Altered ARDS, 266 Right and Left Ventricular Function, 310 PEEP and the Vertical Gradient in ARDS, 267 Coronary Blood Flow With Positive Pressure Ventilation, Lung-Protective Strategies: Setting Tidal Volume and Pressures in ARDS, 267 Factors Influencing Cardiovascular Effects of Positive Long-Term Follow-Up on ARDS, 268 Pressure Ventilation, 311 Pressure-Volume Loops and Recruitment Maneuvers in Compensation in Individuals With Normal Setting PEEP in ARDS, 268 Cardiovascular Function, 311 Patient Evaluation for Lung Recruitment, 268 Effects of Lung and Chest Wall Compliance and Airway Pressure-Volume Loops in Setting PEEP, 269 Resistance, 312

Recruitment Maneuvers, 271

Beneficial Effects of Positive Pressure Ventilation on How Auto-PEEP Occurs, 330 Heart Function in Patients With Left Ventricular Physiological Factors That Lead to Auto-PEEP, 330 Dysfunction, 312 Identifying and Measuring Auto-PEEP, 331 Minimizing the Physiological Effects and Complications of Effect on Ventilator Function, 332 Mechanical Ventilation, 312 Measuring Static Compliance With Auto-PEEP, 332 Mean Airway Pressure and  $P_aO_2$ , 313 Methods of Reducing Auto-PEEP, 332 Reduction in Airway Pressure, 313 Hazards of Oxygen Therapy With Mechanical Inspiratory Flow, 313 Ventilation, 333 Inspiratory-to-Expiratory Ratio, 314 Oxygen Toxicity and the Lower Limits of Hypoxemia, 333 Inflation Hold, 314 Absorption Atelectasis, 333 Depression of Ventilation, 333 Positive End-Expiratory Pressure, 314 High Peak Pressures From Increased Airway Increased Work of Breathing, 333 System-Imposed Work of Breathing, 333 Resistance, 314 Intermittent Mandatory Ventilation, 315 Work of Breathing During Weaning, 333 Measuring Work of Breathing, 334 Effects of Mechanical Ventilation on Intracranial Pressure, Steps to Reduce Work of Breathing During Mechanical Renal Function, Liver Function, and Gastrointestinal Function, 315 Ventilation, 334 Effects of Mechanical Ventilation on Intracranial Pressure Ventilator Mechanical and Operational Hazards, 339 and Cerebral Perfusion, 315 Complications of the Artificial Airway, 339 Renal Effects of Mechanical Ventilation, 316 Summary, 341 Renal Response to Hemodynamic Changes, 316 References, 343 Endocrine Effects of Positive Pressure Ventilation on **Troubleshooting and Problem Solving, 346** Renal Function, 316 Definition of the Term Problem, 347 Arterial Blood Gases and Kidney Function, 317 Solving Ventilation Problems, 347 Implications of Impaired Renal Effects, 317 Protecting the Patient, 347 Effects of Mechanical Ventilation on Liver and Identifying the Patient in Sudden Distress, 348 Gastrointestinal Function, 317 Patient-Related Problems, 349 **Nutritional Complications During Mechanical** Airway Problems, 349 Ventilation, 317 Pneumothorax, 350 Summary, 318 Bronchospasm, 350 References, 319 Secretions, 350 Effects of Positive Pressure Ventilation on the Pulmonary Edema, 350 **Pulmonary System, 320** Dynamic Hyperinflation, 350 Lung Injury With Mechanical Ventilation, 321 Abnormalities in Respiratory Drive, 351 Ventilator-Associated Lung Injury Versus Change in Body Position, 351 Ventilator-Induced Lung Injury, 321 Drug-Induced Distress, 351 Barotrauma or Extraalveolar Air, 321 Abdominal Distention, 351 Barotrauma or Volutrauma, 323 Pulmonary Embolism, 351 Atelectrauma, 324 Ventilator-Related Problems, 351 Biotrauma, 325 Leaks, 352 Multiple Organ Dysfunction Syndrome, 325 Inadequate Oxygenation, 352 Vascular Endothelial Injury, 325 Inadequate Ventilatory Support, 352 Historic Webb and Tierney Study, 326 Trigger Sensitivity, 352 Role of PEEP in Lung Protection, 326 Inadequate Flow Setting, 352 Ventilator-Induced Respiratory Muscle Weakness, 326 Other Examples of Patient-Ventilator Asynchrony, 352 Effects of Mechanical Ventilation on Gas Distribution and Common Alarm Situations, 353 Pulmonary Blood Flow, 327 Low-Pressure Alarm, 353 Ventilation to Nondependent Lung, 327 High-Pressure Alarm, 354 Ventilation-to-Lung Periphery, 327 Low-PEEP/CPAP Alarms, 355 Increase in Dead Space, 327 Apnea Alarm, 356 Redistribution of Pulmonary Blood Flow, 327 Low-Source Gas Pressure or Power Input Alarm, 356 Ventilator Inoperative Alarm and Technical Error Effects of Positive Pressure on Pulmonary Vascular Resistance, 328 Message, 356 Respiratory and Metabolic Acid-Base Status in Mechanical Operator Settings Incompatible With Machine Ventilation, 328 Parameters, 356 Hypoventilation, 328 Inspiratory-to-Expiratory Ratio Indicator and Alarm, 356 Hyperventilation, 329 Other Alarms, 356 Metabolic Acid-Base Imbalances and Mechanical Use of Graphics to Identify Ventilator Problems, 356 Ventilation, 329 Leaks, 358 Air Trapping (Auto-PEEP), 330 Inadequate Flow, 358

Comparison of Traditional Weaning Methods, 395 Inadequate Sensitivity Setting for Patient Triggering, 359 Overinflation, 359 Closed-Loop Control Modes for Ventilator Auto-PEEP, 359 Discontinuation, 395 Inadequate Inspiratory Time During Pressure Automatic Tube Compensation, 395 Ventilation, 359 Volume-Targeted Pressure Support Ventilation, 397 Waveform Ringing, 359 Automode and Variable Pressure Support/Variable Expiratory Portion of Volume-Time Curve Below Pressure Control, 397 Baseline, 360 Mandatory Minute Ventilation, 397 Patient-Ventilator Asynchrony, 360 Adaptive Support Ventilation, 398 Unexpected Ventilator Responses, 361 Artificial Intelligence Systems, 398 Evidence-Based Weaning, 398 Unseated or Obstructed Expiratory Valve, 361 Excessive CPAP/PEEP, 362 Evaluation of Clinical Criteria for Weaning, 398 Nebulizer Impairment of Patient's Ability to Triager a Recommendation 1: Pathology of Ventilator Pressure-Supported Breath, 362 Dependence, 398 High Tidal Volume Delivery, 363 Weaning Criteria, 400 Altered Alarm Function, 364 Patient Ventilatory Performance and Muscle Electromagnetic Interference, 364 Strength, 401 Operator's Manuals, 364 Measurement of Drive to Breathe, 402 Summary, 364 Work of Breathing, 402 References, 367 Adequacy of Oxygenation, 403 Recommendation 2: Assessment of Readiness for **Basic Concepts of Noninvasive Positive** Weaning Using Evaluation Criteria, 403 **Pressure Ventilation, 368** Recommendation 3: Assessment During a Spontaneous Types of Noninvasive Ventilation Techniques, 369 Breathing Trial, 403 Negative Pressure Ventilation, 369 Recommendation 4: Removal of the Artificial Airway, 403 Positive Pressure Ventilation, 370 Postextubation Difficulties, 404 Goals of and Indications for Noninvasive Positive Pressure Noninvasive Positive Pressure Ventilation After Ventilation, 370 Extubation, 405 Acute Care Setting, 370 Factors in Weaning Failure, 405 Chronic Care Setting, 372 Recommendation 5: Spontaneous Breathing Other Indications for Noninvasive Ventilation, 373 Trial Failure, 405 Facilitation of Weaning From Invasive Ventilation, 373 Nonrespiratory Factors That May Complicate Weaning, 405 "Do Not Intubate" Patients, 373 Cardiac Factors, 406 Patient Selection Criteria, 373 Acid-Base Factors, 408 Acute Care Setting, 374 Metabolic Factors, 408 Chronic Care Setting, 374 Effect of Pharmacological Agents, 408 Equipment Selection for Noninvasive Ventilation, 375 Nutritional Status and Exercise, 408 Types of Ventilators, 375 Psychological Factors, 409 Humidification During Noninvasive Ventilation, 378 Recommendation 6: Maintaining Ventilation in Patients Patient Interfaces, 378 With Spontaneous Breathing Trial Failure, 409 Nasal Interfaces, 378 Final Recommendations, 410 Setup and Preparation for Noninvasive Ventilation, 382 Recommendation 7: Anesthesia and Sedation Strategies Monitoring and Adjustment of Noninvasive and Protocols, 410 Ventilation, 382 Recommendation 8: Weaning Protocols, 410 Aerosol Delivery in Noninvasive Ventilation, 384 Recommendation 9: Role of Tracheostomy in Complications of Noninvasive Ventilation, 384 Weaning, 410 Discontinuing Noninvasive Ventilation, 386 Recommendation 10: Long-Term Care Facilities for Patient Care Team Concerns, 386 Patients Requiring Prolonged Ventilation, 412 Summary, 386 Recommendation 11: Clinician Familiarity With References, 388 Long-Term Care Facilities, 412 Recommendation 12: Weaning in Long-Term Ventilation 20 Weaning From and Discontinuation of Units, 412 Mechanical Ventilation, 391 American Thoracic Society/American College of Weaning Techniques, 392 Chest Physicians Clinical Practice Guideline: Liberation Methods of Titrating Ventilator Support During From Mechanical Ventilation, 412 Weaning, 393 Ethical Dilemma: Withholding and Withdrawing Intermittent Mandatory Ventilation, 393 Ventilatory Support, 412 Pressure Support Ventilation, 394 Summary, 413

