Bronchoscopy: Diagnosis of Tumors of the Lung

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ETIOLOGY AND INCIDENCE

The lungs have both benign and malignant tumors, as well as metastases from primary cancers of other organs and tissues. Bronchogenic carcinoma is the most common and accounts for 90 percent of all lung tumors. (Figure 1.) In women 20 percent are primary lung carcinoma, while in men 35 percent are primary lung carcinoma. Patients with a localized tumor show only a five-year survival. In males the rate is 30 percent, and in females the rate is 50 percent. Statistics indicate that the number of female smokers is rising so the survival rate is predicted to drop. Occurrence of lung cancer is most common between the ages of 45 and 70.

Cigarette smoking is associated with 70 percent of the cases in women and 90 percent in men, with a strong dose relationship and regression of incidence after quitting. The four most common types of lung cancer are squamous cell, small cell, large cell and adenocarcinoma. Other causes of lung cancer are related to occupational agents such as asbestos, radiation, arsenic, chromates, nickel, chlorethyl ethers, mustard gases and coke-oven emissions. In addition, heredity remains a critical but poorly understood factor.

At the time of diagnosis, 20 percent of the patients will have local disease (cancer occurring in one location only). In 25 percent of the patients, the cancer has spread to regional lymph nodes and in 55 percent of the cases, the patients will have distant metastatic cancer.

Carcinoma of the lung represents a major health challenge with a generally negative prognosis. However, an orderly approach to diagnosis, staging and treatment, based on knowledge of the clinical behavior of lung cancer, allows selection of the best therapy for either potential cure or optimal palliation of individual patients.

ANATOMY OF THE CHEST

Composed of soft, spongy tissue, the two cone-shaped lungs are located inside the thoracic cavity (chest wall) and consume most of the space. The right and left lungs are separated medially at the heart and mediastinum. A serous membrane, the parietal pleura, lines the inner walls of the thoracic cavity, and a similar membrane, the visceral pleura, covers the lungs. Separating the two membranes is a thin layer of watery liquid that serves to lubricate the adjacent membranes and reduces friction as they move against each other during breathing. Each lung includes an apex projecting into the neck area and a concave base that sits above the dome of the diaphragm. (Figure 3.)

The two lungs are differing sizes. The slightly larger right lung has three lobes: superior, middle and inferior; whereas, the smaller left lung contains only the superior and inferior lobes. Supplying each lobe with air are the primary bronchi, two branched airways that lead from the trachea and convey oxygen to the alveoli, the microscopic air sacs contained in each lung.

Inside the lungs, each primary bronchus is subdivided into smaller branches known as lobar bronchi (three on the right and two on the left). Progressively, these branches become even smaller. Tertiary or segmental bronchi supply a part of the lung called a bronchopulmonary segment. (In most people, the right lung contains 10 segments and the left has eight segments.)

Bronchioles are smaller branches of the segmental bronchi and lead to the lobule, the smallest component of the lung. Branching from the bronchiole are the terminal bronchioles. Fifty to 80 terminal bronchioles reside within each lobule of the lung.

Two or more respiratory bronchioles extend from each terminal bronchiole and are the first structures to engage in gas exchange. From each respiratory bronchiole are two to 10 long alveolar ducts. Extruding from the alveolar ducts are the crowded alveolar sacs that are adjacent to the primary bronchi's final destination, alveoli. Alveoli are small, thin-walled air sacs that open to the alveolar sac. Air diffuses from the alveolar ducts to the alveolar sacs and, ultimately, to the alveoli.

