

22

Dynamic Lung and Chest Wall Mechanics

LEARNING OBJECTIVES

Upon completion of this chapter, the student should be able to answer the following questions:

1. What is the difference between laminar and turbulent air flow?
2. How does airway resistance affect airflow?
3. What factors contribute to airway resistance?
4. When reviewing a spirogram, what is the FEV1? What is the FVC? Where is the peak flow measured? Why is the inspiratory flow-volume curve different from the expiratory flow-volume curve?
5. What is the expiratory equal pressure point? What are common causes of expiratory flow limitation?
6. Under what conditions might work of breathing increase? How can work of breathing be assessed?
7. How is dynamic compliance different from static compliance?

Dynamic Lung Mechanics

In this chapter, the principles that control air movement into and out of the lungs are examined. *Dynamic mechanics* is the study of physical systems in motion, and for the respiratory system it is the study of the properties of a lung whose volume is changing with time.

Airflow in Airways

Air flows into and out of an airway when there is a pressure difference at the two ends of the airway. By way of review, during inspiration the diaphragm contracts, pleural pressure becomes more negative, and gas flows into the lung (see Fig. 21.2). To meet the changing metabolic needs of the body, gas exchange depends on the speed at which fresh gas is brought to the alveoli and the rapidity with which the metabolic products of respiration (i.e., CO₂) are removed. Two major factors determine the speed at which gas flows into the airways for a given pressure change: the pattern of airflow and the resistance to airflow by the airways.

Patterns of Airflow

There are two major patterns of airflow in the airways—laminar and turbulent. *Laminar flow* is parallel to the airway walls and is present at low flow rates. As the flow rate

increases and particularly as the airways divide, the flow stream becomes unsteady and small eddies develop. At higher flow rates the flow stream is disorganized, and turbulence occurs.

The pressure-flow characteristics of laminar flow were first described by the French physician Poiseuille and apply to both liquids and air. In straight circular tubes the flow rate (\dot{V}) is defined by the following equation:

Equation 22.1

$$\dot{V} = \frac{P\pi r^4}{8\eta l}$$

where P is the driving pressure, r is the radius of the tube, η is the viscosity of the fluid, and l is the length of the tube. It can be seen that driving pressure (P) is proportional to the flow rate (\dot{V}); thus the greater the pressure, the greater the flow.

The flow resistance (R) across a set of tubes is defined as the change in driving pressure (ΔP) divided by the flow rate, or:

Equation 22.2

$$R = \frac{\Delta P}{\dot{V}} = \frac{8\eta l}{\pi r^4}$$

The units of resistance are cm H₂O/L•second. This equation is for laminar flow and demonstrates that the radius of the tube is the most important determinant of resistance. If the radius of the tube is reduced by half, the resistance will increase 16-fold. If, however, tube length is increased twofold, the resistance will increase only twofold. Thus, the radius of the tube is the principal determinant of resistance. Stated another way, resistance is inversely proportional to the fourth power of the radius, and it is directly proportional to the length of the tube and to the viscosity of the gas. To increase flow, increase the radius of the tube, shorten the tube, or decrease the viscosity of the flowing compound.

In *turbulent flow*, gas movement occurs both parallel and perpendicular to the axis of the tube. Pressure is proportional to the flow rate squared. The viscosity of the gas increases with increasing gas density, and therefore the pressure drop increases for a given flow. Overall, gas velocity is blunted because energy is consumed in the process of generating eddies and chaotic movement. As a consequence, higher driving pressure is needed to support a given turbulent flow than to support a similar laminar flow.

Whether flow through a tube is laminar or turbulent depends on the Reynolds number. The *Reynolds number* (R_c) is a dimensionless value that expresses the ratio of two dimensionally equivalent terms (kinematic/viscosity), as seen in the equation:

Equation 22.3

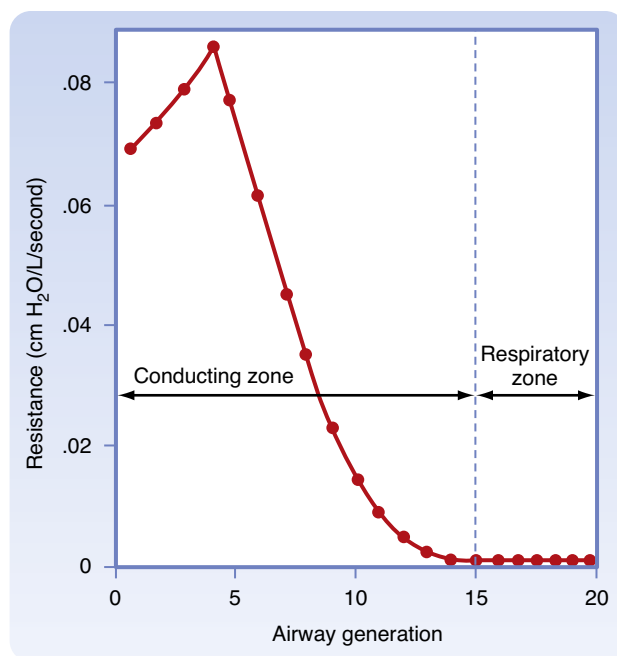
$$R_c = \frac{2rvd}{\eta}$$

where d is the fluid density, v is the average velocity, r is the radius, and η is the viscosity. In straight tubes, turbulence occurs when the Reynolds number is greater than 2000. From this relationship it can be seen that turbulence is most likely to occur when the average velocity of the gas flow is high and the radius is large. In contrast, a low-density gas such as helium is less likely to cause turbulent flow. This is clinically relevant in states of increased airway resistance where a decrease in gas density can improve airflow (e.g., by substituting helium for nitrogen in inspired air with a compound called Heliox). The increased rate of airflow also causes audible changes in voice pitch when breathing helium.

