EIGHTH EDITION

PILBEAM'S MECHANICAL VENTILATION

PHYSIOLOGICAL AND CLINICAL APPLICATIONS

J.M. CAIRO

ELSEVIER

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!

ELSEVIER

You can now purchase Elsevier products on Evolve!
Go to evolve.elsevier.com/shop to search and browse for products.

2019v1.0

PILBEAM'S

MECHANICAL VENTILATION

PHYSIOLOGICAL AND CLINICAL APPLICATIONS

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

Elsevier
3251 Riverport Lane
St. Louis, Missouri 63043

PILBEAM'S MECHANICAL VENTILATION: PHYSIOLOGICAL AND CLINICAL APPLICATIONS, EIGHTH EDITION ISBN: 978-0-323-87164-8

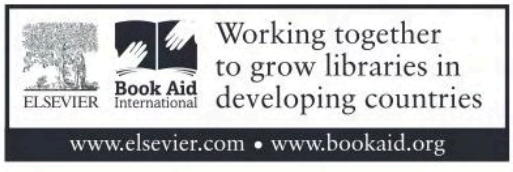
Notices

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.

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



To Rhonda and Allyson

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

The 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

A 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

This page intentionally left blank

CHAPTER 1

Basic Terms and Concepts of Mechanical Ventilation

OUTLINE

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.*
4. Provide the value for intraalveolar pressure throughout inspiration and expiration during normal, quiet breathing.
5. Write the formulas for calculating compliance and resistance.
6. Explain how changes in lung compliance affect the peak pressure measured during inspiration with a mechanical ventilator.
7. 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.
9. Using a figure showing abnormal compliance or airway resistance, determine which lung unit will fill more quickly or with a greater volume.
10. 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.
12. Briefly discuss the principle of operation of negative pressure, positive pressure, and high-frequency mechanical ventilators.
13. Define peak inspiratory pressure, baseline pressure, positive end-expiratory 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 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.

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

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_2O) 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_2O (1 mm Hg = 1.36 cm H_2O) at sea level, atmospheric pressure is designated as 0 cm H_2O. For example, when airway pressure increases by +20 cm H_2O during a positive pressure breath, the pressure actually increases from 760 mm Hg to 780 mm Hg (1034 to 1054 cm H_2O). Other units of measure that are becoming more widely used for gas pressures, such as arterial oxygen pressure (P_aO_2) and arterial carbon dioxide pressure (P_aCO_2), 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

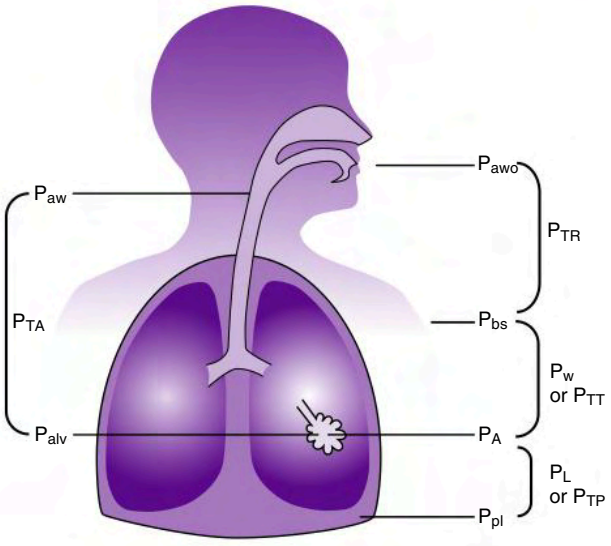
Airway opening pressure (P_{awo}) is most often called **mouth pressure** (P_M) or **airway pressure** (P_{aw}) (Fig. 1.1). Other terms that are often used to describe the airway opening pressure include **upper-airway pressure**, **mask pressure**, and **proximal airway pressure**.[1] Unless pressure is applied at the airway opening, P_{awo} is zero or atmospheric pressure.