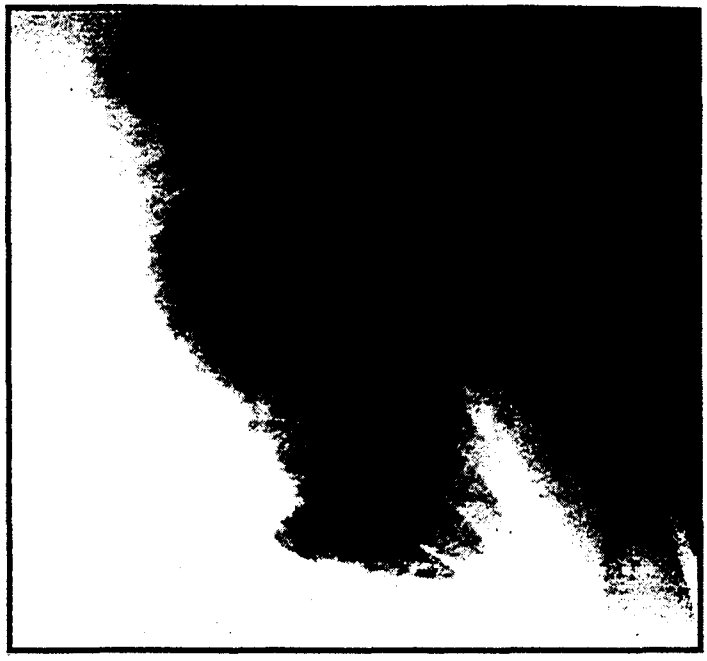

FIGURE 1—Anterior view of bronchogenic carcinoma in the left chest.

THE EXCHANGE OF GASES

Inside an adult lung are an estimated 300 million alveoli with a 750-foot total surface area.[5] This large surface area of thin epithelial cells provides an ideal arena for the exchange of gases (oxygen and carbon dioxide), accomplished by a process called diffusion. Oxygen travels through the walls of the alveoli and moves into the blood of the capillaries, while carbon dioxide diffuses from the blood in the capillaries into the alveoli. Respectively, the molecules of oxygen and carbon dioxide are moving in two directions from areas of higher concentration to areas of lower concentration.

One factor that influences the amount of oxygen present is the amount of hemoglobin in the blood. Because oxygen forms such a strong chemical bond with hemoglobin, an increase in hemoglobin will be accompanied by an increase in oxygen. Normally, there are 12-15 grams of hemoglobin in the blood. Conversely, carbon dioxide also bonds to hemoglobin and again the chemical attraction makes the gas exchange more efficient.

Carbon dioxide diffuses from the tissue when it is formed by cellular metabolism and enters the capillary blood. Approximately 90 percent of the carbon dioxide enters the red blood cells and combines with water to form carbonic acid. This carbonic acid will disassociate into bicarbonate ions and diffuse out of the cell and back into the plasma. Most of the carbon dioxide is transported as bicarbonate. The carbon dioxide is picked up by the hemoglobin (deoxyhemoglobin) and becomes active in the gas exchange.

This deoxygenated blood containing the carbon dioxide originates in the pulmonary arteries. During the gas exchange, the carbon dioxide travels to the alveolar capillaries where the blood is oxygenated and subsequently flows into the pulmonary veins and on to the left atrium. The lungs receive their blood supply from the bronchial arteries, branches of the aorta. This blood supply feeds the connective tissue of the lung and the visceral pleura.

THE FUNCTIONS OF BREATHING

The two functions of breathing by the lungs are inspiration and expiration. Breathing, also called pulmonary ventilation, consists of two processes: the collection of air from outside the body to inside the body through the bronchial tree to the alveoli and the reverse. Inspiration is the action of contraction of the inspiratory muscles, primarily the diaphragm. Expansion of the diaphragm increases the size of the chest cavity. As the diaphragm descends, the muscles elevate the lower ribs causing a forward movement of the sternum. (Figure 2.) Expiration occurs when the muscles used during inspiration relax to allow the elastic forces of the lungs to return lung volume to normal.

