Manual of CRITICAL CARE NURSING

NURSING INTERVENTIONS AND COLLABORATIVE MANAGEMENT

SIXTH EDITION

MARIANNE SAUNORUS BAIRD
SUSAN BETHEL

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MOSBY

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MANUAL OF CRITICAL CARE NURSING: NURSING INTERVENTIONS AND COLLABORATIVE MANAGEMENT, SIXTH EDITION
ISBN 978-0-323-06376-0

Library of Congress Cataloging-in-Publication Data
Baird, Marianne Saunorus.
Manual of critical care nursing : nursing interventions and collaborative management/Marianne Saunorus Baird, Susan Bethel.—6th ed.
p. ; cm.
Rev. ed. of: Manual of critical care nursing. 5th ed. c2005.
Includes bibliographical references and index.
ISBN 978-0-323-06376-0 (hardcover : alk. paper)
1. Intensive care nursing. 2. Nursing diagnosis. I. Bethel, Susan. II. Manual of critical care nursing. III. Title.
[DNLM: 1. Critical Care. 2. Nursing Assessment. 3. Nursing Care.
WY 154]
RT120.I5M3644 2011
616.02'8–dc22

2010031645

Managing Editor: Maureen Iannuzzi
Senior Developmental Editor: Robin Richman
Developmental Editor: Laurie Sparks
Publishing Services Manager: Pat Joiner-Myers
Senior Project Manager: Joy Moore
Senior Book Designer: Amy Buxton

Printed in The United States of America

Last digit is the print number: 9 8 7 6 5 4 3 2 1

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Preface

Manual of Critical Care Nursing is a clinical reference for both practicing and student critical care nurses. It is the most comprehensive of the critical care handbooks available, yet is concise and easy to use because of its abbreviated outline format and its portable trim size. This handbook provides quick information for more than 75 clinical phenomena seen in critical care and can be used in the clinical setting to plan nursing care.

WHO WILL BENEFIT FROM THIS BOOK?

Nurses from novice to expert will derive help with assessing, managing, and evaluating their acutely ill patients. The textual information and numerous tables will serve as a quick review for the practicing nurse. Academicians can use the book in teaching students to apply theoretical concepts to clinical practice. Students will find the book to be an excellent tool for assessing the patient systematically, as well as for learning how to set priorities for nursing interventions.

WHY IS THIS BOOK IMPORTANT?

Given the increasing acuity of hospitalized patients, information previously considered exclusive to critical care such as managing acid-base balance and arterial blood gas interpretation is becoming common knowledge in progressive care, telemetry, stepdown units, and high-acuity medical-surgical units. Accordingly, the care outlined here is applicable across the spectrum of high-acuity care, from high-acuity medical-surgical to critical care.

BENEFITS OF USING THIS BOOK

Our primary goal is to present the information necessary to provide patient-centered care in a technologically advanced environment in a quick and easy-to-use format. Throughout the text, we strive to consider the whole patient with care recommendations that address the physical, emotional, mental, and spiritual distress involved in illness. The prevention of potentially life-threatening complications is of primary importance and therefore addressed through assessment, planning, implementation, and evaluation of interdisciplinary collaborative care, and nursing plans of care.

To best assess changes in status, knowledge of the patient's condition before acute, critical illness is essential. This book offers many interventions for each disorder, but not all interventions are appropriate for every patient. Our intent is to offer a thorough selection of prioritized actions that can be chosen as needed in planning individualized care.

HOW TO USE THIS BOOK

Manual of Critical Care Nursing has been reorganized for easy access and logical presentation. Information regarding general concepts of patient care, including those unique to the critical care environment, is presented in the first two chapters, General Concepts in Caring for the Critically Ill and Managing the Critical Care Environment. Following is a chapter on Trauma and related disorders. Chapters 4 through 10 cover disorders classified by body system, and Chapter 11 addresses Complex Special Situations, such as high-risk obstetrics and organ transplantation.

Each body system–specific chapter includes a general physical assessment, and several chapters include generic plans of care applicable to patients with all disease processes affecting that body system. Each disorder includes a brief review of pathophysiology, physical assessment, diagnostic testing, collaborative management, NANDA-approved nursing diagnoses and nursing interventions, patient/significant other teaching, desired outcomes, and disease-specific discharge planning considerations. Gerontologic icons highlight material relevant to the care of older adults. Desired nursing care outcomes and interventions are based on the University of

Iowa's Nursing Intervention Classification (NIC) and Nursing Outcomes Classification (NOC) systems and are highlighted throughout the text. Nursing interventions are linked to nursing diagnoses, and suggested outcomes include specific measurement criteria for physical parameters and time frames for attainment of expected outcomes. The time frames are guidelines, because each patient's response time to both illness and intervention is unique.

