

## 8 Pulmonology

### Physiological Principles

#### ■ Components of Pulmonary Function

The three components of pulmonary function are (Fig. 8.1):

- Ventilation
- Perfusion, i.e., blood flow
- Diffusion, i.e., gas exchange

#### Ventilation

##### Inspiration

Gases flow from regions of high pressure to regions of low pressure. Thus at inhalation, the intrapulmonary pressure must be lower than atmospheric pressure. The drop in pressure is produced by increasing the volume of the lungs. To do this, the diaphragm moves downward and the ribs are raised by contraction of the external intercostal muscles. During forced breathing, the auxiliary breathing muscles are also called into play.

##### Expiration

Unforced exhalation is a passive event. The diaphragm relaxes, the ribs return to their lower position, and the lungs follow their natural tendency to contract. As a result, the intrapulmonary volume falls and the intrapulmonary pressure rises under the influence of the ambient pressure. Air streams outward. In forced breathing, on the other hand, the expiratory breathing muscles are activated.

#### Pleura

The lung must be able to follow the breathing movements without being completely held fast to the diaphragm and the rib cage. This is enabled by the two pleural layers:

- The visceral pleura covers the lung.
- The parietal pleura lines the thorax.

Between the two pleural layers is a thin liquid film through which the lung adheres to the thorax and the diaphragm by surface tension. Except during

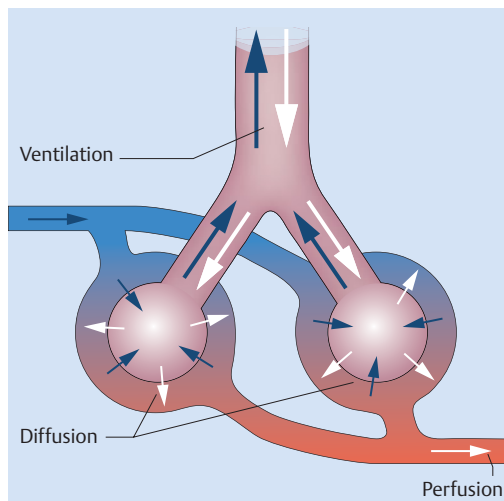


Fig. 8.1 Components of pulmonary function.

forced expiration, there is always negative pressure in the pleural cavity.

*Clinical relevance:* If, for example, air enters the pleural cavity as the result of a thoracic injury, the negative intrapleural pressure is eliminated and the lung collapses as a result of its elasticity. This condition is called pneumothorax (p. 166).

#### Perfusion

The lung is perfused by two vascular systems:

- The bronchial arteries, part of the systemic circulation, that supply oxygen-rich blood to the lungs
- The pulmonary arteries, part of the pulmonary circulation, that carry oxygen-poor blood from the right heart to the lungs, where it can be recharged with oxygen

#### Low-Pressure System

In connection with respiration, the pulmonary veins and arteries are of interest. They are part of the low-pressure system (p. 85) where pressures are only 5–20 mmHg. The myocardium of the right ventricle is correspondingly thin.

### Ventilation–Perfusion Relationship

Physiologically, ventilated lung areas are well perfused. In order to maintain an effective ventilation–perfusion relationship, blood flow is reduced by vasoconstriction in lung parts with decreased ventilation.

**Clinical relevance:** The Euler–Liljestrand reflex explains how a ventilation disorder also leads to a perfusion disorder. Vasoconstriction increases the pressure in the pulmonary circulation, creating stress on the right heart.

### Diffusion

The ideal conditions for diffusion through a membrane are:

- A large membrane surface
- A thin membrane
- A large concentration gradient
- A substance that “likes” to diffuse, i.e., that has a high diffusion coefficient

Alveoli and respiratory gases create favorable conditions for gas exchange in the lung.

### Alveoli

- The lungs consist of about 300 million alveoli, i.e., small air sacks, each with a diameter of about 0.3 mm. This provides an exchange surface of 100 m<sup>2</sup> for diffusion.
- The alveolar membrane is only 1–2 μm thick and does not impede gas exchange.
- The alveoli are protected from collapsing by a very thin film of phospholipids, which is called *surfactant* and decreases the surface tension.

