Pulmonary Medicine

Editor

Clarence A. Guenter, M.D.

Professor and Head,
Division of Internal Medicine
University of Calgary;
Director, Department of Internal Medicine
Foothills Provincial General Hospital
Calgary, Alberta

Associate Editor

Martin H. Welch, M.D.

Associate Professor of Internal Medicine
Wichita State University Branch
University of Kansas School of Medicine;
Chief, Medical Service
Veterans Administration Center
Wichita, Kansas

With Six Contributors

J. B. Lippincott Company
Philadelphia • Toronto

ISBN 0-397-50364-4

Library of Congress Catalog Card Number 76-49517

Printed in the United States of America

3 5 4

Library of Congress Cataloging in Publication Data

Main entry under title:

Pulmonary medicine.

Bibliography: p.
Includes index.
1. Respiratory organs—Diseases. 2. Respiration.
I. Guenter, Clarence A. II. Welch, Martin H.
DNLM: 1. Respiratory tract disease. WF140 P983
RC731.P84 616.2 76-49517
ISBN 0-397-50364-4

Preface

This book offers an integrated approach to pulmonary medicine. Consequently, the clinical recognition, evaluation, and management of the diseases of the respiratory system are presented within the framework of lung function, pathophysiology, diagnostic radiology, and microbiology. Within the discussions of clinical recognition, evaluation, and management, the reader will find a strong emphasis upon practical guidelines. Because the spectrum of diseases in patients cared for by the internist and the chest physician today involves new areas of special emphasis, we have included full discussion of environmental factors, pre- and postoperative management, chest trauma, and respiratory failure in all of its aspects.

So that one may approach diagnosis and treatment on the basis of an understanding of functional changes, we begin with an introduction to respirology. In Part I, the respiratory consequences of environmental changes, airways function, ventilatory function, and of the respiratory function of the lungs and blood must now more than ever be fully understood prior to the specific management of respiratory illness.

Part II centers upon respiratory disease itself, with the emphasis being placed upon the presenting problem of the patient with respiratory illness. This problem-oriented approach is especially necessary in pulmonary medicine, when the specific etiology is not immediately apparent, and permits reasonable and effective management, while the precise cause of the disease is being delineated. Patients with acute respiratory tract infections, chest trauma, or pleural disease commonly present with just those problems.

A number of problems do not fall into a common clinical presentation. These include intrathoracic tumors, chest wall disorders, and pulmonary vascular disease (particularly the ubiquitous process of pulmonary thromboembolism). These are covered in separate sections for ease of comprehension.

In order to provide a sharp focus and unify presentation, the editors restricted contributions to a small number of institutions, availing themselves of the collaboration of authors with recognized eminence in clinical management who could provide standardization and cohesiveness. Each of the contributors also has an ongoing interest in clinical investigation and education in pulmonary medicine.

No attempt has been made in this volume to be exhaustive in areas covered frequently and well in the developing literature. We offer, rather, a distillation of what is known, so that what is presented can be understood readily—and applied to patient management effectively.

The bibliographies have been abbreviated and will reveal to the reader an emphasis upon recent articles which offer a further expansion into current thinking and practice. Sufficient documentation of historical work exists to remind us of the

heritage upon which our knowledge is based. Consequently, we have not credited individual investigators for many important contributions, even though those contributions are reflected in the information presented herein.

Clinical medicine is an unspecified mixture of scientific fact, judgment, and personal bias. Upon dealing with subjects about which there is recognized controversy, we have made a deliberate effort to point out those aspects which are based on scientific information and those which rely more exclusively upon clinical judgment. It is hoped that the student, recognizing incongruities that are not apparent to us, will himself search for better evidence for clinical approach.

CLARENCE A. GUENTER, M.D.
MARTIN H. WELCH, M.D.

Contents

PART ONE

Introduction to Respirology

1 The Respiratory Environment

Clarence A. Guenter, M.D.