For clarity and consistency throughout the book, normal values are given for hemodynamic monitoring and other measurements. All values should be individualized to each patient's baseline health status.

NEW TO THIS EDITION

The sixth edition has been extensively revised and reorganized to mirror a practicing nurse's approach to patient care and allow even easier access to information. Changes include:

- New information on patient safety, organ transplantation, emotional and spiritual support of the patient and significant others, peripheral vascular disease, continuous renal replacement therapy, brain death, neuromuscular diseases, hyperglycemia, and oncologic emergencies.
- Updated guidelines and recommendations for mechanical ventilation, hemodynamic monitoring, and the management of heart and respiratory failure including evolving mechanical devices.
- A composite chapter reflecting the evolution of management of hyperglycemia and associated emergency conditions.
- Enhanced information on acid-base balance, acute asthma, burns, sepsis, cardiogenic shock, aortic dissection, and the management of delirium.
- Physical assessment and generic plans of care for disorders of each body system.
- Collaborative care and nursing care plans with prioritized interventions and outcomes based on the Nursing Outcomes Classification (NOC).
- Organization of information by alphabetical order, with color tabs for easy access.
- Appropriate resuscitation interventions within the section on Dysrhythmias and Conduction Disturbances.

We hope that critical care practitioners, students, and academicians will find that the new edition of *Manual of Critical Care Nursing* provides them with a wealth of easy-to-access knowledge to apply in practice and in the classroom.

ACKNOWLEDGMENTS

We want to thank many individuals who supported the development of this manuscript. In particular, we are grateful for the time and efforts of Laurie Sparks, Developmental Editor, and Jeff Somers. We appreciate the guidance of Maureen Iannuzzi, Editor, and Robin Levin Richman, Senior Developmental Editor. We thank all the contributors for their hard work and attention to detail, as well as all the reviewers whose comments helped guide our revisions. All are recognized as shining stars in their own right. Both perseverance and patience are the fundamental characteristics inherent in all participants.

We extend special recognition to Barbara McLean, for "going the extra mile" to enhance her content; to Phyllis Gordon and Cynthia Rebik Christensen for their excellent job in creating the new Peripheral Vascular Disease section; Patricia Weiskittel for taking on several additional sections; Elizabeth Scruth for her extensive enhancement to the Cardiogenic Shock section; Gretchen J. Carrougher, Shari Honari, and Carolyn Blayney for extensive work on the Burn section; and Vicki Good for creating the new Patient Safety section.

Marianne Saunorus Baird and Susan Bethel

I acknowledge the support of my daughter Rachel, my husband Thom, and my mother, Irene Saunorus. I could not have done it without all of you.

MSB

I acknowledge the support of my husband Terry, and the team effort of my team of authors within the Greenville Health System. You are the best!

SB

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CHAPTER

1 General Concepts in Caring for the Critically Ill

ACID-BASE IMBALANCES

Cells must transport ions, metabolites and gases in order to respond appropriately. For this to occur, the bloodstream's chemical environment must be electrically stable. *The stability of the environment is measured by the arterial pH* and must be *chemically neutral (pH 7.40)* for all systems to function properly. *The arterial blood gas (ABG) is the most commonly used analysis to measure acid-base balance and to assess the efficacy of oxygenation.* Respiratory (CO_2) and metabolic acids (H^+) are generated as cells work and must be buffered or eliminated to maintain a neutral chemical environment. When the chemical environment is no longer neutral, the patient has an acid-base imbalance. Ineffective metabolism (tissue level), renal dysfunction, and/or problems with ventilation (breathing gasses effectively) are often the cause of acid-base imbalance.

There are two main types of acid-base imbalance: *acidosis* and *alkalosis*. The kidneys and lungs work in tandem to maintain chemical neutrality, but it is actually cellular function which produces acid. When either the kidneys or lungs are over or under functioning, the other system is designed to have the opposite response in order to compensate and bring the pH back to a normal range. When the kidneys fail to regulate metabolic acids (H^+), the lungs must compensate. When the lungs fail to regulate respiratory acid (CO_2), the kidneys must compensate. Additional buffering mechanisms are also available to help regulate the accumulation of acids. Control of alkaline states, resulting from accumulation of bases or loss of acids, is maintained in a similar fashion between the lungs and kidneys.