References, 415

T-Piece Weaning, 395

21 Long-T	'erm V	'entil	ation,	418
-----------	--------	--------	--------	-----

Goals of Long-Term Mechanical Ventilation, 419

Sites for Ventilator-Dependent Patients, 420

Acute Care Sites, 420

Intermediate Care Sites, 420

Long-Term Care Sites, 420

Patient Selection, 420

Disease Process and Clinical Stability, 420

Psychosocial Factors, 421

Financial Considerations, 422

Preparation for Discharge to the Home, 422

Geographical and Home Assessment, 423

Family Education, 423

Additional Preparation, 424

Follow-Up and Evaluation, 424

Adequate Nutrition, 424

Family Issues, 424

Equipment Selection for Home Ventilation, 426

Tracheostomy Tubes, 426

Ventilator Selection, 426

Complications of Long-Term Positive Pressure

Ventilation, 429

Alternatives to Invasive Mechanical Ventilation

at Home, 430

Noninvasive Positive Pressure Ventilation, 430

Negative Pressure Ventilation, 430

Additional Noninvasive Devices, 431

Diaphragm Pacing, 433

Continuous Positive Airway Pressure for Obstructive Sleep

Apnea, 433

Glossopharyngeal Breathing, 434

Expiratory Muscle AIDS and Secretion Clearance, 434

Assisted Coughing, 434

Mechanical Oscillation, 435

Mechanical Insufflation-Exsufflation, 435

Tracheostomy Tubes, Speaking Valves, and Tracheal

Buttons, 435

Tracheostomy Tube Selection and Benefits, 435

Loss of Speech, 436

Speaking With Tracheostomy Tubes During

Ventilation, 436

Speaking Tracheostomy Tubes, 436

Tracheostomy Speaking Valves, 437

Concerns With Speaking Tubes and Valves, 439

Tracheal Buttons and Decannulation, 439

Ancillary Equipment and Equipment Cleaning for Home

Mechanical Ventilation, 440

Disinfection Procedures, 440

Humidifiers, 441

Summary, 441

References, 444

# 22 Neonatal and Pediatric Mechanical Ventilation, 447

Recognizing the Need for Mechanical Ventilatory

Support, 448

Clinical Indications for Respiratory Failure, 448

Determining Effective Oxygenation and Ventilation, 449

Goals of Newborn and Pediatric Ventilatory

Support, 450

Noninvasive Respiratory Support, 450

Noninvasive Nasal Continuous Positive Airway Pressure in Neonates. 450

Noninvasive Positive Pressure Ventilation in

Neonates, 454

Continuous Positive Airway Pressure and Bilevel Positive

Airway Pressure in Pediatric Patients, 455

Conventional Mechanical Ventilation, 457

Indications for Ventilatory Support of Neonates, 457

Indications for Ventilatory Support of Pediatric

Patients, 458

The Pediatric Ventilator, 458

Pressure Control Mode, 461

Positive End-Expiratory Pressure, 462

Volume Control Mode, 467

Pressure Support Ventilation, 467

Dual-Control Mode, 468

Volume Support Ventilation, 472

Airway Pressure Release Ventilation, 472

Lung-Protective Strategies in Conventional

Ventilation, 472

High-Frequency Ventilation, 474

Indications for High-Frequency Ventilation, 475

Contraindications and Complications of High-Frequency

Ventilation, 475

High-Frequency Ventilation Techniques, 475

High-Frequency Positive Pressure Ventilation, 475

High-Frequency Flow Interruption, 476

High-Frequency Percussive Ventilation, 476

High-Frequency Oscillatory Ventilation, 476

High-Frequency Jet Ventilation, 477

Physiology of High-Frequency Ventilation, 478

Management Strategies for High-Frequency

Ventilation, 480

Weaning and Extubation, 482

Adjunctive Forms of Respiratory Support, 483

Surfactant Replacement Therapy, 483

Prone Positionina, 484

Inhaled Nitric Oxide Therapy, 484

Summary, 485

References, 487

### 23 Special Techniques Used in Ventilatory Support, 492

Airway Pressure Release Ventilation, 493

Other Names, 493

Advantages of Airway Pressure Release Compared with

Conventional Ventilation, 494

Preserving Spontaneous Ventilation, 494

APRV and Airway Pressures During Spontaneous

Breathing, 495

Disadvantages, 495 Initial Settings, 495

Setting High Pressure, 495

Setting Low Pressure, 496

Setting High Time, 496

Setting Low Time, 496

Adjusting Ventilation and Oxygenation, 496

Discontinuation, 497

High-Frequency Oscillatory Ventilation in the Adult, 497

Technical Aspects, 498

Initial Control Settings, 499

Mean Airway Pressure, 499 Amplitude, 499 Frequency, 500 Inspiratory Time Percentage, 500 Bias Flow, 500 Additional Settings, 500 Indication and Exclusion Criteria, 501 Monitoring, Assessment, and Adjustment, 501 Adjusting Settings to Maintain Arterial Blood Gas Goals, 502 Returning to Conventional Ventilation, 503 Heliox Therapy and Mechanical Ventilation, 503 Gas Flow Through the Airways, 504 Heliox in Avoiding Intubation and During Mechanical Ventilation, 504 Postextubation Stridor, 505 Devices for Delivering Heliox in Spontaneously Breathing Patients, 505

Mask Heliox, 505

Cost and Gas Consumption During Heliox Therapy, 505 Heliox and Aerosol Delivery, 506

Manufactured Heliox Delivery System, 506 Heliox and Aerosol Delivery During Mechanical Ventilation, 507 Heliox With a Mechanical Ventilator, 507 Technical Considerations in Heliox Delivery, 508

Heliox and NIV, 509

Monitoring the Electrical Activity of the Diaphragm and Neurally Adjusted Ventilatory Assist, 509

Review of Neural Control of Ventilation, 510 Diaphragm Electrical Activity Monitoring, 510 History of Diaphragm Electrical Activity Monitoring, 510 The Edi Catheter: Its Characteristics and Placement, 510 Detecting Patient-Ventilator Asynchrony Using the Edi Catheter, 512 Using the Edi Waveform to Interpret Ventilator Synchrony, 513 Neurally Adjusted Ventilatory Assist, 514 Using NAVA Ventilation, 514 Alarms and Safety Features in NAVA, 515 Results of Initiating NAVA Ventilation, 516