Although these relationships apply well to smooth cylindrical tubes, application of these principles to a complicated system of tubes such as the airways is difficult. As a result, much of the flow in the airways demonstrates characteristics of both laminar and turbulent flow. In the trachea, for example, even during quiet breathing the Reynolds number is greater than 2000. Hence turbulent flow occurs in the trachea even during quiet breathing. Turbulence is also promoted by the glottis and vocal cords, which produce some irregularity and obstruction in the airways. As gas flows distally, the total cross-sectional area increases dramatically, and gas velocities decrease significantly. As a result, gas flow becomes more laminar in the smaller airways even during maximal ventilation. Overall, the gas flow in the larger airways (nose, mouth, glottis, and bronchi) is turbulent, whereas the gas flow in the smaller airways is laminar. Breath sounds heard with a stethoscope reflect turbulent airflow. Laminar flow is silent, which is why it is difficult to “hear” small airway disease with a stethoscope.

Airway Resistance

Airflow resistance is the second major factor that determines rates of airflow in the airways. Airflow resistance in the airways (R_{aw}) differs in airways of different size. In moving from the trachea toward the alveolus, individual airways become smaller while the number of airway branches increases dramatically. R_{aw} is equal to the sum of the resistance of each of these airways (i.e., $R_{aw} = R_{large} + R_{medium} + R_{small}$). From Poiseuille's equation, one might conclude that the major site of airway resistance is in the smallest airways. In fact, however, the major site of resistance along the bronchial tree is in the first eight generations of airways. The smallest airways



• **Fig. 22.1** Airway resistance as a function of the airway generation. In a normal lung, most of the resistance to airflow occurs in the first eight airway generations.

contribute very little to the overall total resistance of the bronchial tree (Fig. 22.1). The reason for this is twofold: (1) airflow velocity decreases substantially as the effective cross-sectional area increases (i.e., flow becomes laminar); and (2) most importantly, the airway branches in each generation exist in parallel rather than in series. The resistance of airways in parallel is the inverse of the sum of the individual resistances; therefore, the overall contribution to resistance of the small airways is very small. As an example, assume that each of three tubes has a resistance of 3 cm H₂O. If the tubes are in series, the total resistance (R_{tot}) is the sum of the individual resistances:

Equation 22.4

$$R_{tot} = R_1 + R_2 + R_3 = 3 + 3 + 3 = 9 \text{ cm H}_2\text{O/L} \cdot \text{sec}$$

If the tubes are in parallel (as they are in small airways), the total resistance is the sum of the inverse of the individual resistances:

Equation 22.5

$$\begin{aligned} 1/R_{tot} &= 1/R_1 + 1/R_2 + 1/R_3 = 1/3 + 1/3 + 1/3 \\ R_{tot} &= 1 \text{ cm H}_2\text{O/L} \cdot \text{sec} \end{aligned}$$

This relationship is in marked contrast to the pulmonary blood vessels, in which most of the resistance is located in the small vessels (see Chapter 23). Thus, as airway diameter decreases, the resistance offered by each individual airway increases, but the large increase in the number of parallel pathways and cross-sectional area reduces the small airway resistance at each generation of branching.

During normal breathing, approximately 80% of the resistance to airflow at functional residual capacity (FRC)

occurs in airways with diameters greater than 2 mm. Because the small airways contribute so little to total lung resistance, measurement of airway resistance is a poor test for detecting small airway obstruction.

Factors That Contribute to Airway Resistance

In healthy individuals, airway resistance is approximately $1 \text{ cm H}_2\text{O/L} \cdot \text{second}$. One of the most important factors affecting resistance is lung volume. Increasing lung volume increases the caliber of the airways because it creates a positive transairway pressure. As a result, resistance to airflow decreases with increasing lung volume and increases with decreasing lung volume. If the reciprocal of resistance (i.e., conductance) is plotted against lung volume, the relationship between lung volume and conductance is linear (Fig. 22.2). Other factors that increase airway resistance include airway mucus, edema, and contraction of bronchial smooth muscle, all of which decrease the caliber of the airways.

The density and viscosity of the inspired gas also affect airway resistance. When scuba diving, gas density rises and results in an increase in airway resistance; this increase can cause problems for individuals with asthma and obstructive pulmonary disease. Breathing a low-density gas such as an oxygen-helium mixture results in a decrease in airway resistance and has been exploited in the treatment of **status asthmaticus**, a condition associated with a sudden increased airway resistance due to a combination of bronchospasm, airway inflammation, and hypersecretion of mucus.

Neurohumoral Regulation of Airway Resistance

In addition to the effects of disease, airway resistance is regulated by various neural and humoral agents. Stimulation of

efferent vagal fibers, either directly or reflexively, increases airway resistance and decreases anatomic dead space (see Chapter 23) secondary to airway constriction (recall that the vagus nerve innervates airway smooth muscle). In contrast, stimulation of sympathetic nerves and release of the postganglionic neurotransmitter norepinephrine inhibits airway constriction. Reflex stimulation of the vagus nerve by inhalation of smoke, dust, cold air, or other irritants can also result in airway constriction and coughing. Agents such as histamine, acetylcholine, thromboxane A_2 , prostaglandin F_2 , and leukotrienes (LTB_4 , LTC_4 , and LTD_4) are released by resident airway cells (e.g., mast cells, airway epithelial cells) and recruited cells (e.g., neutrophils, eosinophils) in response to various triggers such as allergens and viral infections. These agents act directly on airway smooth muscle to cause constriction and an increase in airway resistance. Inhalation of methacholine, a derivative of acetylcholine, is used to diagnose airway hyperresponsiveness, which is one of the cardinal features of certain asthma phenotypes. Although everyone is capable of responding to methacholine, airway obstruction develops in individuals with asthma at much lower concentrations of inhaled methacholine.