GASES IN THE RESPIRATORY PATHWAY

Atmospheric Gases

The ubiquitous nature of oxygen, man's most immediately life-sustaining natural resource, may diminish the intrigue of the study of environmental gases. Curiosity is frequently stimulated by the threat of extinction of natural resources, and no satisfactory evidence can be accumulated as to a significant loss of available oxygen in our atmosphere. How the relatively constant concentration of gases (Table 1-1) is maintained in the atmosphere has not been established. Estimates indicate that the proportion of oxygen has been stable for several millennia.[2] The major source of oxygen for environmental replenishment is photosynthesis by plants. The major utilization of oxygen is by land and marine animals and by the combustion of fossil fuels. The enormous reserves of oxygen in the earth's atmosphere and in the oceans suggest that there will be no global life-limiting oxygen deficiency in the foreseeable future. A recent estimate indicated that if all the fossil fuels were completely burned instantaneously, a decrease of 3 per cent of the earth's global oxygen resources would occur. Variably contested evolutionary hypotheses attempt to explain these atmospheric conditions; however, there are major inadequacies in these hypotheses.

The convenience of unlimited oxygen reserves provides the respirologist with eternal optimism. Fortunately, the other major atmospheric gases are as conveniently arranged. Nitrogen and argon, the major inert gases, have no known biologi-

Table 1-1. Composition of Atmospheric Gas (Dry, Sea Level)

Gas	*mm. Hg*	*% Total*
Nitrogen	590	78.09
Oxygen	158	20.95
Carbon Dioxide	0.2	0.03
Other Inert Gases*	7.0	0.93
Water Vapor†	—	—
Total	760.2	100.00

*Predominantly argon.

†Water vapor pressure varies greatly with temperature (see Factors Affecting Alveolar Gas Partial Pressures, p. 5), but minimally with altitude. Although no figure is included in the above table, water vapor is never completely absent from the environment.

(Adapted from Altman, P. L., and Dittmer, D. S.: Biological Handbooks, Respiration and Circulation. Federation of the American Society of Experimental Biologists, 1971[1])

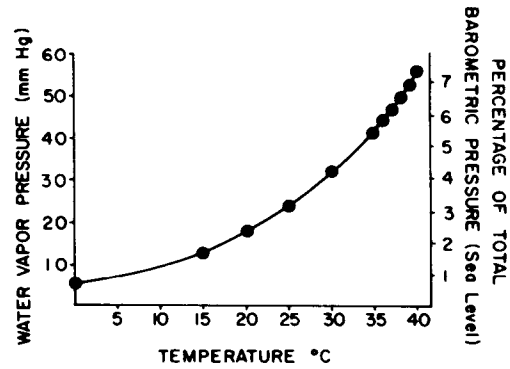

Fig. 1-1. The water vapor pressure, in mm. Hg, is plotted on the ordinate on the left, and water vapor pressure as a percentage of total barometric pressure (at sea level) on the ordinate on the right. All values are for fully saturated gas. Note that water vapor pressure in cold environments approaches zero. Sub-zero temperatures frequently encountered in winter in northern climates result in water vapor pressure of less than 5 mm. Hg, even when the atmosphere is fully saturated. As a result, warming of such air in heated buildings produces an atmosphere with a relative humidity of approximately 10 per cent. Humidification of air under these circumstances may be a major objective of inside air conditioning. Even when air that is fully saturated with respect to vapor pressure is warmed from room temperature to body temperature (approximately 20° to 37° C.), it becomes less than 50 per cent saturated. (Adapted from Altman, P. L., and Dittmer, D. S.: Biological Handbooks. Respiration and Circulation. Federation of the American Society of Experimental Biologists, 1971[3])

cal effects in concentrations that occur in the atmosphere. Carbon dioxide is suitably present in trace amounts only, permitting the metabolizing body to efficiently discharge its accumulated carbon dioxide into the atmosphere.

At sea level, the barometric pressure is about 760 mm. Hg. This pressure is comprised of the pressures of all the gases that are present in the atmosphere, regardless of temperature (see Table 1-1). (Dalton's law of partial pressures states that the partial pressure of each gas in a mixture is independent of the other gases present; therefore, the total pressure equals the sum of the partial pressures of all gases present.)

Since man's environment embraces a wide range of atmospheric pressures, functional amounts of the gases are expressed more conveniently as partial pressures than as concentrations. For example, a person traveling from sea level, at a barometric pressure of 760 mm. Hg, to a high altitude, with a barometric pressure of 380 mm. Hg, is still breathing 20.95 per cent oxygen; however, the 50 per cent decrease in available oxygen per unit of atmospheric volume is reflected in the partial pressure of oxygen (Po_2), which has decreased from 160 to 80 mm. Hg.

