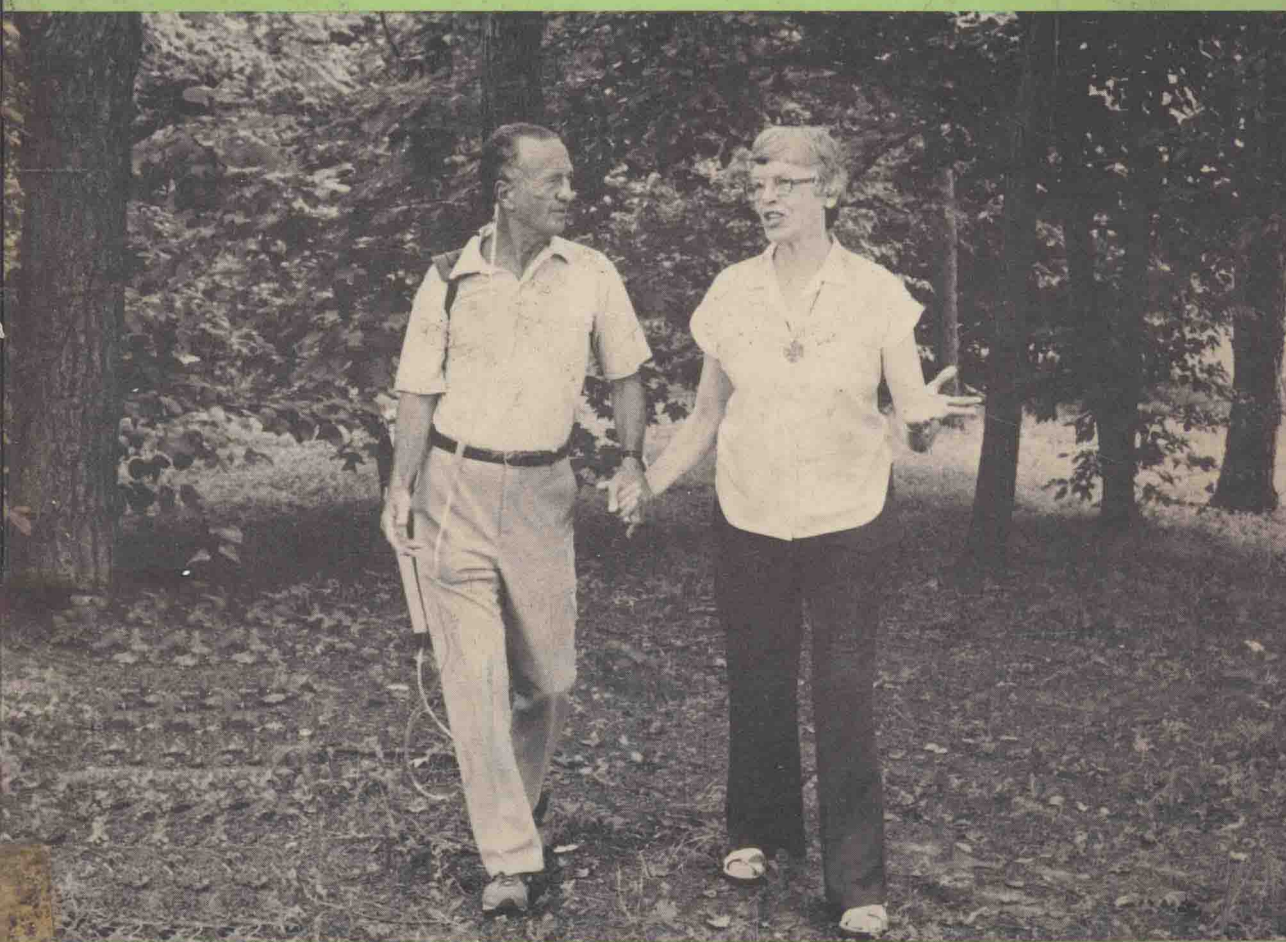
APPLETON PATIENT EDUCATION SERIES

Pulmonary Care: A Guide for Patient Education



Margaret Devany Burns

Pulmonary Care: A Guide for Patient Education

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Introduction to the Patient Education Series

A great deal of development has occurred in patient education in the last decade. Programs are much more widespread; valuable experience has been gained in applying general educational theory and practice in many areas of clinical practice.

