

**Chapter 28: The Chest**

**Pulmonary Volumes and Capacities**

Tidal volume (TV) = depth of breathing = volume of gas inspired or expired during each normal respiratory cycle = 0.5 L on the average.

Inspired reserve volume (IRV) = maximum volume that can be inspired from end-inspiratory position = 3.3 L on the average.

Expired reserve volume (ERV) = maximum volume that can be expired from end-respiratory level = 0.7-1 L on the average.

Residual volume (RV) = volume left in lungs after maximum expiration = 1.1 L on the average.

Forced expiratory volume in 1 second = FEV1 (FEV1 should be 80% or more of predicted value from a normative chart).

Forced vital capacity = FVC (FVC should be 80% or more of predicted value from a normative chart).

The ratio of FEV1/FVC should be greater than 0.75 for young patients and 0.70 for older individuals.

Total lung capacity (6 L for males; 4.2 L for females)

$$\mathbf{TLC = IRV + TV + ERV + RV}$$

(Total volume contained in the lungs after maximum inspiration).

Vital capacity (4.8 L for males; 3.1 L for females)

$$\mathbf{VC = IRV + TV + ERV}$$

(Maximum volume that can be expelled from the lungs by forceful effort following maximum inspiration).

Functional residual capacity (2.2 L for males; 1.8 L for females)

$$\mathbf{FRC = RV + ERV}$$

(Volume in the lungs at resting expiratory level).

Physiological dead space (dead space of upper airway bypassed by tracheotomy 70-100 cc).

Physiological dead space = Anatomical dead space + the volume of gas that ventilates the alveoli that have no capillary blood flow + the volume of gas that ventilates the alveoli in excess of that required to arteriolize the capillary blood.

### Mean Normal Blood Gas and Acid Base Values

	Arterial Blood	Mixed Venous Blood
pH	7.40	7.37
pCO <sub>2</sub>	41 mm Hg	46.5 mm Hg
pO <sub>2</sub>	95 mm Hg	40 mm Hg
O <sub>2</sub> sat	97.1%	75.0%
bicarbonate	24. mEq/L	25.0 mEq/L

### Miscellaneous

1. Silo-filler's disease (bronchiolitis obliterans) is a pathologic entity consisting of a collection of exudate in the bronchioles, obliterating the lumen. This complication often follows inhalation of nitrogen dioxide, exposure to open bottles of nitric acid, and exposure to silos. The diagnosis is made on history of exposure, dyspnea, cough, and x-ray findings similar to miliary tuberculosis. Treatment is symptomatic. Prognosis is poor since the majority of these patients eventually succumb to this disease.

2. Bronchogenic cysts are congenital, arise from the bronchi, and are lined with epithelial cells. Furthermore, their walls may contain glands, smooth muscles, and cartilage. In the absence of infection they may remain asymptomatic. Otherwise, they give a productive cough, hemoptysis, and fever. The recommended treatment is surgical excision.

3. Blebs or bullae: These are air-containing structures resembling cysts but their walls are not epithelium lined.

4. Anthracosilicosis: Coal miner's pneumoconiosis.

5. Berylliosis: This condition is characterized by an infiltration of lungs by beryllium. It often is found in workers at fluorescent lamp factories.

6. Bagassosis: This condition is characterized by an infiltration of the lungs by sugar cane fibers.

7. Byssinosis: This condition is characterized by an infiltration of the lungs by cotton dust.

8. Adenocarcinoma of the bronchus is the leading primary pulmonary carcinoma in females while bronchogenic (squamous cell) is most common in males.

9. Pancoast syndrome (superior sulcus tumor) is a syndrome caused by any process of the apex of the lung which can invade the pleural layers, infiltrate between the lower cords of the brachial plexus, and may involve the cervical sympathetic nerve chain, phrenic, and recurrent laryngeal nerves. It is usually secondary to a benign or malignant tumor. However, a large inflammatory process may cause this syndrome as well. The symptoms are:

a. Pain in shoulder and arm, particularly in the axilla and inner arm.

b. Intrinsic hand muscle atrophy.

c. Horner's syndrome (enophthalmos, ptosis of the upper lid, constriction of the pupil with narrowing of the palpebral fissure, and decreased sweating homolaterally).