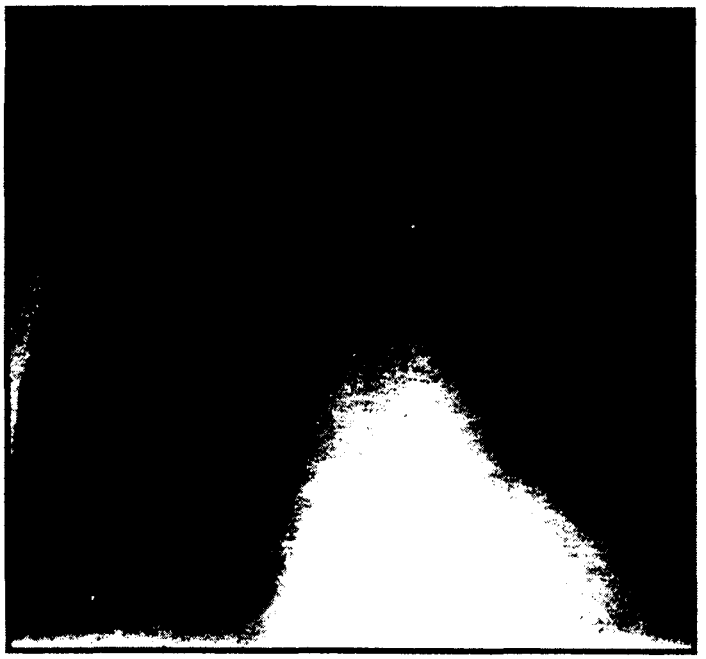

FIGURE 2—Anterior view of chest showing lung and ribs.

The surface forces of the lung area, or surface tension, is controlled by a mixture of phospholipids, cholesterol and proteins. The major component is dipalmitoyl phosphatidylcholine (surfactant). Surfactant reduces surface tension at the alveolar air interface. This reduction increases the compliance and decreases the work of respiration.

In elderly people who have reduced traction exerted on the walls of the lungs, there may be a reduction in airway size. This reduction may lead to hypoxia, which is bluish color of the tissues caused by the presence of deoxyhemoglobin (a reduced hemoglobin count). Evidence of this condition is most readily seen in the lips, mucous membrane, nail beds and ear lobes.

The neural mechanism for respiration is found in the medullary centers and is dependent on the rhythmic impulses generated within the brain stem. These impulses are transmitted to the respiratory muscles. The pneumotaxic center within the rosulate pons is very sensitive to various stimuli and will place limits on the duration of inspiration. During exercise the increase in respiratory stimulus is second only to hyperventilation (voluntary). While exercising, the blood acid levels increase due to an increase in lactic acid concentration, and this causes increases in ventilation as the body compensates to maintain hemostasis. The brain will receive neural impulses and cause reflex increases in respiration. As the blood acid levels decrease by the effect of increased lactic acid, the change will create an increase in respiration to remove the lactic acid through increasing the oxygen being distributed to the tissues. During exercise, ventilation will remain increased due to increased body temperature. As the body temperature drops, a parallel drop in ventilation occurs until normal levels are reached.

When inspired air enters the nasal area, it usually adjusts to body temperature. If breathing through the mouth occurs, cool dry air will reach the lower respiratory airways and lead to drying of the secretions. In turn, damage to the mucosal lining may occur and impaction of the mucous plugs.

Most particles are removed from the inspired gas exchange by impaction on the walls of the upper airways. Particles that are irritating can stimulate bronchial receptors in the large bronchi and lead to a reflex bronchoconstriction cough. This cause will increase secretion of mucous. Toxic material taken in by the lungs can cause paralysis of the cilia (little hair-like fibers that generate the motion to move material) and lead to increased statis of secretion, predisposing the individual to lung infections, including tumors.

CLINICAL COURSE

Lung cancer is one of the most aggressive neoplasms in medical oncology. The major presenting complaints are cough (75 percent), weight loss (40 percent), chest pain (40 percent) and dyspnea (20 percent). The tumor is discovered by secondary spread in the course of investigation of an apparent primary neoplasm elsewhere. Not more than 20 to 30 percent of lung cancer patients have lesions sufficiently localized to even permit resection. Adenocarcinoma and squamous cell patterns tend to remain localized longer and have a slightly better prognosis than the undifferentiated cancers.

The most intriguing clinical aspects of bronchogenic carcinoma is its association with a series of hormonal and systemic syndromes, some of which may antedate the development of a gross pulmonary lesion.[3] The hormones involved are: (1) antidiuretic, (2) ACTH, producing Cushing's Syndrome, (3) parathyroid causing hypocalcemia, (4) calcitonin, causing hypocalcemia, (5) gonadotropins causing gynecomastia, and (6) serotonin, associated with the carcinoid syndrome.[3] Any one of the histologic types of tumors may occasionally produce any one of the hormones, but tumors producing ACTH and antidiuretic hormone are predominantly oat cell (small

cell) carcinomas, while those inducing hypercalcemia are mostly squamous cell tumors.[3]

Other systemic manifestations include: myopathy (muscle weakness); peripheral neuropathy (sensory); hematologic abnormalities such as leukemoid reactions; and hypertrophic pulmonary osteoarthropathy (clubbing of the fingers). There is much to be learned about the activity of these cancers but most important is an understanding of their causation.

