

NONTUBERCULOUS DISEASES OF THE CHEST

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FOREWORD

DISEASES of the chest are taking a tremendous toll of human lives throughout the world. Nontuberculous morbid conditions of the lung, pleura, esophagus and other structures of the chest represent an important, often grave, problem clinically as well as individually and socially.

The basic motivation of preparing this volume has been to offer to the practicing physician, the chest specialist and to students of medicine, up-to-date, competent information relative to the manifold questions in the field of chest diseases.

Great progress has been made in the realm of diagnosis in recent years. Significant advances have augmented the potentialities of therapy during the past decade. Because the writers of this book have realized that there was an urgent need for a comprehensive, informative guide to the better understanding and more efficient treatment of chest diseases, this volume presents the essence of the vast clinical experience of men who devote their time to the practice of teaching of this specialty.

In order to assure an encompassing, precise and modern presentation of data concerning the multitude of diseases of the chest, this book has been written as a collective and cooperative project by 37 contributors from the United States of America, as well as Argentina, England, India, and Mexico.

For the adequate management of various ailments of the lung it is mandatory to acquire working knowledge of the physiology and pathologic physiology of respiration. For this reason, a complete chapter has been assigned to this particular subject.

In addition to a critical review of available diagnostic procedures, new methods are offered for the management of various clinical entities. The rapidity of modern transportation and the facility of long-distance travel make it obligatory that physicians familiarize themselves with so-called tropical diseases and with diseases endemic in distant geographic locations. When it comes to puzzling diagnostic problems, one must have a wide perspective. This volume is aimed to be of assistance in this respect.

For the sake of clarifying difficult diagnostic and therapeutic questions peculiar to certain age groups, it has been deemed expedient to deal with allergic bronchial asthma in children and in adults in two separate chapters.

Congenital diseases of the chest, pathologic conditions resulting from metabolic disturbances, diseases of the hemopoietic system, lymphomatoid diseases, lung changes secondary to trauma, collagen disease and to cardiovascular diseases are given due attention.

Today a large contingent of the adult population is employed in industrial work. With this situation in mind, considerable space has been given in the text to the discussion of industrial hazards and to the assaying of lung diseases caused by exposure to noxious fumes, gases and dusts. In the chapter on industrial diseases of the lung, valuable data are outlined on the estimation of disability in and the prevention of pneumoconiosis.

The pathology, symptomatology, diagnosis and treatment of foreign bodies in the air and food passages are covered in a well illustrated chapter.

This book has been prepared as a companion volume to FUNDA-MENTALS OF PULMONARY TUBERCULOSIS AND ITS COM-PLICATIONS published by Charles C Thomas, Publisher. Both of these volumes are sponsored by the American College of Chest Physicians.

It is the ardent hope and desire of the writers of this volume that it will be used by those interested in diseases of the chest to the best advantage to themselves as well as to their patients.

I take this opportunity to express my heartfelt appreciation and gratitude to all those whose inspiration, encouragement, helpful advice and technical assistance have stimulated and aided the writing and publication of this book.

Andrew L. Banyai, M.D., F.C.C.P., Editor

Milwaukee, Wisconsin

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NONTUBERCULOUS DISEASES

OF

THE CHEST

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CHAPTER I

THE PHYSIOLOGY AND PATHOLOGIC PHYSIOLOGY OF RESPIRATION

By G. G. Ornstein, M.D. and E. H. Robitzek, M.D.

AN EVALUATION of respiratory physiology in the human requires study of an elaborate process whereby reciprocal transport of oxygen from atmosphere to tissues and carbon dioxide in the reverse direction is accomplished. Rhythmic thoracic motion, under nervous and humoral control, brings atmospheric air into periodic contact with the alveolar membrane for further transport to distant tissues via the circulating blood. The anatomic structures and the physiologic mechanisms involved are so thoroughly integrated that no one can properly be separated and all must therefore be considered under combined and descriptive headings.