10. Congenital agenesis of the lung has been classified by Schneider as follows:

Class I: Total agenesis.

Class II: Only the trachea is present.

Class III: Trachea and bronchi are present without any pulmonary tissue.

11. Apnea can occur after tracheotomy. This is due to carbon dioxide narcosis causing the medulla to be depressed. Prior to the tracheotomy, the patient was breathing secondary to the lack of oxygen. After the tracheotomy this oxygen drive is removed and hence the patient remains apneic. Treatment for this is to ventilate the patient until the excess carbon dioxide level is reduced. Mediastinal emphysema and pneumothorax are the most common complications of tracheotomy. (For other complications, see Chap. 15).

12. Hypoxemia is defined as less than 75% oxygen saturation or less than 40 mm Hg pO<sub>2</sub>. A level greater than 5 mg% of met-Hb gives cyanosis.

13. Bronchogenic cyst is a defect at the fourth week of gestation. It constitutes less than 5% of all mediastinal cysts and tumors.

14. The bronchial tree ring is cartilaginous till it reaches 1 mm in diameter. These small bronchioles without cartilaginous rings are held patent by the elastic property of the lung. The bronchial tree is lined by pseudostratified columnar ciliated epithelium as well as nonciliated cuboidal epithelium.

15. The adult trachea measures 10-12 cm and has 16-20 rings. The diameter is approximately 20 mm x 15 mm.

16. The larynx descends on inspiration and ascends on expiration. It also ascends in the process of swallowing and in the production of a high-pitched note.

17. The esophageal lumen widens on inspiration.

18. The total lung surface measures 70 m<sup>2</sup>. The lung contains 300 million alveoli. The lung secretes 200 mL of fluid per day.

19. During inspiration the nose constitutes 79% of the total respiratory resistance, the larynx 6%, and the bronchial tree 15%. During expiration the nose constitutes 74% of resistance, the larynx 3%, and the bronchial tree 23%.

20. Tracheopathia osteoplastica is a rare disease characterized by growths of cartilage and bone within the walls of the trachea and bronchi that produce sessile plaques that project into the lumen. There is no specific treatment other than supportive. It is of unknown etiology. The serum calcium is normal and there are no other calcium deposits.

21. Calcification found in a pulmonary nodule usually implies that it is a benign nodule.

22. Middle lobe syndrome (see Chap. 23).

23. The right upper lobe and its bronchus is the lobe that is most susceptible to congenital anomaly.

24. Cystic fibrosis (mucoviscidosis) is familial, and may be autosomal recessive. The patient presents with multiple polyps, pulmonary infiltration with abscesses, and rectal prolapse. The pancreas is afflicted with a fibrocystic process and produces no enzymes. Trypsin is lacking in the gastric secretion. Ten to fifteen percent of the patients pass trypsin in the stool. There is general malabsorption of liposoluble vitamins. Treatment consists of a high-protein, low-fat diet with water-soluble vitamins and pancreatic extracts. Many patients die of pulmonary abscesses.

25. If a person is ventilated with pure oxygen for 7 minutes, he is cleared of 90% of the nitrogen and can withstand 5-8 minutes without further oxygenation.

### **The Mediastinum**

1. Suprasternal fossa:

a. This is the region in which the sternocleidomastoid muscles converge toward their sternal attachments. Bound inferiorly by the suprasternal notch, they, however, have no superior boundary.

b. The deep cervical fascia splits into an anterior and a posterior portion. These are attached respectively to the anterior and posterior margins of the manubrium.

c. The space between these fascial layers is the small suprasternal space containing:

1) Anterior jugular veins

2) Fatty connective tissues

d. Behind this space lies the pretracheal fascia.

e. Laterally on each side are the medial borders of the sternohyoid and sternothyroid

muscles.

2. In the adult, the innominate artery crosses in front of the trachea, behind the upper half of the manubrium. In the child, it crosses over the level of the superior border of the sternum.

3. The trachea enters the mediastinum on the right side.

4. The trachea bifurcates at T4-5 or about 6 cm from the suprasternal notch. As a person approaches 65 years of age or more, it is possible that the trachea bifurcates at T6.