### Respiratory Gases

Energy is generated in the tissues by oxidation of glucose. The resulting carbon dioxide is carried to the lungs by the blood and diffuses from the capillary to the alveolus in accordance with the concentration gradient. Oxygen diffuses in the other direction and is carried to the tissues in the bloodstream. The different partial pressures of O<sub>2</sub> and CO<sub>2</sub>, which provide the driving force of diffusion, are shown in **Table 8.1**.

In spite of the small differences in concentration, enough CO<sub>2</sub> diffuses because CO<sub>2</sub> has a high diffusion coefficient. In fact, CO<sub>2</sub> diffuses 23 times as rapidly as O<sub>2</sub>.

**Clinical relevance:** Deviations from normal values are classified as follows:

- **Hypoxemia:** Decline in the partial pressure of O<sub>2</sub>
- **Hypercapnia:** Increase in the partial pressure of CO<sub>2</sub>
- **Hypocapnia:** Decline in the partial pressure of CO<sub>2</sub>

When gas exchange is impeded, CO<sub>2</sub> can still be exhaled fairly well at first because of its high diffusion coefficient; i.e., hypoxemia sets in first and hypercapnia only sets in later (respiratory insufficiency, p. 140).

**Table 8.1** O<sub>2</sub> and CO<sub>2</sub> partial pressures (mmHg) in alveoli and blood

Partial pressure	O <sub>2</sub> -poor blood	Alveoli	O <sub>2</sub> -rich blood
pO <sub>2</sub>	40	100	95
pCO <sub>2</sub>	46	40	40

### Blood pH

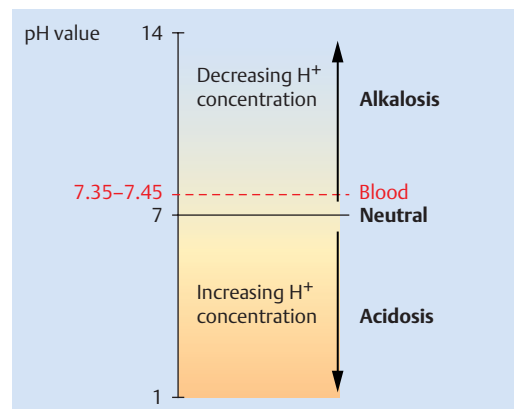
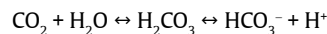
The pH value indicates the hydrogen ion (H<sup>+</sup>) concentration of a solution:

$$\text{pH} = -\log [\text{H}^+]$$

This definition makes it clear that:

- The pH value falls with rising H<sup>+</sup> concentration. This leads to acidosis (**Fig. 8.2**).
- The pH value rises with decreasing H<sup>+</sup> concentration. This leads to alkalosis.

The following chemical equation shows that the carbon dioxide generated in the tissue reacts with water to form carbonic acid. This is unstable and dissociates into bicarbonate and hydrogen ions:



**Fig. 8.2** pH value

$\text{CO}_2$  and  $\text{H}^+$  ions are in equilibrium, so the pH value of the blood is dependent, among other things, on the  $\text{CO}_2$  concentration:

$\text{CO}_2 \uparrow \rightarrow \text{H}^+ \uparrow \rightarrow \text{pH} \downarrow \rightarrow \text{respiratory acidosis}$

$\text{CO}_2 \downarrow \rightarrow \text{H}^+ \downarrow \rightarrow \text{pH} \uparrow \rightarrow \text{respiratory alkalosis}$

### ■ Regulation of Breathing

Breathing is controlled from the breathing center in the medulla oblongata. The breathing center reacts to numerous influences, especially to changes in the partial pressure of  $\text{O}_2$  and  $\text{CO}_2$  in the blood.

#### Chemical Breathing Stimulus

- Peripheral chemoreceptors in the aortic arch and the carotid artery measure the partial  $\text{O}_2$  pressure in the blood and mediate the peripheral breathing stimulus. When the partial pressure of  $\text{O}_2$  drops, a reflex stimulates breathing.
- Central chemical receptors in the medulla oblongata measure the partial pressure of  $\text{CO}_2$  and the pH value of the spinal fluid, which are in equilibrium with the values in the blood. They mediate the central breathing impulse. When the partial pressure of  $\text{CO}_2$  rises or the pH value falls, breathing becomes more rapid and deeper.

Figure 8.3a–c shows that rising partial pressure of  $\text{CO}_2$  has the greatest influence on respiratory minute volume, so that  $\text{CO}_2$  represents the strongest breathing stimulus. At the same time, it is clear that above a certain partial pressure of  $\text{CO}_2$  the respiratory minute volume decreases, which is to say that  $\text{CO}_2$  has a narcotic effect.