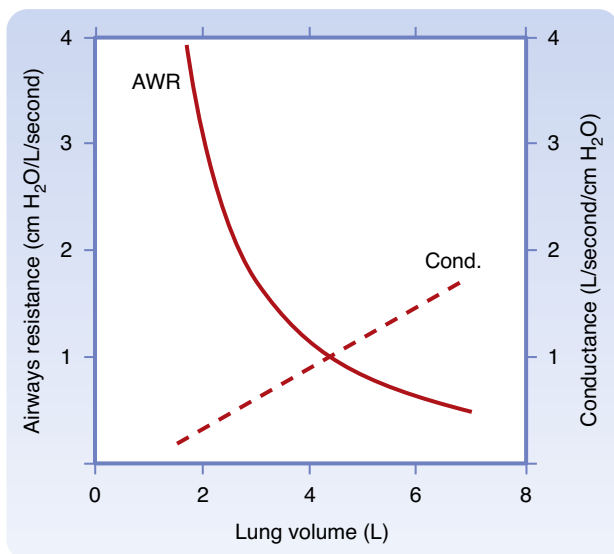
Measurement of Expiratory Flow

Measurement of expiratory flow rates and expiratory volumes is an important clinical tool for evaluating and monitoring respiratory diseases. Commonly used clinical tests have the patient inhale maximally to total lung capacity (TLC) and then exhale as rapidly and completely as possible to residual volume (RV). The test results are displayed either as a **spirogram** (Fig. 22.3A) or as a **flow-volume loop** (Fig. 22.3B). Results from individuals with suspected lung disease are compared with results predicted from normal healthy volunteers. Predicted or normal values vary with age, sex, ethnicity, height, and, to a lesser extent, weight (Table 22.1). Abnormalities in values indicate abnormal pulmonary function and can be used to predict abnormalities in gas exchange. These values can detect the presence of abnormal lung function long before respiratory symptoms develop, and they can be used to determine disease severity and the response to therapy.

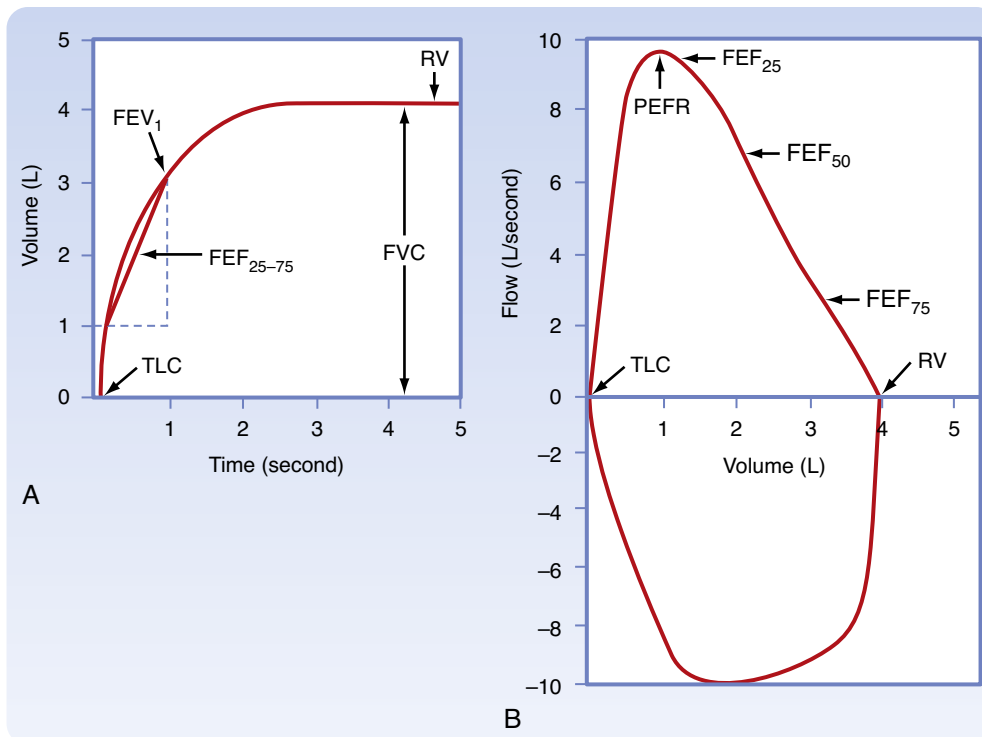
The Spirogram

A spirogram displays the volume of gas exhaled as a function of time (see Fig. 22.3A) and measures: (1) **forced vital capacity (FVC)**, (2) **forced expiratory volume in 1 second (FEV_1)**, (3) the **ratio of FEV_1 to FVC (FEV_1/FVC)**, and (4) the **average midmaximal expiratory flow (FEF_{25-75})**.

The total volume of air that is exhaled during a maximal forced exhalation from TLC to RV is called the *FVC*. The volume of air that is exhaled in the first second during the maneuver is called the FEV_1 . In normal individuals, 70% to 85% (depending on age) of the FVC can be exhaled in the first second. Thus, the normal FEV_1/FVC ratio is greater than 70% in healthy adults.



• **Fig. 22.2** Airway resistance (*AWR*) and conductance (*Cond.*) as a function of lung volume.



• **Fig. 22.3** The clinical spirogram (A) and flow-volume loop (B). The individual takes a maximal inspiration to TLC and then exhales rapidly and forcibly for as long as possible until RV is reached. The volume exhaled is plotted as a function of time. In the spirogram that is reported in clinical settings, exhaled volume increases from the bottom of the trace to the top (A). This is in contrast to the physiologist's view of the same maneuver (see Fig. 21.3), in which the exhaled volume increases from the top to the bottom of the trace. In the flow-volume loop (B), exhaled volume is plotted as a function of the instantaneous flow rate, which is measured using a pneumotachometer. The maximal expiratory flow rate achieved during the maneuver is called the *peak expiratory flow rate*. Note the locations of TLC and RV on both tracings. *FEV₁*, Forced expiratory volume in 1 second; *FVC*, forced vital capacity; *PEFR*, peak expiratory flow rate; *RV*, residual volume; *TLC*, total lung capacity.

