West's Pulmonary Pathophysiology

The Essentials

NINTH EDITION

John B. West
Andrew M. Luks

Wolters Kluwer

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WEST'S PULMONARY PATHOPHYSIOLOGY

THE ESSENTIALS

John B. West, MD, PhD

Distinguished Professor of Medicine and Physiology
University of California, San Diego
School of Medicine
San Diego, California

Andrew M. Luks, MD

Professor of Medicine
University of Washington School of Medicine
Seattle, Washington

Wolters Kluwer

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WEST'S PULMONARY PATHOPHYSIOLOGY

THE ESSENTIALS

To R.B.W.

—John B. West

To all my students.

—Andrew M. Luks

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PREFACE

This book is a companion to *West's Respiratory Physiology, 10th edition* (Wolters Kluwer, 2016) and is about the function of the diseased lung as opposed to the normal lung. It was first published 40 years ago and has therefore served several generations of students. It has been translated into a number of languages. This ninth edition has many extensive changes. The most significant is that Andrew M. Luks, MD, has become a coauthor. Dr. Luks obtained his MD at the School of Medicine, University of California San Diego, and was therefore exposed to much of this material as a medical student. He is now on the faculty at the University of Washington School of Medicine where he enjoys a reputation as an excellent teacher. As he was when he became a coauthor on the 10th edition of *West's Respiratory Physiology*, he has been responsible for many of the important changes in this new edition, particularly the clinical vignettes, many new multiple choice questions, and a number of new illustrations.

Each chapter now has a clinical vignette that emphasizes how the pathophysiology described in the chapter is used in the practice of clinical medicine. At the end of the vignette are several questions and answers to these are in an appendix. Another addition is over 30 new multiple choice questions in the format used by the USMLE. The stems of these questions have a clinical orientation, and their purpose is to test the broader understanding of a topic rather than a simple factual recall. The illustrative material in the book has been considerably expanded with eight new radiographs and CT images as well as color histopathologic sections graciously provided to us by Corinne Fligner, MD, from the University of Washington School of Medicine and Edward Klatt, MD, from Mercer University School of Medicine. The text of the book has been updated in many areas particularly in the sections dealing with modern therapy. Another new development has been the production of seven 50-minute video lectures based on the book. These are freely available on YouTube and have proved to be very popular.

As a result of all these innovations, the length of the book has been increased, but its primary purpose has not changed. As before, it serves as an introductory text for medical students in their second and subsequent years. However, a concise, amply illustrated account of respiratory function in disease will prove useful to the increasingly large number of physicians (such as anesthesiologists and cardiologists) and other medical personnel (including

intensive care nurses and respiratory therapists) who come into contact with respiratory patients.

The authors are grateful for any comments on the selection of material or any factual errors. We will respond to any e-mails on these subjects.

John B. West, jwest@ucsd.edu

Andrew M. Luks, aluks@u.washington.edu

CONTENTS

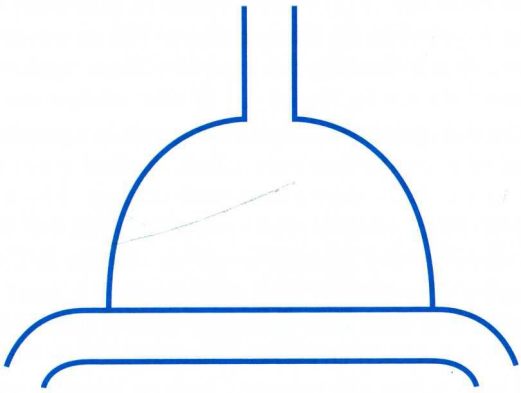
PART ONE

LUNG FUNCTION TESTS AND WHAT THEY MEAN

We learn how diseased lungs work by doing pulmonary function tests. Accordingly, Part One is devoted to a description of the most important tests and their interpretation. It is assumed that the reader is familiar with the basic physiology of the lung as contained in the companion volume, West JB, Luks AM. *West's Respiratory Physiology: The Essentials*. 10th ed. Philadelphia, PA: Wolters Kluwer, 2016.

VENTILATION

1



The simplest test of lung function is a forced expiration. It is also one of the most informative tests, and it requires minimal equipment and trivial calculations. The majority of patients with lung disease have an abnormal forced expiration volume, and, very often, the information obtained from this test is useful in their management. The test has great utility in primary care clinics when patients present for evaluation of chronic dyspnea. For example, it can be valuable in detecting asthma and chronic obstructive pulmonary diseases, extremely common and important conditions. This chapter also discusses a simple test of uneven ventilation.

TESTS OF VENTILATORY CAPACITY

Forced Expiratory Volume

The *forced expiratory volume* (FEV_1) is the volume of gas exhaled in *1 second* by a forced expiration from full inspiration. The *vital capacity* is the *total* volume of gas that can be exhaled after a full inspiration.