A similar measurement is the pressure at the body surface (P_{bs}) 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_{awo} - Mouth or airway opening pressure
P_{alv} - Alveolar pressure
P_{pl} - Intrapleural pressure
P_{bs} - Body surface pressure
P_{aw} - Airway pressure (= P_{awo})
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_{TR} = Transrespiratory pressure ($P_{awo} - P_{bs}$)

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**.[2-4] (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	
C	Compliance	
R	Resistance	
R_{aw}	Airway resistance	
P_M	Pressure at the mouth (same as P_{awo})	
P_{aw}	Airway pressure (usually upper airway)	
P_{awo}	Pressure at the airway opening; mouth pressure; mask pressure	
P_{bs}	Pressure at the body surface	
P_{alv}	Alveolar pressure (also P_A)	
P_{pl}	Intrapleural pressure	
C_{st}	Static compliance	
C_{dyn}	Dynamic compliance	
Pressure gradients		
Transairway pressure (P_{TA})	Airway pressure − alveolar pressure	$P_{TA} = P_{aw} - P_{alv}$
Transthoracic pressure (P_W)	Alveolar pressure − body surface pressure	P_W (or P_{TT}) $= P_{alv} - P_{bs}$
Transpulmonary pressure (P_L)	Alveolar pressure − pleural pressure (also defined as the *transalveolar pressure*)	P_L (or P_{TP}) $= P_{alv} - P_{pl}$
Transrespiratory pressure (P_{TR})	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 cm H_2O. Notice that the pressure inside the alveolus during exhalation increases and becomes slightly positive (+5 cm H_2O). 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.*

*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.[4]

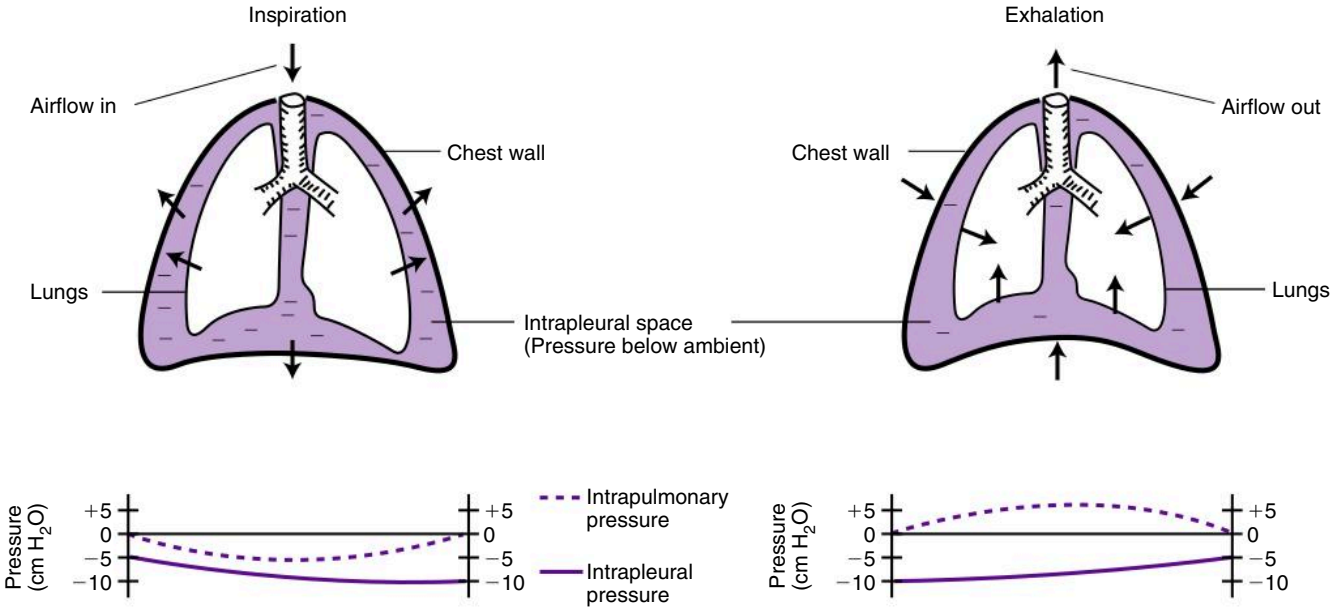


Fig. 1.2 The mechanics of spontaneous ventilation and the resulting pressure waves (approximately normal values). During inspiration, intrapleural pressure (P_{pl}) decreases to −10 cm H_2O. During exhalation, P_{pl} increases from −10 to −5 cm H_2O. (See the text for further description.)

TABLE 1.2 Changes in transpulmonary pressure[a] under varying conditions.