TABLE 22.1 Patterns of Pulmonary Function Test Abnormalities

Pulmonary Function Measurement	Obstructive Pulmonary Disease	Restrictive Pulmonary Disease
FVC (L)	Decreased	Decreased
FEV ₁ (L)	Decreased	Decreased
FEV ₁ /FVC	Decreased	Normal
FEF ₂₅₋₇₅ (L/second)	Decreased	Normal to increased
PEFR (L/second)	Decreased	Normal
FEF ₅₀ (L/second)	Decreased	Normal
FEF ₇₅ (L/second)	Decreased	Normal
Slope of FV curve	Decreased	Normal to increased

A ratio less than 70% suggests difficulty exhaling because of obstruction and is a hallmark of obstructive pulmonary disease. One expiratory flow rate—the average flow rate over the middle section of the VC—can be calculated from the spirogram. This expiratory flow rate has several names, including **MMEF (mid-maximal expiratory flow)** and **FEF₂₅₋₇₅** (forced expiratory flow from 25%–75% of VC). Although it can be identified

from the spirogram tracing, today's spirometers automatically calculate FEF₂₅₋₇₅.

Flow-Volume Loop

Another way of measuring lung function clinically is the flow-volume loop. A flow-volume loop is created by displaying the instantaneous flow rate during a forced maneuver as a function of the volume of inhaled or exhaled gas. This instantaneous flow rate can be displayed both during exhalation (expiratory flow-volume curve) and during inspiration (inspiratory flow-volume curve) (see Fig. 22.3B). By convention, expiratory flow rates are displayed above the horizontal line, and inspiratory flow rates are displayed below the horizontal line. The flow-volume loop measures: (1) the FVC; (2) the greatest flow rate achieved during the expiratory maneuver, called the **peak expiratory flow rate (PEFR)**; and (3) multiple expiratory flow rates at various lung volumes. When the expiratory flow-volume curve is divided into quarters, the instantaneous flow rate at which 50% of the VC remains to be exhaled is called the **FEF₅₀** (also known as the \dot{V}_{max50}), the instantaneous flow rate at which 75% of the VC has been exhaled is called the **FEF₇₅** (\dot{V}_{max75}),

and the instantaneous flow rate at which 25% of the VC has been exhaled is called the FEF_{25} ($\dot{V}_{\max 25}$). Peak flow (PEFR) may decrease in clinical situations when the conducting airways are narrowed. A peak flow meter can be used at home as a tool to track bulk airflow. Measurements of peak flow can be used in asthma treatment to guide protocol-based medication adjustments.



IN THE CLINIC

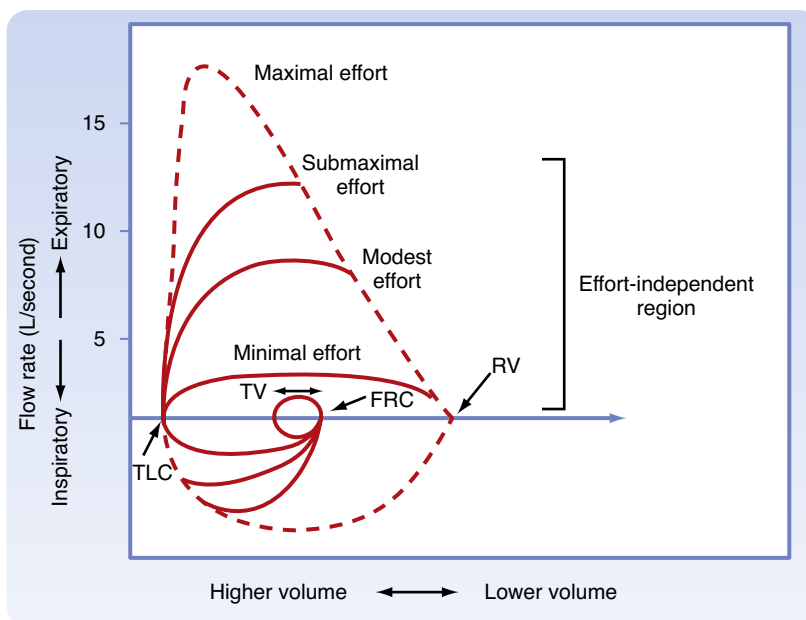
A methacholine challenge test (also called a bronchoprovocation test) is used to evaluate how reactive the airways are. This can help with the diagnosis of asthma. Methacholine, a muscarinic agonist, causes bronchoconstriction, or narrowing of the airways. In this test, the patient inhales increasing concentrations of methacholine. At baseline and after each dose increment, spirometry measurements are obtained. The test is stopped when FEV₁ falls by 20% or more or when a maximum concentration (25 mg/mL) of methacholine has been inhaled. The concentration of methacholine that produces a 20% decrease in FEV₁ is called the *provocation concentration (PC)20*. The lower the PC20, the more sensitive an individual is to methacholine. Most individuals with asthma have a PC20 less than 8 mg/mL of methacholine.

Determinants of Maximal Flow

The shape of the flow-volume loop reveals important information about normal lung physiology that can be altered by disease. Inspection of the flow-volume loop reveals that the maximum inspiratory flow is the same or slightly

greater than the maximum expiratory flow. Three factors are responsible for the maximum inspiratory flow. First, the force generated by the inspiratory muscles decreases as lung volume increases above RV. Second, the recoil pressure of the lung increases as the lung volume increases above RV. This opposes the force generated by the inspiratory muscles and reduces maximum inspiratory flow. Third, airway resistance decreases with increasing lung volume as the airway caliber increases. The combination of inspiratory muscle force, recoil of the lung, and changes in airway resistance causes maximal inspiratory flow to occur about halfway between TLC and RV.

During exhalation, maximal flow occurs early (in the first 20%) in the maneuver, and flow rates decrease progressively toward RV. Even with increasing effort, maximal flow decreases as RV is approached. This is known as *expiratory flow limitation* and can be demonstrated by asking an individual to perform three forced expiratory maneuvers with increasing effort. Fig. 22.4 shows the results of these three maneuvers. As effort increases, peak expiratory flow increases. However, the flow rates at lower lung volumes converge; this indicates that with modest effort, maximal expiratory flow is achieved. No amount of effort will increase the flow rates as lung volume decreases. For this reason, expiratory flow rates at lower lung volumes are said to be *effort independent* and *flow limited* because maximal flow is achieved with modest effort, and no amount of additional effort can increase the flow rate beyond this limit. In contrast, events early in the expiratory maneuver are said to be *effort dependent*; that is, increasing effort generates increasing flow rates. In general, the first 20%



• **Fig. 22.4** Isovolume curves. Three superimposed expiratory flow maneuvers are made with increasing effort. Note that peak inspiratory and expiratory flow rates are dependent on effort, whereas expiratory flow rates later in expiration are independent of effort. *FRC*, Functional residual capacity; *TLC*, total lung capacity; *RV*, residual volume; *TV* (or V_T), tidal volume.