**Clinical relevance:** It is possible that patients with marked pulmonary diseases have a high partial pressure of  $\text{CO}_2$  and have thus lost their central breathing stimulus (global respiratory insufficiency, p. 140).

## Classification of Pulmonary Diseases

Pulmonary diseases can affect one or more of the functional components of the lung and thus lead to ventilation, perfusion, or diffusion disorders.

### Ventilation Disorders

In a ventilation disorder, the lungs or individual areas of the lungs will be ventilated less efficiently. The differences between obstructive and restrictive ventilation disorders as well as their causes are presented in Table 8.2.

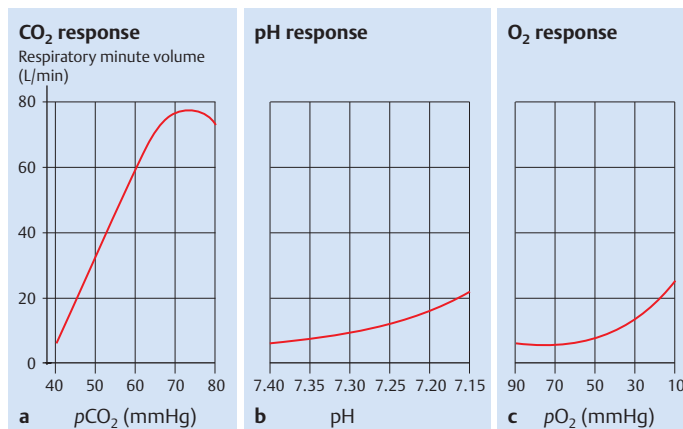
### Perfusion Disorders

A direct effect on pulmonary circulation is exerted by:

- Disruption of arterial blood supply, especially in cases of pulmonary embolism (p. 163)
- Disruption of venous drainage, especially in left heart failure (p. 87)

A ventilation disorder can lead to a perfusion disorder via the Euler–Liljestrand reflex since in less well-ventilated sections of the lung the vessels are constricted in order to optimize the ventilation–perfusion relationship.

Perfusion disorders stress the right heart and can lead to a state of *cor pulmonale* (p. 142).



**Fig. 8.3a–c** Chemical respiratory stimuli: **a**  $\text{CO}_2$  response; **b** pH response; **c**  $\text{O}_2$  response.

**Table 8.2** Causes of ventilation disorders

Type of disorder	Definition	Localization of cause	Examples
Obstructive ventilation disorder	Airways are narrowed or obstructed	• Upper airways	<ul style="list-style-type: none"> <li>• Malformations</li> <li>• Rhinitis (cold)</li> <li>• Foreign body aspiration</li> <li>• Epiglottitis</li> <li>• Sleep apnea syndrome (p. 165)</li> </ul>
		• Lower airways	<ul style="list-style-type: none"> <li>• Acute bronchitis (p. 148)</li> <li>• Chronic bronchitis (p. 148)</li> <li>• Pulmonary emphysema (p. 148)</li> <li>• Bronchial asthma (p. 153)</li> </ul>
Restrictive ventilation disorder	Reduced elasticity of the lung–thorax–diaphragm system	• Pulmonary	<ul style="list-style-type: none"> <li>• Status post partial resection of the lung</li> <li>• Pulmonary fibrosis (p. 160)</li> <li>• Pleural effusion (p. 167)</li> </ul>
		• Extrapulmonary	<ul style="list-style-type: none"> <li>• Deformities of the thorax, e.g., scoliosis, Bechterew disease</li> <li>• Neuromuscular diseases</li> </ul>

## Diffusion Disorders

Diffusion disorders disrupt gas exchange between alveoli and capillaries. Since CO<sub>2</sub> can diffuse significantly faster than O<sub>2</sub>, the first effect is hypoxemia, a decrease in the partial pressure of O<sub>2</sub> in the blood, and then subsequently hypercapnia, an increase of the partial pressure of CO<sub>2</sub> in the blood. There are functional barriers between the alveoli and capillaries in:

- Pulmonary fibrosis (p. 160)
- Pulmonary emphysema (p. 148)
- Pneumonia (p. 155)
- Pulmonary edema (p. 87)