5. To the left of the trachea are: aorta, left recurrent laryngeal nerve, left subclavian artery.

To the right of the trachea are: superior vena cava, azygos vein, right vagus, right lung pleura.

6. The innominate and left carotid arteries lie anterior to the trachea near their origin. As they ascend, the innominate artery lies to the right of the trachea.

7. The pulmonary artery passes anterior to the bronchi and assumes a superior position to the bronchi at the hilus with the exception that the right upper lobe bronchus is superior to the right pulmonary artery.

8. The left main bronchus crosses in front of the esophagus. It presses on the esophagus and together with the aorta forms the bronchoaortic constriction. The first part of the aorta is to the left of the esophagus. As it descends it assumes a left posterolateral position to the esophagus.

9. The course of the esophagus is as shown in Fig. 28-1. The esophagus has four constricting points.

- a. Cricopharyngeus muscle
- b. Aorta crossing
- c. Left main stem bronchus crossing
- d. Diaphragm

(a<b=c<d)

At the level of c the esophagus passes from superior mediastinum to the posterior mediastinum.

10. The following structures are found within the concavity of the aorta:

- a. Left main stem bronchus

- b. Left recurrent laryngeal nerve
- c. Tracheobronchial nodes
- d. Superficial part of the cardiac plexus.

11. The right main stem bronchus is wider, shorter, and follows a more vertical course than the left one.

12. The inferior thyroid vein is immediately in front of the trachea in its infraisthmic portion.

13. Ten percent of the population has a thyroidea ima artery. It arises from either the innominate artery or the aorta. It passes upward along the anterior aspect of the trachea.

### **The Course of the Vagus**

Left:

1. It passes inferiorly between the left subclavian and the left carotid.
2. It follows the subclavian to its origin.
3. It passes to the left of the arch of the aorta.
4. It gives off the recurrent laryngeal nerve which passes superiorly along the left border of the tracheoesophageal groove (between the esophagus and trachea).
5. The main vagus continues to descend behind the left main stem bronchus.

Right:

1. It descends anterior to the subclavian where it gives off the recurrent laryngeal nerve which loops around the subclavian artery and ascends posteromedial to the right common carotid artery to reach the tracheoesophageal groove (between the esophagus and the trachea)
2. The main trunk descends posteriorly along the right side of the trachea, between the trachea and right pleura.
3. It descends posterior to the right bronchus.

### **Fascia of the Mediastinum**

The space between the various organs is occupied by loose areolar tissue. The fascial layers of the mediastinum are a direct continuation of the cervical fascia. A portion of the cervical fascia, the perivisceral fascia, encloses the larynx, pharynx, trachea, esophagus, thyroid, thymus, and carotid sheath contents. This space enclosed by this perivascular fascia

extends to the bifurcation of the trachea. Anteriorly it is bound by pretracheal fascia. The pretracheal fascia is an important landmark in mediastinoscopy in that dissection should be done only beneath this layer.

### **Boundaries of the Mediastinum (Fig. 28-2)**

1. Lateral: parietal pleura
2. Anterior: sternum
3. Posterior: vertebra
4. Inferior: diaphragm
5. Superior: superior aperture of the thorax.

### **Superior Mediastinum**

1. Boundaries:

Superior: superior aperture of the thorax

Anterior: manubrium with sternothyroid and sternohyoid muscles

Posterior: upper thoracic vertebra

Inferior: plane from manubrium to IV vertebra.

2. Structures of the superior mediastinum: thymus, innominate veins, aorta, vagus, recurrent laryngeal nerve, phrenic nerve, azygos vein, esophagus, thoracic duct.

### **Anterior Mediastinum**

It lies between the body of the sternum and the pericardium and contains:

1. Loose areolar tissue
2. Lymphatics
3. Lymph nodes
4. Thymus gland.

### **Middle Mediastinum**

It contains the heart, ascending aorta, superior vena cava, azygos vein, bifurcation of the main bronchus, pulmonary artery trunk, right and left pulmonary veins, phrenic nerves, and the tracheal-bronchial lymph nodes.

## **Posterior Mediastinum**

Anteriorly lies the bifurcation of the trachea, the pulmonary vein, the pericardium, and the posterior part of the upper surface of the diaphragm. Posteriorly lies the vertebral column from T-4 to T-12. Laterally lies the mediastinal pleura.

