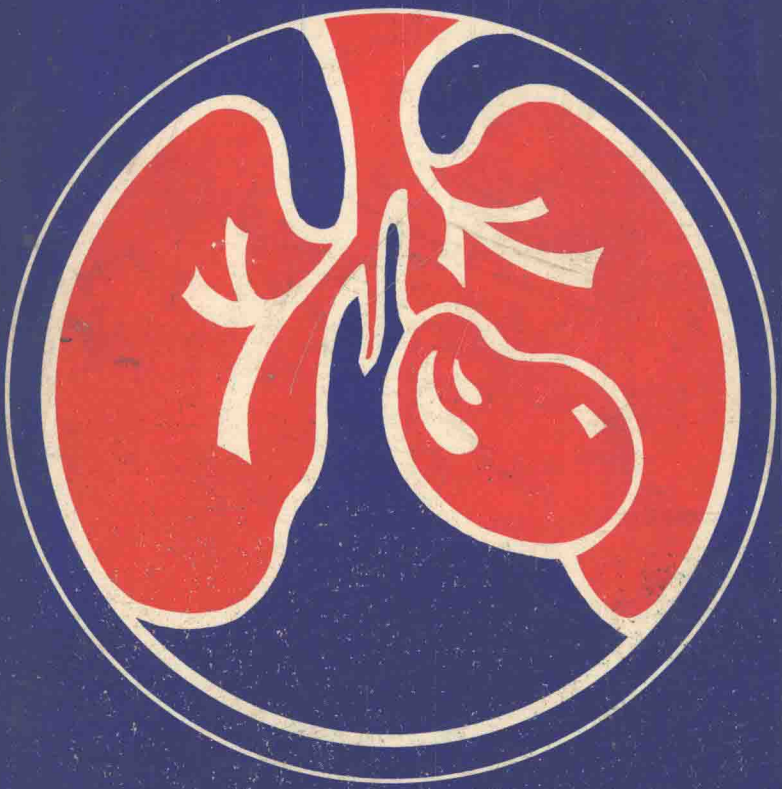
MANUAL OF RESPIRATORY THERAPY

JOAN P. TAYLOR



• SECOND EDITION •

MANUAL OF
RESPIRATORY THERAPY

JOAN P. TAYLOR, B.A., R.R.T.
Formerly Director of Respiratory Therapy Education
University of Kansas Medical Center
College of Health Sciences and Hospital
Kansas City, Kansas

SECOND EDITION

with 63 *illustrations*

THE C. V. MOSBY COMPANY
Saint Louis 1978

SECOND EDITION

Printed in the United States of America

Distributed in Great Britain by Henry Kimpton, London

The C. V. Mosby Company
11830 Westline Industrial Drive, St. Louis, Missouri 63141

Library of Congress Cataloging in Publication Data

Taylor, Joan P 1949-
Manual of respiratory therapy.

Edition of 1973 by C. D. Bryan and J. P. Taylor.
Bibliography: p.
Includes index.
1. Respiratory organs—Diseases. 2. Inhalation therapy. I. Bryan, Clifford D., 1941- Manual of respiratory therapy. II. Title. [DNLM: 1. Inhalation therapy. WB342 T243m]
RC735.I5T39 1978 616.2'004'636 77-22882
ISBN 0-8016-0836-8

CB/CB/CB 9 8 7 6 5 4 3 2 1

MANUAL OF RESPIRATORY THERAPY

PREFACE

Throughout the years that I have been involved in training respiratory therapists, resources available for both students and instructors have increased steadily. However, there is a frustrating lack of reference and resource data for the all-important area of clinical application.

Procedure manuals promote continuity and safety in patient care. Although these manuals do provide an important resource for students, they lack data on the rationale and skills required for intelligent patient application; they are also cumbersome and uninteresting.

Textbooks give students mountains of data concerning pathophysiology, rationale of therapeutics, and organized thoughts concerning technique. They do not provide quick reference nor give any data on sequence of techniques. Because they contain so much material that cannot be utilized at bedside, they are rarely useful for day-by-day clinical instruction.

This manual combines many of the beneficial aspects of procedural guides and textbooks in one portable package and can serve the student and the clinical instructor, at bedside as well as in conferences.