It seemed propitious to reflect that development in a series of books aimed at sharing the emerging body of knowledge gained by practitioners in specialized fields. The authors are pioneers in developing practical tools to evaluate and document patient learning. The emphasis is on clinical usability, focusing on patient assessment for desired behavior, educational interventions, evaluation and outcome criteria, and recording. Also in the series is a book on issues and concepts in patient education with an abbreviated review of teaching, learning and motivation theory, and a focus on issues I perceive to be important in the development of the field. A second book focuses on examples of strategies for institutional change and of systems for delivery of patient education. Strategies of this kind have been very important in dealing with systems that have been resistant and unresponsive to incorporation of patient education into practice.

Producing the series has been educative for those involved. By history and tradition, patient education has been better developed conceptually and more completely accepted and incorporated into some clinical areas than into others. One realizes that this has sometimes occurred because of patterns of delivery of services and because of the predominant medical approaches to problems, and not necessarily on the basis of patient need and ability to profit from education. I hope that the series helps to identify areas needing development and that the imaginative clinicians with whom we are blessed will find the ideas of use in further maturation of the field.

It is important not to underestimate the significance of the patient education movement. Along with other related practices and philosophies, it reflects a very basic change in the standard of care and in the relationship between providers and clients. Like all basic changes, its processes for development are sometimes agonizingly slow and are conglomerations of rational and political processes, and its structures are often loosely coupled and disconnected. However, the movement does reflect the commitment of many lay people and professionals who believe patient education yields more humane care and is also effective as a treatment modality.

A word must be said about the contribution to the series of Leslie Boyer at Appleton-Century-Crofts. The series was her idea, and she was a major partner in its development at every stage.

Barbara Klug Redman
Washington, D.C.

Preface

The underlying theme of this book is the need for a holistic, individualized approach to the educational plan for each patient. The information in this book represents a baseline of knowledge for the health care professional. Yet even as this book is being published, studies in health care facilities across the country are researching new and better ways to prevent or ameliorate difficulties in persons with respiratory disease. These studies are important because they serve as models for determining appropriate evaluation criteria. It is the responsibility of all health care personnel to evaluate and reevaluate the effectiveness of the individual treatment plan. As an extension of that evaluation all health care workers need to familiarize themselves with ongoing research and readjust their own treatment approaches accordingly. Clinical experience and research findings form the basis of this book with theoretical approaches from education, nursing, and many other disciplines guiding the direction for the individual educational plan.

This book draws extensively from nursing experience. Each chapter is authored by at least one person with considerable clinical experience in the area addressed. I am indebted to the contributors in this book for this sharing of experience and insights. I am also indebted to the many other persons who supported the writing and editing of this book.

As graduate students Lisbeth Dryer and myself became interested in the area of evaluation of pulmonary rehabilitative efforts. Lisbeth has served not only as co-author of three chapters with me in this book, but she has also served as willing mentor, painstakingly reading and evaluating every chapter and sharing her insights with me throughout the preparation of this book.

When I was several months into the preparation of this book our family moved to another state. I was most fortunate to meet a pulmonary physician in my new home town who willingly and at times on very short notice, proofread each chapter. To this physician, Dr. Charles Bruton of Oak Ridge, Tennessee, I owe a special thanks. His suggestions regarding Chapters 1 and 6 were particularly helpful.

I believe that the mobilization chapter, Chapter 2, contains one of the most important messages in this book. Mobility is a key to wellness in both health and disease. I wish to express my appreciation not only to my co-author, Dr. James Dooley, but also to the many persons who reviewed this chapter as well as Chapter 1: thanks to Dr. Maureen Groër of the University of Tennessee College of Nursing and Health, to Dr. Dan Miles of the Physiology Department at Wright State University, and to Joan Lynch who also reviewed and thoughtfully commented on other areas of this manuscript.

