Pathophysiology

Edited by

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With contributions by

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Translated by Terry Telger

Includes 74 figures

重庆医科大学附属第一医院图书馆
馆藏专用章

Springer-Verlag
New York • Heidelberg • Berlin

Prof. Dr. med. Albert A. Buehlmann†
Prof. Dr. med. E. Rudolf Froesch
Departement für Innere Medizin, Universität Zürich
CH-8091 Zürich

Prof. Dr. med. Günter Baumgartner
Neurologische Klinik und Poliklinik, Universität Zürich
CH-8091 Zürich

Prof. Dr. med. Paul G. Frick
Priv.-Doz. Dr. med. Walter A. Scheitlin†
Departement für Innere Medizin, Universität Zürich
CH-8091 Zürich

Priv.-Doz. Dr. med. Markus Knoblauch
Kreisspital, CH-8708 Männedorf

Prof. Dr. med. Paul Lichtlen
Medizinische Hochschule, 3000 Hannover

Prof. Dr. med. Martin Schmid
Medizinische Klinik, Stadtspital Waid, CH-8006 Zürich

Prof. Dr. med. P. Werner Straub
Medizinische Klinik, Universität Bern
CH-3010 Bern

† Deceased.

Library of Congress Cataloging in Publication Data

Buehlmann, Albert A. 1923–
Pathophysiology.

Translation of Pathophysiologie.
Bibliography: p.
Includes index.
1. Physiology, Pathological. I. Froesch, E. Rudolf, 1929– joint author. II. Title.
RB113.B8413 616.07 79-408

Title of the German Original Edition: *Pathophysiologie*. Springer-Verlag, Berlin–Heidelberg–New York, 1972, 1974, 1976.

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Printed in the United States of America.

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9 8 7 6 5 4 3 2 1

ISBN 0-387-90370-4 Springer-Verlag New York Heidelberg Berlin
ISBN 3-540-90370-4 Springer-Verlag Berlin Heidelberg New York

Preface

No doubt, there are many ways to practice "good" medicine, whatever this may be. Forty years ago the history, observation, and clinical examination of a patient led to diagnosis and therapy. Since then, we have learned a great deal about the biochemical and physiologic processes in the human body and about the pathogenetic mechanisms by which they are disturbed and lead to disease.

Today, the basis of clinical judgment and patient management is the understanding of pathogenetic mechanisms of disease. This concise and basic text of pathophysiology introduces the medical student to the basic pathophysiologic mechanisms. Each chapter begins with a short outline of the general physiologic and biochemical principles of an organ, a system, or a metabolic process; their possible derangements are described, with emphasis on the more important and more frequently occurring diseases. Although the primary purpose is to convey a basic understanding of clinical medicine to first-year medical students, many students have used this book as a convenient reference up to and after graduation.

In many medical schools, the student's introduction to clinical medicine is a pathophysiology course, lying between biochemistry and physiology on the one hand and bedside teaching on the other. This course has proved to be particularly useful when given in conjunction with courses covering other aspects of pathogenesis, such as immunology, pathology, and psychology, as well as basic principles of patient care. In addition, nurses, dieticians, laboratory technicians, and other medical personnel involved in patient care have found this book rewarding; the course of teaching in these professions too often does not impart sufficient insight into pathogenetic mechanisms.

We hope that this book will help medical students and physicians understand pathogenetic mechanisms in general terms. Its content is limited to what we consider essential: It is an introduction and must be followed by the study of more thorough textbooks, reviews, and original articles.

A. A. Buehlmann
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The Lungs and Respiration

A. A. Buehlmann

Physiology

The exchange of O_2 and CO_2 between air and blood is made possible by pulmonary ventilation and pulmonary blood flow. With the release of CO_2, the lungs also help regulate the acid–base balance.

Five factors are involved in the performance of these tasks. Abnormalities can occur in each of these factors, with resulting disturbances of pulmonary function:

1. The regulation of breathing, innervation of respiratory musculature, and the contractility of respiratory muscles
2. Pulmonary ventilation and its regional distribution as the result of airway resistance and the distensibility of the pulmonary parenchyma
3. The gas-exchange surface area
4. Resistance to diffusion between alveolar gases and the blood
5. Pulmonary blood flow and its regional distribution

The control circuit encompassing the arterial blood gases, respiratory centers, and respiratory musculature regulates the ventilation of the lungs via supplementary afferent nerves in such a way that the P_{O_2}, P_{CO_2}, and pH of the arterial blood remain fairly constant. At the same time, the uptake of O_2 and release of CO_2 may vary considerably in accordance with muscular

activity. The autonomous regulation of breathing can be voluntarily suspended for a limited time and is subject to psychic influences as well.