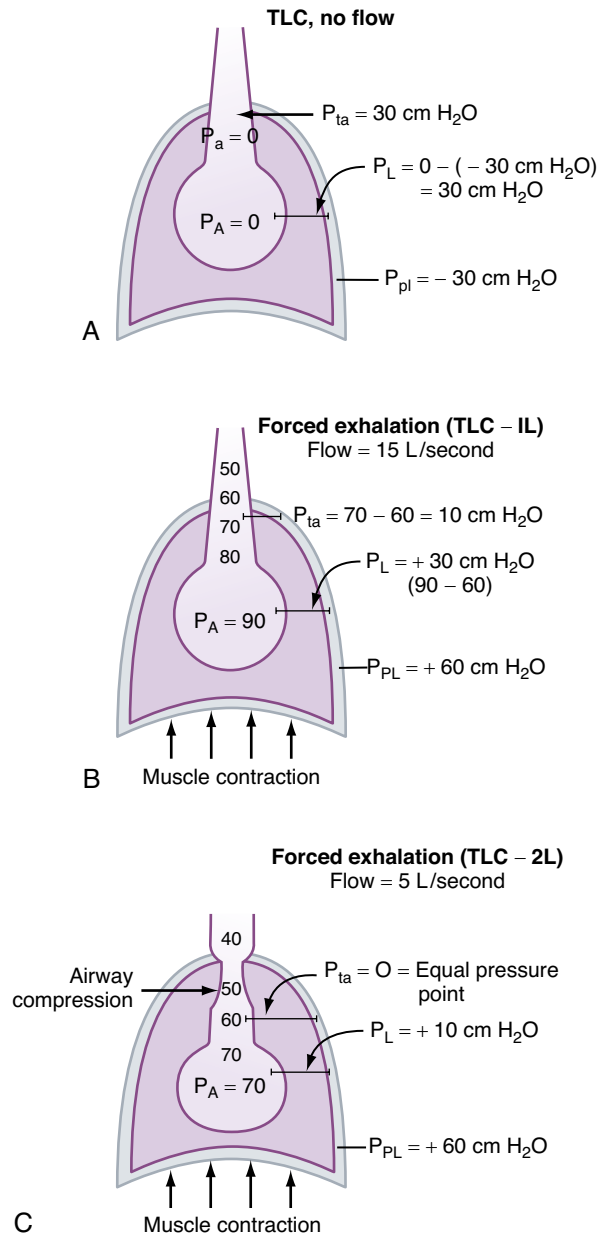
of the flow in the expiratory flow-volume loop is effort dependent.

Flow Limitation and the Equal Pressure Point

Why is expiratory flow limited and reasonably effort independent? Factors that limit expiratory flow are important because many lung diseases affect these factors and thus affect the volume and speed with which air is moved into and out of the lung. Flow limitation occurs when the airways, which are intrinsically floppy distensible tubes, become compressed. The airways become compressed when the pressure outside the airway exceeds the pressure inside the airway. How and when this occurs is important to understanding lung disease. Fig. 22.5 shows the events that occur during expiratory flow limitation at two different lung volumes. The airways and alveoli are surrounded by the pleural space and the chest wall. The airways are shown as tapered tubes because the total or collective airway cross-sectional area decreases from the alveoli to the trachea. At the start of exhalation but before any gas flow occurs, the pressure inside the alveolus (P_A) is zero (no airflow) and pleural pressure (in this example) is -30 cm H₂O. Transpulmonary pressure is thus $+30$ cm H₂O ($P_L = P_A - P_{pl}$). Because there is no flow, the pressure inside the airways is zero and the pressure across the airways (P_{ta} , transairway pressure) is $+30$ cm H₂O ($P_{ta} = P_{airway} - P_{pl} = 0 - [-30 \text{ cm H}_2\text{O}]$). This positive transpulmonary and transairway pressure holds the alveoli and airways open.

When an active exhalation begins and the expiratory muscles contract, pleural pressure rises to $+60$ cm H₂O (in this example). Alveolar pressure also rises, in part because of the increase in pleural pressure ($+60$ cm H₂O) and in part because of the elastic recoil pressure of the lung at that lung volume (which in this case is 30 cm H₂O). Alveolar pressure is the sum of pleural pressure and elastic recoil pressure (i.e., $P_A = P_{el} + P_{pl} = 30 \text{ cm H}_2\text{O} + 60 \text{ cm H}_2\text{O} = 90 \text{ cm H}_2\text{O}$ in this example). This is the driving pressure for expiratory gas flow. Because alveolar pressure exceeds atmospheric pressure, gas begins to flow from the alveolus to the mouth when the glottis opens. As gas flows out of the alveoli, the transmural pressure across the airways decreases (i.e., the pressure head for expiratory gas flow dissipates). This occurs for three reasons: (1) there is a resistive pressure drop caused by the frictional pressure loss associated with flow (expiratory airflow resistance); (2) as the cross-sectional area of the airways decreases toward the trachea, gas velocity increases and this acceleration of gas flow further decreases the pressure; and (3) as lung volume decreases, the elastic recoil pressure decreases.