Chapter 3 was also reviewed by a good friend, Irene Boyd, who lived for many years with the emotional trauma of supporting a much loved family member who eventually died of a chronic respiratory illness. I owe Irene particular thanks for her sharing of those difficult experiences.

Chapter 4 was the first chapter that was written in the form of a script for the patient and initial drafts were reviewed by persons without specific knowledge or experience with respiratory disease. I particularly wish to thank Marjorie Swann of Beavercreek, Ohio, and members of Karen Cardin's Writing for Publication Class at the University of Tennessee for their comments.

Chapter 5 is based heavily on my clinical

experience with patients afflicted with chronic lung disease. I am grateful to each of those individuals and their families for sharing their experiences with me. This chapter was a particular labor of love not only because of the many hours spent collaborating with Lisbeth Dryer, but also because it represents the fruits of my clinical experiences.

I wish to thank the staff of the pulmonary rehabilitation team at Miami Valley Hospital, Dayton, Ohio, and in particular I wish to acknowledge the assistance and mentorship during my position there of Dr. James Graham from whom I learned much about the needs of respiratory patients. Many of the assessment tools in Chapter 5 were developed in collaboration with the team and with Dr. Graham. Other clinical experiences which served as the core for developing Chapter 5 came from working with baccalaureate students, patients and staff on the respiratory unit at the Veterans Administration Hospital in Dayton, Ohio. Lisbeth Dryer, my co-author, wishes to acknowledge the support that she received from four individuals: Dr. Richard Honicky, a pulmonary physician and Professor of Medicine at Michigan State University, and Patricia Peek, Assistant Professor of Nursing at Michigan State University, for their review of the cystic fibrosis section; Robert Wheeler, R.R.T., for his review of home oxygen usage; and Phoebe Dotterer, coordinator of the Pulmonary Rehabilitation Program at Lansing General Hospital, for her consultation on the chronic bronchitis and emphysema sections of Chapter 5. Last but not least, special thanks to Dr. Thomas Dryer for his consultation and support.

Chapter 9 was collaboratively written with Margaret Pierce, who has extensive clinical experience with patients who have lung cancer. She wishes to acknowledge the following professionals who proofread and offered constructive criticism of the manuscript as it was developed: Lois Doane, RN, MSN, Oncology Coordinator at University of Tennessee Hospital, Dr. Johnie Mozingo, Associate Professor at the University of Tennessee College of Nursing, and Dr. Glen Farr of the University of Tennessee School of Pharmacy.

No multi-authored work of this dimension can be put together without the help of an efficient secretarial staff. My thanks and appreciation to Lois Walker for her typing and suggestions in the early phases of this book and for her most efficient work with several of the collaborating authors after my move to Tennessee; also to Judy Wyrick for her typing, proofreading and moral support; and to Mary Jane Johnson an R.N. with a word processing business who helped expedite Chapter 6 and the indexing of this book in record time. One of the most tedious tasks in writing a book is the proofreading of drafts of the manuscript. I particularly wish to thank my good friend Betty Culver for the time that she spent assisting with the proofreading of this manuscript. In terms of library support I give thanks to the educational and the library staff at Oak Ridge Hospital and to Martha Childs, Librarian, at the Preston Medical Library at the University of Tennessee Memorial Hospital and Research Center.

And finally, sincere thanks to Richard Lampert, Editor-in-Chief at ACC, Dr. Barbara Redman, series editor, and to Anne Friedman, my production editor, for their encouragement and advice during the preparation of this book.

MARGARET DEVANY BURNS
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Pulmonary Care

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PART I
Knowledge Base Review for the Nurse

Chapter 1
ANATOMY AND PHYSIOLOGY OF RESPIRATION

Margaret Devany Burns and James Dooley

INTRODUCTION

In order to understand disease processes, the nurse needs to have an understanding of the normal function of the organ system that is impaired by the disease. By understanding the normal function and relevant pathophysiology, the nurse will be able to provide an integrated yet simplified approach to concepts which the patient needs to learn in order to participate actively in his or her own care.