DIAGNOSIS

Once signs, symptoms and studies suggest lung cancer, it will be necessary to establish a tissue diagnosis of malignancy; determine the histologic cell type; and stage the appropriate treatment.

Tumor tissue should be obtained so that a histologic diagnosis of cancer and tumor cell type can be determined precisely. Therefore, cytologic diagnosis from washings or needle aspirates can be obtained from: bronchoscopy; node biopsy; a mediastinoscopy; from the operative specimen at the time of definitive surgical resection; biopsy of an enlarged lymph node; and an adequate cell block from a malignant pleural effusion.

The choice of diagnostic procedures is determined by the patient's symptoms. Usually a physician will begin with a less invasive option, such as the bronchoscopy. The bronchoscope can be moved deeply into the bronchial tubes to obtain a biopsy and bronchial washings that will give the physician the most accurate results. Other diagnostic procedures such as a node biopsy will only indicate the presence of cancer cells but will not pinpoint the site of the tumor. A mediastinoscopy involves opening the chest and requires a longer time for the patient's recovery.

BRONCHOSCOPY PROCEDURE

The patient is placed in a supine position. The bronchoscope is inserted over the surface of the tongue, usually through the right corner of the mouth. The patient's lip is retracted from the upper teeth with the finger of the endoscopist's left hand.

