Pulmonary Function Testing for Preoperative Study of Patients for Anesthesia and Surgery*

ORHAN MUREN, M.D.

Associate Professor of Medicine,
Medical College of Virginia, Richmond, Virginia

Pulmonary complications are the most prevalent causes of morbidity and mortality in patients who undergo surgical procedures. Simple pulmonary function tests which can detect pulmonary dysfunction may be performed preoperatively. If proper measures are taken (chest physiotherapy, postural drainage, bronchodilating agents, antibiotics, humidification of inspired gases, cessation of cigarette smoking), morbidity and mortality can be reduced significantly.

Pulmonary function tests are especially important in patients with borderline pulmonary reserve who need major surgery. After objective evaluation, the anesthesiologist and the surgeon can provide better care for the patient. These studies may also suggest the patients who are more likely to develop postoperative complications.

In many patients, a thorough medical history and physical examination, in addition to a complete blood count with differential, urinalysis, electrolytes, BUN, and chest x-ray, often will give satisfactory information, and many patients will not need pulmonary function testing prior to anesthesia and surgery.

Should history and physical examination show that pulmonary function tests are indicated, they will also indicate the type of tests to be carried out. A history of chronic productive cough, dyspnea, pleuritic chest pain, occupational respiratory hazards, frequent past episodes of pneumonia, pleurisy, chest colds, and sudden reduction in strength should strongly suggest possible pulmonary dysfunction.

Findings on examination of the chest such as prolonged expiration, diffuse or localized wheezing, absent breath sounds, rales, chest-wall deformities and scars, hypertrophy of scalene muscles, and pulmonary osteoarthropathy again should suggest possible abnormalities in lung function, and proper pulmonary function tests should be performed.

There are a variety of pulmonary function tests which may be used to detect dysfunction. In general, they can be divided into two groups:

1. Those relating to the ventilatory function of the lungs and thoracic wall.
2. Those relating to pulmonary gas exchange.

Ventilatory Function Tests. Ventilatory function is determined by measurement of static lung volumes. These are somewhat indicative of the elastic resistance of the lungs and chest wall and dynamic lung compartments; these tests are mainly a reflection of nonelasticity.

Static Lung Volumes. Static lung volumes are assessed by measuring the vital capacity which changes according to height, age, and sex. Full cooperation of the patient during the procedure is essential. Integrity of the entire respiratory system (respiratory centers, thorax, the connections between these two, chest wall, pleura, lung paranchyma, and airways) is necessary for normal vital capacity.

If a person has less than predicted normal vital capacity, by definition he has restrictive lung disease. Normal values range $\pm 15\%$ of the predicted. As an isolated test, vital capacity is of limited clinical value in differential diagnosis of diseases of the chest. However, serial measurements of vital capacity may be helpful in assessing the degree of improvement or worsening in certain conditions such as neuromuscular disorders, or diffuse interstitial pulmonary fibrosis.

Dynamic Lung Volumes. More important information could be obtained by measuring timed vital capacity or forced vital capacity. A young or middle-aged normal person should be able to exhale 80% of his vital capacity in one second and 97% in three seconds. If the ratio between one-

* Presented at the 25th Annual Stoneburner Lecture Series, February 26, 1972, at the Medical College of Virginia, Richmond.

second forced expiratory volume ($FEV_{1.0}$), over forced vital capacity (FVC) is less than 80%, he is considered to have obstructive lung disease. Diminution in $FEV_{1.0}$ to a liter or less indicates severe impairment of ventilatory function and a somewhat poor surgical risk.

Expiratory flow rates can be determined at different points during forced expiratory curve. The forces tending to reduce the size of airways (peribronchial pressure and the force generated by constriction of bronchiol muscles) are balanced by those attempting to increase the size of airways (intraluminal pressure and radial traction of elastic fibers). During forced exhalation, the alveolar pressure and the intrathoracic pressure are greater than atmospheric pressure. The intramural pressure decreases gradually from peripheral airways to atmospheric pressure at the airway opening. At some distance in the airway, intraluminal and pleural pressures are equal. This is called the point of equal pressures. Beyond this point, intraluminal pressure is less than pleural pressure; this would tend to narrow airways, thus limiting flow.