PATHOPHYSIOLOGY OF ACID-BASE REGULATION

Arterial pH is an indirect measurement of CO_2 and H^+ concentration, which reflects the overall level of acid and effectiveness of maintaining the balance. The normal acid-base ratio is 1:20—1 part acid (the H^+ and CO_2 component of H_2CO_3) to 20 parts base (HCO_3^-). If the ratio is altered through an increase or a decrease in either acid H^+ or CO_2 or the base, HCO_3^-, the pH changes. Chemically, the CO_2 does not contain H^+, but when dissolved in water (plasma), $CO_2 + H_2O$ yields H_2CO_3 (carbonic acid). CO_2, when combined with H_2O, becomes the largest contributor of H^+ (acids), which must be eliminated or buffered to maintain normal pH. Too many H^+ ions in the plasma creates *acidemia (pH less than 7.35)*, while too few H^+ ions creates *alkalemia (pH greater than 7.45)*.

Maintaining the 1:20 ratio ("the balance") depends on the ability of the lungs and kidneys to help normalize concentrations of carbonic acid (H_2CO_3) a product of hydrogen ion (H^+) plus bicarbonate buffer (HCO_3). Both the kidney and lung are designed to eliminate carbonic acid effectively and therefore the pH should always be in the range of normal. A pH change is a symptom that there is a significant problem with one or both of the systems.

- *Acidosis:* Extra acids are present or base is lost, with a pH less than 7.35.
  1. *Cellular acidosis:* When cells are hypoxic or processing proteins to yield glucose, there is an increase in lactic acid or ketoacid.
  2. *Respiratory acidosis:* If the function of the lungs is inadequate, such as in COPD disease, the failure to effectively ventilate results in the inability to excrete CO_2, and that failure causes carbonic acid to go up (more acid) and pH to go down.

3. *Renal acidosis:* If the kidney function is inadequate, the ability to break carbonic acid from H_2CO_3 into hydrogen ions (H^+) and HCO_3^-. When this failure occurs, carbonic acid goes up (more acid) and pH goes down.

- Buffering of acid or compensation for an acid state, occurs in three primary ways:
  1. *Plasma and cellular buffering:* Using bicarbonate, proteins, intracellular electrolytes, and chloride to buffer H^+. Most common is the marriage of H^+ to $HC0_3^-$ which yields carbonic acid H_2CO_3.
  2. *Hyperventilation (lungs):* The presence of increased carbonic acid stimulates a hyperventilation response. This allows for exhaling ("blow off") more of the CO_2 component of carbonic acid. This compensatory response for metabolic acidosis occurs within minutes and should bring pH to a normal range.
  3. *Acid excretion (kidneys):* A functional kidney will utilize increased carbonic acid by breaking the H_2CO_3 into bicarbonate and H^+, excreting the H^+ and retaining the bicarbonate. This should compensate for the increased respiratory acidosis but is very slow, taking 4 to 48 hours for compensation to occur.
- *Alkalosis:* Extra base is present or there is loss of acid, with a pH greater than 7.45.
  1. *Respiratory alkalosis:* When hyperventilation is the primary problem, there is a very rapid removal of $C0_2$, causing carbonic acid to go down (less acid) and pH to go up.
  2. *Renal alkalosis:* If the kidney function is overstimulated (for example with aggressive diuresis), there may be excessive loss of hydrogen ions (H^+), causing carbonic acid to go down (less acid) and pH to go up.
  3. *Other contributors:* Gastric and intestinal removal of acids may occur when patients have diarrhea, vomiting, or when excessive gastric drainage influences the acid-base balance.
- *Alkalosis:* Compensating for an alkaline state occurs in two ways:
  1. *Hypoventilation:* The respiratory system responds by slowing ventilation and retaining CO_2 (acid), to help compensate for metabolic alkalosis from any cause. This response occurs within minutes.
  2. *Renal response:* The kidneys respond by retaining more acid (H^+) and excreting more bicarbonate to help correct respiratory alkalosis. This response occurs within 4 to 48 hours.

Example of Compensation (pH Regulation):

When metabolic acids accumulate, they are attracted to bicarbonate. The marriage of H^+ and HCO_3^- buffers the acid. This yields an increase in carbonic acid and causes the pH to go down. Chemoreceptors are stimulated by this acid presence and the hypothalamus, if not damaged, triggers a hyperventilation response. Since H^+ is not measured directly, the indirect calculation of bicarbonate or the base is used to evaluate the presence or absence of metabolic acid. As H^+ goes up, the bicarbonate or base goes down. When evaluating the acid base balance, it is simplest to look at bicarbonate but to think in terms of H^+. They travel in completely opposite directions (when bicarbonate is down, H^+ is up and vice versa).