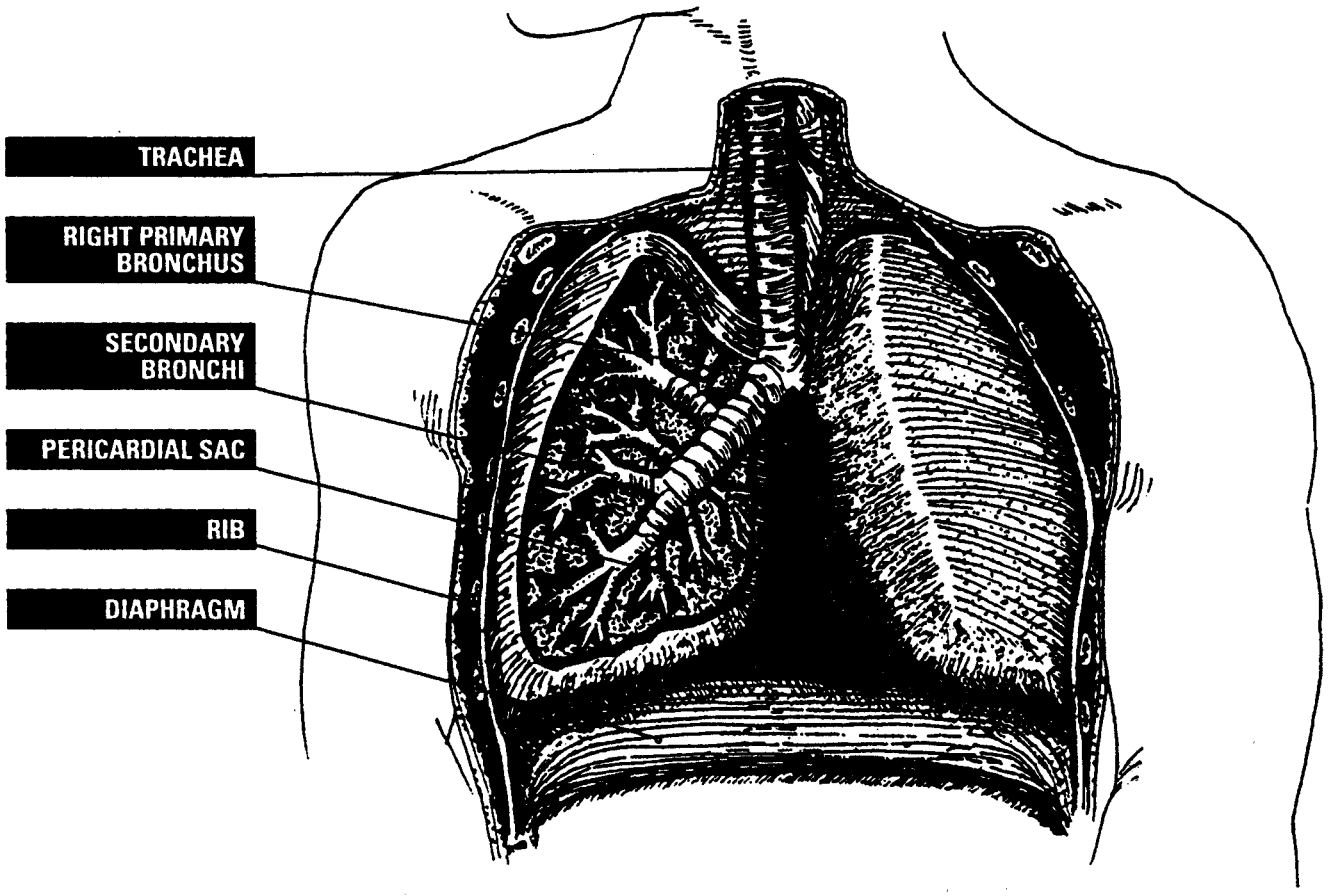

FIGURE 3—General view of the respiratory system.

The epiglottis is identified and elevated with the top of the bronchoscope.[4]

The distal end of the scope is passed through the glottis and the upper tracheal rings are viewed. At this time, a small amount of anesthetic solution may be sprayed through the tube on the carina and into the bronchus by means of a bronchial atomizer or spray.

The patient's head is moved to the left to obtain a view of the right bronchi. A right-angled (Broyles) telescope, which has been already adjusted, is inserted into the head of the bronchoscope. A few seconds are allowed for the optical system to become free of precipitated moisture.[4]

The segmental bronchial orifices of the upper right lobe bronchi are viewed, and the telescope is removed. Suction and aspirating tubes are used to provide a clean, dry field of vision. The scope is advanced to insect the middle of the lobe branches by means of insertion of an oblique 30-degree angle telescope or right-angled telescope. The patient's head may be lowered so that the right middle lobe orifices can be viewed or head turned to the right so that the left main bronchus may be viewed.[4] Aspiration of secretions for study are performed, if necessary. A biopsy may be obtained using suitable forceps for histological diagnosis of a thoracic disorder. Foreign bodies are removed.

The bronchoscope is withdrawn and the patient's face is cleansed. The patient is permitted to sit upon the table for a few minutes before moving to the stretcher. An emesis basin and compresses must be available.[4]

STAGING

Lung cancer staging consists of two parts: first, determination of the location of the tumor (anatomic staging) and, second, assessment of a patient's ability to withstand various antitumor treatments (physiologic staging).

CLASSIFICATION OF LUNG CANCER

Tumor (T)

- ***To*** No evidence
- ***Tx*** Cancer seen in bronchial washing, not by X-ray or fiberoptic bronchoscopy
- ***TIX*** Carcinoma insitu
- ***T1*** Tumor ≤ 3 cm surrounded by lung or visceral pleura, without evidence of invasion proximal to lobar bronchus at bronchoscopy
- ***T2*** Tumor ≥ 3 cm tumor of any size that either invades the visceral pleura
- ***T3*** Tumor of any size with direct extension into the chest wall or a tumor in the main Bronchus within 2 cm of the carina
- ***T4*** A tumor of any size with invasion of the mediastinum

Regional Lymph Nodes (N)

- ***No*** No metastasis to regional lymph nodes
- ***N1*** Metastasis to lymph nodes in the peribronchial or ipsilateral hilar region
- ***N2*** Metastasis to ipsilateral mediastinal or subcarinal lymph nodes
- ***N3*** Metastasis to contralateral mediastinal, contralateral hilar ipsilateral or contralateral scalene

Distant Metastasis (M)

- ***Mo*** No known distant metastasis
- ***N1*** Distant metastasis present with site specified

In almost all patients with small cell lung cancer the cells will have spread beyond the primary site at the time of diagnosis. The treatment for small cell lung cancer is chemotherapy with or without irradiation.