Water vapor is a significant variable in atmospheric gas composition. Water vapor pressure (P_{H_2O}) is dependent on available water and (H_2O) capacity at fixed temperature. Thus, at 0° C., air that is fully saturated has a water vapor pressure of 4.6 mm. Hg, whereas at body temperature (37° C.), the water vapor pressure is 47 mm. Hg (Fig. 1-1). In the human body, all gases are saturated with water vapor. When dry air is inhaled, it is humidified; the increase in temperature in the body increases the capacity of the gases to carry water vapor. Water vapor is added to gases in the respiratory airways by the moist linings of the respiratory tract.

Gases in the Body

The interrelationship of atmospheric gases and, in particular, the effect of water vapor pressure on the partial pressures of the gases in the respiratory passages is demonstrated in Table 1-2. Nitrogen and oxygen are the only gases present in noteworthy concentrations in dry air; however, moist, warm tracheal air contains significant amounts of water vapor. Because the total barometric pressure is unchanged in the trachea, water vapor displaces each of the other gases, thereby decreasing their partial pressures. Thus, the partial pressure of oxygen is decreased from 159.1 to 149.2 mm. Hg, simply by the warming and humidification of air. As the gas advances to the lower respiratory tract, where gas exchange takes place, there are additional changes in its composition as indicated in the alveolar air column (Table 1-2). These changes in gas composition do

Table 1-2. Composition of Respired Gases

Gas	*Dry Air*		*Moist Tracheal Air*		*Alveolar Air*	
	Partial Pressure (mm. Hg)	*Percent Total*	*Partial Pressure (mm. Hg)*	*Percent Total*	*Partial Pressure (mm. Hg)*	*Percent Total*
Nitrogen*	600.7	79.02	563.6	74.16	568.0	74.74
Oxygen	159.1	20.95	149.2	19.63	105.0	13.82
Carbon dioxide	0.2	.03	0.2	.03	40.0	5.26
Water vapor	0.0	0.00	47.0	6.18	47.0	6.18
Total†	760.0	100	760.0	100	760.0	100

*This value includes other inert gases separately itemized in Table 1-1.
†Values may be calculated for other altitudes by utilizing local barometric pressures (e.g., mean barometric pressure in Denver of 640 mm. Hg, or in Calgary, Alberta, 660 mm. Hg). For dry gas, the Po_2 at a barometric pressure of 660 mm. Hg: 20.95 × 660 = 138 mm. Hg. For saturated gas at 37° C.: 20.95 × (660 − 47) = 128 mm. Hg.

not merely reflect physical changes in temperature and water vapor pressure, but result from the actual uptake of oxygen from the alveolar space and the release of carbon dioxide into the alveolar space through the gas-exchanging surfaces of the peripheral lung units (see Chap. 4). Under normal circumstances, oxygen is taken up by the blood in the lungs in proportion to the utilization of oxygen by the tissues; carbon dioxide is released by the blood flowing through the lungs in proportion to the production of carbon dioxide by the tissues.

Figure 1-2 demonstrates the changes in oxygen and carbon dioxide along the entire respiratory pathway under various conditions. Each stage or physiological compartment is completely dependent on the gas pressures in the stage that precedes it and in the stage that follows it. The gases are predominantly moved by convection from the atmosphere to the alveolar space, and vice versa. Figure 1-2A illustrates the normal progression of oxygen and carbon dioxide partial pressures through the respiratory pathway.

The availability of oxygen to the blood is most immediately dependent on oxygen in the alveolar compartment; similarly, the release of carbon dioxide from the blood is most immediately dependent on the level of carbon dioxide in the alveolar compartment. Therefore, an understanding of factors that determine the partial pressures of these gases at the alveolar level is fundamental. Sequentially, the first important factor is the partial pressure of the inhaled gases. It is apparent that if the inhaled Po_2 were decreased, it would necessarily result in a decreased Po_2 of moist tracheal air. This in turn would result in a decrease in alveolar Po_2. High-altitude environments characteristically result in a decreased barometric pressure and a decreased partial pressure of inspired oxygen, compared with sea-level values.