In patients with altered and more collapsible airways, flow is limited at lower levels of pleural pressure. The maximal flow rate produced by forced exhalation depends upon the level of lung inflation. At maximum lung inflation, expiratory flow increases as the pressure increases. At lower lung volumes, expiratory flow increases as pressure increases up to a certain level, after which more effort cannot increase and may even decrease flow rate. This explains why the maximum mid-expiratory flow rate (MMFR or FEF 25%–75%) is the most sensitive test for expiratory flow rate determination since the initial 25% of the FVC is primarily effort-dependent and the last 25% affected by diminished lung volumes.

The MMFR measures the maximum rate of flow in the mid portion of FVC curve (FEF 25%–75%). (The length of time necessary to exhale the middle 50% of the forced vital capacity is measured and flow rate is calculated.) Normal values are 3–4.5L per second. It should be emphasized that small airways (2 mm in diameter) are only responsible for approximately 15%–20% of the total airway resistance, so marked increases in peripheral or small airways will not be detected by the conventional tests such as $FEV_{1.0}$ or MMFR.

In the early stages of chronic bronchitis, pulmonary emphysema, and bronchiectasis there is a significant degree of involvement of the small airways. In the early stages of these conditions, frequency-dependent dynamic compliance and alveolar arterial oxygen tension gradient, A-a PO_2, may detect pulmonary dysfunction. For clinical purposes, however, simple spirometric studies with measurements of FVC, $FEV_{1.0}$, and MMFR are satisfactory. Improvements in flow rates after administration of bronchodilating agents suggest the presence of partially reversible obstruction. One-second forced expiratory volume $\times$ 30 gives indirect measurement of maximum voluntary ventilation (MVV).

If a bedside study of ventilatory function is desired, a Wright Respirometer may be used for measuring tidal volume and inspiratory capacity. A Peak Flow Meter may also be used for measuring peak flow rates.

Failure to blow out a lighted match held six inches away from the wide-open mouth again suggests significant reduction in flow rates.

In general, patients with obstructive lung disease are more prone to develop postoperative pulmonary complications such as atelectasis and broncho-pneumonia. This is why pulmonary function testing is important for determining the type of physiologic abnormality present so that proper measures can be taken to improve potentially reversible abnormalities.

Pulmonary Gas Exchange. One of the most important functions of the lungs is alveolar gas exchange which involves the following processes:

1. Ventilation
2. Uniform distribution of inspired air
3. Diffusion
4. Pulmonary capillary blood flow

Normal alveolar gas exchange maintains partial pressures of oxygen and carbon dioxide in the arterial blood within normal limits not only at rest, but also during increased physical activity and body metabolism. Abnormality in any of the above processes or in any combinations of the four, results in hypoxemia or hypoxemia and hypercapnia if the person is breathing room air.

Arterial gas studies indicate:

1. The level of oxygenation (PO_2 in mm of Hg)
2. The level of ventilation (PCO_2 in mm of Hg.)
3. The hydrogen ion activity (pH)

The above determinations are extremely important in all seriously ill patients.

It is often impossible to judge the levels of ventilation and oxygenation by clinical evaluation alone. There is much overlapping between signs and symptoms related to hypoxemia and hypercapnia: nervousness, headache, irritability, confusion, coma, altered blood pressure, tachycardia, etc. Sometimes signs and symptoms related to hypoxemia and hypercapnia are absent and only by measurements of arterial gas studies can the level of ventilation and oxygenation be determined and appropriate treatment initiated.

An A-a PO_2 gradient greater than 10 mm Hg on room-air breathing indicates a defect in blood gas equilibrium. The three primary mechanisms of increased A-a PO_2 gradient are impaired diffusion, venous-to-arterial shunting of blood, and abnormal ventilation/perfusion ratios in the lung.