The posterior mediastinum contains the thoracic aorta, azygos vein, hemiazygos vein, cranial nerve X, splanchnic nerve, esophagus, thoracic duct, posterior mediastinal lymph nodes, and the intercostal arteries.

### **Lymph Nodes of the Thorax (Fig. 28-3)**

1. Parietal nodes are inconsequential clinically. They are grouped into intercostal, sternal, and phrenic nodes.

2. Visceral nodes are of greater clinical importance. They are grouped as follows:

a. Peritracheobronchial

1) Paratracheal

2) Pretracheal

3) Superior tracheobronchial

4) Inferior tracheobronchial

b. Bronchopulmonary

c. Anterior mediastinal or prevascular

d. Pulmonary

e. Posterior mediastinal.

### **Lymphatic Drainage of the Lung**

1. Right

Superior area (anteromedial area of the right upper lobe): right paratracheal nodes.

Middle area (posterolateral area of right upper lobe, right middle lobe, and superior right lower lobe): right paratracheal nodes and inferior tracheal-bronchial nodes.

Inferior area (lower half of right lower lobe): inferior tracheal-bronchial nodes and posterior mediastinal nodes.

## 2. Left

Superior area (upper left upper lobe): left paratracheal, anterior mediastinal, and subaortic nodes.

Middle area (lower left upper lobe and upper left lower lobe): left paratracheal, inferior tracheobronchial, and anterior mediastinal nodes.

Inferior area (inferior part of the left lower lobe): inferior tracheobronchial nodes.

The left inferior tracheobronchial nodes drain into the right paratracheal nodes.

3. Right upper lung --> right neck.

Right lower lung --> right neck.

Left lower lung --> right neck.

Left upper lung --> left neck.

Lingular lobe --> both sides of the neck.

### **Purposes of Mediastinoscopy**

Barium swallow and tracheogram are usually obtained before mediastinoscopy if indicated.

1. Histologic diagnosis
2. To determine which nodes are involved
3. To make the diagnosis of sarcoidosis.

### **Mediastinal Tumors**

1. One-third of all mediastinal tumors are malignant. Among the malignant ones, lymphoma is most commonly encountered.

2. Superior mediastinum: Thyroid, neurinoma, thymoma, parathyroid.

Anterior mediastinum: Dermoid, teratoma, thyroid, thymoma.

Low anterior mediastinum: Pericardial cyst.

Middle mediastinum: Pericardial cyst, bronchial cyst, lymphoma, carcinoma.

Posterior mediastinum: Neurinoma and enterogenous cyst.

## Superior Vena Cava Syndrome

1. Etiology: Malignant metastasis, mediastinal tumors, mediastinal fibrosis, vena cava thrombosis.

2. Signs and symptoms include: Edema and cyanosis of the face, neck, and upper extremities. Venous hypertension with dilated veins. Normal venous pressure of lower extremities. Visible venous circulation of the anterior chest wall.

### Endoscopy

#### *Size of Tracheotomy Tubes and Bronchoscopes*

Age	Tracheotomy	Bronchoscope
Premature	No. 000 x 26 to No. 00 x 33 mm	3 mm
6 months	No. 0 x 33 mm to No. 0 x 40 mm	3.5 mm
18 months	No. 1 x 46 mm	4 mm
5 years	No. 2 x 50 mm	5 mm
10 years	No. 3 x 50 mm to No. 4 x 68 mm	6 mm
Adult		7 mm

#### Size of Esophagoscope

Child	5 x 35 or 6 x 35 cm
Adult	9 x 50 cm.

During esophagoscopy, the average distance from the incisor teeth to the:

	Adult	3 years	1 years	Birth
Cricopharyngeus muscle	16 cm	10	9	7
Aorta	23 cm	15	14	12
Left bronchus	27 cm	16	15	13
Hiatus	38 cm	23	21	19
Cardiac	40 cm	25	23	21
Greater curvature of the stomach	53 cm	30	27	23.