Regulation of Breathing

Two functions that share many of the same regulatory centers and pathways can be distinguished in the regulation of breathing:

1. The coordination of muscular innervation for rhythmic breathing
2. The regulation of ventilation to keep arterial blood gases constant

Unlike the myocardium, the respiratory musculature has no intrinsic rhythm. The interaction of various nerve centers is necessary for its coordinated periodic innervation. The "apneusis center" located in the lower pons region prolongs the activity of inspiratory stimulation. The "pneumotaxic center," which is located in the upper pons region and is influenced by numerous afferent inputs, excites the expiratory and inhibits the inspiratory stimulation of the bulbar respiratory center. Expansion of the lungs sends inhibitory impulses to the apneusis center via the vagus nerve, thereby increasing the stimulation of expiration. Excision of the pneumotaxic center combined with vagal transection results in cessation of breathing following inspiration.

A fall of the arterial P_{O_2} below 70 mm Hg leads to a marked increase in ventilation by excitation of chemoreceptors in the carotid bodies. The P_{CO_2} acts peripherally at the same sites as the P_{O_2}, but also exerts a central effect. Ventilation is increased by a rise in the P_{CO_2}. A decrease in pH has the same effect. Humoral regulation by the P_{O_2} and P_{CO_2}, is probably accomplished by intracellular pH changes in the peripheral and central receptors. The two phrenic nerves arising from cervical segments C3 to C5 supply the diaphragm, which is essential for inspiration, while intercostal nerves I to XII innervate the intercostal muscles, which are actively involved in both inspiration and expiration.

The initial adaptation of breathing during muscular efforts is probably controlled by mechanical receptors in joints and muscles. During steady state the ventilation is regulated by the arterial P_{CO_2} and P_{O_2}. Oxygen breathing during exercise reduces the ventilation more than during rest.

Pregnancy, fever, and thyrotoxicosis elevate the threshold for O_2, increase the sensitivity to O_2, and increase the stimulation by the carotid bodies at a given P_{O_2}, so that ventilation is augmented and arterial P_{CO_2} decreased. Myxedema, hypothermia, and starvation have the opposite effect.

Pulmonary Volumes and Distensibility of Lungs and Thorax

The uniform unfolding of the lungs at the onset of spontaneous respiration after birth is facilitated by a phospholipid surface film known

as the *surfactant*. A deficiency of surfactant promotes the formation of *hyaline membranes*, which may cause severe respiratory disorders in infants.

The *vital capacity*, or the volume of air between maximal inspiration and expiration, can be measured with a simple spirometer. This parameter is of great practical importance in assessing the ventilatory reserves. The *residual volume*, or the volume of gas remaining in the lungs after maximal expiration, is measured indirectly by a gas mixing method or by body plethysmography. Vital capacity plus residual volume give the *total lung capacity*. Normal values for total lung capacity depend primarily upon age, body size, and gender. Total lung capacity and vital capacity are about 15% lower in women than in men of equal size and age. These capacities continue to increase after the cessation of longitudinal growth, attain a maximum at age 23–25, and remain more or less constant through age 50 (Fig. 1-1).

Aging is normally accompanied by an increase in the compliance of the lungs. The declining recoil force of the pulmonary parenchyma leads to an increase in the residual volume and the gas content of the alveoli, which impairs gas mixing (Fig. 1-1).

The recoil tendency of the lungs is always expiratory, while that of the thoracic cage is expiratory during deep inspiration and inspiratory during deep expiration. This results in a resting position that corresponds to the *functional residual capacity*. The functional residual capacity is normally equal to 40%–50% of the total lung capacity and is greater in a sitting or standing position than in recumbency. It normally decreases with increasing abdominal content, as during pregnancy.

The distensibility, or *compliance*, of the pulmonary parenchyma is described in terms of the quotient dV/dP_{el}. In the absence of gas flow in the airways, the *pleural pressure* is equal to the elastic pressure, P_{el}. The quotient is not constant over the entire range of vital capacity, but decreases

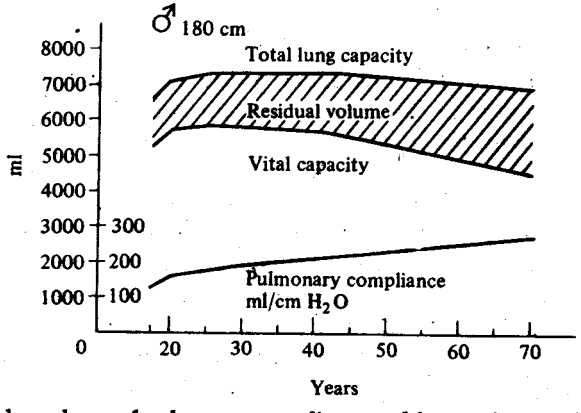

FIG. 1-1. Age dependence of pulmonary compliance and lung volumes.

with increasing inflation of the lungs. However, the surfactant between the alveolar gases and lung tissues alters the surface tension of pulmonary fluids in such a way that this volume/pressure ratio remains nearly linear for the individual alveolae and thus for the lung as a whole in the range of functional residual capacity with a respiratory volume less than one-half the vital capacity. Pulmonary compliance increases during growth, paralleling the increase in total lung capacity and vital capacity, and continues to increase with age in the adult (Fig. 1-1).