Pressure	End Expiration	End Inspiration
Passive spontaneous ventilation		
Intraalveolar (intrapulmonary)	0 cm H_2O	0 cm H_2O
Intrapleural	−5 cm H_2O	−10 cm H_2O
Transpulmonary	$P_L = 0 - (-5) = +5$ cm H_2O	$P_L = 0 - (-10) = 10$ cm H_2O
Negative pressure ventilation		
Intraalveolar (intrapulmonary)	0 cm H_2O	0 cm H_2O
Intrapleural	−5 cm H_2O	−10 cm H_2O
Transpulmonary	$P_L = 0 - (-5) = +5$ cm H_2O	$P_L = 0 - (-10) = 10$ cm H_2O
Positive pressure ventilation		
Intraalveolar (intrapulmonary)	0 cm H_2O	9-12 cm H_2O[b]
Intrapleural	−5 cm H_2O	2-5 cm H_2O[b]
Transpulmonary	$P_L = 0 - (-5) = +5$ cm H_2O	$P_L = 10 - (2) = +8$ cm H_2O[b]

[a] $P_L = P_{alv} - P_{pl}$.
[b] Applied pressure is +15 cm H_2O.

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 (Δ) 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_2O (100 mL/cm H_2O); 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_2O (50 to 170 mL/cm H_2O). For intubated and mechanically ventilated patients with normal lungs and a normal chest wall, compliance

Key Point 1.1

Normal compliance in spontaneously breathing patients: 0.05 to 0.17 L/cm H_2O or 50 to 170 mL/cm H_2O

Normal compliance in intubated patients: Males: 40 to 50 mL/cm H_2O, up to 100 mL/cm H_2O; Females: 35 to 45 mL/cm H_2O, 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_2O.

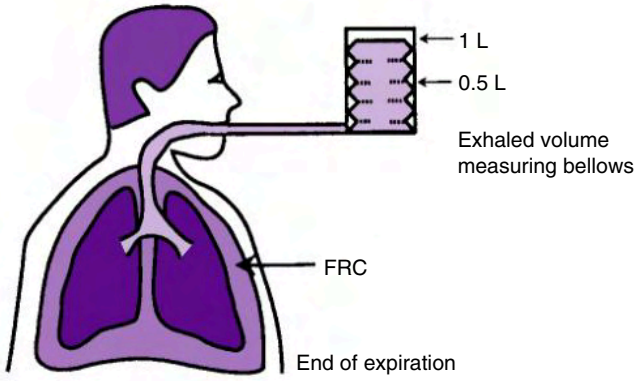


Fig. 1.3 A volume device (bellows) is used to illustrate the measurement of exhaled volume. Ventilators typically use a flow transducer to measure the exhaled tidal volume. The functional residual capacity is the amount of air that remains in the lungs after a normal exhalation.

BOX 1.3 Equation for Calculating Static Compliance

C_S = (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.

varies from 40 to 50 mL/cm H_2O in men and 35 to 45 mL/cm H_2O in women to as high as 100 mL/cm H_2O 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_S) 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 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

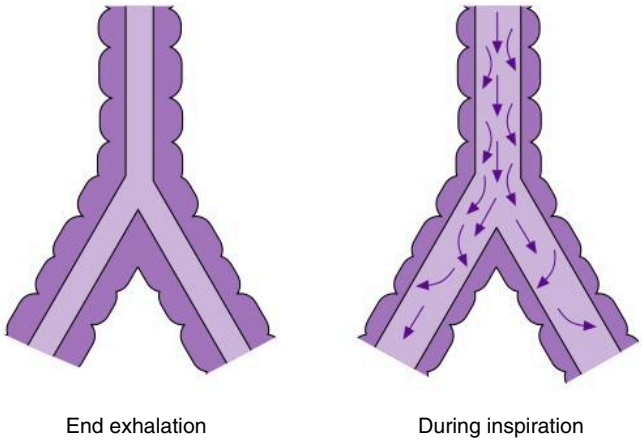


Fig. 1.4 Expansion of the airways during inspiration. (See the text for further explanation.)