Factors Affecting Alveolar Gas Partial Pressures

1. Inspired Po_2, Pco_2
2. Volume of oxygen uptake and carbon dioxide release in lungs
   a. Oxygen consumption and carbon dioxide production at tissue level (metabolic rate)
   b. Fuel being utilized by tissues and metabolic respiratory quotient
3. Volume of alveolar gas exchange (alveolar ventilation)

The second important factor determining the alveolar gas partial pressures is the oxygen uptake and carbon dioxide release in the lungs. It is clear that the decrease in oxygen would be precisely the same as the increase in carbon dioxide if the oxygen consumption and carbon dioxide production were equal. Although this is true under certain circumstances, the alveolar oxygen uptake and carbon dioxide release

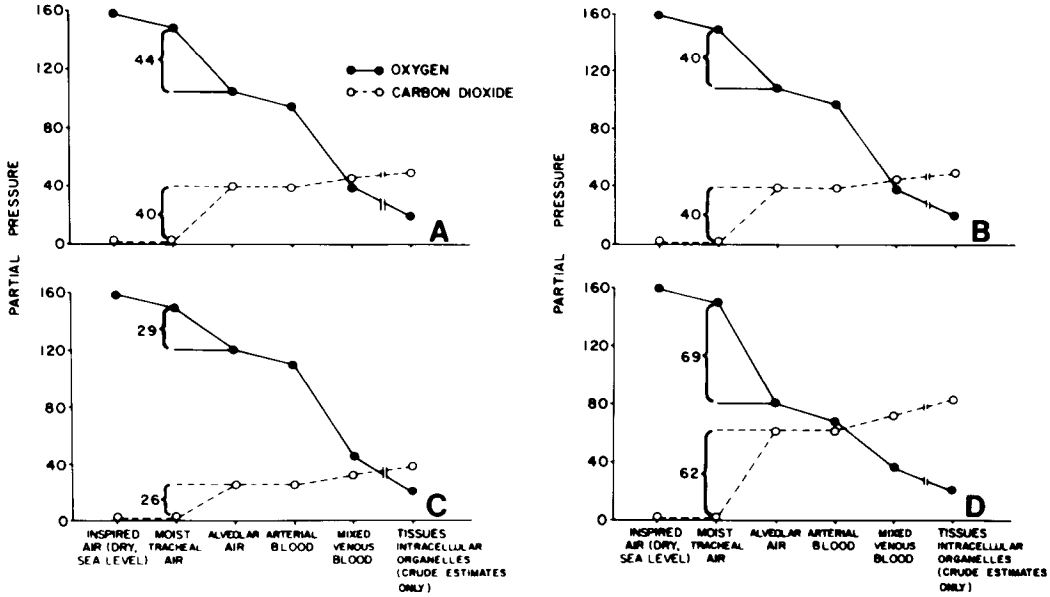

Fig. 1-2. Partial pressures of oxygen and carbon dioxide in the respiratory pathway steady-state conditions. (*A*) Normal resting. The solid line indicates the partial pressure of oxygen beginning in the inspired air on the left and progressing to the moist tracheal air, alveolar air, arterial blood, mixed venous blood, and, ultimately, tissues. The values represented indicate the average value in each region of the pathway, but are entirely speculative at the final level of the tissues. Movement of oxygen from the inspired air to the trachea, and from the trachea to the alveolar spaces is almost entirely by convection. Movement of oxygen from the alveolar spaces to the blood is by diffusion. Movement of oxygen within the arterial blood to the systemic capillaries is, again, by convection, as a result of blood flow. Movement of oxygen from the blood to the tissues is by diffusion. The interrupted line demonstrates the partial pressures of carbon dioxide in each region of the respiratory pathway; the same transport processes apply as for oxygen.

(*B*) Normal, resting, metabolic fuel—carbohydrate. The partial pressures for oxygen and carbon dioxide are again illustrated. Note that the alveolar extraction of oxygen and release of carbon dioxide are equal, thus resulting in a respiratory exchange ratio of 1.0. Thus, the alveolar ventilation and the partial pressures of carbon dioxide and oxygen become somewhat dependent on the fuel being metabolized.