TREATMENT

The treatment of choice for non-small cell lung cancer stages I and II is surgical resection; patients with stage IV are not candidates for surgery. Patients with classification of T3NoMo should be considered for surgical resection. Radiation therapy is a proven benefit for controlling bone pain, spinal cord compressions, brain metastasis and bronchial obstruction. Drug treatment is not usually recommended, although some improved results with drugs have been reported. There is no effective specific drug for bronchogenic carcinoma.

The poor prognosis for patients with bronchogenic carcinoma requires emphasis on prevention. Cigarette tobacco should be avoided and exposure to potentially carcinogenic substances in industry must be reduced below dangerous levels.

AUTHORS

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REFERENCES

1. *The Merck Manual*. Vol. 1. 16th ed. Merck, Sharp, and Dohme Research Laboratories; 1987:732-736.
2. Wilson, Martin. *Harrison's Principles of Internal Medicine*. 13th ed. New York: McGraw-Hill; 1994:1164-1165, 1221-1229.
3. Robbins SL. *Pathologic Basis of Disease*. 2nd ed. Philadelphia: WB Saunders; 1997.
4. Ballinger WF, Treybal, JC, and Vose AB. *Alexander's Care of the Patient in Surgery*. 5th ed. CV Mosby Company; 1972:572-574.
5. Hole JW. *Human Anatomy and Physiology*. 4th ed. Dubuque, Iowa: WC Brown Publishers; 1987.

BIBLIOGRAPHY

- *Essentials of Medicine*. 2nd ed. Philadelphia: WB Saunders Company; 1995.
- Shoemaker WC. *Textbook of Critical Care*. 2nd ed. Philadelphia: WB Saunders Company; 1989.
- Conn HF, Conn RB. *Current Diagnosis 6*. Philadelphia: WB Saunders Company; 1980.
- Sodeman WA, Sodeman TM. *Pathologic Physiology*. 6th ed. Philadelphia: WB Saunders Company; 1979.
- Walter JB, Israel MS. *General Pathology*. 6th ed. New York: Churchill; 1987.
- Sidransky H. *Nutritional Pathology*. Vol 10. New York: Dekker Inc; 1985.
- Thomas SJ. *Manual of Cardiac Anesthesia*. New York: Churchill; 1984.
- Fulicic JR. *Surgical Technology*. 2nd ed. Philadelphia: WB Saunders Company; 1986.
- Schwartz SI. *Principles of Surgery*. 3rd ed. New York: McGraw-Hill; 1979.
- Spence AP. *Basic Human Anatomy*. 2nd ed. Menlow Park, Calif: Benjamin/Cummings Company; 1986.
- Hollinshead WH, Rosse C. *Textbook of Anatomy*. 4th ed. Philadelphia: Harper and Row; 1985.
- Weiss L. *Cell and Tissue Biology*. 6th ed. Baltimore: Urban and Schwarzenberg; 1988.
- Guyton AC. *Physiology of the Human Body*. 6th ed. Philadelphia: Saunders College Publishing; 1984.
- Hole JW. *Human Anatomy and Physiology*. Dubuque, Iowa: WC Brown Co; 1978.
- Milnor WR. *Cardiovascular Physiology*. New York: Oxford University Press; 1990.
- Little RC. *Physiology of the Heart and Circulation*. Chicago: Year Book Medical Publishers; 1971.
- Martin DW, Mayes PA, Rodwell VW, Granner DK. *Harpers Review of Biochemistry*. 20th ed. Los Altos, Ca: Lange Medical Publications; 1985.
- White A. *Principles of Biochemistry*. 5th ed. New York: McGraw-Hill; 1973.
- Weiss L. *Cell and Tissue Biology*. 6th ed. Baltimore: Urban and Schwarzenberg; 1988.
- Tortora G, Evans R. *Principles of Human Physiology*. 2nd ed. New York: Harper and Row; 1986.

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