The main function of the lungs is ventilation. Ventilation is the conduction of oxygen into and carbon dioxide out of the lungs. Oxygen is required continuously by every living cell in the body and is delivered to the bloodstream by the lungs. Carbon dioxide, a waste product of cellular metabolism, is excreted by the cells, and eventually by the lungs. In order to maintain a healthy environment within the body, proper levels of these two gases, oxygen and carbon dioxide, must be maintained. The respiratory system is one of several body systems which regulate these levels. In addition to transporting gases the respiratory system assists in the regulation of the body's level of acidity. The body can only live and function within a certain narrow pH range. The body handles excess acid on a short-term basis by converting the acid to carbon dioxide in the bloodstream which the lungs then excrete to a level compatible with homeostasis.[4,17] The first step in understanding pathologic functioning of the respiratory system is to review the normal anatomy and physiology of respiration.

ANATOMY AND PHYSIOLOGY OF THE RESPIRATORY SYSTEM

The respiratory system is made up of the pulmonary system and its associated skeletal, muscular, vascular, and nervous system contingents. In this section the anatomy and physiology of the respiratory system are reviewed with respect to structure and function of the subsystems involved.

The pulmonary system is composed of upper and lower airways which lead from the external environment to the alveoli where gas exchange takes place. The lungs are sponge-like structures which are covered by a paper-thin membrane called the visceral pleura. The lungs are divided by the trachea into the right and left lung. The right lung has three divisions: an upper, middle, and lower lobe. The left lung has two divisions: an upper and lower lobe. The presence of only two lobes on the left side may relate to the fact that space is taken

up by the heart in the middle and left chest cavity. The airways have two major subdivisions: 1. the upper airway, and 2. the lower airway. The larynx, a transitional airway, is considered anatomically as a lower airway structure. Its functioning is consistent with that of the upper airway and it will be discussed as an upper airway structure (Figure 1).

Upper Airway

The upper airway consists of the nose, nasopharynx oropharynx, mouth, laryngopharynx, and the larynx. These passageways serve as conduits for transporting gases back and forth from the environment to the lower airways. They also condition the air by filtering, humidifying, and regulating the temperature of the air as it descends into the lower airways. The nasal cavity in particular contains specialized structures which maximize the functions of filtration, humidification, and temperature regulation. It is for this reason that inspiration of air through the nose rather than through the mouth is preferred. During rest, ventilation can easily be accomplished through the nose unless some pathology of the nasal passageway is present. Increased ventilation, as occurs during exercise, results in breathing through the larger and less advantageous oral cavity, although the work of breathing is reduced.

The larynx is an important transitional organ between the upper and lower airways. The opening of the larynx, called the glottis, is covered by the epiglottis, a flexible elastic cartilage. This covering prevents food or other foreign substances from getting into the lungs during the act of swallowing. The larynx also participates in coughing, which is one of the respiratory system's essential defense mechanisms.

Lower Airway

The lower airway includes the trachea, bronchi, bronchioles, and lung parenchyma. In order to better understand the function of the lower airway let us divide these airways into two parts. The proximal part of the lower airway is referred to as the conductive zone. The conductive zone is composed of the trachea and the first sixteen divisions of the tracheobronchial tree. A primary function of this zone is to conduct air between the upper airway structures and the lung parenchyma. The structures found in the conducting zone do not participate in gas exchange and are referred to as anatomical dead space. The concept of dead space is important because if one breathes only enough air to fill the conducting zones then gas exchange will be inhibited. A person who is breathing in a shallow manner due to drug overdose, for example, will not bring in sufficient air to fill the air sacs, therefore acute arterial hypoxemia may ensue. Dead space can be artificially increased as in the case of a person using a snorkel tube. The dead space is increased by the length of the tube. Using a snorkel tube to swim underwater, a person will need to take in a larger volume of air through the tube in order to fill his or her air sacs to a degree equivalent to that amount which he or she would take in under similar conditions out of water and without the snorkel tube.[6] In addition to the anatomic dead space of the conducting airways, there is a potential *physiologic dead space,* that portion of the lung parenchyma that does not exchange gases. Normally, anatomic dead space is essentially equal to physiologic dead space, that is about 150 ml. In the diseased lung, however, the physiologic dead space may be as much as ten times the anatomic dead space (1 to 2 liters).[8]