### Left Lung

Lobes	Segments
Upper division of upper lobe	a. Apical-posterior b. Anterior
Lower division of upper lobe	a. Superior b. Inferior

Lower lobe	<ul style="list-style-type: none"> <li>a. Superior</li> <li>b. Anteromedial basal</li> <li>c. Lateral basal</li> <li>d. Posterior basal</li> </ul>
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### **Right Lung**

Lobes	Segments
Upper lobe	<ul style="list-style-type: none"> <li>a. Apical</li> <li>b. Posterior</li> <li>c. Anterior</li> </ul>
Middle lobe	<ul style="list-style-type: none"> <li>a. Lateral</li> <li>b. Medial</li> </ul>
Lower lobe	<ul style="list-style-type: none"> <li>a. Superior</li> <li>b. Medial basal</li> <li>c. Anterior basal</li> <li>d. Lateral basal</li> <li>e. Posterior basal.</li> </ul>

Relative contraindications for esophagoscopy:

1. Aneurysm of the aorta.
2. Spinal deformities, osteophytes.
3. Esophageal burns and being treated with steroids.

Relative contraindications for bronchogram:

1. Acute infection.
2. Acute asthmatic attacks.
3. Acute cardiac failure.

Causes for hemoptysis: (in order of decreasing frequency)

1. Bronchiectasis.
2. Adenoma.
3. Tracheobronchitis.
4. Tuberculosis.

5. Mitral stenosis.

Foreign bodies:

Right upper lobe bronchus:	most common
Left upper lobe bronchus:	second most common
Trachea:	least likely

Most common site for esophageal foreign bodies is the cervical esophagus.

Most common foreign bodies in children are peanuts, safety pins, coins.

Most common foreign bodies in adults are meat and bone.

**Vascular Anomalies** (See Chap. 11, Fig. 11-2a, b, c)

1. Double aortic arch: This is a true vascular ring. It is due to the persistence of the right fourth branchial arch vessel. The symptoms include stridor, intermittent dysphagia, and aspiration pneumonia. The right posterior arch is usually the largest of the two arches.

2. Right aortic arch with ligamentum arteriosum: This is due to the persistence of the right fourth branchial arch vessel becoming the aorta instead of the left fourth arch vessel. This crosses the trachea causing an anterior compression.

3. Anomalous right subclavian artery: This is due to the right subclavian artery arising from the dorsal aorta giving a posterior compression of the esophagus. There is no constriction over the trachea.

4. Anomalous innominate and/or left common carotid: The innominate arises too far left from the aorta. It crosses the trachea anteriorly causing an anterior compression. The left common carotid arises from the aorta on the right or from the innominate artery. It also causes an anterior compression of the trachea. In another variant of this anomaly, the innominate and the right common carotid arise from the same trunk and in dividing, encircle the trachea and esophagus causing airway obstruction as well as dysphagia.

5. Patent ductus arteriosus.

6. Coarctation of the aorta.

7. An enlarged heart especially with mitral insufficiency can compress on the left bronchus.

8. Dysphagia lusoria is a term used to include dysphagia caused by any aberrant great vessel. The common cause is an abnormal subclavian artery arising from the descending aorta.

9. Anomalous innominate arteries have been estimated to be the most common vascular anomaly. They cause an anterior compression on the trachea. During bronchoscopy if the pulsation is obliterated with the bronchoscope, the radial pulse on the right arm is

reduced while the temporal pulse is also reduced. In the case of a subclavian anomaly the bronchoscope compressing the abnormal subclavian produces a decrease of the radial pulse but the temporal pulse will remain normal. A bronchoscope compressing a double aortic arch pulsation will produce no pulse changes in either the radial or temporal pulse.

### **Basic Pulmonary Physiology**

To most physicians, pulmonary physiology is a complex discipline whose subject matter lies beyond the grasp of all but those specifically involved in pulmonary medicine. However, all practitioners should be acquainted with the fundamentals of lung function. In particular, otolaryngologists should have more than a cursory comprehension of pulmonary physiology since many of their patients will have diseases affecting both the upper airway and the lungs themselves.

This section will attempt to review the basic concepts of pulmonary physiology in a simple and concise manner.

### **Definitions**

To follow the text below, the reader will need to be familiar with the following definitions:

### **Lung Volumes**

Lung volumes can be divided into "primary volumes" and "capacities".

#### 1. Volumes

a. Tidal volume (TV): the tidal volume is the volume of gas which is either inspired or expired during each respiratory cycle.

b. Residual volume (RV): the residual volume is the amount of gas which remains in the lungs at the end of a maximal expiratory effort.