Thus, as air moves out of the lung, the driving pressure for expiratory gas flow decreases. In addition, the mechanical tethering that holds the airways open at high lung volumes diminishes as lung volume decreases. There is a point between the alveoli and the mouth at which the pressure



• **Fig. 22.5** Flow limitation. **A**, End inspiration, before the start of exhalation. **B**, At the start of a forced exhalation. **C**, Expiratory flow limitation later in a forced exhalation. Expiratory flow limitation occurs at locations where airway diameter is narrowed as a result of negative transmural pressure. See text for details. TLC, Total lung capacity.

inside the airways equals the pressure that surrounds the airways. This point is called the **equal pressure point**. Airways toward the mouth but still inside the chest wall become compressed because the pressure outside is greater than the pressure inside (**dynamic airway compression**). As a consequence, the transairway pressure now becomes negative ($P_{ta} = P_{aw} - P_{pl} = 58 - [+60] = -2 \text{ cm H}_2\text{O}$) just beyond the equal pressure point. No amount of effort will increase the flow further because the higher pleural pressure tends to collapse the airway at the equal pressure point, just as it also tends to increase the gradient for expiratory

gas flow. Under these conditions, airflow is independent of the total driving pressure. Hence the expiratory flow is effort independent and flow limited. It is also why airway resistance is greater during exhalation than during inspiration. In the absence of lung disease, the equal pressure point occurs in airways that contain cartilage, and thus they resist collapse. The equal pressure point, however, is not static. As lung volume decreases and as elastic recoil pressure decreases, the equal pressure point moves closer to the alveoli.

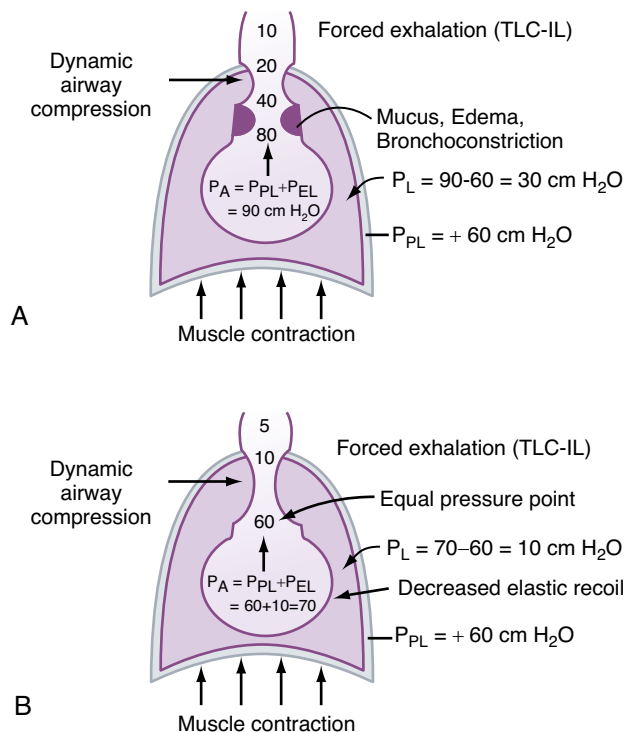


IN THE CLINIC

What happens in individuals with lung disease? Imagine an individual with airway obstruction secondary to a combination of mucus accumulation and airway inflammation (Fig. 22.6A). At the start of exhalation, the driving pressure for expiratory gas flow is the same as in a normal individual; that is, the driving pressure is the sum of the elastic recoil pressure and pleural pressure. As exhalation proceeds, however, the resistive drop in pressure is greater than in the normal individual because of the greater decrease in airway radius secondary to the accumulation of mucus and the inflammation. As a result, the equal pressure point now occurs in small airways that are devoid of cartilage. These airways collapse. This collapse is known as **premature airway closure**, which results in a less-than-maximal expiratory volume and produces an increase in lung volume known as *air trapping*. The increase in lung volume initially helps offset the increase in airway resistance caused by the accumulation of mucus and inflammation because it results in an increase in airway caliber and elastic recoil. As the disease progresses, however, inflammation and accumulation of mucus increase further, there is a greater increase in expiratory resistance, and maximal expiratory flow rates decrease.

Now imagine an individual with emphysema and a loss of elastic recoil (see Fig. 22.6B). At the start of exhalation the driving pressure for expiratory gas flow is reduced secondary to a loss of elastic recoil. While the resistive drop in pressure is normal, the smaller initial driving pressure results in an equal pressure point that occurs closer to the alveolus in airways that do not contain cartilage. Premature airway closure again occurs but for a very different reason than the premature airway closure observed in individuals with an increase in airway resistance.

Individuals with premature airway closure frequently have **crackles**, also sometimes called **rales**, a popping sound usually heard during inspiration on auscultation. These crackles are due to the opening of airways during inspiration that closed (i.e., were compressed) during the previous exhalation. Crackles can be due to mucus accumulation, airway inflammation, fluid in the airways, or any mechanism responsible for airway narrowing or compression. They are also heard in individuals with emphysema, in which there is a decrease in lung elastic recoil. In fact, acute and chronic lung diseases can change the expiratory flow-volume relationship by changes in (1) static lung recoil pressure, (2) airway resistance and the distribution of resistance along the airways, (3) loss of mechanical tethering of intraparenchymal airways, (4) changes in the stiffness or mechanical properties of the airways, and (5) differences in severity of the aforementioned changes in various lung regions.



• **Fig. 22.6 A**, Flow limitation in the presence of increased airway resistance. **B**, Flow limitation in the presence of a loss of elastic recoil. TLC, Total lung capacity.

Dynamic Compliance

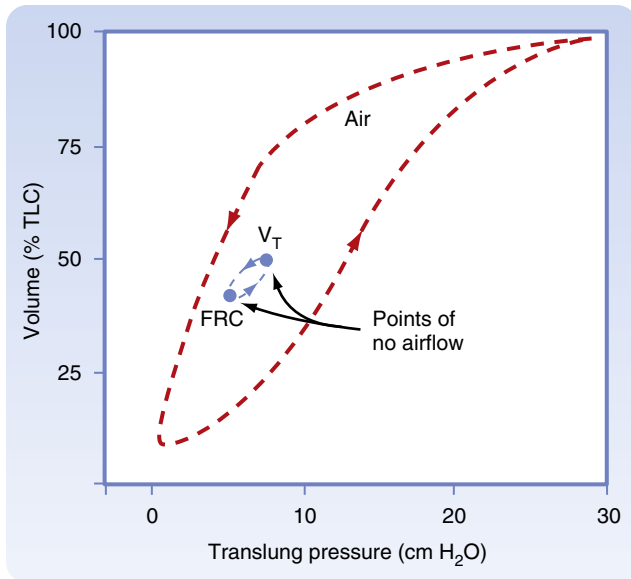
One additional measurement of dynamic lung mechanics should be mentioned, and this is the measurement of dynamic compliance. A dynamic pressure-volume curve can be created by having an individual breathe over a normal lung volume range (usually from FRC to FRC + 1 L). The mean dynamic compliance of the lung (dyn C₁) is calculated as the slope of the line that joins the end-inspiratory and end-expiratory points of no flow (Fig. 22.7).