The amount of true venous-to-arterial shunt is determined by having the person breathe 100% oxygen for 15 to 20 minutes. Normally, arterial PO_2 will increase to about 600 mm Hg. The observation of increased A-a PO_2 gradient on breathing room air and of a PO_2 greater than 550 mm Hg while breathing oxygen indicates that mismatching of the distribution of ventilation and perfusion in the lungs is the cause of hypoxemia.

The observation of high A-a PO_2 gradient on room air in the presence of normal spirometric studies may also suggest peripheral airway disease.

Whenever possible, arterial blood gas studies should be obtained at rest, after exercise, and sometimes after the administration of 100% oxygen for 15 to 20 minutes. The above studies may give valuable information in the evaluation and follow-up of patients with respiratory disease.

Normal arterial blood O_2 at sea level is found in two forms:

1. Dissolved O_2 (0.3 ml per 100 ml per 100 mm Hg)
2. Oxyhemoglobin (Hb O_2 19.7 ml per 100 ml)

Normally, the total O_2 content of arterial blood is approximately 20 ml per 100 ml. The amount of dissolved O_2 present is linearly related to arterial O_2 tension. If partial pressure of O_2 in arterial blood is 600 mm Hg, there is approximately 1.8 ml of physically dissolved O_2 per 100 ml blood.

On the other hand, the relation between oxyhemoglobin and partial pressure of O_2 is S-shaped. If PO_2 is 100 mm Hg, oxygen saturation is 96% to 98%. If PO_2 is 60 mm Hg, oxygen saturation is approximately 90%. In the flat portion of this curve, a large decrease in O_2 tension causes only a small drop in oxygen saturation. However, at the steep portion of this curve, a small reduction in O_2 tension causes a large diminution of O_2 saturation.

For instance, a drop of O_2 tension from 40 mm Hg to 27 mm Hg causes a reduction of saturation or O_2 content of approximately 25%. A high CO_2 tension and increased H^+ ion activity shifts this curve to the right. This is called the Bohr effect. Increased temperature and increased 2–3 DPG (2–3 Diphosphoglycerate) of the red blood cells can also shift this curve to the right. A low PCO_2, a high pH, a low temperature, and a low 2–3 DPG causes a shift to the left. Normally, a shift to the left in the lungs helps the loading of O_2 with hemoglobin. A shift to the right at tissue levels helps unloading of O_2 to tissues.

There are four causes of hypoxemia on breathing ambient air at sea level:

1. Uneven $\dot{V}/\dot{Q}$
2. Venous-to-arterial shunt
3. Alveolar hypoventilation
4. Impaired diffusion

In clinical medicine, uneven $\dot{V}/\dot{Q}$ is the most common cause of hypoxemia. However, in a given case, there is more than one factor responsible for hypoxemia.

Arterial PCO_2 indicates the balance between CO_2 produced at the tissue level and CO_2 eliminated by alveolar ventilation.

$$PaCO_2 \propto \frac{\dot{V}CO_2}{\dot{V}A}$$

Normally, alveolar ventilation is proportional to CO_2 production which is related to the rate of metabolism. In any given person at any given time, CO_2 production is at a certain level and only alveolar ventilation could change arterial PCO_2. In conclusion, we can say that the level of PCO_2 indicates the level of ventilation.

Hydrogen ion activity is determined by measurement of pH. According to the Henderson-Hasselbalch equation:

$$pH = PK^1 + \log \frac{(HCO_3)}{PCO_2 \times 0.03}$$

$$pH = 6.1 + \log \frac{24}{1.2}$$

$$pH = 6.1 + \log \frac{20}{1}$$

$$pH = 6.1 + 1.3 = 7.40$$

The ratio of HCO_3 and dissolved CO_2 determine the pH. As long as this ratio is 20, the pH should be normal.