#### 2. Capacities

a. Total lung capacity (TLC): the total lung capacity is the amount of gas contained in the lungs at the end of a maximal inspiratory effort.

b. Vital capacity (VC): the vital capacity is the maximum volume of gas which is expelled when a patient makes a forceful effort after inspiring to his total lung capacity.

c. Functional residual capacity (FRC): the functional residual capacity is the volume of gas which remains in the lungs at resting expiratory level.

## **Dynamic Lung Volumes**

1. Forced expiratory volume in 1 second (FEV1): FEV1 is the volume of gas which is expelled from the lungs during the first second after the initiation of a forceful effort following a maximal inspiration.

2. FEV1/FVC ratio: The FEV1/FVC ratio is the ratio of the volume of gas expelled from the lungs 1 second after forceful effort following a maximal inspiration divided by the maximum volume of gas which is expelled from the lungs by forceful effort following a maximal inspiration.

With the above definitions in mind, we can now turn our attention to pulmonary function testing and what it can reveal to us about the status of a patient's lungs.

## **Spirometry**

The spirometer is the most useful piece of equipment in a pulmonary function testing laboratory because, using a spirometer and a nitrogen or helium analyzer, one can obtain values for the lung volumes, capacities, and dynamic lung volumes. By analyzing the data obtained, a physician can determine whether a patient has normal or abnormal lung function. Abnormal lung function has generally been divided into two main categories: (1) airflow limitation, commonly referred to as obstructive lung diseases, and (2) restrictive lung disease.

### **Diseases Producing Obstruction to Air Flow**

Any disease process which impedes the flow of air through an airway of over 2 mm in size will produce spirometric evidence for limitation to airflow. This limitation to airflow will often be seen in a reduction in the vital capacity and in the FEV1. In addition, one will find a diminution of the FEV1/FVC ration below the predicted normal. There often is an elevation in the functional residual capacity and the residual volume, and in severe cases, air trapping with an increase in total lung capacity may be seen. The finding of airflow limitation on pulmonary function testing does not indicate the cause of the patient's disease. Asthma, chronic bronchitis, and pulmonary emphysema will all produce evidence for airflow limitation. When a tracing reveals evidence for airflow limitation, the patient should be given a bronchodilator. Patients with asthma most often demonstrate a reversible component to their airway narrowing. When these patients are given a bronchodilator in the laboratory and the pulmonary function is repeated, an improvement is often seen in their studies. Most laboratories regard an improvement in FVC or FEV1 of 20% or more as an indication of reversibility. Lack of response to the bronchodilator does not mean that a patient does not have an element of reversibility, but on the other hand, an improvement in function after bronchodilator does indicate that there is a reversible component to the airflow limitation.

Patients with diseases considered to be nonreversible, such as pulmonary emphysema, most often will not demonstrate any reversibility in airflow limitation after the administration of a bronchodilator. Patients scheduled for surgery, who demonstrate evidence for airflow limitation on initial testing, should universally be given a bronchodilator. Preoperative bronchodilator therapy may greatly improve their pulmonary function, and hence reduce their operative risk if used for a significant time in the preoperative and postoperative period.

Patients with restrictive lung disease demonstrate a different pattern of pulmonary function abnormality. In patients with restrictive disease, there is a reduction in the volume of air that can be inspired. There are many disease entities that will lead to restriction. Surgical removal of lung tissue, restricted expansion of the lungs (such as may be seen in scoliosis or fibrothorax), or neurologic disorders (such as amyotrophic lateral sclerosis) which produce muscular weakness, demonstrate a restrictive pattern. In addition, diseases which replace lung tissue with granulomas also produce a restrictive pattern. This pattern is characterized in general by a reduction in vital capacity, forced vital capacity, and FEV1, but with preservation of the FEV1/FVC ratio. In reviewing pulmonary function data, it is important to evaluate the FEV1/FVC ratio since both obstructive and restrictive disease will produce reductions in forced vital capacity and FEV1. However, in airflow limitation, the FEV1/FVC ratio will be reduced, whereas in restriction, the FEV1/FVC ratio will be preserved.