## Cardinal Pulmonary Symptoms

### ■ Overview

Important cardinal pulmonary symptoms are:

- Dyspnea (p. 49)
- Cyanosis (p. 43)
- Chest pain (p. 50)
- Coughing and expectoration

### ■ Coughing and Expectoration

Coughing is a physiological protective mechanism to clear the airways, but it can also be a sign of pulmonary disease. Depending on duration, distinctions are made among:

- Acute cough lasting up to 3 weeks, e.g., in acute bronchitis, pneumonia, pulmonary embolism, and aspiration of a foreign body
- Spasmodic coughing as in bronchial asthma
- Chronic coughing lasting longer than 3 weeks, e.g., in chronic bronchitis, tumors, or bronchiectases.

In addition, a distinction is made between:

- Coughing without expectoration, also called dry or irritated cough
- Coughing with expectoration, also called productive cough.

Expectoration can involve bronchial secretions (sputum) or blood (**Table 8.3**).

■ *Chronic cough and bloody expectoration must be diagnostically clarified.*

## Complications of Pulmonary Diseases

In this section we will discuss complications that can occur in a number of pulmonary diseases:

- Respiratory insufficiency
- Pulmonary hypertension and cor pulmonale
- Atelectases
- Bronchiectases

### ■ Respiratory Insufficiency

Bronchopulmonary diseases can reduce respiratory efficiency to such an extent that there is a change

**Table 8.3** Diagnostic indications provided by sputum quality

Sputum	Possible causes
Cough urge without expectoration	<ul style="list-style-type: none"> <li>• Interstitial processes, e.g., in:               <ul style="list-style-type: none"> <li>– Interstitial pneumonia (p. 155)</li> <li>– Interstitial pulmonary disease and pulmonary fibrosis (p. 160)</li> </ul> </li> <li>• Side-effects of medication, e.g. in ACE inhibitors</li> </ul>
Thick, glassy secretion	Bronchial asthma (p. 153);
Serous secretion	Virus infection
Yellowish secretion	Bacterial superinfection
Hemoptysis	Sputum stained with a small amount of blood, e.g., in: <ul style="list-style-type: none"> <li>• Bronchitis (p. 148)</li> <li>• Pneumonia (p. 155)</li> <li>• Bronchiectases (p. 143)</li> <li>• Bronchial carcinoma (p. 157)</li> <li>• Tuberculosis (p. 268)</li> </ul>
Hemoptoe	Coughing up of large quantities of blood, e.g., in: <ul style="list-style-type: none"> <li>• Bronchiectases (p. 143)</li> <li>• Tuberculosis (p. 268)</li> <li>• Central bronchial carcinoma (p. 157)</li> <li>• Trauma</li> </ul>

in the blood gases. Blood gas analysis (BGA, p. 147) provides information as to whether there is a partial or a global respiratory insufficiency.

- Partial respiratory insufficiency:
  - Hypoxemia
  - The partial pressure of CO<sub>2</sub> is normal or diminished because of increased respiratory activity
- Global respiratory insufficiency:
  - Hypoxemia
  - Hypercapnia
  - Respiratory acidosis

*There is a difference between partial and global respiratory insufficiency.*

In global respiratory insufficiency the central respiratory drive is absent (p. 139), whereas the peripheral respiratory drive is still activated by lack of oxygen. Uncontrolled administration of oxygen deprives the patient of the remaining respiratory drive and thus puts them in a life-threatening situation.

*In global respiratory insufficiency, oxygen is administered with BGA monitoring.*

## Symptoms

### Partial Respiratory Insufficiency

- Dyspnea
- Tachycardia
- Possible central cyanosis (p. 43)
- Possible confusion and altered consciousness
- Possible clubbed digits resulting from chronic lack of oxygen (p. 49)

### Global Respiratory Insufficiency

In addition to the symptoms of partial insufficiency there are headache, vertigo, and sweating as expression of hypercapnia. As the disorder progresses, CO<sub>2</sub> narcosis can develop (p. 139).

## Therapy

Treatment of the underlying disease is a significant component of therapy. The goal of symptomatic therapy is to improve oxygenation and elimination of CO<sub>2</sub>. In addition to physical therapy and respiratory therapy, treatment with oxygen and, if appropriate, the use of respiratory aids are options for symptomatic therapy.

### Treatment with Oxygen

Patients with partial respiratory insufficiency can receive oxygen without danger, either through nasal prongs or via a face mask. Oxygen can also be administered at home over the long term.