Some procedural information included will not be applicable to every situation. However, wherever possible, I have attempted to provide general guidelines rather than specific techniques so that the book can be adapted to individual needs.

The data included are primarily intended for respiratory therapists; however, medical students, nurses, and other personnel interested in respiratory care may also find parts of the text useful.

I realize that this book is by no means exhaustive on any subject included. A list of selected references is appended for further study. Manufacturers' operation manuals should be used to supplement the information included on apparatus.

In the revision of the manual, emphasis has been given to the increased activities of respiratory therapists in critical care areas. Sections concerning positive-pressure therapeutics, airway care, and patient monitoring have been added.

Special acknowledgment is due Ms. Dori Nelson for her work in preparing the revised manuscript.

Joan P. Taylor

MANUAL OF RESPIRATORY THERAPY

CONTENTS

1

RESPIRATORY FAILURE

Patients suffering from a wide variety of disorders, not only cardiopulmonary but of other body systems as well, exhibit symptoms of respiratory insufficiency. Clinicians may now detect acute respiratory failure by determining the arterial blood gas values. It is, however, much more difficult to forsee the development of respiratory failure in the patient exhibiting subclinical signs of moderate respiratory insufficiency. The incidence of acute respiratory failure would be reduced if more attention were given to the early treatment of subacute hypoxia and hypoventilation. This is particularly true of the patient who is not thought of as having a "pulmonary problem" but who may gravitate into decompensation following surgical procedures, depressant drug administration, or other therapeutic measures.

Definition

Respiratory failure is the stage of decompensation of respiratory function at which the arterial blood values are approximately as follows: Pa_{O_2} less than 50 mm Hg, Pa_{CO_2} greater than 60 mm Hg, and pH less than 7.25.

Obviously, some patients will be in severe respiratory failure before the above values are reached, whereas others may remain relatively symptom free with one or more of the above values even further deranged.

Classification

Hypoxia alone

Severe hypoxia may occur without accompanying hypercapnia and acidosis in a number of intrinsic disorders affecting the pulmonary parenchyma. Since it is treated symptomatically with insufflation of small increments of supplemental oxygen, it does

not assume as important a role in this chapter as hypoxia with accompanying hypercapnia. Rather, it is classified below, and treatment is discussed in Chapter 6.

1. Decreased pulmonary diffusing capacity.
   a. Alveolar capillary block, diffuse interstitial infiltrative disease, or inflammation of the alveolar wall will lengthen the diffusion pathway for oxygen. A similar effect is seen with any extensive fibrotic disease or in pulmonary edema.
   b. Reduced pulmonary capillary bed, pulmonary embolism, or resection of the lung will impair oxygen uptake, since there is a decrease in surface area for diffusion.
2. Arteriovenous shunt. Lung compression or diffuse atelectasis will result in a venous admixture with attendant hypoxemia.
3. Decreased circulating hemoglobin. Various anemias, hemolytic processes, and hemoglobin abnormalities may result in normal saturation but decreased oxygen-carrying capacity.

Hypoxia with hypercapnia

Alveolar hypoventilation from any cause may produce respiratory failure. Ignoring this critical situation for even a short time will result in cerebral depression, hypoxia, coma, and death.

1. Intrinsic lung disease. An acute exacerbation of any chronic lung disease, but particularly one with an obstructive component, may precipitate respiratory failure.
   a. Chronic obstructive pulmonary disease (COPD).
      (1) Generally, a superimposed infection causing increased bronchial spasm, bronchorrhea, and mucosal edema will further reduce alveolar ventilation.
      (2) The patient with chronic bronchitis will respond to any increase in airway resistance or major insult such as atelectasis, pneumothorax, pneumonia, pulmonary embolism, or cardiac failure with increasing dyspnea, leading to fatigue and respiratory failure.
   b. Parenchymal disease.
      (1) Any of the parenchymal diseases causing hypoxia alone may lead to alveolar hypoventilation if inadequately treated.
      (2) Complications of preexisting parenchymal lung disease may cause a further deterioration of pulmonary mechanics, resulting in severe alveolar hypoventilation.
2. Extrinsic causes of hypoventilation. Any patient with normal pulmonary function who experiences a loss of innervation of

the respiratory musculature or a change in thoracic wall or mediastinal structure may develop acute respiratory failure.