Dynamic compliance is always less than static compliance, and it increases during exercise. This is because during tidal volume breathing, a small change in alveolar surface area is insufficient to bring additional surfactant molecules to the surface, and thus the lung is less compliant. During exercise the opposite occurs; there are large changes in tidal volume, and more surfactant material is incorporated into the air-liquid interface. Therefore, the lung is more compliant.

Sighing and yawning increase dynamic compliance by increasing tidal volume and restoring the normal surfactant layer. Both of these respiratory activities are important for maintaining normal lung compliance. In contrast to the lung, the dynamic compliance of the chest wall is not significantly different from its static compliance.

Work of Breathing

Breathing requires the use of respiratory muscles (diaphragm, intercostals, etc.), which expends energy. Work is



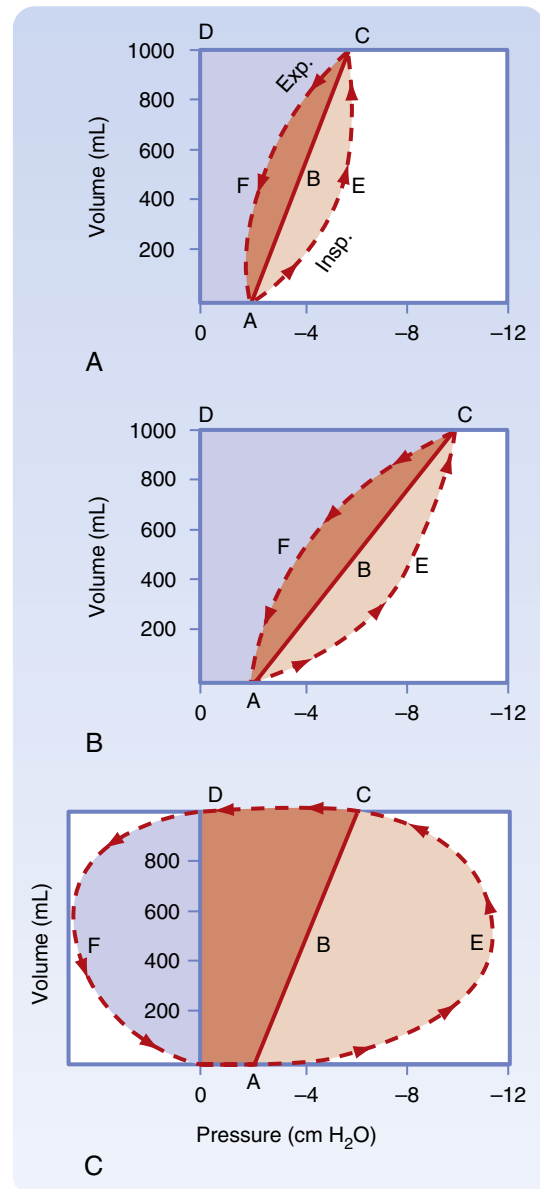
• **Fig. 22.7** Inflation-deflation pressure-volume curve. The direction of inspiration and exhalation is shown by the arrows. The difference between the inflation and deflation pressure-volume curves is due to the variation in surface tension with changes in lung volume. Note the slope of the line joining points of no airflow. This slope is less steep than the slope from the deflation pressure-volume curve at the same lung volume. *FRC*, Functional residual capacity; *TLC*, total lung capacity.

required to overcome the inherent mechanical properties of the lung (i.e., elastic and flow-resistive forces) and to move both the lungs and the chest wall. This work is known as the **work of breathing**. Changes in the mechanical properties of the lung or chest wall (or both) in the presence of disease result in an increase in the work of breathing. Respiratory muscles can perform increased work over long periods. However, like other skeletal muscles, they can fatigue, and respiratory failure may ensue. Respiratory muscle fatigue is the most common cause of **respiratory failure**, a process in which gas exchange is inadequate to meet the metabolic needs of the body. In the respiratory system, the work of breathing is calculated by multiplying the change in volume by the pressure exerted across the respiratory system:

$$\text{Work of breathing (W)} = \text{Pressure (P)} \times \text{Change in volume } (\Delta V)$$

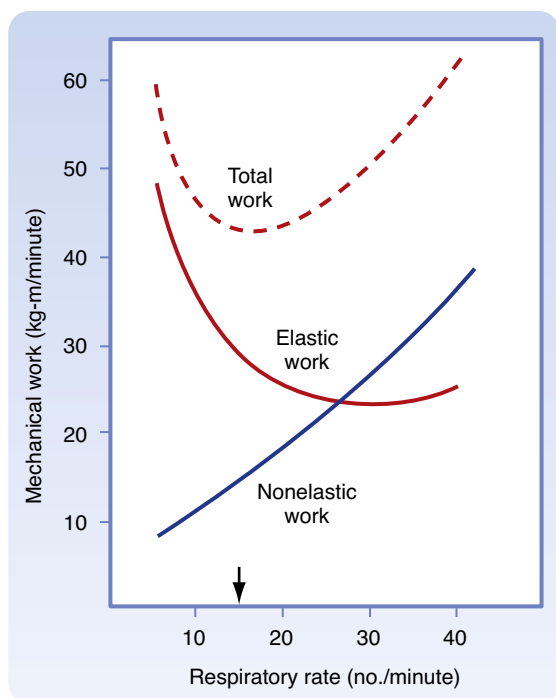
Although methods are not available to measure the total amount of work involved in breathing, one can estimate the mechanical work by measuring the volume and pressure changes during a respiratory cycle. Analysis of pressure-volume curves can be used to illustrate these points. **Fig. 22.8A** represents a respiratory cycle of a normal lung. The static inflation-deflation curve is represented by line *ABC*. The total mechanical workload is represented by the trapezoidal area *OAECD*.