(*C*) Effect of increased ventilation without increased metabolic rate. In a hyperventilating individual, the partial pressure of oxygen may be increased throughout the respiratory pathway, and the partial pressure of carbon dioxide decreased. The values illustrated apply only to an individual who has reached a steady state or equilibrium throughout the respiratory pathway.

(*D*) Effect of increased metabolic rate without increased alveolar ventilation. This illustration demonstrates the marked reduction of oxygen and the marked elevation of carbon dioxide throughout the body's respiratory pathways, as a result of the metabolic rate which is in excess of the level of ventilation.

(*B* adapted from Saltzman, H. A., and Salzano, J. V.: Effects of carbohydrate metabolism upon respiratory gas exchange in normal men. J. Appl. Physiol., *30*:228, 1971[4])

are generally not equal. As indicated in Figure 1-2A, the decrease in Po_2 of 44 mm. Hg which takes place at the alveolar level is compared with the increase in carbon dioxide of 40 mm. Hg. If oxygen uptake and carbon dioxide release were precisely equal, these changes would also be of equal value.

The obvious discrepancy between oxygen utilization and carbon dioxide produc-

tion referred to above is a result of the particular fuel being metabolized. The ratio of CO_2 production to oxygen consumption has been termed the *metabolic respiratory quotient*. For example, when pure carbohydrate is metabolized, the oxidation equation may be summarized as follows: $C_6H_{12}O_6+6O_2 \rightarrow 6CO_2+6H_2O$ (+ energy).

It is apparent that when pure carbohydrate, such as glucose, is being metabolized, precisely the same amount of carbon dioxide is produced as oxygen utilized. Thus, the metabolic respiratory quotient is 1.0. Similarly, one can calculate the metabolic respiratory quotient for protein (approximately 0.8), fat (approximately 0.7), and ethanol (0.67). Therefore, the alveolar gas composition may be influenced by the fuel that the individual is metabolizing. For example, persons utilizing carbohydrate extract less oxygen in relation to the carbon dioxide production. Because the carbon dioxide controls ventilation, there is an increase in the alveolar oxygen partial pressure.[4] When the alveolar gas, blood flow, and tissue metabolism are in equilibrium, this results in an oxygen extraction from inspired air to alveolar gas of 40 mm. Hg and a carbon dioxide washout from alveolar gas to atmospheric air of 40 mm. Hg (Fig. 1-2B). The precise fuel being metabolized at any time by the body, or by specific tissues, cannot be ascertained. The oxygen extraction at the alveolar level, and the carbon dioxide washout from the alveoli, however, can be readily assessed. This indirect measurement of the average metabolic respiratory quotient has been termed the *respiratory exchange ratio* (R). It is clear that when the entire respiratory pathway is in equilibrium, this measurement accurately reflects the average of the total body metabolic respiratory quotient. Frequently, however, steady-state conditions do not prevail, and conditions such as transient hypoventilation or hyperventilation alter the respiratory exchange ratio, so that it does not reflect tissue metabolism accurately.

Several important circumstances that result in altered respiratory exchange ratios should be noted. Completely normal increases in the respiratory exchange ratio are seen at the onset and immediately following cessation of exercise. During these times, alveolar ventilation exceeds tissue metabolic requirements. This does not result in increased oxygen extraction from the alveoli, because oxygen stores cannot be significantly supersaturated. It does result, however, in depletion of carbon dioxide from tissue carbon dioxide stores. Thus, the carbon dioxide washout is greater than the oxygen consumption, and the respiratory exchange ratio may exceed 1.0. Similarly, during transient periods of metabolic acidosis, such as occur with anaerobic metabolism, the oxygen utilization may be relatively fixed, but due to circulating organic acids, body carbon-dioxide stores in the form of bicarbonate and blood carbonic acid are dissociated to H_2O and CO_2, with exhalation of the CO_2. The availability of large body-CO_2 stores thus permits temporary depletion of carbon dioxide, resulting in a respiratory exchange ratio that may exceed 1.0. This abnormal release of CO_2 has been studied as an early indication of tissue hypoxia. The level of exercise (as a determinant of the amount of metabolism) that produces such an increase in the respiratory exchange ratio has been suggested as a sensitive test for metabolic acidosis resulting from limited oxygen delivery (reflecting cardiorespiratory disease).[5] Conversely, circumstances under which ventilation is depressed and the carbon dioxide washout diminished, such as transient alveolar hypoventilation due to breath holding or sudden sedation, result in a respiratory exchange ratio less than that which would be appropriate for the fuel being metabolized at the time, e.g., < 0.70.