Airway Structures Related to Function. From an anatomic view, several structural characteristics of the tracheobronchial tree that have an important bearing on lung function require review. These characteristics are the *mucociliary escalator, bronchial smooth muscle activity, airflow resistance in the airways, the anatomic placement of the right and left mainstem bronchus, and airway support.* The trachea lies directly anterior to the esophagus and extends to a length of about 12 cm to the area where it divides into the right and left bronchi. The walls of the trachea, as well as the bronchi, are composed of an epithelial lining which contains numerous mucus-secreting glands. The epithelial mucosa is coated with cilia which extend from

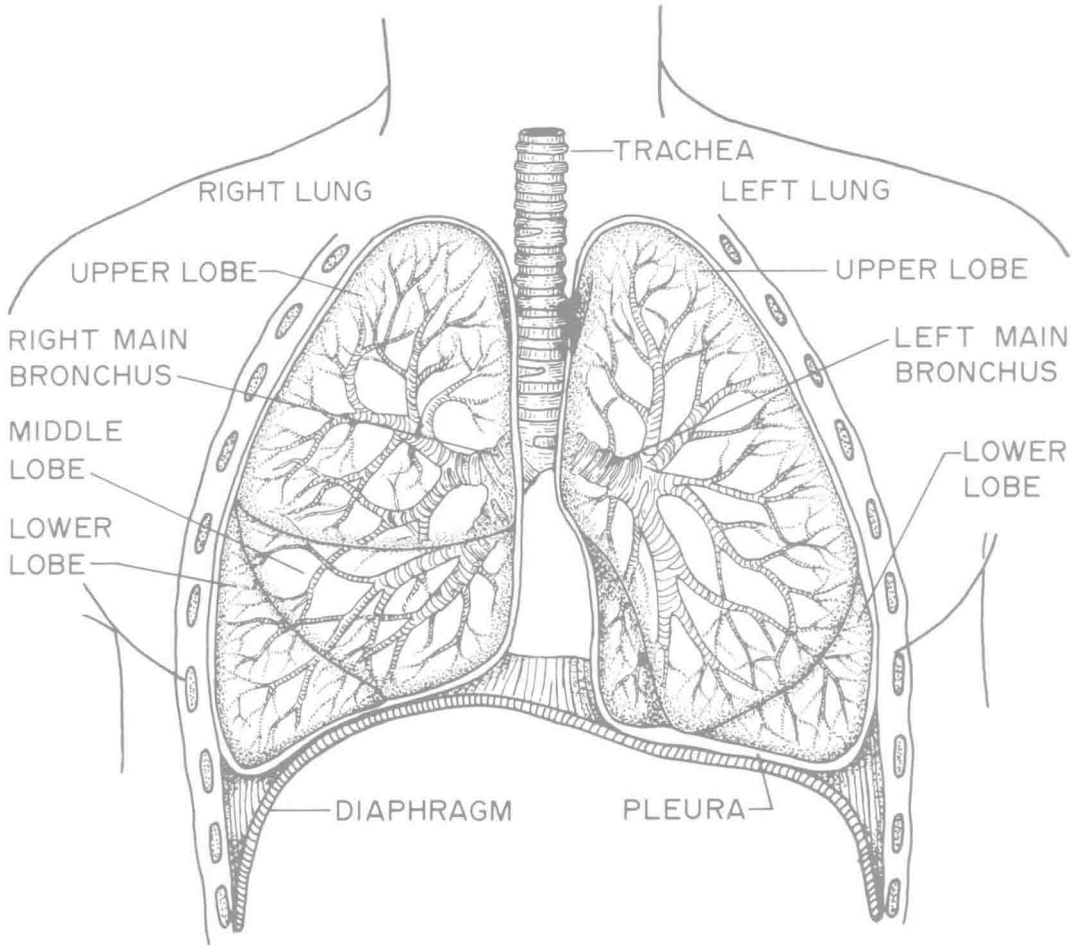

Figure 1. Anatomy of the lung

the larynx to, *but not including*, the respiratory bronchioles. Cilia are hairlike projections which move in a distinctive wave pattern, beating mucus and the inhaled foreign matter that becomes imbeded in the mucus, up to the airways where it is swallowed or expectorated. These structures, the cilia and the mucus-secreting glands, make up the major component parts of the mucociliary escalator.