In patients with global respiratory insufficiency, uncontrolled administration of oxygen is life-threatening because hypercapnia has already disabled the respiratory drive, so that now the drive is only activated by a lack of oxygen. If such patients receive uncontrolled oxygen, they are deprived of the remaining stimulus to breathing. In such cases, oxygen must be administered under meticulous BGA monitoring. If the pCO<sub>2</sub> continues to rise, respiratory support is required.

### Use of Respiratory Support and Ventilation

Respiratory support is indicated when the patient can no longer exert the respiratory effort required for adequate gas exchange. Of the many procedures that support breathing, two important forms are described here:

- *Continuous positive airway pressure* (CPAP) using a CPAP mask can reduce the patient's respiratory effort and support spontaneous breathing. This procedure is indicated if the respiratory mus-

cles are exhausted and there is a threat of global respiratory insufficiency. The patient can use this noninvasive ventilatory procedure at home, for instance at night.

- If the patient no longer spontaneously breathes enough or if there is a gas exchange disorder that cannot be overcome in any other way, *controlled mechanical ventilation* in an intensive care unit is required. For this procedure, the patient is sedated and intubated with an endotracheal tube. The ventilation apparatus takes over the entire work of respiration.

### Lung Transplantation

Lung transplantation (LTX) is the last resort for the most severe respiratory insufficiency resulting from a variety of irreversible diseases, for example, in advanced pulmonary fibrosis or mucoviscidosis.

### ■ Pulmonary Hypertension and Cor Pulmonale

#### Pathomechanism of Pulmonary Hypertension

The pulmonary circulation is part of the low-pressure system (p. 85). The myocardium of the right ventricle is of sufficient strength to overcome the low maximal pressure of 20 mmHg.

Any factor that reduces the overall vascular diameter of the pulmonary circulation raises the flow resistance of the pulmonary arteries and thus the pressure in the pulmonary circulation. This results in pulmonary hypertension, which places a stress on the right ventricle.

■ *Total diameter ↓ → resistance to flow in the pulmonary arteries ↑ → pulmonary hypertension*

#### Cor Pulmonale

Enlargement of the right heart caused by stress resulting from a disorder in pulmonary function is called cor pulmonale. This can develop suddenly or over a long period. The clinical signs of right heart failure are given on page 87.

#### Acute Cor Pulmonale

When parts of the pulmonary arterial circulation are suddenly blocked by an embolus (p. 163), the pressure and thus the resistance to flow in the pulmonary circulation are abruptly increased. The right ventricle is not able to adapt to the new conditions rapidly enough and acute cor pulmonale results.

■ *Pulmonary embolism → acute cor pulmonale*

### Chronic Cor Pulmonale

Chronic cor pulmonale is often caused by ventilation disorders that lead to pulmonary vasoconstriction. The physiological background is the Euler-Liljestrand reflex (p. 137).

Through this mechanism, diseases like bronchial asthma, COPD, and pulmonary fibrosis develop a gradual increase of pressure in the pulmonary circulation, to which the right ventricle can adapt itself at first by hypertrophy. Chronic cor pulmonale can also develop in perfusion disorders such as relapsing small pulmonary embolisms.

■ *Ventilation disorders → perfusion disorders → slow rise in pressure → chronic cor pulmonale*

### ■ Atelectasis

#### Definition and Causes

Atelectasis is defined as lung tissue empty of air but without inflammatory changes.

*Primary atelectasis* is present when sections of the lungs have never been ventilated. It can occur in newborn or premature infants if the lungs did not expand completely with the first breath, for example, when there is:

- Lack of surfactant (p. 137), especially in premature infants
- Aspiration of embryonic fluid
- Damage to the respiratory center

*Secondary atelectasis* is produced by the collapse of alveoli that have already been ventilated. Three different forms are distinguished (they are given in **Table 8.4** with examples):

- In *obstructive atelectasis*, also known as *resorption atelectasis*, the airways are blocked and the sections downstream of the obstruction can no longer be ventilated. The gas mixture that is still present is absorbed in time and the alveoli collapse.
- In *compression atelectasis*, pressure prevents adequate ventilation.
- *Relaxation atelectasis* results when the lung collapses as a result of pneumothorax (p. 166).