Figure 1-2C illustrates changes in partial pressures of oxygen and carbon dioxide in the respiratory pathway as a result of an increase in ventilation above body requirements, under steady-state conditions. Note that under these circumstances, the partial pressures of the inspired gases in moist tracheal air are unchanged. As a result of a marked increase in alveolar ventilation (hyperventilation), an increased amount of oxygen is available at the alveolar level when compared with

normal conditions illustrated in Figure 1-2A. Consequently, in this example, the alveolar P_{O_2} is 120 mm. Hg. This permits increased oxygen availability for diffusion into the arterial blood and throughout the delivery system, with eventual increased oxygen availability at the tissue level. Carbon dioxide partial pressures are comparably affected. Due to the increase in alveolar gas exchange, the amount of carbon dioxide remaining in the alveoli is decreased. Thus, the alveolar P_{CO_2} is decreased to 26 mm. Hg, the arterial P_{CO_2} is approximately the same, and the tissue CO_2 is washed out to a somewhat lower level than indicated under normal circumstances in Figure 1-2A. Under these steady-state circumstances, the extraction of oxygen from alveolar gas results in a P_{O_2} decrease of 29 mm. Hg from inspired air to alveolar gas, and a P_{CO_2} decrease of 26 mm. Hg from the alveolar gas to the atmosphere.

The partial pressures depicted in Figure 1-2A represent approximate values seen throughout the respiratory tract, under normal resting steady-state conditions. It is quite clear that if tissue metabolism were suddenly accelerated, as during exercise, oxygen utilization and carbon dioxide production would be increased. Under these circumstances, the body generally responds by delivering more oxygen to the alveoli and by removing the carbon dioxide more rapidly by increasing alveolar ventilation. If ventilation were constant, however, the increased extraction of oxygen and production of carbon dioxide would severely alter alveolar gas levels. This situation is illustrated in Figure 1-2D, where alveolar ventilation has been held constant but the metabolic rate has increased. Note that the marked decrease in tissue oxygen tension results in increased extraction of oxygen from the blood at the tissue level, so that the mixed venous blood P_{O_2} is diminished. Because alveolar ventilation and, consequently, alveolar oxygen delivery are fixed, there is a decrease in alveolar P_{O_2}. Similarly, the high metabolic rate results in a high tissue P_{CO_2}, increased transport of carbon dioxide from the tissues, and increased mixed venous P_{CO_2}. Because the alveolar ventilation is fixed, removal of carbon dioxide from the alveoli is inadequate, and the alveolar P_{CO_2} is elevated. The P_{CO_2} of blood leaving the alveoli (arterial blood P_{CO_2}) is elevated. This represents an inadequate response to tissue respiratory requirements, and because the response is inadequate primarily with respect to ventilation, it is termed *alveolar hypoventilation*. Note that the alveolar extraction of oxygen results in a drop of P_{O_2} from inspired air to alveolar gas of 69 mm. Hg, and the washout of carbon dioxide at the alveolar level results in a decrease in P_{CO_2} from alveolar gas to the atmosphere of 62 mm. Hg. This, then, represents a new steady state in which inadequate alveolar ventilation is responsible for marked alterations in alveolar gas partial pressures.

In each of the above circumstances, the person has developed an equilibrium with respect to carbon dioxide and oxygen partial pressures throughout the respiratory pathway. Thus, alveolar gas exchange, blood transport of gases, and tissue metabolism reflect a continuous and stable exchange of oxygen and carbon dioxide. Within wide physiological ranges, once an equilibrium has been established, the actual rate of blood flow (cardiac output) is not a determinant of the gas levels in the alveoli or respiratory airways.