Thoracic Movements

The essential skeletal framework upon which the thoracic motion is accomplished is composed of the ribs, sternum, the vertebral column and the scapulae. The ribs connect posteriorly to the spine through a flexible system of multiple hinges, the costo-vertebral joints, and anteriorly to the sternum through the semi-flexible costo-sternal cartilages. Expansion of the skeletal cage occurs on inspiration and is a complex of upward, forward and lateral motions. It is accomplished through the contractions of the external intercostal muscles, the scaleni, the pectorals, the serrati and the trapezius and rhomboids. Further increase in the capacity of what has been called the thoracic "bell jar" is obtained by the descent of the diaphragm which exerts most of its thrust in a downward and anterior direction. Expiration is fundamentally passive except when the individual is under stress. Then the internal intercostals and the abdominals, facilitated by the abdominal viscera and a relaxed diaphragm, contract to diminish the volume of the thoracic cage. The anterior, inferior portions of the thorax are most expansile; the posterior, apical and apico-mediastinal portions are least so.

The lungs follow this scheme of expansion insofar as the essentially non-expansile root zone allows. There is relatively unhampered motion in the lung areas situated anteriorly and below the root regions; the apical and posterior areas may descend and come forward and laterally only in accordance with the descent, forward and lateral motion of the lung root. This motion is dependent upon a resilient bronchial tree the elasticity of which is derived from its muscular, elastic and cartilagenous components. On inspiration the motion is largely one of descent and a "fanning out" with concomitant elongation and widening. The reverse occurs on expiration.

The role of the diaphragm can scarcely be overemphasized as it relates to the efficacy of overall ventilation. Observations on normal subjects and upon pre- and post-operative patients by clinical, fluoroscopic and ventilatory studies indicate that satisfactory ventilation is overwhelmingly dependent upon proper diaphragmatic function. It is for this reason that surgical procedures for the lower lobes must be planned carefully and as carefully executed in order to preserve the integrity of the diaphragm.

The motivating factor in the control of the thoracic movements is the presence of the respiratory center situated in the medulla. In addition to its inherent rhythmicity this center is influenced by nervous impulses from the lung parenchyma via the vagus which stimulate deflation on full inspiration and inflation on full expiration (Hering-Breuer reflex). The medullary center is futher stimulated by impulses originating in the cortex, the carotid sinus, the aortic node, the skin, and many peripheral and cranial sensory nerves, the diaphragm and other respiratory muscles. Aortic hypertension, acting through receptors in the aortic arch and the carotid sinus causes depression of respiration; the converse is also true. Further influence upon the respiratory center is provided by humoral elements, notably carbon dioxide excess, oxygen deficit and acid-base alterations. Of these, the effect of carbon dioxide is probably most important; the factor of oxygen lack, operating largely through the carotid sinus, is probably the least. By virtue, then, of these complicated respiratory movements is the alveolus assured of a constant and adequate supply of oxygen. In the phase of inspiration the increased volume of the thoracic cage, in accordance with Boyle's law, reduces the intrathoracic pressure and establishes a gradient which induces an

influx of air from the relatively higher pressured atmosphere. In expiration the converse operates.

Ventilation

The logical purpose of rhythmical thoracic movements is the provision of atmospheric air into intimate contact with respiratory epithelium and the removal of excess carbon dioxide. This process is known as ventilation. A number of conventional terms have been utilized and are generally accepted in the description of the ventilatory fractions composing total ventilation.

Tidal air: This fraction is usually described as the volume of air that is inhaled and exhaled during a single quiet respiratory cycle. Values of from 200 to 800 cc. in clinically normal individuals reduce the significance of this determination and complicate its interpretation. It is important to record the conditions under which any "quiet respiratory cycle" is measured since wide variations obtain, even in the same individual, depending upon whether the subject is basal, recumbent, upright, and emotionally stimulated or depressed. The values obtained are found to vary with habitus, with the efficiency of gas exchange and with the chemico-circulatory status of the blood. In the average man the tidal air roughly approximates 500 cc.

Complemental air: Some authors take the total volume of air that can be inhaled from the beginning of a quiet respiration as the complemental air. Others begin measurement at the conclusion of a quiet inspiration. This volume averages 3000 cc. in the former instances, 2500 cc. in the latter.

Supplemental or reserve air: This fraction, averaging about 1000 cc., represents the volume of air which can be forcibly expired following a quiet expiration.

Vital capacity: The sum of the tidal, complemental and supplemental volumes is known as the vital capacity. The average volume usually given is 4000 cc.