Weaning from NAVA, 516 Evaluating NAVA, 516 Summary, 516

References, 519

Appendix A Answer Key, 522

Appendix B Review of Abnormal Physiological Processes, 539

**Appendix C Graphics Exercises, 544** 

Glossary, 548

Index, 555

This page intentionally left blank

# Basic Terms and Concepts of Mechanical Ventilation

### **OUTLINE**

PHYSIOLOGICAL TERMS AND CONCEPTS RELATED TO MECHANICAL VENTILATION, 2

**NORMAL MECHANICS OF SPONTANEOUS VENTILATION, 2** 

Ventilation and Respiration, 2

Gas Flow and Pressure Gradients During Ventilation, 2 Units of Pressure, 3

Definitions of Pressures and Gradients in the Lungs, 3

Transairway Pressure, 3

Transthoracic Pressure, 3

Transpulmonary Pressure, 3

Transrespiratory Pressure, 4

**LUNG CHARACTERISTICS, 4** 

Compliance, 5 Resistance, 6

Measuring Airway Resistance, 7

TIME CONSTANTS, 8

TYPES OF VENTILATORS AND TERMS USED IN MECHANICAL VENTILATION, 10

**TYPES OF MECHANICAL VENTILATION, 10** 

Negative Pressure Ventilation, 10

Positive Pressure Ventilation, 10

High-Frequency Ventilation, 11
DEFINITION OF PRESSURES IN POSITIVE PRESSURE

**VENTILATION, 11** 

Baseline Pressure, 12

Peak Pressure, 13 Plateau Pressure, 13

Pressure at the End of Exhalation, 13

**SUMMARY, 13** 

### **KEY TERMS**

- Acinus
- Airway opening pressure
- Airway pressure
- Alveolar distending pressure
- Alveolar pressure
- Ascites
- Auto-PEEP
- Bronchopleural fistulas
- Compliance
- · Critical opening pressure
- Elastance
- Esophageal pressure
- External respiration
- Extrinsic PEEP
- Fast lung unit
- Functional residual capacity

- Heterogeneous
- High-frequency jet ventilation
- High-frequency oscillatory ventilation
- High-frequency percussive ventilation
- Homogeneous
- Internal respiration
- Intrapulmonary pressure
- Intrinsic PEEP
- Manometer
- Mask pressure
- Mouth pressure
- Peak airway pressure
- Peak inspiratory pressure
- Peak pressure
- Plateau pressure
- Positive end-expiratory pressure (PEEP)

- Pressure gradient
- Proximal airway pressure
- Resistance
- Respiration
- Slow lung unit
- Static compliance/static effective compliance
- Time constant
- Transairway pressure
- Transpulmonary pressure
- Transrespiratory pressure
- Transthoracic pressure
- Upper airway pressure
- Ventilation

### **LEARNING OBJECTIVES**

On completion of this chapter, the reader will be able to do the following:

- 1. Define ventilation, external respiration, and internal respiration.
- 2. Draw a graph showing how intrapleural and alveolar (intrapulmonary) pressures change during spontaneous ventilation and during a positive pressure breath.
- 3. Define the terms transpulmonary pressure, transrespiratory pressure, transairway pressure, transthoracic pressure, elastance, compliance, and resistance.
- Provide the value for intraalveolar pressure throughout inspiration and expiration during normal, quiet breathing.
- 5. Write the formulas for calculating compliance and resistance.
- Explain how changes in lung compliance affect the peak pressure measured during inspiration with a mechanical ventilator.
- Describe the changes in airway conditions that can lead to increased resistance.

- 8. Calculate the airway resistance given the peak inspiratory pressure, a plateau pressure, and the flow rate.
- Using a figure showing abnormal compliance or airway resistance, determine which lung unit will fill more quickly or with a greater volume.
- Compare several time constants, and explain how different time constants will affect volume distribution during inspiration.
- 11. Give the percentage of passive filling (or emptying) for one, two, three, and five time constants.
- Briefly discuss the principle of operation of negative pressure, positive pressure, and high-frequency mechanical ventilators.
- Define peak inspiratory pressure, baseline pressure, positive endexpiratory pressure, and plateau pressure.
- 14. Describe the measurement of plateau pressure.

# PHYSIOLOGICAL TERMS AND CONCEPTS RELATED TO MECHANICAL VENTILATION

The purpose of this chapter is to provide a brief review of the physiology of breathing and a description of the pressure, volume, and flow events that occur during the respiratory cycle. The effects of changes in lung characteristics (e.g., respiratory compliance and airway resistance) on the mechanics of breathing are also discussed.

# NORMAL MECHANICS OF SPONTANEOUS VENTILATION

### **Ventilation and Respiration**

The primary function of the respiratory system is gas exchange (i.e., the exchange of oxygen and carbon dioxide between an organism and its environment). Two terms that are used to describe this movement of air into and out of the lungs are ventilation and respiration. Spontaneous ventilation or breathing is typically defined as the movement of air into and out of the lungs. The driving force required to accomplish a spontaneous breath involves establishing a pressure difference between the atmosphere and the pressure inside of the lungs. As discussed in the following section on gas flow and pressure gradients, it is important to recognize that establishing this pressure gradient during a spontaneous inspiration is not a passive event but rather an active event involving "work" performed by the respiratory muscles. Specifically, it is the contraction of the muscles of inspiration during a spontaneous breath that causes expansion of the thorax, which in turn produces a decrease in the intrapulmonary pressure. It is important to recognize that establishing this pressure gradient during spontaneous inspiration is not a passive event but rather an active event involving "work" performed by the respiratory muscles due to the elastic properties of the lungs and chest wall surrounding the lungs.

During a quiet inspiration, the diaphragm descends and enlarges the longitudinal length of the thoracic cavity while the external intercostal muscles raise the ribs slightly, increasing the circumference of the thorax. Contraction of the diaphragm and external intercostal muscles provides the energy to move air into the lungs and therefore perform the work of breathing required to overcome the impedance offered by the lungs and chest wall. Note that during a maximal spontaneous inspiration, the accessory muscles of inspiration must also be recruited to increase the volume of the thorax.

During a quiet expiration, the inspiratory muscles simply relax, the diaphragm moves upward, and the ribs return to their resting position. The volume of the thoracic cavity decreases, and air is forced out of the alveoli. To achieve a maximum expiration (below the end-tidal expiratory level), the accessory muscles of expiration must be used to compress the thorax. Box 1.1 lists the various accessory muscles of breathing.

Respiration involves the exchange of oxygen and carbon dioxide between an organism and its environment. Respiration is typically divided into two components: **external respiration** and **internal respiration**. External respiration involves the diffusion of oxygen and carbon dioxide between the alveoli and the pulmonary capillaries. Oxygenated blood leaving the pulmonary capillaries is carried by the pulmonary veins to the left heart and distributed to

### BOX **1.1**

### **Accessory Muscles of Breathing**

### Inspiration

- · Scalene (anterior, medial, and posterior)
- · Sternocleidomastoids
- Pectoralis (major and minor)
- Trapezius

### **Expiration**

- Rectus abdominis
- External oblique
- Internal oblique
- Transverse abdominal
- Serratus (anterior, posterior)
- · Latissimus dorsi

the cells of the body via the systemic arteries and capillaries. Simultaneously, carbon dioxide dissolved in blood returning via the systemic veins from the systemic organs diffuses from the pulmonary capillaries into the alveoli and is ultimately exhaled to the atmosphere during expiration.

Internal respiration occurs at the cellular level and involves the exchange of oxygen and carbon dioxide between the systemic capillaries and the cells of the body. At the cellular level, oxygen diffuses into the cells, where it is used in the oxidation of available substrates (e.g., carbohydrates and lipids) to produce energy. Carbon dioxide, which is a major by-product of aerobic metabolism, diffuses out of the cells into the systemic capillaries and is returned by bulk flow via the systemic veins back to the right heart, the pulmonary arteries, and the pulmonary capillaries.

# Gas Flow and Pressure Gradients During Ventilation

For air to flow through a tube or airway, a **pressure gradient** must exist (i.e., pressure at one end of the tube must be higher than pressure at the other end of the tube). Air will always flow from the high-pressure point to the low-pressure point.