An important concept is *the mechanism of action of bronchial smooth muscle.* It is this smooth muscle that is responsible for the dilation and constriction of the airways. This is a particularly important concept as it is the inhibition of the contraction of these muscles by biochemical manipulation that forms the basis for bronchodilator drug action. The smooth muscle of the air passageways are innervated by sympathetic and parasympathetic nerve supply. The sympathetic nervous system possesses adrenergic activity involving alpha and beta receptors while the parasympathetic nervous system functions by cholinergic activity. Beta receptors are divided into two subgroups, beta-1 and beta-2. Beta-2 receptors are believed to be present in glands, smooth muscle and mucosal vessels of the bronchial tree. Adrenergic stimulation of beta-2 receptors results in bronchial smooth muscle dilation. Stimulation of cholinergic activity results in

bronchial smooth muscle constriction. With both systems active the bronchi have an intermediate size. The beta-2 receptor is hypothesized to be an enzyme which after adrenergic stimulation catalyzes the reaction converting adenosine triphosphate (ATP) to cyclic 3′–5′ adenosine monophosphate (cyclic AMP). Cyclic AMP produces bronchodilation. The major pharmocologic means of inducing bronchodilation is to increase cyclic AMP or inhibit its breakdown. Another drug action to achieve bronchodilation is to inhibit parasympathetic cholinergic activity.[3,15]

Another concept which requires review is that of the concept of *resistance to air flow*. Resistance to airflow (R) in general, is determined by the radius (r) and length (L) of the tube, the characteristics of the airflow (turbulent versus laminar), and the viscosity (N) of the gas flowing through the system. The relationship of the variables involved in resistance to laminar airflow is expressed mathematically in Poiseuille's equation:

$$R = \frac{8NL}{\pi r^2}$$

R = resistance to airflow
N = viscosity of the gas
L = length of the tube
r = radius (8 and π are constants)

From this equation the effect of the radius of the tube can be seen. If the length and viscosity of the gas remain constant and the radius increases then airway resistance decreases. Thus, the airflow resistance has an inverse relationship to the radius. The smaller the radius, the higher the resistance to airflow (when viscosity and length of the tube remain constant). When the airflow is turbulent this mathematical equation does not apply. In turbulent airflow gas molecules interact in a random manner. If all other factors remain unchanged, resistance to airflow increases when the flow of air changes from laminar to turbulent flow.

There is in fact a balance that exists in the tracheobronchial tree between two opposing factors. As the diameter of the channels of the tracheobronchial tree becomes more narrow, theoretically, airway resistance should increase and reach its greatest resistance point in the smallest diameter tubes. This does not happen in the lungs because as the tubes become smaller they also become more numerous—the total cross sectional area increases markedly in the small airways. This lowers resistance to airflow. Instead, the greatest area of resistance to airflow takes place in the middle-size tubes found in the conducting airways. It is for this reason that small-airway damage can be present and not be detected by measurements of airway resistance.[13] The effect of airway resistance is to change the rapidity of the gas flow by changing the nature of the flow from laminar to turbulent. Laminar flow is a streamlined flow where little friction occurs between the molecules. In turbulent flow, however, there is random movement of the molecules of air. Figure 2 shows how the nature of airflow is altered by the presence of a partial obstruction in an airway (Figure 2).