a. Respiratory musculature.
   (1) Failure of respiratory musculature associated with central nervous system depression secondary to trauma, drug use, or brain disease will produce alveolar hypoventilation.
   (2) Paralysis of respiratory muscles secondary to spinal cord trauma, peripheral nervous system disease, muscle relaxant drugs, or myoneural junction abnormalities will cause impairment or loss of ventilatory movements.

b. Decreased thoracic compliance.
   (1) Splinting or compensatory postural changes caused by pain, traumatic flail chest, or diaphragmatic trauma will impair ventilatory mechanics.
   (2) Skeletal deformities, obesity, or abdominal distention or ascites may severely reduce thoracic compliance and thus compromise alveolar ventilation.

c. Lung compression.
   (1) Pleural effusion, empyema, or pneumothorax will produce an increase in the work of breathing with a resultant decrease in alveolar ventilation.
   (2) Mediastinal or pleural neoplasms may reduce effective alveolar ventilation by compression of adjacent healthy lung tissue.

Diagnosis

Clinical examination

1. Physical signs of hypoxia.
   a. Moderate hypoxia. Increasing mental stimulation is followed by progressive irritability, drowsiness, headache, and depression states.
   b. More severe hypoxia. Tachycardia, dyspnea, progressive muscle weakness, loss of coordination, judgmental errors, and double vision are characteristic.
   c. Acute hypoxia. Loss of consciousness, hypoventilation states progressing to apnea, and cardiac arrhythmias occur. If sufficient hemoglobin is present, cyanosis may be detected at the nailbeds, lips, and conjunctiva.
2. Physical signs of hypoventilation.
   a. Moderate hypoventilation. Early hypoventilation states without loss of lung function (e.g., postanesthetic depression) may

result only in hypoxia without an elevation of carbon dioxide. Because of its greater diffusibility, carbon dioxide levels do not increase as rapidly as oxygen tension falls in these patients.

b. Acute hypoventilation. Severe hypercapnia with resultant acidosis produces dyspnea, loss of cerebral function, blood pressure elevation, and all the symptoms of acute hypoxia, since Pa_{O_2} always falls precipitously in advance of Pa_{CO_2} and pH changes.

3. Auscultation. Diminished breath sounds, moist rales, and dyspnea are the cardinal signs of increased work of breathing. Particular attention should be given to posterior basal segments as an indication of the degree of alveolar hypoventilation.

Laboratory values

1. Pa_{O_2}.
   a. Textbook normals for arterial oxygen tensions are rarely noted in this institution; instead, Pa_{O_2} levels of 70 to 90 mm Hg are commonly seen in quiet breathing of room air. In general, the older patient's normal Pa_{O_2} will fall somewhere between 70 and 75 mm Hg if he does not exhibit cardiopulmonary disorders.
   b. Hypoxic levels of 60 to 65 mm Hg generally produce few symptoms and only require watching. As illustrated by the oxyhemoglobin dissociation curve, 60 mm Hg is approximately the point at which hemoglobin saturation begins to fall abruptly. Fever, acidosis, or an elevated P_{CO_2} (Bohr effect) shifts the curve to the right, resulting in lower hemoglobin saturation at any P_{O_2}. (See Figs. 1-1 and 1-2.)
   c. A Pa_{O_2} of less than 50 mm Hg indicates severe hypoxia. Pa_{O_2}'s of 20 mm Hg have been noted in conscious, although severely decompensated, patients.
2. Pa_{CO_2}.
   a. The arterial CO_2 tension is a very sensitive indicator of the effectiveness of alveolar ventilation. Breathing room air, a normal, healthy subject at rest will have a Pa_{CO_2} in the range of 40 to 45 mm Hg.
   b. Pa_{CO_2}'s of greater than 100 mm Hg have been observed in conscious patients with chronic pulmonary disease. Overzealous ventilatory assistance or voluntary hyperventilation will occasionally decrease the Pa_{CO_2} below 20 mm Hg, but