■ *Postoperative pain therapy and physical therapy encourage diaphragmatic breathing and thus prevent formation of atelectasis.*

### Complications

- Respiratory insufficiency
- Infections and abscesses

### Diagnosis and Therapy

Chest radiography gives diagnostic information (Fig. 8.4). In addition to causal treatment such as removal of foreign bodies, the following symptomatic measures can be considered:

- Respiratory therapy
- Antibiotics for infections
- Segment or lobe resection in chronic atelectasis

**Table 8.4** Types and causes of secondary atelectasis

Type	Causes
Obstruction atelectasis	<ul style="list-style-type: none"> <li>• Bronchial carcinoma (p. 157)</li> <li>• Foreign bodies</li> <li>• Secretion</li> </ul>
Compression atelectasis	<ul style="list-style-type: none"> <li>• Reduced diaphragmatic breathing, e.g., postoperatively</li> <li>• Thoracic deformities, e.g., marked scoliosis</li> <li>• Pleural effusion (p. 167)</li> <li>• Pulmonary embolism resulting from reduction of surfactant (p. 163)</li> </ul>
Relaxation atelectasis	<ul style="list-style-type: none"> <li>• Pneumothorax (p. 166)</li> </ul>



**Fig. 8.4** Chest radiograph: atelectasis in the right upper lobe.

### ■ Bronchiectasis

#### Definition and Causes

Bronchiectases are irreversible pouch-shaped or cylindrical distensions of the bronchi, principally in consequence of an underlying pulmonary disease (Fig. 8.5). As a result of the chronic inflammation, the walls of the bronchi disintegrate and elastic fibers and smooth muscles are destroyed. Bronchial obstruction produces unphysiological pressures that distend the weakened airways.

Possible congenital causes are:

- Mucoviscidosis (p. 161)
- Immune deficiencies such as IgA deficiency
- Primary ciliary dyskinesia, an autosomal recessive disease in which the mobility of the cilia in the airways is reduced. The reduced mucociliary clearance favors bronchopulmonary infections.

Examples of acquired causes are:

- COPD (page 148)
- Bronchial stenosis caused by tumors and foreign bodies

#### Symptoms and Complications

The distended bronchi collect secretions that can be cleared by coughing after a change in position and



**Fig. 8.5** Autopsy findings: extended bronchiectases filled with secretion. Healthy lung tissue is hardly recognizable. (Riede 2004.)



that present an ideal environment for microorganisms. Typical signs of disease are:

- A high volume of expectoration
- Three-layered, sweetish smelling sputum consisting of pus, slime, and foam

Some important complications are:

- Relapsing bronchopulmonary infections, lung abscesses, and fungal infestations
- Pulmonary bleeding
- Respiratory insufficiency

### Diagnosis and Therapy

Bronchiectases can be visualized by imaging procedures, such as computed tomography, which has replaced bronchography. Therapy consists of both surgical and conservative measures:

- Segment or lobe resection in locally limited bronchiectases
- Mobilization of secretions
- Inoculations
- Where appropriate, bronchospasmolysis and specific antibiotic therapy

## Pulmonological Diagnosis

### ■ Physical Examination

#### Inspection

Inspection reveals principally:

- The shape of the thorax, e.g., deformation of the thorax or barrel chest in hyperdistension
- The respiratory excursion, e.g., symmetry, thoracic or abdominal breathing
- Indications of dyspnea such as elevated respiratory rate, choosing a bodily position to facilitate breathing, and use of the auxiliary respiratory muscles
- Cyanosis, etc.

#### Percussion

Percussion of the thorax can determine the borders of the lungs in inspiration and expiration and thus the respiratory displacement. In a healthy person, this is 3–5 cm.

In addition, percussion yields a characteristic sound that is described as sonorous. Differences in sound on percussion reveal pathological deviations:

- A loud, deep sound on percussion is called hyper-sonorous and is the result of an elevated air content in the lungs, such as in pulmonary emphysema.
- A muted sound on percussion indicates infiltration, e.g., in pneumonia or pleural effusion.

### Auscultation

#### Physiological Auscultation Findings

In a healthy person, auscultation with the stethoscope yields:

- A relatively loud, rough sound over the trachea and the large bronchi, caused by turbulent flow and called bronchial breathing
- On the periphery, a soft, low inspiratory sound caused by laminar flow, called vesicular breathing

#### Pathological Auscultation Findings

A weakened or absent breathing sound can occur, for example, in emphysema or pneumothorax. Stridor, wheezing, and crepitation are pathological breath sounds.