Moreover, in obstructive disease, the total lung capacity is often normal or high, whereas in restriction, the total lung capacity will be found to be reduced. Patients with restrictive disease do not demonstrate improvement after the administration of a bronchodilator since the defect in restrictive disease does not lie in narrowing of the airways.

In addition to spirometric data, other techniques have recently been used to study basic pulmonary function.

### **Flow Volume Loops**

The maximal expiratory flow volume curve has been in use for the past several years. In contrast to the spirometer, in which volume is plotted against time, in the flow volume loop one measures flow as a function of lung volume. Conditions that produce airflow limitation will demonstrate reduction in flow throughout the patient's forced vital capacity maneuver. This can be seen in the accompanying Fig. 28-6, and usually presents as a concave curve in contrast to the normal convex curve seen in patients without disease. Note that the flow volume loop also provides data about inspiration. This will be discussed in subsequent sections. A bronchodilator also can be administered to a patient following a flow volume loop. An improvement in the loop also is indicative of reversible airflow limitation.

Patients with restrictive disease demonstrate a reduction in vital capacity but, as can be seen in the accompanying diagram (Fig. 28-7), the slope of the expiratory loop is normal, indicating that there is no reduction in flow rate. Many devices employing flow volume loop techniques are available for "pulmonary function screening". It should be noted that a number of these devices do not contain the additional equipment needed to determine lung volumes such as FRC and RV. Consequently these cannot provide information regarding total lung capacity.

### **Additional Values of the Flow-Volume Curve**

In addition to providing evidence concerning basic lung function, the flow-volume loop can be used to detect upper airway obstruction - a most useful test for the otolaryngologist.

The ability of the flow-volume loops to detect upper airway obstruction is based on the following physiologic principle: When a patient inspires, atmospheric pressure is greater than intratracheal pressure. For this reason, when a patient inspires, the atmospheric pressure tends to narrow the trachea. If there is an "extrathoracic" (or upper airway) obstruction, any narrowing of the extrathoracic lumen will be magnified on inspiration. Hence, the inspiratory loop of the flow volume curve will be flattened. On the expiratory portion of the flow volume loop, the intratracheal pressure is greater than atmospheric pressure. Thus, any narrowing produced by a variable resistance will be eliminated and the expiratory loop of the flow volume loop will be normal. Extra thoracic obstruction is therefore characterized by a flattened inspiratory loop with a relatively normal expiratory loop (Fig. 28-8).

In the intrathoracic cavity, the airways enlarge on inspiration. This is because inspiration produces higher lung volumes, and because the lung parenchyma exerts a tethering action on airways, lung inflation produces an increase in airway diameter. On expiration, the intrathoracic pressure increases, lung volumes decrease, and the airways narrow. Hence, any lesion which produces intrathoracic obstruction will be most manifest on expiration. Intrathoracic obstructions will be characterized by a normal inspiratory loop but an abnormal expiratory loop (Fig. 28-9).

### **Lung Compliance**

One of the most important concepts in pulmonary physiology relates to the compliance of the lungs. Compliance is regarded as a measure of distensibility. A structure that is easily distended by small changes in pressure is felt to be highly compliant. For example, a lung whose elastic tissue has been destroyed, such as an emphysematous lung, will be easily distended by small changes in pressure. This will produce a compliance curve shown in Fig. 28-10. Highly distensible lungs are inelastic, as opposed to a "stiff lung", which is poorly distensible and highly elastic. It will take a large pressure change to evoke a significant change in the lung volume. As an example, patients with pulmonary fibrosis have stiff lungs. These lungs require high pressure gradients to produce significant changes in lung volume. Thus pressure volume relationship can be seen in Fig. 28-10.

Since the adequacy of ventilation is determined in part by alveolar ventilation, it is important for the physician to consider the compliance characteristics of the lung in any patient who is being mechanically ventilated. Patients with highly compliant lungs will require small pressure changes to induce large volume changes, whereas in patients with stiff lungs, high pressures may be needed to generate adequate volumes for ventilatory demands.