There are four primary or simple acid-base disturbances as well as combined or mixed acid-base disturbances. Primary increase or decrease in PCO_2 indicates respiratory acidosis or alkalosis respectively. Primary increase or decrease in HCO_3 indicates metabolic alkalosis or acidosis respectively.

The following are some indications for pulmonary function testing:

1. To determine the type of physiologic alteration present.
2. To quantitate the degree of functional impairment.
3. To initiate therapy on a more rational physiological basis and objectively follow the efficacy of therapy.
4. Preoperative risks evaluation. Re: anesthesia and surgery.

The following arterial gas studies were obtained on room air in a 60-year-old man with chronic obstructive pulmonary disease (COPD):

PO_2:60 mm Hg

PCO_2:55 mm Hg

pH:7.34

HCO_3:30 m Eq/L

The above studies indicate mild hypoxemia and partially compensated respiratory acidosis.

Spirometric studies revealed moderately severe airway obstructive disease. One day after admission, elective cholecystectomy was performed. One day after surgery, arterial gas studies on ambient air were obtained.

PO_2:45 mm Hg

PCO_2:80 mm Hg

pH:7.26

HCO_3:35 m Eq/L

The above studies indicate severe hypoxemia and partially compensated severe respiratory acidosis. It took approximately 12 days to correct arterial gas studies and acid-base disturbance.

On the other hand, in another patient with COPD and more or less the same arterial gas abnormalities, elective gall bladder surgery was postponed for seven days and the patient was given chest physiotherapy, postural drainage, bronchodilating agents, antibiotics, and humidified inspired air. He stopped smoking. Postoperative status was entirely unremarkable.

In conclusion, if history and physical examination indicate pulmonary dysfunction, simple pulmonary function tests should be obtained and proper measures taken to reduce morbidity and mortality in surgery patients, especially those with underlying chronic lung disease.

REFERENCES

BATES, D. V., MACHLEM, P. T., AND CHRISTIE, R. V. *Respiratory Function in Disease*. Philadelphia: W. B. Saunders, 1971.

CAMPBELL, E. J. M., AGOSTONI, E., AND DAVIS, J. N. *The Respiratory Muscles, Mechanics and Neurol. Control*. Philadelphia: W. B. Saunders, 1970.

CHERNIACK, R. M. AND CHERNIACK, L. *Respiration in Health and Disease*. Philadelphia: W. B. Saunders, 1961.

COMROE, J. H., FORSTER, R. E., DUBOIS, A. B., BRISCOE, W. A., AND CARLSEN, E., JR. *The Lung*. Chicago: Yearbook Medical Publications, 1962.

FENN, W. O. AND RAHN, H. *Handbook of Physiology, Section 3: Respiration*. Vol. 1 and 2. Baltimore: Waverly Press, 1965.

MEAD, J. Mechanical properties of lungs. *Physiol. Rev.* 41:281, 1961.

MEAD, J., TURNER, J. M., MACKLEM, P. T., AND LITTLE, J. B. Significance of the relationship between lung recoil and maximum expiratory flow. *J. Appl. Physiol.* 22:95, 1967.

SCURR, C. AND FELDMAN, S. *Scientific Foundations of Anesthesia*. Philadelphia: F. A. Davis Company, 1970.

SLONIM, N. B. AND HAMILTON, L. H. *Respiratory Physiology*. Saint Louis: The C. V. Mosby Company, 1971.

SYKES, M. K., MCNICOL, M. W., AND CAMPBELL, E. J. M. *Respiratory Failure*. Oxford: Blackwell Scientific, 1969.

WEST, J. B. *Ventilation/Blood Flow and Gas Exchange*. Philadelphia: F. A. Davis Company, 1970.

WOOLCOCK, A. V., VENCENT, N. J., AND MACHLEM, P. T. Frequency dependence of compliance as a test for obstruction in the small airways. *J. Clin. Invest.* 48:1097, 1969.