Consider what happens during a normal quiet breath. Lung volumes change as a result of gas flow into and out of the airways caused by changes in the pressure gradient between the airway opening and the alveoli. During a spontaneous inspiration, contraction of the inspiratory muscles causes enlargement of the thorax resulting in a decrease (more negative) in intrapleural and alveolar pressure. The alveolar pressure therefore becomes less than the pressure at the airway opening (i.e., the mouth and nose), and gas flows into the lungs. Conversely, during a quiet expiration, relaxation of the inspiratory muscles results in a decrease in thoracic volume (i.e., diaphragm and external intercostal muscles return to their resting position) and an increase in alveolar pressure. Gas flows out of the lungs during expiration because the pressure in the alveoli is higher than the pressure at the airway opening. It is important to recognize that when the pressure at the airway opening and the pressure in the alveoli are the same, as occurs at the end of expiration, bulk gas flow does not occur because the pressures across the conductive airways are equal (i.e., there is no pressure gradient).

### **BOX 1.2 Pressure Equivalents**

```
1 mm Hg = 1.36 cm H_2O

1 kPa = 7.5 mm Hg

1 Torr = 1 mm Hg

1 atm = 760 mm Hg = 1034 cm H_2O
```

### **Units of Pressure**

Ventilating pressures are commonly measured in centimeters of water pressure (cm H<sub>2</sub>O) or mm of mercury pressure (mm Hg). These pressures are referenced to atmospheric pressure, which is assigned a baseline value of zero. In other words, although atmospheric pressure is 760 mm Hg or 1034 cm H<sub>2</sub>O (1 mm Hg = 1.36 cm H<sub>2</sub>O) at sea level, atmospheric pressure is designated as 0 cm H<sub>2</sub>O. For example, when airway pressure increases by +20 cm H<sub>2</sub>O during a positive pressure breath, the pressure actually increases from 760 mm Hg to 780 mm Hg (1034 to 1054 cm H<sub>2</sub>O). Other units of measure that are becoming more widely used for gas pressures, such as arterial oxygen pressure (P<sub>a</sub>O<sub>2</sub>) and arterial carbon dioxide pressure (P<sub>a</sub>CO<sub>2</sub>), are the torr (1 Torr = 1 mm Hg) and the kilopascal ([kPa]; 1 kPa = 7.5 mm Hg). The kilopascal is used in the International System of Units. (Box 1.2 provides a summary of common units of measurement for pressure.)

### Definitions of Pressures and Gradients in the Lungs

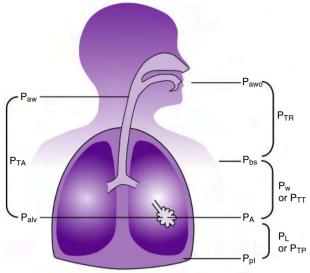
Airway opening pressure  $(P_{awo})$  is most often called mouth pressure  $(P_M)$  or airway pressure  $(P_{aw})$  (Fig. 1.1). Other terms that are often used to describe the airway opening pressure include upper-airway pressure, mask pressure, and proximal airway pressure. Unless pressure is applied at the airway opening,  $P_{awo}$  is zero or atmospheric pressure.

A similar measurement is the pressure at the body surface (P<sub>bs</sub>) equal to zero (atmospheric pressure) unless the person is placed in a pressurized chamber (e.g., hyperbaric chamber) or a negative pressure ventilator (e.g., iron lung).

Intrapleural pressure  $(P_{pl})$  is the pressure in the potential space between the parietal and visceral pleurae.  $P_{pl}$  is normally about -5 cm  $H_2O$  at the end of expiration during spontaneous breathing. It is about -10 cm  $H_2O$  at the end of inspiration. Because  $P_{pl}$  is often difficult to measure in a patient, a related measurement is used, the **esophageal pressure**  $(P_{es})$ , which is obtained by placing a specially designed balloon in the esophagus; changes in the balloon pressure are used to estimate pressure and pressure changes in the pleural space. (See Chapter 10 for more information about esophageal pressure measurements.)

Another commonly measured pressure is alveolar pressure  $(P_{alv})$ . This pressure is also called *intrapulmonary pressure* or *lung pressure*. Alveolar pressure normally changes as the intrapleural pressure changes. During spontaneous inspiration,  $P_{alv}$  is about -1 cm  $H_2O$ , and during exhalation it is about +1 cm  $H_2O$ .

Four basic pressure gradients are used to describe normal ventilation: transairway pressure, transthoracic pressure, transpulmonary pressure (or transalveolar pressure), and transrespiratory pressure (Table 1.1; see also Fig. 1.1).



P<sub>awo</sub> - Mouth or airway opening pressure
P<sub>alv</sub> - Alveolar pressure
P<sub>pl</sub> - Intrapleural pressure
P<sub>bs</sub> - Body surface pressure
P<sub>aw</sub> - Airway pressure (= P<sub>awo</sub>)

 $P_L$  or  $P_{TP}$  = Transpulmonary pressure  $(P_L = P_{alv} - P_{pl})$   $P_w$  or  $P_{TT}$  = Transthoracic pressure  $(P_{alv} - P_{bs})$   $P_{TA}$  = Transairway pressure  $(P_{aw} - P_{alv})$  $P_{TB}$  = Transrespiratory pressure

Fig. 1.1 Various pressures and pressure gradients of the respiratory system. (From Kacmarek RM, Stoller JK, Heuer AJ, editors: *Egan's fundamentals of respiratory care*, ed 11, St. Louis, MO, 2017, Elsevier.)

### **Transairway Pressure**

Transairway pressure ( $P_{TA}$ ) is the pressure difference between the airway opening and the alveolus:  $P_{TA} = P_{awo} - P_{alv}$ . It is therefore the pressure gradient required to produce airflow in the conductive airways. It represents the pressure that must be generated to overcome resistance to gas flow in the airways (i.e., airway resistance).

### **Transthoracic Pressure**

**Transthoracic pressure** ( $P_W$  or  $P_{TT}$ ) is the pressure difference between the alveolar space or lung and the body's surface ( $P_{bs}$ ):  $P_W$  (or  $P_{TT}$ ) =  $P_{alv}$  –  $P_{bs}$ . It represents the pressure required to expand or contract the lungs and the chest wall at the same time.

### **Transpulmonary Pressure**

Transpulmonary pressure or transalveolar pressure ( $P_L$  or  $P_{TP}$ ) is the pressure difference between the alveolar space and the pleural space ( $P_{pl}$ ):  $P_L$  (or  $P_{TP}$ ) =  $P_{alv}$  –  $P_{pl}$ .  $P_L$  is the pressure required to maintain alveolar inflation and is therefore sometimes called the **alveolar distending pressure**. (NOTE: An airway pressure measurement called the **plateau pressure** [ $P_{plat}$ ] is sometimes substituted for  $P_{alv}$ .  $P_{plat}$  is measured during a breath-hold maneuver during mechanical ventilation, and the value is read from the ventilator manometer. ( $P_{plat}$  is discussed in more detail later in this chapter.)

All modes of ventilation increase  $P_{TP}$  during inspiration, by either decreasing  $P_{pl}$  (negative pressure ventilators) or increasing  $P_{alv}$  by increasing pressure at the upper airway (positive pressure ventilators). During negative pressure ventilation, the pressure at

TABLE 1.1 Terms, abbreviations, and pressure gradients for the respiratory system.