### **Diffusing Capacity**

When pulmonary physicians talk about diffusing capacity of the lung, they are referring to the quantity of a specific gas that diffuses across the alveolar capillary per unit time. A full discussion of diffusing capacity is beyond the scope of this text. In most pulmonary laboratories, the gas used to measure the diffusing capacity of the lung is carbon monoxide. Carbon monoxide is rapidly bound to hemoglobin and in clinical practice, the diffusion capacity is felt to represent the volume of capillary blood into which the carbon monoxide can dissolve. Hence, diseases such as emphysema, characterized by a reduction in capillary blood volume, will have associated low diffusion capacities.

## Blood Gas Interpretation

It must be emphasized that total ventilation (tidal volume x respiratory frequency) is not the same as alveolar ventilation (volume of gas in each breath which participates in gas exchange x respiratory frequency). For the purposes of clinical medicine, alveolar and arterial carbon dioxide tensions are equal. Since alveolar ventilation is the factor which determines the level of arterial  $p\text{CO}_2$ , the adequacy of ventilation is assessed by measuring the arterial  $p\text{CO}_2$ .

### Hypoxemia

In clinical practice the most common causes of hypoxemia include two main factors: (1) simple hypoventilation and (2) ventilation-perfusion inequality. Other causes for hypoventilation include anatomic shunts and abnormalities of diffusion. The latter two are rarely found in a clinical practice and will not be discussed.

A useful technique for evaluating the presence of intrinsic lung disease is determination of the alveolar-arterial or A-a gradient. One simple way to do this is to assume that the alveolar  $p\text{O}_2$  is equal to  $148 - \text{the arterial } p\text{CO}_2 \times 1.2$ . If, in general, an arterial oxygen tension is below normal but the A-a gradient is measured as less than 10 mm Hg, it is most likely that alveolar hypoventilation is the sole abnormality producing hypoxemia. In hypoventilation that is caused by primary lung disease per se, the A-a gradient is elevated. Abnormally low arterial oxygen tensions produced by uncomplicated alveolar hypoventilation can be corrected merely by improving the level of the alveolar ventilation. Diseases that produce widened A-a gradients often will produce hypoxemia that cannot be completely corrected by simply increasing the level of alveolar ventilation. The most common cause of hypoxemia in patients is a maldistribution of alveolar ventilation and pulmonary blood flow. Diseases such as asthma and bronchitis impair ventilation and hence disturb ventilation/blood flow relationships. Our treatment of hypoxemia caused by ventilation-perfusion abnormalities may be illustrated in Fig. 28-11. If the alveolus 1 has a reduction in ventilation due to airway narrowing, then the arterial oxygen tension in blood vessel I flowing past alveolus 1 will be reduced. Alveolus 2, which receives normal ventilation, will fully saturate the hemoglobin that flows past it in blood vessel II. As a result, the arterial oxygen tension in blood vessel III will be reduced since it is a composite of blood from blood vessel I and II. However, ventilation-perfusion relationships cannot be compensated by simple hyperventilation; since simple hyperventilation cannot improve the saturation of blood in vessel II, nor significantly raise the arterial oxygen tension in alveolus 1. If, however, the tension of inspired oxygen is raised, then the oxygen tension in alveolus 1 will rise and thus increase the saturation of hemoglobin in vessel I. As a result, the arterial oxygen tension of vessel III will be increased. It is for this reason that diseases characterized by ventilation-perfusion mismatching do show improvement with higher inspired oxygen tensions. It should be apparent that if there is a true anatomic shunt present around alveolus 1 (Fig. 28-12), raising the inspired oxygen tension will not result in any improvement in arterial oxygen tension since the hemoglobin molecules in vessel II are already fully saturated and there is no blood gas exchange in alveolus 1. True shunts, therefore, do not respond to increases in inspired oxygen tension.

It is important to emphasize at this juncture that patients with longstanding ventilation-perfusion mismatching with advanced pulmonary disease often have had chronic hypoxemia

and CO<sub>2</sub> retention. These patients no longer have their respiratory drive determined by hydrogen ion concentration and are deriving their respiratory drive in a large part from their arterial oxygen tensions. The injudicious correction of arterial oxygen tension may, therefore, lead to a cessation of respiratory drive. It is for this reason that venti-masks using high-flow system that controls the inspired oxygen tension closely, or low-flow oxygen systems using 1 or 2 L of flow, should be employed in the treatment of patients with significant ventilation-perfusion abnormalities.