In restrictive lung diseases, such as pulmonary fibrosis, lung compliance is decreased and the pressure-volume curve is shifted to the right. This results in a significant increase in the work of breathing (see **Fig. 22.8B**), as indicated by the increase in the trapezoidal area of *OAECD*. In obstructive lung diseases,



• **Fig. 22.8** Mechanical work done during a respiratory cycle in a normal lung (**A**), a lung with reduced compliance (**B**), and a lung with increased airway resistance (**C**). Breakdown of the trapezoidal areas enables one to appreciate the individual aspects of the mechanical workload, which include the following: *OABCD*, Work necessary to overcome elastic resistance; *AECF*, work necessary to overcome nonelastic resistance; *AECB*, work necessary to overcome nonelastic resistance during inspiration; *ABCF*, work necessary to overcome nonelastic resistance during exhalation (represents stored elastic energy from inspiration).

such as asthma during an exacerbation or chronic bronchitis, airway resistance is elevated (see **Fig. 22.8C**) and greater negative pleural pressure is needed to maintain normal inspiratory flow rates. In addition to the increase in total inspiratory work (*OAECD*), individuals with obstructive lung disease have an increase in positive pleural pressure during exhalation because of the increase in resistance and the increased expiratory workload, which is visualized as area *DFO*. The stored elastic energy, represented by area *ABCF* of **Fig. 22.8A**, is not sufficient, and



• **Fig. 22.9** Effect of the respiratory rate on the elastic, nonelastic, and total mechanical work of breathing at a given level of alveolar ventilation. Individuals tend to adopt the respiratory rate at which the total work of breathing is minimal (*arrow*) for those without lung disease.

additional energy is needed for exhalation. With time or disease progression, these respiratory muscles can fatigue and result in respiratory failure. The work of breathing is also increased when deeper breaths are taken (an increase in tidal volume requires more elastic work to overcome) and when the respiratory rate increases (an increase in minute ventilation requires more flow resistance force to overcome) (Fig. 22.9). Normal individuals and individuals with lung disease adopt respiratory patterns that minimize the work of breathing. For this reason, individuals with pulmonary fibrosis (increased elastic work)

Key Concepts

1. There are two major patterns of airflow in the airways: turbulent and laminar.
2. *Resistance* to airflow is the change in pressure per unit of flow. Airway resistance varies with the inverse of the fourth power of the radius and is higher in turbulent than in laminar flow. The major site of airway resistance is the first eight airway generations. Airway resistance decreases with increases in lung volume and with decreases in gas density (such as when nitrogen is replaced with helium). Airway resistance is also regulated by neural and humoral agents.
3. Pulmonary function tests (spirometry, flow-volume loop, body plethysmography) can detect abnormalities in lung function before individuals become symptomatic. Test results are compared with results obtained in normal individuals and vary with sex, ethnicity, age,

breathe more shallowly and rapidly, and those with obstructive lung disease (normal elastic work but increased resistive work) breathe more slowly and deeply.



IN THE CLINIC

Chronic obstructive pulmonary disease (COPD) is a general term that includes diseases such as emphysema and chronic bronchitis. COPD most commonly occurs in individuals who smoke, in whom pathologic changes in the lung consistent with both emphysema and chronic bronchitis can coexist. Other long term environmental exposures (biomass fuel smoke or other air pollution) may also contribute to COPD, as may genetic conditions or abnormal lung development. For individuals with COPD in whom emphysema is a major component, the elastic tissue in the alveolar and capillary walls is progressively destroyed, which results in increased lung compliance and decreased elastic recoil. The decrease in elastic recoil results in movement of the equal pressure point toward the alveolus and premature airway closure. This produces air trapping and increases in RV, FRC, and TLC. Airway resistance is also increased. These increases in lung volumes increase the work of breathing by stretching the respiratory muscles and decreasing their efficiency.

In chronic bronchitis, accumulation of mucus and airway inflammation cause the equal pressure point to move toward the alveolus, which leads to premature airway closure and increases in RV, FRC, and TLC. Airway resistance and the work of breathing are increased, but lung compliance is normal.

In restrictive lung diseases such as pulmonary fibrosis, lung compliance is decreased. Lung volumes are decreased, but flow rates are reasonably normal. Some of the changes in pulmonary function values in obstructive and restrictive pulmonary diseases are shown in Table 22.1.

In the third trimester of pregnancy, the enlarged uterus increases intra-abdominal pressure and restricts movement of the diaphragm. As a result, FRC decreases. Additionally, progesterone causes increased airway blood flow (hyperemia) and mucosal edema leading to increased airway resistance. These changes result in decreased lung volume, decreased lung compliance, and increased airway resistance in otherwise healthy women.

- and height. COPD is characterized by increases in lung volumes and airway resistance and by decreases in expiratory flow rates. Emphysema, a specific type of COPD, is further characterized by increased lung compliance. Restrictive lung diseases are characterized by decreases in lung volume, normal expiratory flow rates and resistance, and a marked decrease in lung compliance.
4. The *equal pressure point* is the point at which the pressure inside and surrounding the airway is the same. The location of the equal pressure point is dynamic. Specifically, as lung volume and elastic recoil decrease, the equal pressure point moves toward the alveolus in normal individuals. In individuals with chronic obstructive pulmonary disease (COPD), the equal pressure point at any lung volume is closer to the

alveolus. Expiratory flow limitation occurs at the equal pressure point.

5. Energy is expended during breathing to overcome the inherent mechanical properties of the lung. Respiratory muscle fatigue is the most common cause of respiratory failure. Individuals breathe at a respiratory rate to minimize work while maintaining adequate oxygenation and ventilation. For individuals with increased airway resistance, work is minimized by using larger-volume breaths at lower frequencies. For individuals with restrictive lung diseases, work is minimized by taking smaller volume breaths at higher respiratory rates.
6. The dynamic compliance of the lung is always less than the static compliance and increases during exercise, sighing, and yawning.