Abbreviation	Term			
С	Compliance			
R	Resistance			
R <sub>aw</sub>	Airway resistance			
P <sub>M</sub>	Pressure at the mouth (same as P <sub>awo</sub> )			
P <sub>aw</sub>	Airway pressure (usually upper airway)			
P <sub>awo</sub>	Pressure at the airway opening; mouth pressure; mask pressure			
P <sub>bs</sub>	Pressure at the body surface			
Palv	Alveolar pressure (also P <sub>A</sub> )			
P <sub>pl</sub>	Intrapleural pressure			
C <sub>st</sub>	Static compliance			
C <sub>dyn</sub>	Dynamic compliance			
Pressure gradients				
Transairway pressure (P <sub>TA</sub> )	Airway pressure – alveolar pressure	$P_{TA} = P_{aw} - P_{alv}$		
Transthoracic pressure (P <sub>w</sub> )	Alveolar pressure — body surface pressure	$P_W$ (or $P_{TT}$ ) = $P_{alv} - P_{bc}$		
Transpulmonary pressure (P <sub>L</sub> )	Alveolar pressure — pleural pressure (also defined as the <i>transalveolar pressure</i> )	$P_L$ (or $P_{TP}$ ) = $P_{alv} - P_{pl}$		
Transrespiratory pressure (P <sub>TR</sub> )	Airway opening pressure — body surface pressure	$P_{TR} = P_{awo} - P_{bs}$		

the body surface  $(P_{bs})$  becomes negative and this pressure is transmitted to the pleural space, resulting in a decrease (more negative) in intrapleural pressure  $(P_{pl})$  and an increase in transpulmonary pressure  $(P_L)$ . During positive pressure ventilation, the  $P_{bs}$  remains atmospheric, but the pressures at the airway opening  $(P_{awo})$  and in the conductive airways (airway pressure, or  $P_{aw}$ ) become positive. Alveolar pressure  $(P_{alv})$  then becomes positive, and transpulmonary pressure  $(P_L)$  is increased.\*

### **Transrespiratory Pressure**

Transrespiratory pressure ( $P_{TR}$ ) is the pressure difference between the airway opening and the body surface:  $P_{TR} = P_{awo} - P_{bs}$ . Transrespiratory pressure is used to describe the pressure required to inflate the lungs during positive pressure ventilation. In this situation, the body surface pressure ( $P_{bs}$ ) is atmospheric and usually is given the value zero; thus  $P_{awo}$  becomes the pressure reading on a ventilator gauge ( $P_{aw}$ ).

Transrespiratory pressure has two components: transthoracic pressure (the pressure required to overcome elastic recoil of the lungs and chest wall) and transairway pressure (the pressure required to overcome airway resistance). Transrespiratory pressure can therefore be described by the equations  $P_{TR} = P_{TT} + P_{TA}$  and  $(P_{awo} - P_{bs}) = (P_{alv} - P_{bs}) + (P_{aw} - P_{alv})$ .

Consider what happens during a normal, spontaneous inspiration (Fig. 1.2). As the volume of the thoracic space increases, the pressure in the pleural space (intrapleural pressure) becomes more negative in relation to atmospheric pressures. (This is an expected result according to Boyle's law. For a constant temperature, as the volume increases, the pressure decreases.) The intrapleural pressure drops from about -5 cm  $H_2O$  at end expiration to

about -10 cm  $H_2O$  at end inspiration. The negative intrapleural pressure is transmitted to the alveolar space, and the intrapulmonary, or alveolar ( $P_{alv}$ ), pressure becomes more negative relative to atmospheric pressure. The transpulmonary pressure ( $P_L$ ), or the pressure gradient across the lung, widens (Table 1.2). As a result, the alveoli have a negative pressure during spontaneous inspiration.

The pressure at the airway opening or body surface is still atmospheric, creating a pressure gradient between the mouth (zero) and the alveolus of about -3 to -5 cm  $H_2O$ . The transairway pressure gradient ( $P_{TA}$ ) is approximately (0 - [-5]), or 5 cm  $H_2O$ . Air flows from the mouth or nose into the lungs and the alveoli expand. When the volume of gas builds up in the alveoli and the pressure returns to zero, airflow stops. This marks the end of inspiration; no more gas moves into the lungs because the pressure at the mouth and in the alveoli equals zero (i.e., atmospheric pressure) (see Fig. 1.2).

During expiration, the muscles relax and the elastic recoil of the lung tissue results in a decrease in lung volume. The thoracic volume decreases to resting, and the intrapleural pressure returns to about  $-5~{\rm cm}~H_2{\rm O}$ . Notice that the pressure inside the alveolus during exhalation increases and becomes slightly positive (+5 cm  $H_2{\rm O}$ ). As a result, pressure is now lower at the mouth than inside the alveoli, and the transairway pressure gradient causes air to move out of the lungs. When the pressure in the alveoli and mouth is equal, exhalation ends.

### **LUNG CHARACTERISTICS**

Normally, two types of forces oppose inflation of the lungs: elastic forces and frictional forces. Elastic forces arise from the elastic properties of the lungs and chest wall. Frictional forces are the result of two factors: the resistance of the tissues and organs as they become displaced during breathing and the resistance to gas flow through the airways.

Two parameters are often used to describe the mechanical properties of the respiratory system and the elastic and frictional forces opposing lung inflation: *compliance* and *resistance*.

<sup>\*</sup>The definition of transpulmonary pressure varies in research articles and textbooks. Some authors define it as the difference between airway opening pressure and pleural pressure, whereas others define transpulmonary pressure as the pressure difference between airway pressure and pleural pressure. This latter definition implies that airway pressure is the pressure exerted by the lungs during a breath-hold maneuver, that is, under static (no-flow) conditions.<sup>4</sup>

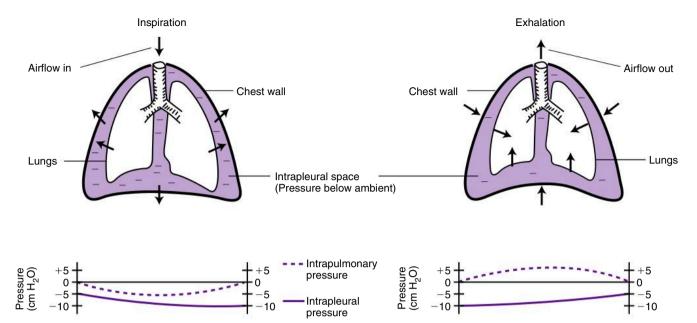


Fig. 1.2 The mechanics of spontaneous ventilation and the resulting pressure waves (approximately normal values). During inspiration, intrapleural pressure  $(P_{pl})$  decreases to -10 cm  $H_2O$ . During exhalation,  $P_{pl}$  increases from -10 to -5 cm  $H_2O$ . (See the text for further description.)

### TABLE 1.2 Changes in transpulmonary pressure under varying conditions.

Pressure	End Expiration	End Inspiration
Passive spontaneous ventilation		
Intraalveolar (intrapulmonary)	0 cm H <sub>2</sub> O	0 cm H₂O
Intrapleural	-5 cm H <sub>2</sub> O	-10 cm H <sub>2</sub> O
Transpulmonary	$P_1 = 0 - (-5) = +5 \text{ cm H}_2O$	$P_1 = 0 - (-10) = 10 \text{ cm H}_2\text{C}$
Negative pressure ventilation	_ · · · · ·	
Intraalveolar (intrapulmonary)	0 cm H <sub>2</sub> O	0 cm H <sub>2</sub> O
Intrapleural	−5 cm H <sub>2</sub> O	-10 cm H <sub>2</sub> O
Transpulmonary	$P_1 = 0 - (-5) = +5 \text{ cm H}_2O$	$P_1 = 0 - (-10) = 10 \text{ cm H}_2\text{C}$
Positive pressure ventilation	_ · · · · ·	
Intraalveolar (intrapulmonary)	0 cm H <sub>2</sub> O	9-12 cm H <sub>2</sub> O <sup>b</sup>
Intrapleural	−5 cm H <sub>2</sub> O	2-5 cm H <sub>2</sub> Õ <sup>b</sup>
Transpulmonary	$P_1 = 0 - (-5) = +5 \text{ cm H}_2O$	$P_L = 10 - (2) = +8 \text{ cm H}_2O^b$

 $<sup>^{</sup>a}P_{L}=P_{alv}-P_{pl}$ 

### **Compliance**

The **compliance** (C) of any structure can be described as the relative ease with which the structure distends. It can be defined as the inverse of **elastance** (e), where *elastance* is the tendency of a structure to return to its original form after being stretched or acted on by an outside force. Thus C = 1/e or e = 1/C. The following examples illustrate this principle. A balloon that is easy to inflate is said to be compliant (it demonstrates reduced elasticity), whereas a balloon that is difficult to inflate is considered not compliant (it has increased elasticity). In a similar way, consider the comparison of a golf ball and a tennis ball. The golf ball is more elastic than the tennis ball because it tends to retain its original form; a considerable amount of force must be applied to the golf ball to compress it. A tennis ball, on the other hand, can be compressed more easily than the golf ball, so it can be described as less elastic and more compliant.