VARIATIONS IN ATMOSPHERIC CONDITIONS

Decreased Barometric Pressure

Human awareness of adverse effects of the low barometric pressure of high altitudes is recorded in literature regarding the Spanish invasion of the Western Hemisphere. As early as the 1500's, the Spanish complained of the "thinness of the air" in the high mining areas of South America. Their concern about the inability to have offspring suggested that this, too, was a result of environmental hazard. Probably, detailed analysis of folk literature from other high-lying areas in the world would produce additional historical evidence of recognition of these atmo-

spheric problems. Acute mountain sickness, at an elevation of about 10,000 feet, was first described in 1671 by the physiologist, Borelli. Subsequently, many additional descriptions of illness were recorded, and we now recognize an entire spectrum, ranging from mild alterations in judgment at relatively low altitudes, to severe acute or chronic altitude disease at higher altitudes. Although persons with previous cardiorespiratory disease may be significantly less well at altitudes as low as 5,000 feet, more than 3,000,000 persons in South America live above 13,000 feet, and there are communities in the United States at altitudes above 7,000 feet. Furthermore, recent wars have been fought between the Indians and the Chinese at altitudes in excess of 15,000 feet.

Intriguing studies of human adaptation to high altitudes describe life on the Andean Plateau, from Colombia to Chile in South America.[6] This plateau rises above 8,000 feet and is suitable for human habitation up to the permanent snow line (17,500 feet). There are more than 10,000,000 persons living in this zone, and historical and archeological records indicate that it has been densely populated for a long time. The Inca Empire had its center in this zone, and it is clear that reproduction by the natives effectively perpetuated the race. Studies of selected communities in this area indicate that although fertility is reasonably maintained (certainly compatible with a population increase), cultural patterns favor reproductive function. The native population has a high birth rate and a high death rate, with peculiarly high prenatal and postnatal death rates for females. This results in a high ratio of males to females in the adult population. Birth weights are low, postnatal growth is quite slow relative to other populations throughout the world, and the adolescent growth spurt is less than that for other groups. Nevertheless, environmental factors appear to be less important than socioeconomic factors in determining the ability of the natives to thrive.

Effects of Altitude on Lung Function. In view of the constant concentration of oxygen in air, availability of oxygen to the individual is dependent on barometric pressure. Large changes in barometric pressure are observed with varying altitudes. Figure 1-3 demonstrates the decrease in barometric pressure and the decrease in inhaled Po_2 with increasing altitude (an exponential relationship). Thus, the inspired oxygen partial pressure may be predicted on the basis of the change in barometric pressure resulting from the altitude. Figure 1-4 illustrates the approximate values for inspired Po_2 at several different altitudes, with approximate changes in Po_2 of moist tracheal air, alveolar gas, arterial blood, mixed venous blood, and the directional changes in tissue oxygen partial pressure.

The immediate effects of a sudden change in barometric pressure have been studied in persons transported to high altitudes and have been simulated in low-pressure chambers. Among the earliest outstanding and comprehensive studies

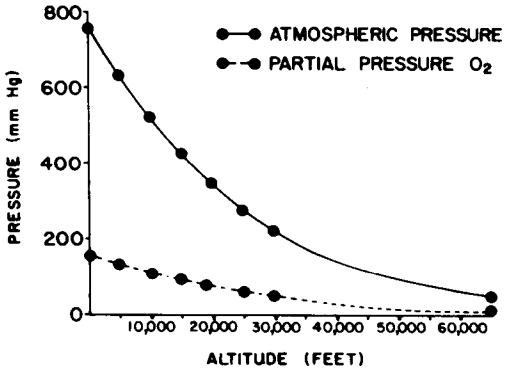

Fig. 1-3. Effect of altitude on barometric pressure and partial pressure of oxygen. The barometric pressure at various geometric elevations is plotted by the solid line. Note the exponential relationship. The partial pressure of oxygen is a fixed proportion (20.95%) of the total barometric pressure and is plotted by the interrupted line. Whereas the atmospheric partial pressure of oxygen at sea level is approximately 160 mm. Hg, it is reduced to half that value at 18,000 feet. (Adapted from Altman, P. L., and Dittmer, D. S.: Biological Handbooks. Environmental Biology. Federation of the American Society of Experimental Biologists, 1966[7])