The lungs increase buffering *to compensate* for a failure of the kidneys or a cellular excess acid production to keep the pH balanced. The lungs do this by effectively exhaling more CO_2 than usual, breaking down the carbonic acid and therefore bringing pH back towards normal.

When CO_2 is retained or increased because of respiratory failure, the kidneys should, in turn, respond by processing the increased H_2CO_3. The kidneys separate the carbonic acid into H^+ and HCO_3^- and excrete the H^+ while retaining HCO_3^- bicarbonate. If either the kidneys or lungs do not respond to a pH change (*no compensation*) or they provide an ineffective response (*partial compensation*), the patient will remain in acid-base imbalance. If the pH is outside of the range of normal, then there is a primary problem and *compensation* is inadequate or has failed. Patients may have a pure acidosis or alkalosis and the overall problem may be masked by compensation or two problems presenting at the same time.

It is essential to understand that unless the patient has ingested acid (aspirin, ethanol, etc.), all acid in the bloodstream was produced at the cellular level (Table 1-1). When evaluating patients, care providers must have a basic understanding of the acid-base balancing system. The main formula for maintenance of acid-base balance is the following:

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow HCO_3^- \text{ and } H^+$$

Table 1-1 PRODUCERS AND REGULATORS OF ACID

Acid Pathways	Cause	Measure
Cells produce acid (acid production increases).	Hypermetabolic states, such as pain, hyperthermia, or inflammation. The respiratory and heart rates increase, and bicarbonate is initially consumed by buffering.	$HCO_3 \downarrow$
	Tissues are hypoxic; anaerobic metabolism ensues resulting in lactic acidosis.	Lactate level $\uparrow$
	Absolute insulin deficiency results in failure of glucose to be transported into cells.	Blood glucose level $\uparrow$ Ketoacids $\uparrow$
Cells regulate acids.	When acid production (H^+) increases, pH decreases, bicarbonate is initially consumed by buffering, and CO_2 is exhaled in larger amounts, and H^+ exchanges for K^+ as cells buffer acid.	pH $\downarrow$ $HCO_3 \downarrow$ $K^+ \uparrow$ Total serum $CO_2 \downarrow$
Lungs regulate acid.	When acid increases due to hypermetabolic states such as pain, hyperthermia, or inflammation, carbonic acid (H_2CO_3) increases and rapidly converts to CO_2 and H_2O. The respiratory rate increases to blow off CO_2.	$Paco_2 \downarrow$
Kidneys regulate acid.	When acid increases, tubules are affected by low blood pH, and work to neutralize increased carbonic acid (H_2CO_3) by separating it into H^+ and bicarbonate HCO_3. Kidneys excrete what is necessary to sustain normal pH if renal function is normal. If abnormal, kidneys may not perform this task.	$HCO_3 \uparrow$ Kidney function is assessed by serum BUN and creatinine; elevated BUN and creatinine indicate abnormal kidney function.

The most important component identified is the H_2CO_3, or carbonic acid. As carbonic acid increases ("goes up"), the pH decreases ("goes down"), reflecting the presence of acid. If the carbonic acid decreases ("goes down"), the pH increases ("goes up"), reflecting the absence of acid. The equation is constantly shifting from left to right and right to left to maintain a normal H_2CO_3 and therefore a normal pH. Whatever causes the change of carbonic acid concentration (may be related to a regulation failure by either the lungs or kidneys or a metabolic acid production state) is the "primary culprit." Identifying the origin or cause of the change in pH direction identifies the problem. Therefore, if the problem is too much acid (either increased CO_2 or H^+), the carbonic acid goes up and the pH goes down. The primary problem is acidosis. Further evaluation is needed to determine whether failure to regulate the acid was ineffective regulation by the lungs, the kidneys or an increase in cellular acid production (ketoacidosis or lactic acidosis).

Safety Alert *Changes in pH are associated with changes in the potassium level. As the plasma level of nonvolatile or metabolic acid (H^+) increases, H^+ moves into the cells in order to buffer the acid effect. In this case H^+ "exchanges places" with the intracellular potassium (K^+), resulting in a measured serum hyperkalemia but is actually an intracellular hypokalemia. The positively charged intracellular potassium ions are replaced by positively charged hydrogen ions. During an alkalotic state, K^+ may shift into cells as H^+ is released into the serum, creating a transient hypokalemia. As pH changes, it is imperative for the care providers to observe the corresponding changes in the K^+ level, and manage K^+ carefully. When the pH normalizes, the K^+ will shift back to its original location. If a transient K^+ change is managed too aggressively, the patient may experience dangerous hypokalemia or hyperkalemia when pH normalizes.*