The simple, classic way of making these measurements is shown in **Figure 1.1**. The patient is comfortably seated in front of a spirometer having a low resistance. He or she breathes in maximally and then exhales as hard and as far as possible. As the spirometer bell moves up, the kymograph pen moves down, thus indicating the expired volume against time. The water-filled spirometer shown in Figure 1.1 is now seldom used and has been replaced by electronic spirometers that often provide a graph to be filed with the patient's chart or in the patient's electronic medical record.

Figure 1.2A shows a normal tracing. The volume exhaled in 1 second was 4.0 liters, and the total volume exhaled was 5.0 liters. These two volumes are therefore the forced expiratory volume in 1 second (FEV_1) and the vital capacity, respectively. The vital capacity measured with a forced expiration may be less than that measured with a slower exhalation, so that the term *forced vital capacity* (FVC) is generally used.

These values are reported both as absolute values and as a percentage of what one would predict for an individual of the same age, gender, and height.

The ratio of the FEV_1 to FVC (FEV_1/FVC) is also reported. The normal value is approximately 80% but decreases with age (see Appendix A for

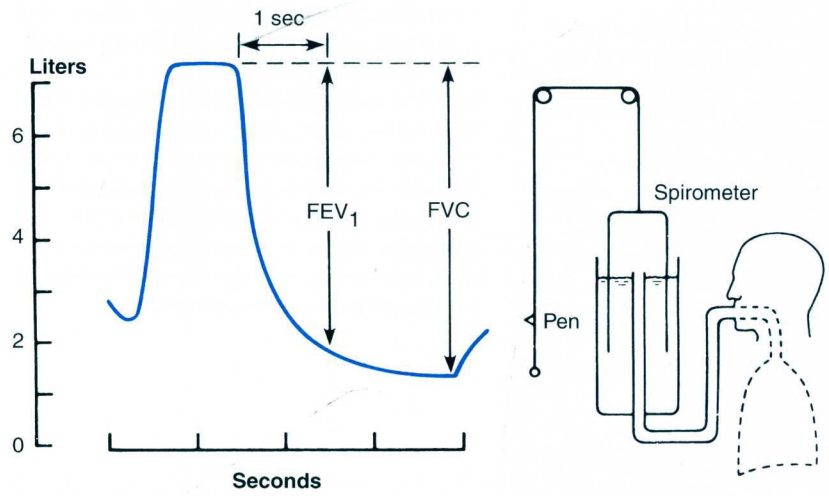


Figure 1.1. Measurement of forced expiratory volume (FEV_1) and forced vital capacity (FVC).

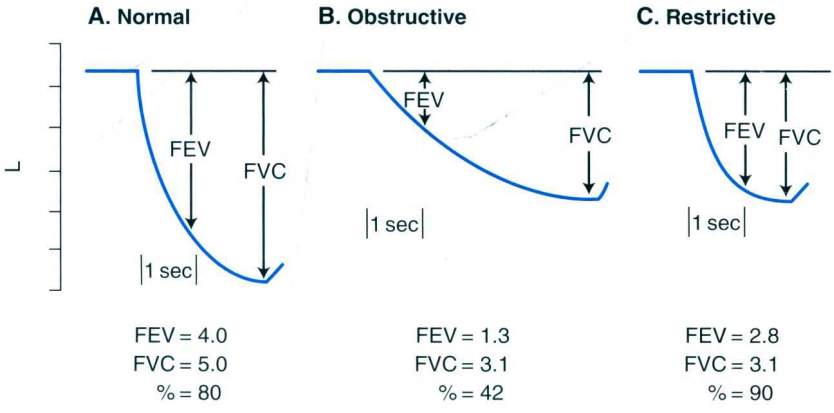


Figure 1.2. Normal, obstructive, and restrictive patterns of a forced expiration.

normal values). Expert guidelines put forth by various organizations include more refined definitions for the lower limit of normal for the FEV_1/FVC ratio, but the 80% cutoff is a useful threshold for the beginning student.

The FEV can be measured over other times, such as 2 or 3 seconds, but the 1-second value is the most informative. When the subscript is omitted, the time is 1 second.

Figure 1.2B shows the type of tracing obtained from a patient with chronic obstructive pulmonary disease (COPD). Note that the rate at which the air was exhaled was much slower, so that only 1.3 liters were blown out in the first second. In addition, the total volume exhaled was only 3.1 liters. FEV_1/FVC was reduced to 42%. These figures are typical of an *obstructive* pattern.

Contrast this pattern with that of **Figure 1.2C**, which shows the type of tracing obtained from a patient with pulmonary fibrosis. Here, the vital capacity was reduced to 3.1 liters, but a large percentage (90%) was exhaled in the first second. These figures mean *restrictive* disease. Note that the specific numerical values in these examples have been inserted for illustrative purposes and will vary between patients, but the general pattern will remain the same between patients with each category of diseases.

The patient should loosen tight clothing, and the mouthpiece should be at a convenient height. One accepted procedure is to allow two practice blows and then record three good test breaths. The highest FEV_1 and FVC from these three breaths are then used. The volumes should be converted to body temperature and pressure (see Appendix A).