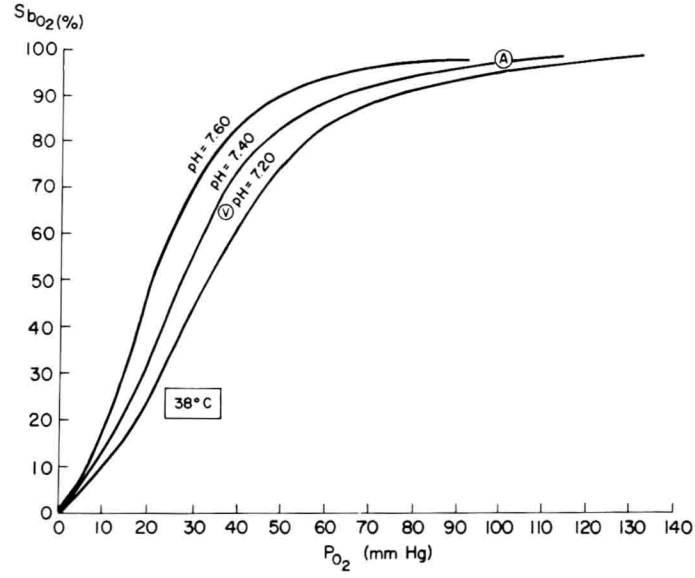


Fig. 1-1. Effect of pH on oxyhemoglobin dissociation curve. (From Slonim, N. Balfour, and Hamilton, Lyle H.: Respiratory physiology, ed. 3, St. Louis, 1976, The C. V. Mosby Co.)

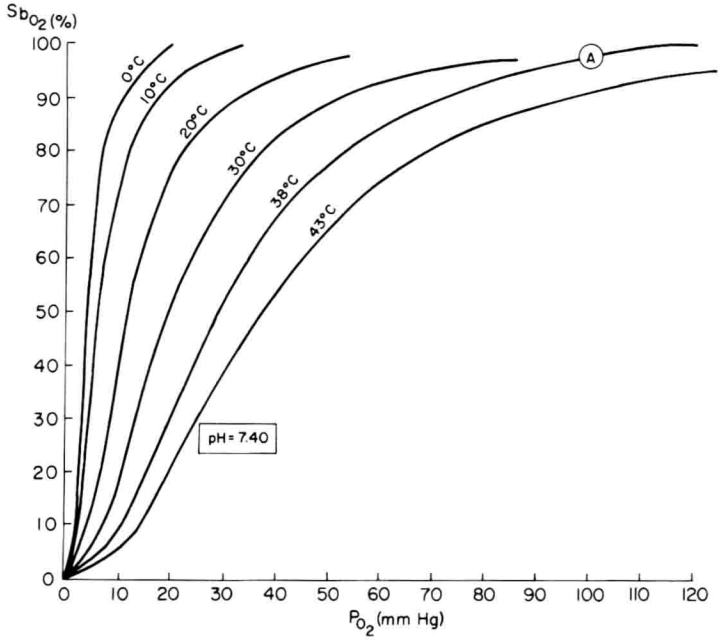


Fig. 1-2. Effect of temperature on oxyhemoglobin dissociation curve. (From Slonim, N. Balfour, and Hamilton, Lyle H.: Respiratory physiology, ed. 3, St. Louis, 1976, The C. V. Mosby Co.)

the resultant alkalotic state may cause convulsions and other cerebral dysfunctions.

c. Severe hypercapnia will produce acidosis. The resultant increases in $[H^+]$ and P_{CO_2} will cause cerebral depression, cardiac dysfunction, circulatory shifts, and electrolyte changes. Acute hypercapnia (without change in $[HCO_3^-]$) should be rapidly corrected by improved alveolar ventilation, but chronic hypercapnia requires close titration of pH levels during gradual correction.

d. Any persistent change in alveolar ventilation will shift plasma CO_2 values along a dissociation curve. Renal retention of bicarbonate shifts the curve upward, whereas excretion of bicarbonate results in a lowered CO_2 dissociation curve. Once a new curve has been established, further changes in ventilation will be reflected along the new curve. In addition, the CO_2 dissociation curve is influenced by the amount of oxygen combined with hemoglobin at any one time. Since oxyhemoglobin is a weak acid, it can be seen from the CO_2 dissociation curve that at high oxygen tensions less CO_2 can be carried by the blood (Haldane effect). (See Fig. 1-3.)