- Stridor is a whistling breath sound, occurring with constricted airways, that can be heard even without a stethoscope. If the upper respiratory airways are constricted by swelling, secretion, or foreign bodies, inspiratory stridor results, i.e., whistling on inhalation. Bronchial obstruction, on the other hand, leads to expiratory stridor.

- *Inspiratory stridor in obstruction of the upper airways*
- *Expiratory stridor in bronchial obstruction*

- Wheezing is a variable breath sound that can be heard as whistling, squeaking, or humming. It is caused by obstruction such as secretion and can sometimes be heard even without a stethoscope.
- Crackling is a background noise of breathing that can be heard with a stethoscope. It is caused by accumulated fluid, e.g., pulmonary edema or accumulation of secretions.

### ■ Pulmonary Function Test

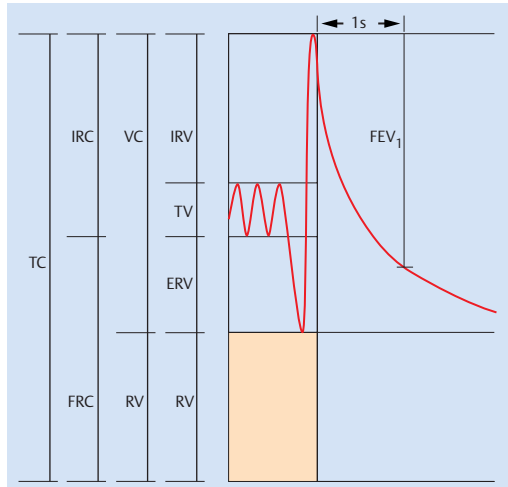
The following procedures are used in a pulmonary function test:

- Spirometry
- Whole-body plethysmography
- Compliance measurement



## Spirometry

Ventilation can be evaluated by means of spirometry. Both static values, as shown in **Fig. 8.6** and summarized in **Table 8.5**, and dynamic values can be determined with spirometry.



**Fig 8.6** Lung volumes and capacities (TV = tidal volume; IRV = inspiratory reserve volume; ERV = expiratory reserve volume; RV = residual volume; VC = vital capacity; IRC = inspiratory reserve capacity; FRC = functional residual capacity; TC = total lung capacity; FEV<sub>1</sub> = forced expiratory volume in 1 second).

**Table 8.5** Lung volumes and capacities

Static size	Definition	Averages for men. Values in women are up to 20% lower
Tidal volume (TV)	Quantity of air that is inhaled and exhaled with each breath at rest	500 mL
Inspiratory reserve volume (IRV)	Additional amount of air that can be inhaled after unforced inspiration	~2500 mL
Expiratory reserve volume (ERV)	Additional amount of air that can be exhaled after unforced expiration	~1500 mL
Residual volume (RV)	<ul style="list-style-type: none"> <li>Amount of air remaining in the lungs after a maximal expiration</li> <li>The RV cannot be measured spirometrically</li> </ul>	<ul style="list-style-type: none"> <li>~1500 mL</li> <li>20% TC</li> </ul>
Vital capacity (VC)	<ul style="list-style-type: none"> <li>Maximal amount of air that can be exhaled after a maximal inspiration</li> <li>TV + IRV + ERV</li> </ul>	<ul style="list-style-type: none"> <li>~4500 mL</li> <li>80% TC</li> </ul>
Total capacity (TC)	<ul style="list-style-type: none"> <li>Amount of air in the lungs after a maximal inspiration</li> <li>VC + RV</li> <li>The TC cannot be measured spirometrically</li> </ul>	~6000 mL
Functional residual capacity (FRC)	<ul style="list-style-type: none"> <li>Amount of air in the lungs after an unforced expiration</li> <li>ERV + RV</li> <li>The FRC cannot be measured spirometrically</li> </ul>	~3000 mL
Inspiratory reserve capacity (IRC)	<ul style="list-style-type: none"> <li>Maximal amount of air that can be inhaled after unforced expiration</li> <li>TV + IRV</li> </ul>	~3000 mL

- The *one-second capacity* is determined with the *Tiffeneau test*. The patient inhales slowly and as deeply as possible and then exhales as rapidly as possible. The spirometer measures how much the patient exhales in the first second. In a person with healthy lungs, the forced expiratory volume in the first second (FEV<sub>1</sub>) is equal to 80% of the vital capacity (VC). This value is lower in patients with obstructive ventilation disorders because of the constricted airways.