The test is often valuable in assessing the efficacy of bronchodilator drugs. If reversible airway obstruction is suspected, the test should be carried out before and after administering the drug (e.g., albuterol by nebulizer or metered-dose inhaler). Both the FEV_1 and FVC usually increase in a patient with bronchospasm.

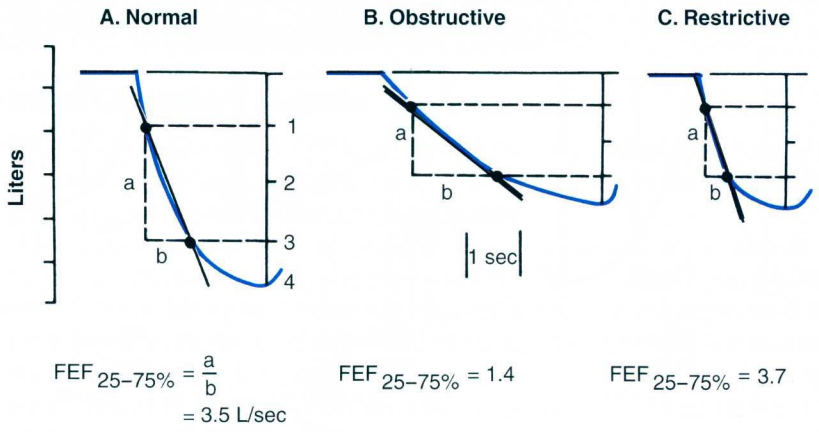


Figure 1.3. Calculation of forced expiratory flow ($FEF_{25-75\%}$) from a forced expiration.

FEV_1 and FVC

The 1-second forced expiratory volume together with the forced vital capacity is

- A simple test
- Often informative
- Abnormal in many patients with lung disease
- Often valuable in assessing the progress of disease

Forced Expiratory Flow

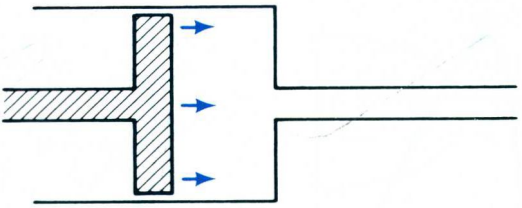
This index is calculated from a forced expiration, as shown in **Figure 1.3**. The middle half (by volume) of the total expiration is marked, and its duration is measured. The $FEF_{25-75\%}$ is the volume in liters divided by the time in seconds.

The correlation between $FEF_{25-75\%}$ and FEV_1 is generally close in patients with obstructive pulmonary disease. The changes in $FEF_{25-75\%}$ are often more striking, but the range of normal values is greater.

Interpretation of Tests of Forced Expiration

In some respects, the lungs and thorax can be regarded as a simple air pump **(Figure 1.4)**. The output of such a pump depends on the stroke volume, the resistance of the airways, and the force applied to the piston. The last factor is relatively unimportant in a forced expiration, as we shall presently see.

The *vital capacity* (or forced vital capacity) is a measure of the stroke volume, and any reduction of it affects the ventilatory capacity. Causes of stroke volume reduction include diseases of the thoracic cage, such as kyphoscoliosis,



↓ Stroke volume

Kyphoscoliosis
Interstitial lung disease
Poliomyelitis
Muscular dystrophy
Pleural disease

↑ Airway resistance

Asthma
Chronic bronchitis

Figure 1.4. Simple model of factors that may reduce the ventilatory capacity. The stroke volume may be reduced by diseases of the chest wall, lung parenchyma, respiratory muscles, and pleura. Airway resistance is increased in asthma and bronchitis.

ankylosing spondylitis, and acute injuries; diseases affecting the nerve supply to the respiratory muscles or the muscles themselves, such as poliomyelitis and muscular dystrophy; abnormalities of the pleural cavity, such as pneumothorax and pleural thickening; disease in the lung itself, such as fibrosis, which reduces its distensibility; space-occupying lesions, such as cysts; or an increased pulmonary blood volume, as in left heart failure. In addition, there are diseases of the airways that cause them to close prematurely during expiration, thus limiting the volume that can be exhaled. This occurs in asthma and chronic bronchitis.

The *forced expiratory volume* (and related indices such as the $FEF_{25-75\%}$) is affected by the airway resistance during forced expiration. Any increase in resistance reduces the ventilatory capacity. Causes include bronchoconstriction, as in asthma or following the inhalation of irritants such as cigarette smoke; structural changes in the airways, as in chronic bronchitis; obstructions within the airways, such as an inhaled foreign body or excess bronchial secretions; and destructive processes in the lung parenchyma that interfere with the radial traction that normally holds the airways open.

The simple model of Figure 1.4 introduces the factors limiting the ventilatory capacity of the diseased lung, but we need to refine the model to obtain a better understanding. For example, the airways are actually *inside*, not *outside*, the pump, as shown in Figure 1.4. Useful additional information comes from the flow–volume curve.

Expiratory Flow–Volume Curve

If we record flow rate and volume during a maximal forced expiration, we obtain a pattern like that shown in **Figure 1.5A**. A curious feature of the flow–volume curve is that it is virtually impossible to get outside it. For example,