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*]

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

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

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

$$R_{aw} = \left[\frac{40 - 25\ \text{cm}H_2O}{1(\text{L/s})}\right] = 15\ \text{cm}\frac{H_2O}{(\text{L/s})}$$

BOX 1.4 Normal Resistance Values

Unintubated Patient
0.6 to 2.4 cm H_2O/(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)

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, $R_{aw} = (PIP - P_{plat})/\text{flow}$:

$$R_{aw} = \left[\frac{40 - 25\ \text{cm}H_2O}{1(\text{L/s})}\right] = 15\ \text{cm}\frac{H_2O}{(\text{L/s})}$$

For an intubated patient, this is an example of elevated airway resistance. The elevated R_{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

Case Study 1.1

Determine Static Compliance (C_S) 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 H_2O, P_{plat} is 19 cm 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_{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.

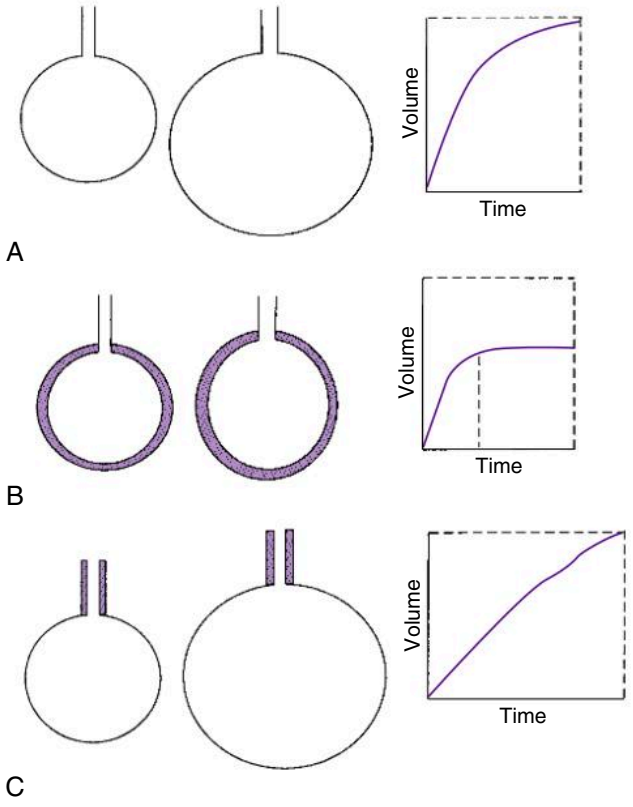


Fig. 1.5 (A) Filling of a normal lung unit. (B) A low-compliance unit, which fills quickly but with less air. (C) Increased resistance; the unit fills slowly. If inspiration were to end at the same time as in (A), the volume in (C) would be lower.

BOX 1.5 Calculation of Time Constant

Time constant = C × R_{aw}
Time constant = 0.1 L/cm H_2O × 1 cm H_2O/(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