An increase in airway resistance requires an increase in the pressure gradient required to move air in the area of most resistance. *In disease states there will be an increase in airway resistance at the area of dysfunction.* This is the basis of tests for obstructive lung disease (OLD) where measures of forced expiration are plotted over time. Generally, the amount of air expelled in 1 sec will be abnormally low if OLD is present.[11]

Anatomically, another point to review is the nature of the *structure of the bronchi*. The trachea bifurcates at the carina to produce the right and left main stem bronchi. Both these tubes perform in a manner similar to the trachea in terms of airway clearance and conduction functions. The right main stem bronchus morphologically appears to be an extension rather than a branch of the trachea. It is wider, shorter, and has more of a vertical angle than the left bronchus. Due to this natural placement of the right bronchus, drainage from the trachea into the right lung field is favored. *This has implications for the development of aspiration pneumonias as well as the likelihood of an endotracheal tube being inadvertently misplaced or slipping too far down the right main stem bronchus impeding or preventing aeration of the left side of the lung.*

Airway support and patency is provided by cartilage in the larger airways. In the transition from larger to smaller airways, this cartilaginous airway support is lost. *Patency in the small airways depends upon smooth muscle*

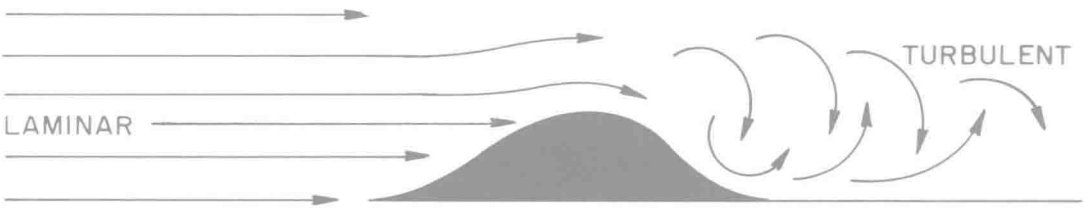

Figure 2. Laminar and turbulent airflow

control and support provided by the elastic recoil of surrounding lung parenchyma. Alveolar elastic recoil exerts a radial pull on the airways that they surround, that is, they tend to pull the airways open as they recoil. This factor has important implications for the emphysema patient who has lost a significant degree of alveolar elastic recoil. *With less airway support, small airways narrow, leading to airway obstruction on expiration.* The bronchi divide and subdivide until the diameter of the tubes reaches a point of less than 1 mm. The noncartilagenous tubes are then referred to as bronchioles. As the bronchioles continue to branch, and the sum of the total cross sectional area increases, airway resistance decreases. *These bronchioles contain no cartilage and are kept open by the elastic recoil of the lung parenchyma. This is an important concept in understanding how airways collapse in emphysema.* At the level of the sixteenth division, the bronchioles have reached a diameter of approximately 0.5 mm. The epithelium flattens, and mucous glands and cilia disappear.

Distal to the terminal bronchioles are the respiratory bronchioles, the alveolar ducts, and the alveolar sacs. The latter three structures make up one acinus. The acinus structures comprise the respiratory zone where gas exchange takes place. Let us look now at Figure 3 showing the conducting and gas exchange zones and their size and function. Note the diameter of these structures gets progressively smaller until the size of about 0.5 mm is reached. All gas exchange airways are approximately the same diameter as the terminal bronchioles.

Lung Parenchyma. Structures within the parenchyma include the respiratory bronchioles, the alveolar ducts and the alveolar sacs. The function of the respiratory zone structures is to allow gas exchange between the alveolar air and the blood. Alveolar ducts and alveolar sacs are designed to greatly increase the surface area of the lungs and together are responsible for all of the alveolar gas exchange. The clusters of alveolar sacs are responsible for the majority of the gas exchange as would be expected from their expansive surface area design. *Pulmonary surfactant acts to prevent the collapse of the alveoli and small airways.* Surfactant is a liquid which lines the alveolar epithelium. It is a protein-bound phospholipid substance, manufactured by type II alveolar epithelial cells, which decreases surface tension and stabilizes the alveoli.[14] During inhalation surfactant provides an increase in surface tension which increases compliance of the lung tissue and favors lung recoil. Surface tension reduction occurs in direct relation to the decreases in alveolar pressure and airflow radius according to the LaPlace equation:

$$P = \frac{2 \times T}{R},$$

in which P represents the pressure that must be generated to overcome the surface tension (T), and R represents the radius of the alveolus.[16] During exhalation, the molecules of surfactant move closer together which contributes to a decrease in surface tension. The interdependence of the alveoli within the parenchyma counters the tendency of the alveolus to collapse which allows for some aeration and facilitates lung reexpansion.[12]