Residual air*: Following a full forced expiration approximately 1200 cc. of air is still present within the lungs. This fraction is known

^{*}Air which is expired is susceptible of easy measurement through the use of any of a number of quantitatively calibrated receptacles. Residual air is determined by analysis of a known mixture of gases after a period of rebreathing. The dilution method of Van Slyke and Binger is satisfactory. More recently greater accuracy has been provided through the use of the oxygen dilution method of Darling et al. Lung volumes at any phases of respiration may similarly be determined.

as the residual air. Included with this volume is the *minimal air* volume which, while present in the alveoli, cannot readily be measured, and the absence of which in atelectasis causes pulmonary tissue to lose its buoyancy. It has been found to amount to about 200 cc.

Total capacity: The total volume of air which the lungs may contain after full inspiration, commonly averaging 5000 cc., is known as the total capacity.

Midcapacity: The quantity of air present within the lungs at the mid point during a quiet respiration is variously known as the midcapacity, the subtidal volume and the functional residual air. Some authors prefer to consider this volume to be more properly measured from the point of expiration after a quiet respiration. It thus represents the sum of the supplemental and residual air volumes.

Resting minute ventilation: The total volume of air ventilated in one minute under conditions of rest is known as the minute volume or the resting minute ventilation. The average minute volume is about 7000 cc. but varies from 4000 to 9000 cc. in normal individuals. This variability has been amply demonstrated in hundreds of determinations; the factors dictating extreme care in interpretation are those noted under "tidal air"—of which, in a sense, the resting minute ventilation is a function.

Maximum minute ventilation: The maximum quantity of air that can be ventilated in one minute under conditions of forced breathing, first described by Hermannsen, is known as the maximum breathing capacity or maximum minute ventilation. It varies widely but averages 154 liters for males and 100 liters for females (Cournand et al.).

Ventilatory factor: The quotient of maximum minute ventilation divided by the resting minute ventilation is known as the ventilatory factor (Ornstein et al.). Composed as it is of the two moderately variable figures referred to in the preceding two paragraphs, this factor is subject to some fluctuation. It is this feature which endows the ventilatory factor with its sensitivity in the early revelation of deficiencies in pulmonary reserve function. The average factor is 20 for men and 13 for women. Cournand and Richards have utilized these figures in another manner in arriving at their estimate of the breathing reserve. By expressing the difference between resting and maximal minute ventilations as a percentage of the maximal minute ventilation they obtain the percentage breathing reserve. The threshold of dyspnoea is usually found in the 60 to 70 per cent range. Essentially similar purposes are served by estimations of the ventilatory factors and breathing reserves; the former is a more

sensitive index and less complicated in its derivation. In practical usage an arbitrary figure of 6 has been determined as the dividing line between an adequate and an inadequate ventilatory factor. Major thoracic surgery is ill-advised for patients whose performance is below this value.

It must be emphasized that all of the above figures are subject to wide variations depending on the height, weight, age, sex, body configuration and state of physical training of any subject. The vital capacity retains a fairly well fixed relationship with the surface skin areas of the body and approximates ten volumes of tidal air. Measurement of the vital capacity, while a rough estimate of the pulmonary ventilation, gives little information about the pulmonary reserve and none of the gaseous exchange across the respiratory epithelium. Thus, an apparently satisfactory vital capacity may be associated with deficient oxygen utilization and an apparently deficient vital capacity with excellent utilization. Consideration of the total of each of these factors is required for the proper evaluation of pulmonary efficiency. This will be considered in the discussion of pathologic physiology.

Gaseous Exchange

We are concerned here principally with gas exchange at the alveolar membrane and in the peripheral capillary bed. The catalytic oxidationreduction reactions involved in cellular metabolism are somewhat oblique to this discussion and will not be considered here. Our prime interest is in the delivery of oxygen to the tissues and the removal of carbon dioxide.

The alveolar membrane constitutes the first barrier to be penetrated. It is composed of alveolar epithelium and capillary endothelium and is thereby two cells in thickness. That the capillary may be uncovered at some points has been pointed out; in these zones the thickness may be unicellular. The membrane has the physical characteristics of any wet membrane. Through it gases diffuse in accordance with the laws of partial pressures, their coefficients of solubility and their rates of diffusion. Alterations of any of these or alterations in the character or surface of the membrane cause changes in the rate or degree of gaseous exchange. On the other side of the membrane is the receptor system, namely, the blood. It plays its role of supplying hemoglobin and bicarbonate as vehicles for the transport of oxygen and carbon dioxide respectively. Alterations in the rate of blood flow, concentration of hemoglobin or concentration of bicarbonate causes alterations in the gas-carrying