■ *One-second capacity or FEV<sub>1</sub> = 80% VC*

- In the *bronchospasmolysis test*, the Tiffeneau test is repeated after inhalation of a bronchodilator. In this way it can be determined whether the previously demonstrated obstruction is reversible, e.g., in bronchial asthma (p. 153).
- The peak expiratory flow is measured with the *peak flow meter*. This is a simple device given to patients so that they can measure and monitor themselves. Normal values depend on sex, age, and height and are presented in **Fig. 8.7**.

## Whole-Body Plethysmography

The whole-body plethysmograph is a closed chamber with a volume of about 1 m<sup>3</sup> in which the patient is seated. In contrast to spirometry, the examination

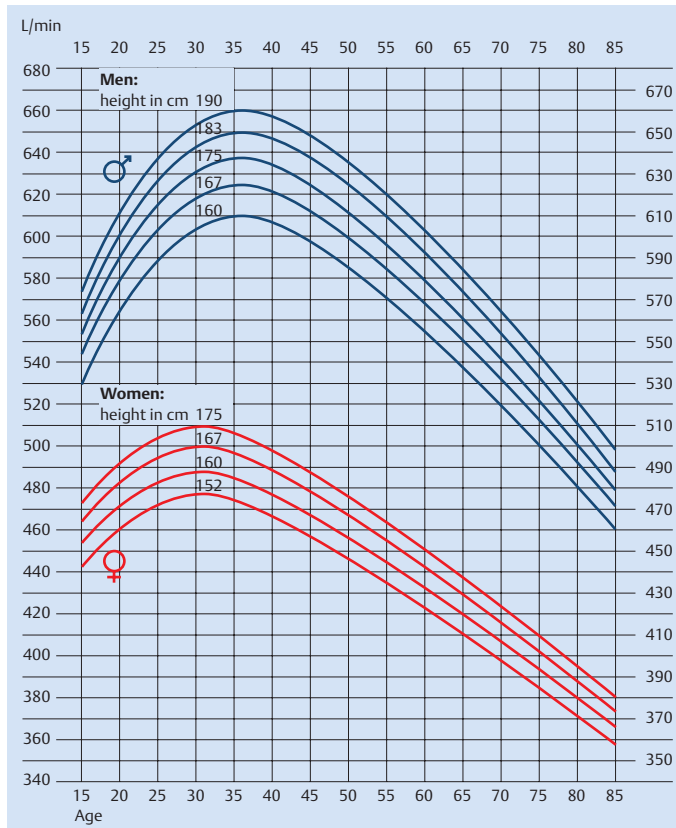


Fig. 8.7 Normal peak flow values.

does not depend on the patient's cooperation. In addition, values can be measured that cannot be recorded with spirometry:

- Residual volume, total capacity, and functional residual capacity
- Airway resistance, known simply as resistance

### Lung Compliance

Compliance is a measure of the expansibility of the lung–thorax–diaphragm system. The quantity measured is the change in volume of the lung per unit of intrathoracic pressure difference. The patient must swallow a pressure probe that measures pressure in the esophagus at the beginning and end of inspiration. The difference corresponds to the intrathoracic pressure difference.

The normal value is 0.03–0.05 L/kPa. Lower values indicate decreased expansibility and thus a restrictive ventilation disorder. This examination is seldom used because it is unpleasant for the patient and equivalent information can be obtained by measuring the vital capacity.

### Pulmonary Function in Ventilation Disorders

The different measurement results in obstructive and restrictive ventilation disorders are summarized in **Table 8.6**.

- The changed values in *obstructive ventilation disorders* can be ascribed to the fact that constricted airways interfere especially with exhalation, which is normally a passive process. Air remains in the alveoli, and this is called *air trapping* (**Fig. 8.8**). Thus an obstruction is expressed, in addition to increased resistance to flow, by decreased  $FEV_1$  and increased residual volume.

Patients and physical therapists can determine the extent of the obstruction themselves, by measuring the expiratory peak flow with a peak flow meter.

- In *restrictive ventilation disorders*, the lung–thorax–diaphragm system is no longer as elastic as in a person with healthy lungs, so that the total capacity and all its constituent values decrease. Decreased compliance is also an expression of decreased expansibility.