Skeletal Structure

Most of the skeletal structure of the thorax is comprised of twelve pairs of *c*-shaped ribs. These ribs are connected posteriorly to the

ANATOMICAL PART	APPROXIMATE DIAMETER OF LUMEN*
Trachea	20mm
Main stem bronchi to bronchioles	10mm of first bifurcation to about 1mm at 11th bifurcation
Terminal bronchioles	1mm at 16th bifurcation
Respiratory bronchioles	0.5mm at 23rd bifurcation
Alveolar ducts	0.5mm
Alveolus	(Surface)

NOTE:
*Different authors give different diameters.

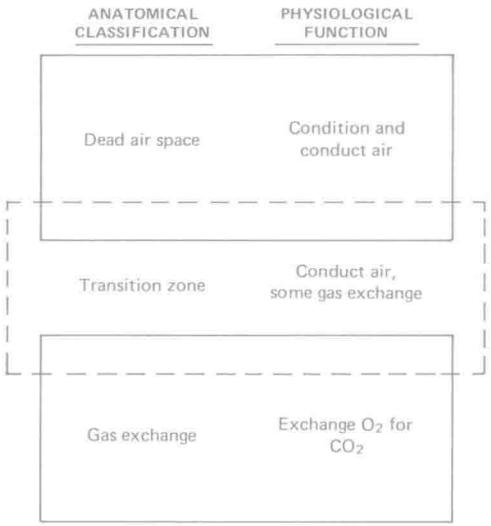

Figure 3. Zones of conduction and gas exchange

thoracic vertebrae which serve as the posterior midline of the thorax. The seven superior pairs of ribs articulate via their costal cartilages to the sternum which serves as the anterior midline of the thorax. The remaining five pairs of ribs do not articulate directly with the sternum but through their costal cartilages attach to the rib above. The thorax serves as a support base for the shoulder girdle and provides attachment for much of the upper body musculature. Within the thorax are the protected vital organs of the cardiopulmonary system.

The lungs join the interior surface of the thorax via the pleura surrounding each lung. Each lung actually lies free in its pleural cavity and responds passively to the changes in thoracic volume brought about by the muscles of ventilation. Pleurae are the tissue-paper-thin membranes lining the outside of the lungs and the inside of the chest cavity. The pleura over the outside of the lungs is referred to as the visceral pleura while the layer inside the chest cavity is referred to as the parietal pleura. Pleural fluid in the area between the linings provides a lubricant which allows them to move back and forth over each other during inspiration and expiration.

Ventilation, Diffusion, and Perfusion

Ventilation. *Ventilation is the process by which air carrying either oxygen or carbon dioxide is moved to and from the lungs.* The purpose of ventilation is to keep the alveolar partial pressure of oxygen higher and carbon dioxide lower than the levels found in the pulmonary capillary blood. This balance is supported by the process of diffusion. Inspired gas velocity in the terminal bronchiole and its associated alveoli is negligible. Molecular gas movement in these "terminal units" is provided solely by diffusion and is greatly affected by the structural arrangement of the alveoli. The varying lengths of bronchial airways and other structural and gravitational stresses imposed on the vertical lung theoretically assure a nonuniform gas distribution, yet, by poorly understood mechanisms, inspired gas is distributed with surprising uniformity in the lungs of young healthy adults.[4] This phenomenon has been evidenced as a result of nitrogen clearance graphs which indicated a nearly uniform distribution of inspired tidal volumes. Similar tests also have defined markedly nonuniform inspired gas distribution in lung disease patients. In diseased