In the clinical setting, compliance measurements are used to describe the elastic forces that oppose lung inflation. More specifically, the compliance of the respiratory system is determined by measuring the change  $(\Delta)$  of volume (V) that occurs when pressure (P) is applied to the system:  $C = \Delta V/\Delta P$ . Volume typically is measured in liters or milliliters and pressure in centimeters of water pressure. It is important to understand that the compliance of the respiratory system is the sum of the compliances of both the lung parenchyma and the surrounding thoracic structures. In a spontaneously breathing individual, the total respiratory system compliance is about 0.1 L/cm H<sub>2</sub>O (100 mL/cm H<sub>2</sub>O); however, it can vary considerably, depending on a person's posture, position, and whether he or she is actively inhaling or exhaling during the measurement. It can range from 0.05 to 0.17 L/cm H<sub>2</sub>O (50 to 170 mL/cm H<sub>2</sub>O). For intubated and mechanically ventilated patients with normal lungs and a normal chest wall, compliance

<sup>&</sup>lt;sup>b</sup>Applied pressure is +15 cm  $H_2O$ .



Normal compliance in spontaneously breathing patients: 0.05 to 0.17 L/cm H<sub>2</sub>O or 50 to 170 mL/cm H<sub>2</sub>O

Normal compliance in intubated patients: Males: 40 to 50 mL/cm H<sub>2</sub>O, up to 100 mL/cm H<sub>2</sub>O; Females: 35 to 45 mL/cm H<sub>2</sub>O, up to 100 mL/cm  $H_2O$ 



### **CRITICAL CARE CONCEPT 1.1**

### Calculate Pressure

Calculate the amount of pressure needed to attain a tidal volume of 0.5 L (500 mL) for a patient with a normal respiratory system compliance of 0.1 L/cm H<sub>2</sub>O.

varies from 40 to 50 mL/cm H<sub>2</sub>O in men and 35 to 45 mL/cm H<sub>2</sub>O in women to as high as 100 mL/cm H<sub>2</sub>O in either gender (Key Point 1.1).

Changes in the condition of the lungs or chest wall (or both) affect total respiratory system compliance and the pressure required to inflate the lungs. Diseases that reduce the compliance of the lungs or chest wall increase the pressure required to inflate the lungs. Acute respiratory distress syndrome and kyphoscoliosis are examples of pathological conditions associated with reductions in lung compliance and thoracic compliance, respectively. Conversely, emphysema is an example of a pulmonary condition in which pulmonary compliance is increased as a result of a loss of lung elasticity. With emphysema, less pressure is required to inflate the lungs.

Critical Care Concept 1.1 presents an exercise in which students can test their understanding of the compliance equation.

For patients receiving mechanical ventilation, compliance measurements are made during static or no-flow conditions (e.g., this is the airway pressure measured at end inspiration; it is designated as the plateau pressure). Thus these compliance measurements are referred to as static compliance or static effective compliance. The tidal volume used in this calculation is determined by measuring the patient's exhaled volume near the patient connector (Fig. 1.3). Box 1.3 shows the formula for calculating static compliance (C<sub>S</sub>) for a ventilated patient. Note that although this calculation technically includes the recoil of the lungs and thorax, thoracic compliance generally does not change significantly in a ventilated patient. (NOTE: It is important to understand that if a patient actively inhales or exhales during measurement of a plateau pressure, the resulting value will be inaccurate. Active breathing can be a particularly difficult issue when patients are tachypneic, such as when a patient is experiencing respiratory distress.)

### Resistance

Resistance is a measurement of the frictional forces that must be overcome during breathing. These frictional forces are the result of the anatomical structure of the airways and the tissue viscous resistance offered by the lungs and adjacent tissues and organs.

As the lungs and thorax move during ventilation, the movement and displacement of structures such as the lungs, abdominal

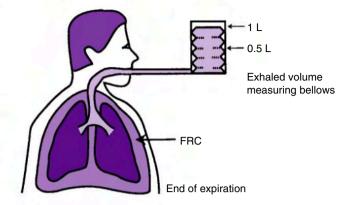


Fig. 1.3 A volume device (bellows) is used to illustrate the measurement of exhaled volume. Ventilators typically use a flow transducer to measure the exhaled tidal volume. The functional residual capacity is the amount of air that remains in the lungs after a normal exhalation.

### **BOX 1.3** Equation for Calculating Static Compliance

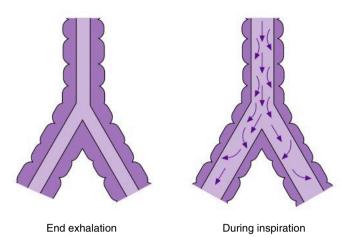
C<sub>S</sub> = (Exhaled tidal volume)/(Plateau pressure – EEP)  $C_S = V_T/(P_{plat} - EEP)^*$ 

\*EEP is the end-expiratory pressure, which some clinicians call the baseline pressure; it is the baseline from which the patient breathes. When positive end-expiratory pressure (PEEP) is administered, it is the EEP value used in this calculation.

organs, rib cage, and diaphragm create resistance to breathing. Tissue viscous resistance remains constant under most circumstances. For example, an obese patient or one with fibrosis has increased tissue resistance, but the tissue resistance usually does not change significantly when these patients are mechanically ventilated. On the other hand, if a patient develops ascites, or fluid accumulation in the peritoneal cavity, tissue resistance increases.

The resistance to airflow through the conductive airways (airway resistance) depends on the gas viscosity, the gas density, the length and diameter of the tube, and the flow rate of the gas through the tube, as defined by Poiseuille's law. During mechanical ventilation, viscosity, density, and tube or airway length remain fairly constant. In contrast, the diameter of the airway lumen can change considerably and affect the flow of the gas into and out of the lungs. The diameter of the airway lumen and the flow of gas into the lungs can decrease as a result of bronchospasm, increased secretions, mucosal edema, or kinks in the endotracheal tube. The rate at which gas flows into the lungs also can be controlled on most mechanical ventilators.

At the end of the expiratory cycle, before the ventilator cycles into inspiration, normally no flow of gas occurs; the alveolar and mouth pressures are equal. Because flow is absent, resistance to flow is also absent. When the ventilator cycles on and creates a positive pressure at the mouth, the gas attempts to move into the lower-pressure zones in the alveoli. However, this movement is impeded or even blocked by having to pass through the endotracheal tube and the upper conductive airways. Some molecules are slowed as they collide with the tube and the bronchial walls; in



**Fig. 1.4** Expansion of the airways during inspiration. (See the text for further explanation.)

doing this, they exert energy (pressure) against the passages, which causes the airways to expand (Fig. 1.4); as a result, some of the gas molecules (pressure) remain in the airway and do not reach the alveoli. In addition, as the gas molecules flow through the airway and the layers of gas flow over each other, resistance to flow, called *viscous resistance*, occurs.

The relationship of gas flow, pressure, and resistance in the airways is described by the equation for airway resistance,  $R_{aw}=P_{TA}/\text{flow}$ , where  $R_{aw}$  is airway resistance and  $P_{TA}$  is the pressure difference between the mouth and the alveolus, or the transairway pressure (Key Point 1.2). Flow is the gas flow measured during inspiration. Resistance is usually expressed in centimeters of water per liter per second (cm  $H_2O/[L/s])$ . In normal, conscious individuals with a gas flow of 0.5 L/s, resistance is about 0.6 to 2.4 cm  $H_2O/(L/s)$  (Box 1.4). The actual amount varies over the entire respiratory cycle. The variation occurs because flow during spontaneous ventilation is usually slower at the beginning and end of the cycle and faster in the middle portion of the cycle.  $^{1\ast}$ 

Airway resistance is increased when an artificial airway is inserted. The smaller internal diameter of the tube creates greater resistance to flow (resistance can be increased to 5 to 7 cm  $\rm H_2O/[L/s]$ ). As mentioned, pathological conditions can also increase airway resistance by decreasing the diameter of the airways. In conscious, unintubated patients with emphysema and asthma, resistance may range from 13 to 18 cm  $\rm H_2O/(L/s)$ . Still higher values can occur with other severe types of obstructive disorders.