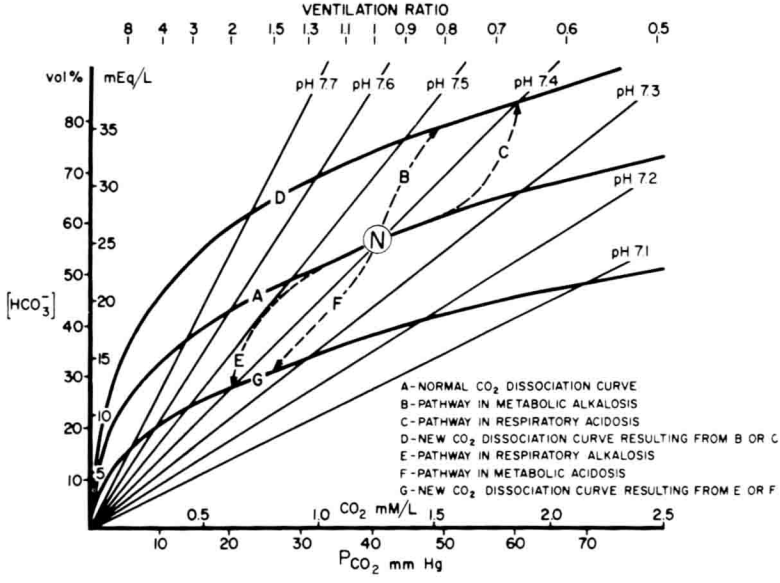


Fig. 1-3. CO_2 dissociation curves. (From Slonim, N. Balfour, and Hamilton, Lyle H.: Respiratory physiology, ed. 3, St. Louis, 1976, The C. V. Mosby Co.)

3. pH. The hydrogen ion concentration in body fluids strongly affects cellular chemistry and metabolic enzyme systems. For this reason, pH homeostasis must be maintained within a very narrow range. An arterial pH of 7.38 to 7.42 is considered "normal." The regulation of pH within the acceptable range is controlled by three interrelated buffering systems: blood buffers, alveolar ventilation, and renal function. Nonpulmonary causes of derangement will produce changes in alveolar ventilation that tend to compensate metabolic changes. Similarly, renal and blood buffering systems will make adjustments for alterations in Pa_{CO_2}. The causes, effects, and compensatory mechanisms are as follows:

Condition	*pH*	*Cause*	*Compensation*
Metabolic acidosis (acidemia)	↓ 7.38	↓ $[HCO_3^-]$	↓ Pa_{CO_2}
Metabolic alkalosis (alkalemia)	↑ 7.42	↑ $[HCO_3^-]$	↑ Pa_{CO_2}
Respiratory acidosis (hypercapnia)	↓ 7.38	↑ Pa_{CO_2}	↑ $[HCO_3^-]$
Respiratory alkalosis (hypocapnia)	↑ 7.42	↓ Pa_{CO_2}	↓ $[HCO_3^-]$

4. Calculation of pH. CO_2 combines with water to form carbonic acid, which dissociates into hydrogen ions and bicarbonate ions:

$$CO_2 + H_2O \leftrightharpoons H_2CO_3 \leftrightharpoons H^+ + HCO_3^-$$

The degree of dissociation is expressed as the ratio between the concentrations of dissociated and undissociated acid:

$$K = \frac{[H^+]\ [HCO_3^-]}{[H_2CO_3]}$$

The value K is termed the dissociation constant. In the preceding equation K is 8×10^{-7} at 38° C. The relationship thus stated is known as the Henderson equation. For convenience $[H^+]$ is often expressed as pH, the logarithm of the reciprocal of the hydrogen ion concentration:

$$pH = \log \frac{1}{[H^+]}$$

The Henderson equation can be modified to accommodate this mode of expression, as follows:

$$K = \frac{[H^+]\ [HCO_3^-]}{[H_2CO_3]} \qquad (1)$$