The compliance of the lungs is also influenced by their blood content. In an upright position the blood-rich basal regions of the lung have a lower compliance than the apical regions, which are characterized by low perfusion but good ventilation during rest.

The pleural pressure corresponds to the *intrathoracic pressure* and exhibits slight local variations. During inspiration it is 1–2 cm H_2O more negative in the basal region than the apical region. The respiratory changes in the intrathoracic pressure are transmitted to the esophagus where they can be measured by the intraesophageal-balloon technique.

The *dynamic compliance* is given by the quotient dV/dP_{el} during spontaneous respiration at respiratory volumes of approximately 1000 ml in the adult. The zero flow occurring at the interphase between respiratory excursions lasts only a fraction of a second. The *static compliance* is the quotient obtained during a prolonged cessation of breathing at the end of an inspiration. The statically measured compliance is greater than the dynamically measured value, especially when strongly divergent airway resistances are juxtaposed.

The compliance of the thoracic cage is of the same order of magnitude as that of the lungs. It is difficult to measure and of no clinical importance. The thoracic compliance is greatly increased during artificial respiration during paralysis or relaxation of respiratory and abdominal muscles by drugs.

Resistance to Flow; Ventilatory Reserves

The resistance to flow (viscance), given by the quotient $(P_{pl} - P_{el})$/ flow rate, is composed of the *aerodynamic airway resistance*, or simply resistance, and the *lung-tissue deformation resistance*. During normal respiration the resistance is 70%–80% of the viscance. The airway resistance corresponds to the quotient alveolar pressure/flow rate and can be measured by whole-body plethysmography with minimal patient discomfort. It is a function of gas viscosity, gas density, and airway geometry. Normally 75% of the resistance is localized in the larynx (glottis). Its numerical value in the adult is of the order of 1.5–2.5 cm H_2O/liter/sec. Flow resistance is doubled during nasal respiration. The primary pressure drop thus takes place in the extrathoracic airways under normal conditions. The diameter of the airways varies with the expansion of the lungs, increasing

somewhat during inspiration and decreasing during expiration. At high rates of flow both the inspiratory and expiratory resistances are increased because of turbulence. For the turbulent component of flow, the resistance increases with the square of the flow rate. When the turbulent component is strong, the resistance at a given flow rate decreases with air density at higher altitudes or if the N_2 in the respiratory air is replaced by the lighter He.

Ventilatory breathing reserves are described in terms of the maximum possible ventilation per minute. The maximum breathing capacity (MBC) is obtained at a respiratory frequency of 40–50/min and it is 25–30 times the vital capacity. During maximal physical exertion for a period of several minutes, ventilation levels off at 65%–75% of the MBC.

In spontaneous respiration the airway pressure is negative during inspiration and positive during expiration. Without external anatomical stabilization the extrathoracic airways would collapse during inspiration while the intrathoracic airways remain open due to the negative intrathoracic pressure. However, the positive intrathoracic and alveolar pressure, which is particularly marked during forced expiration, may lead to collapse of the intrathoracic airways if the primary pressure drop is not extrathoracic (in the larynx and nose) for pathologic reasons.

The flow resistance accompanying forced inspiration and expiration can be determined simply by measuring the forced inspiratory and forced expiratory volume ($FIV_{1.0}$, $FEV_{1.0}$). In this test the patient is instructed to inhale (exhale) as rapidly as possible after maximal expiration (inspiration). From 80%–90% of the vital capacity is normally inhaled in 1 sec, and 70%–80% percent exhaled. These relative values are independent of the vital capacity and generally do not decrease until after age 70. The maximum flow rate, or peak flow, is attained at the onset of forced expiration, the flow rate decreasing steadily thereafter. During forced inspiration a high flow rate is maintained over most of the vital capacity. The peak flow of forced expiration is not attained.

Ventilation and Circulation

The rhythmic changes produced in the intrathoracic, intra-alveolar, and intra-abdominal pressure by the respiratory muscles influence the circulation. The respiratory changes in intrathoracic pressure amount to approximately 5 mm Hg during quiet respiration and are transmitted to the heart, the superior vena cava, the aorta, and the intrapulmonary-prealveolar arteries and the veins. The alveolar capillaries are subjected to alternating positive and negative alveolar pressure. The intrathoracic pressure is positive only during forced expiration. The intra-abdominal pressure transmitted to the inferior vena cava and to its area of blood influx assumes positive values and increases during inspiration.

The venous return to the right heart is promoted during inspiration

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