Several challenges are associated with increased airway resistance. With greater resistance, a greater pressure drop occurs in the conducting airways and less pressure is available to expand the alveoli. As a consequence, a smaller volume of gas is available for gas exchange. The greater resistance also requires that more force be exerted to maintain adequate gas flow. To achieve this force, spontaneously breathing patients use the accessory muscles of inspiration. This generates more negative intrapleural pressures and a greater pressure gradient between the upper airway and the pleural space to achieve gas flow. The same occurs during mechanical ventilation; more pressure must be generated by the

**Key Point** 1.2  $R_{aw} = (PIP - P_{plat})/flow$  (where PIP is peak inspiratory pressure); or  $R_{aw} = P_{TA}/flow$ ; example:

$$R_{aw} \, = \, \left[ \frac{40 - 25 \; cm H_2 O}{1 (L/s)} \right] \, = \, 15 \; cm \frac{H_2 O}{(L/s)} \label{eq:Raw}$$

### **80X 1.4 Normal Resistance Values**

### **Unintubated Patient**

0.6 to 2.4 cm H<sub>2</sub>O/(L/s) at 0.5 L/s flow

### **Intubated Patient**

Approximately 6 cm  $H_2O/(L/s)$  or higher (airway resistance increases as endotracheal tube size decreases)

ventilator to try to "blow" the air into the patient's lungs through obstructed airways or a small endotracheal tube.

### **Measuring Airway Resistance**

Airway resistance pressure is not easily measured; however, the transairway pressure can be calculated:  $P_{TA}=PIP-P_{plat}.$  This allows determination of how much pressure is delivered to the airways and how much to alveoli. For example, if the peak pressure during a mechanical breath is 25 cm  $H_2O$  and the plateau pressure (i.e., pressure at end inspiration using a breath hold) is 20 cm  $H_2O$ , the pressure lost to the airways because of airway resistance is 25 cm  $H_2O-20$  cm  $H_2O=5$  cm  $H_2O$ . In fact, 5 cm  $H_2O$  is about the normal amount of pressure  $(P_{TA})$  lost to airway resistance  $(R_{aw})$  with a proper-sized endotracheal tube in place. In another example, if the peak pressure during a mechanical breath is 40 cm  $H_2O$  and the plateau pressure is 25 cm  $H_2O$ , the pressure lost to airway resistance is 40 cm  $H_2O-25$  cm  $H_2O=15$  cm  $H_2O$ . This value is high and indicates an increase in  $R_{aw}$  (see Box 1.4).

Many mechanical ventilators allow the therapist to choose a specific constant flow setting. Monitors are incorporated into the user interface to display peak airway pressures, plateau pressure, and the actual gas flow during inspiration. With this additional information, airway resistance can be calculated. For example, let us assume that the flow is set at 60 L/min, the peak inspiratory pressure (PIP) is 40 cm  $H_2O$ , and the  $P_{plat}$  is 25 cm  $H_2O$ . The  $P_{TA}$  is therefore 15 cm  $H_2O$ . To calculate airway resistance, flow is converted from liters per minute to liters per second (60 L/min = 60 L/60 s = 1 L/s). The values are then substituted into the equation for airway resistance,  $R_{aw} = (PIP - P_{plat})/flow$ :

$$R_{aw} \, = \, \left[ \frac{40 - 25 \; cm H_2 O}{1 (L/s)} \right] \, = \, 15 \; cm \frac{H_2 O}{(L/s)} \label{eq:Raw}$$

For an intubated patient, this is an example of elevated airway resistance. The elevated  $R_{\rm aw}$  may be caused by increased secretions, mucosal edema, bronchospasm, or an endotracheal tube that is too small

Ventilators with microprocessors can provide real-time calculations of airway resistance. It is important to recognize that where

The transairway pressure  $(P_{TA})$  in this equation is sometimes referred to as  $\triangle P$ , the difference between peak inspiratory pressure (PIP) and  $P_{plat}$ . (See the section on defining pressures in positive pressure ventilation.)



### Case Study 1.1

# Determine Static Compliance (C<sub>S</sub>) and Airway Resistance (Raw)

An intubated, 36-year-old woman diagnosed with pneumonia is being ventilated with a volume of 0.5 L (500 mL). The peak inspiratory pressure is 24 cm  $\rm H_2O$ ,  $\rm P_{plat}$  is 19 cm  $\rm H_2O$ , and baseline pressure is 0. The inspiratory gas flow is constant at 60 L/min (1 L/s).

What are the static compliance and airway resistance values? Are these normal values?

pressure and flow are measured can affect the airway resistance values. Measurements taken inside the ventilator may be less accurate than those obtained at the airway opening. For example, if a ventilator measures flow at the exhalation valve and pressure on the inspiratory side of the ventilator, these values incorporate the resistance to flow through the ventilator circuit and not just patient airway resistance. Clinicians must therefore know how the ventilator obtains measurements to fully understand the resistance calculation that is reported.

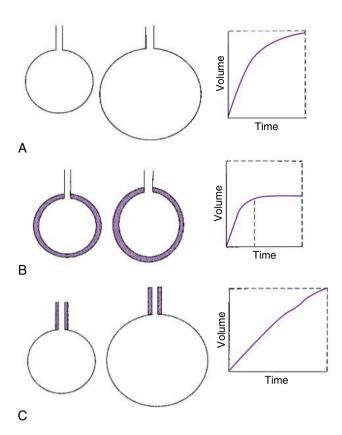
Case Study 1.1 provides an exercise to test your understanding of airway resistance and respiratory compliance measurements.

### **TIME CONSTANTS**

Regional differences in compliance and resistance exist throughout the lungs. That is, the compliance and resistance values of a terminal respiratory unit (acinus) may be considerably different from those of another unit. Thus the characteristics of the lung are heterogeneous, not homogeneous. Indeed, some lung units may have normal compliance and resistance characteristics, whereas others may demonstrate pathophysiological changes such as increased resistance, decreased compliance, or both.

Alterations in C and  $R_{\rm aw}$  affect how rapidly lung units fill and empty. Each small unit of the lung can be pictured as a small, inflatable balloon attached to a short drinking straw. The volume the balloon receives in relation to other small units depends on its compliance and resistance, assuming that other factors are equal (e.g., intrapleural pressures and the location of the units relative to different lung zones).

Fig. 1.5 provides a series of graphs illustrating the filling of the lung during a quiet breath. A lung unit with normal compliance and airway resistance will fill within a normal length of time and with a normal volume (see Fig. 1.5A). If the lung unit has normal resistance but is stiff (low compliance), it will fill rapidly (see Fig. 1.5B). For example, when a new toy balloon is first inflated, considerable effort is required to start the inflation (i.e., high pressure is required to overcome the critical opening pressure of the balloon to allow it to start filling). When the balloon inflates, it does so rapidly at first. It also deflates quickly. Notice, however, that if a given pressure is applied to a stiff lung unit and a normal unit for the same length of time, a much smaller volume will be delivered to the stiff lung unit (compliance equals volume divided by pressure) compared with the volume delivered to the normal unit.



**Fig. 1.5** (A) Filling of a normal lung unit. (B) A low-compliance unit, which fills quickly but with less air. (C) Increased resistance; the unit fills slowly. If inspiration were to end at the same time as in (A), the volume in (C) would be lower.

### **BOX 1.5** Calculation of Time Constant

Time constant = C  $\times$  R<sub>aw</sub> Time constant = 0.1 L/cm H<sub>2</sub>O  $\times$  1 cm H<sub>2</sub>O/(L/s) Time constant = 0.1 s

In a patient with a time constant of 0.1 s, 63% of inhalation (or exhalation) occurs in 0.1 s; that is, 63% of the volume is inhaled (or exhaled) in 0.1 s, and 37% of the volume remains to be exchanged.

Now consider a balloon (lung unit) that has normal compliance but the straw (airway) is narrow (high airway resistance) (see Fig. 1.5C). In this case the balloon (lung unit) fills slowly. The gas takes much longer to flow through the narrow passage and reach the balloon (acinus). If gas flow is applied for the same length of time as in a normal situation, the resulting volume is smaller.