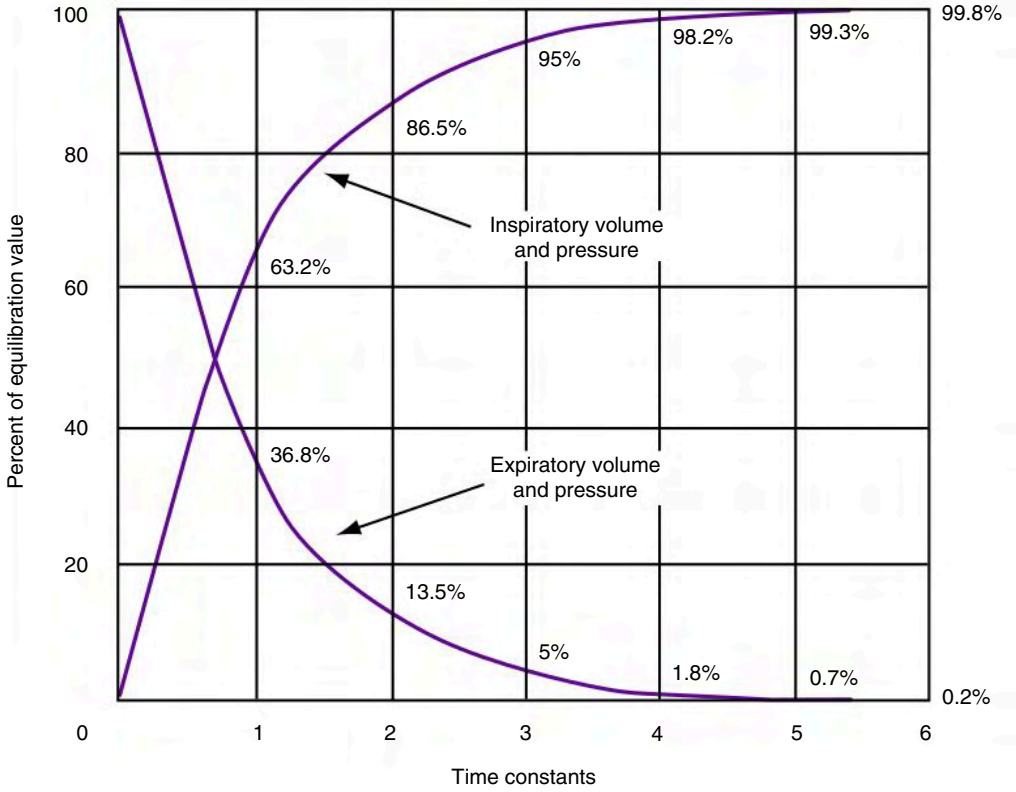


Fig. 1.6 The time constant (compliance × resistance) is a measure of how long the respiratory system takes to passively exhale (deflate) or inhale (inflate). (From Kacmarek RM, Stoller JK, Heuer AJ, editors: *Egan's fundamentals of respiratory care,* ed 12, St. Louis, MO, 2020, Elsevier.)

H_2O and an airway resistance of 1 cm 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 × 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 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.[5,6] Exact time settings require careful observation of the patient and measurement of end-expiratory pressure to determine which time is better tolerated.

***Key Point* 1.3** Time constants approximate the amount of time required to fill or empty a lung unit.

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

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.[7-10] 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.[9,10] 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

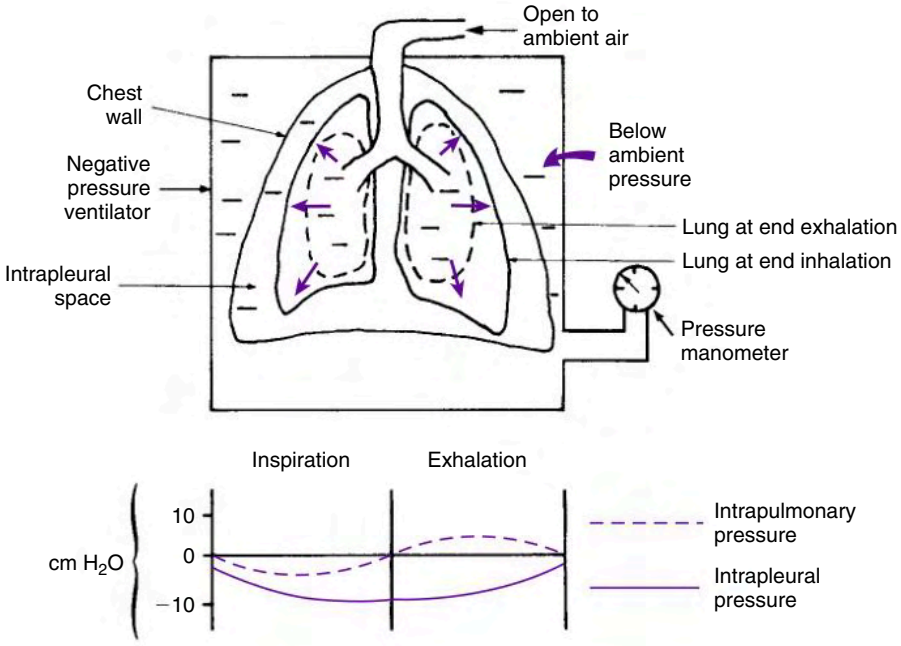


Fig. 1.7 Negative pressure ventilation and the resulting lung mechanics and pressure waves (approximate values). During inspiration, intrapleural pressure drops from about −5 to −10 cm H_2O and alveolar (intrapulmonary) pressure declines from 0 to −5 cm H_2O; as a result, air flows into the lungs. The 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_2O and alveolar (intrapulmonary) pressure increases from 0 to about +5 cm H_2O 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.