The length of time lung units required to fill and empty can be determined. The product of compliance (C) and resistance ( $R_{aw}$ ) is called a **time constant**. For any value of C and  $R_{aw}$ , the time constant always equals the length of time (in seconds) required for the lungs to inflate or deflate to a certain amount (percentage) of their volume. Box 1.5 shows the calculation of one time constant for a lung unit with a compliance of 0.1 L/cm

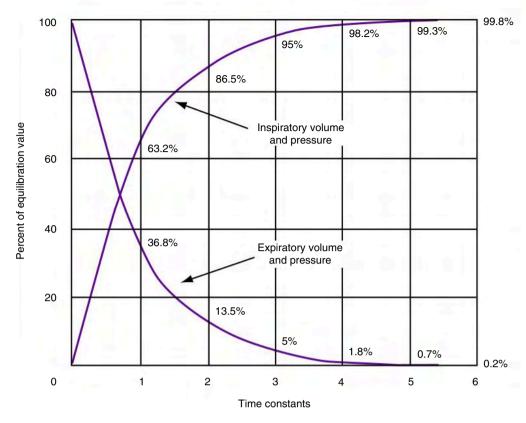


Fig. 1.6 The time constant (compliance × resistance) is a measure of how long the respiratory system takes to passively exhale (deflate) or inhale (inflate). (From Kacmarek RM, Stoller JK, Heuer AJ, editors: Egan's fundamentals of respiratory care, ed 12, St. Louis, MO, 2020, Elsevier.)

 $\rm H_2O$  and an airway resistance of 1 cm  $\rm H_2O/(L/s)$ . One time constant equals the amount of time it takes for 63% of the volume to be inhaled (or exhaled), two time constants represent that amount of time for about 86% of the volume to be inhaled (or exhaled), three time constants equal the time for about 95% to be inhaled (or exhaled), and four time constants are the time required for 98% of the volume to be inhaled (or exhaled) (Fig. 1.6). 4-6 In the example in Box 1.5, with a time constant of 0.1 s, 98% of the volume fills (or empties) the lungs in four time constants, or 0.4 s.

After five time constants, the lung is considered to contain 100% of tidal volume to be inhaled or 100% of tidal volume has been exhaled. In the example in Box 1.5, five time constants would equal  $5 \times 0.1$  s, or 0.5 s. Thus in half a second, a normal lung unit, as described here, would be fully expanded or deflated to its end-expiratory volume (Key Point 1.3).

Calculation of time constants is important when setting the ventilator's inspiratory time and expiratory time. An inspiratory time less than three time constants may result in incomplete delivery of the tidal volume. Prolonging the inspiratory time allows even distribution of ventilation and adequate delivery of tidal volume. Five time constants should be considered for the inspiratory time, particularly in pressure ventilation, to ensure adequate volume delivery (see Chapter 2 for more information on pressure ventilation). It is important to recognize, however, that if the inspiratory time is too long, the respiratory rate may be too low to achieve effective minute ventilation.

An expiratory time of fewer than three time constants may lead to incomplete emptying of the lungs. This can increase the **Key Point** 1.3 Time constants approximate the amount of time required to fill or empty a lung unit.

functional residual capacity and cause trapping of air in the lungs. Some clinicians think that using the 95% to 98% volume-emptying level (three or four time constants) is adequate for exhalation.<sup>5,6</sup> Exact time settings require careful observation of the patient and measurement of end-expiratory pressure to determine which time is better tolerated.

In summary, lung units can be described as fast or slow. Fast lung units have short time constants and take less time to fill and empty. Short time constants are associated with normal or low airway resistance and decreased compliance, such as occurs in a patient with interstitial fibrosis. It is important to recognize, however, that these lung units will typically require increased pressure to achieve a normal volume. In contrast, slow lung units have long time constants, which require more time to fill and empty compared with a normal or fast lung unit. Slow lung units have increased resistance or increased compliance, or both, and are typically found in patients with pulmonary emphysema.

It must be kept in mind that the lung is rarely uniform across ventilating units. Some units fill and empty quickly, whereas others do so more slowly. Clinically, compliance and airway resistance measurements reflect a patient's overall lung function, and clinicians must recognize this fact when using these data to guide treatment decisions.

### TYPES OF VENTILATORS AND TERMS USED IN **MECHANICAL VENTILATION**

Various types of mechanical ventilators are used clinically. The following section provides a brief description of the terms commonly applied to mechanical ventilation.

### **TYPES OF MECHANICAL VENTILATION**

Three basic methods have been developed to mimic or replace the normal mechanisms of breathing: negative pressure ventilation, positive pressure ventilation, and high-frequency ventilation.

### **Negative Pressure Ventilation**

**CHAPTER 1** 

Negative pressure ventilation (NPV) attempts to mimic the function of the respiratory muscles to allow breathing through normal physiological mechanisms. A good example of negative pressure ventilators is the tank ventilator, or "iron lung." With this device, the patient's head and neck are exposed to ambient pressure while the thorax and the rest of the body are enclosed in an airtight container that is subjected to negative pressure (i.e., pressure less than atmospheric pressure). Negative pressure generated around the thoracic area is transmitted across the chest wall, into the intrapleural space, and finally into the intraalveolar space.

With negative pressure ventilators, as the intrapleural space becomes negative, the space inside the alveoli becomes increasingly negative in relation to the pressure at the airway opening (atmospheric pressure). This pressure gradient results in the movement of air into the lungs. In this way, negative pressure ventilators resemble normal lung mechanics. Expiration occurs when the negative pressure around the chest wall is removed. The normal elastic recoil of the lungs and chest wall causes air to flow out of the lungs passively (Fig. 1.7).

Negative pressure ventilators can provide several advantages. The upper airway can be maintained without the use of an endotracheal tube or tracheostomy. Patients receiving negative pressure ventilation can talk and eat while being ventilated. Negative pressure ventilation has fewer physiological disadvantages in patients with normal cardiovascular function than does positive pressure ventilation.<sup>7-10</sup> In hypovolemic patients, however, a normal cardiovascular response is not always present. As a result, patients can have significant pooling of blood in the abdomen and reduced venous return to the heart.<sup>9,10</sup> In addition, difficulty gaining access to the patient can complicate care activities (e.g., bathing and turning).

The use of negative pressure ventilators declined considerably in the early 1980s, and currently they are rarely used in hospitals. Other methods of creating negative pressure (e.g., chest cuirass, Poncho wrap, Porta-Lung) have been used in home care to treat patients with chronic respiratory failure associated with neuromuscular diseases (e.g., polio, amyotrophic lateral sclerosis).8-13 More recently, these devices have been replaced with noninvasive positive pressure ventilators (NIVs) that use a mask, nasal device, or tracheostomy tube as a patient interface. Chapters 19 and 21 provide additional information on the use of NIV and NPV.

### **Positive Pressure Ventilation**

Positive pressure ventilation (PPV) occurs when a mechanical ventilator is used to deliver air into the patient's lungs by way of an endotracheal tube or positive pressure mask. For example, if

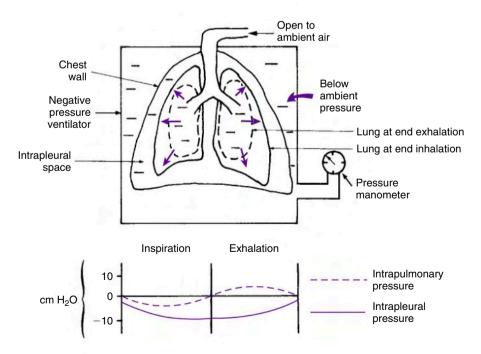


Fig. 1.7 Negative pressure ventilation and the resulting lung mechanics and pressure waves (approximate values). During inspiration, intrapleural pressure drops from about —5 to -10 cm H<sub>2</sub>O and alveolar (intrapulmonary) pressure declines from 0 to -5 cm H<sub>2</sub>O; as a result, air flows into the lungs. The alveolar pressure returns to zero as the lungs fill. Flow stops when pressure between the mouth and the lungs is equal. During exhalation, intrapleural pressure increases from about -10 to -5 cm H<sub>2</sub>O and alveolar (intrapulmonary) pressure increases from 0 to about +5 cm H<sub>2</sub>0 as the chest wall and lung tissue recoil to their normal resting position; as a result, air flows out of the lungs. The alveolar pressure returns to zero, and flow stops.