CARDIAC SURGERY ESSENTIALS FOR CRITICAL CARE NURSING

Sonya R. Hardin

Roberta Kaplow

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Editors

Sonya R. Hardin, PhD, RN, CCRN, ACNS-BC, NP-C

Associate Professor
School of Nursing
College of Health and Human Services
University of North Carolina at Charlotte
Charlotte, North Carolina

Staff Nurse
Davis Regional Medical Center
Statesville, North Carolina

Roberta Kaplow, PhD, RN, AOCNS, CCNS, CCRN

Clinical Nurse Specialist
Emory University Hospital
Atlanta, Georgia

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Dedication

This book is dedicated to Jack, Eleanor, Susan, Ray, Grace, Princess, Pauline, James, and Bria. We are grateful for your love, support, patience, and encouragement as we worked the production of this book.

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Contents

Contributors

Dawn B. Adams, RN, MSN, CCRN
Clinical Nurse Specialist
Project Coordinator
DeKalb Medical
Decatur, GA

Rachel S. Ball, RN, MSN, ACNP
Nurse Practitioner
Cardiac Surgery Critical Care
University of Michigan Medical Center
Ann Arbor, MI

Mary Jane Bowles, RN, MSN, CCRN
Clinical Specialist, CC Dept.
Mary Washington Hospital
Fredericksburg, VA

Susan K. Chase, EdD, APRN, BC
Professor
College of Nursing
University of Central Florida
Orlando, FL

Catherine Drumm, RN-BC, MA
Nurse Manager
Private Ambulatory Practices
Hospital for Special Surgery
New York, NY

Myra F. Ellis, RN, MSN, CCRN-CSC
CTICU, Clinical Nurse IV
Duke University Hospital
Durham, NC

Kathy Halabicky, RN, MSN, ACNP
Nurse Practitioner
Cardiac Surgery Critical Care
University of Michigan Medical Center
Ann Arbor, MI

Brenda Hardin-Wike, RN, MSN, CCRN, CCNS
Unit Director CTICU 7 ICU
University of California at Los Angeles
Ronald Reagan Medical Center
Los Angeles, CA

Laurie Hartman, RN, MSN, ACNP
Coordinator Advanced Practice Team
Cardiac Surgery Critical Care
University of Michigan Medical Center
Ann Arbor, MI

Jenniffer Hughes, RN, CCRN-CSC
Clinical Nurse III
Cardiothoracic ICU
University of North Carolina Hospital
Chapel Hill, NC

Barbara Hutton-Borghardt, RN, MSN, CCRN
Nursing Supervisor
Memorial Sloan-Kettering Cancer Center
New York, NY

Toni Patrice Johnson, RN, MSN, CNS, CPAN
Clinical Nurse Specialist
Perioperative and Endoscopic Services
Emory University Hospital, Midtown
Atlanta, GA

Beverly Jones, RN, MSN, ACNP
University of Michigan Medical Center
Cardiac Surgery Critical Care
Ann Arbor, MI

Erin Lindstrom, RN, MSN, FNP
Nurse Practitioner
Cardiac Surgery Critical Care
University of Michigan Medical Center
Ann Arbor, MI

Susan Lynch, RN, MSN
RN-BSN Coordinator
School of Nursing
University of North Carolina at Charlotte
Charlotte, NC

Carol Isaac MacKusick, RN, MSN, PhD(c), CNN
Assistant Professor of Nursing
Clayton State University
Morrow, GA

Timothy E. McMurry, RN, BSN, CCRN-CSC
Clinical Coordinator, CVRU
Presbyterian Hospital
Charlotte, NC

Julie Miller, RN, BSN, CCRN
Staff Development Educator, Critical Care
Trinity Mother Frances Hospitals and Clinics
Tyler, TX

Vicki Morelock, RN, MN, CCRN
Clinical Nurse Specialist for Cardiovascular Surgical Services
Emory University Hospital, Midtown
Atlanta, GA

Kristine J. Peterson, RN, MSN, CCRN, CCNS
Clinical Nurse Specialist, CV and Critical Care
Park Nicollet Methodist Hospital
St Louis Park, MN

Kirsten Platt, RN, BSN, CCRN
Clinical Nurse II
Cardiothoracic ICU
University of North Carolina Hospital
Chapel Hill, NC

Tracey Romans, RN, MSN, FNP
Nurse Practitioner
Cardiac Surgery Critical Care
University of Michigan Medical Center
Ann Arbor, MI

Mary Zellinger, RN, MN, ANP, CCRN-CSC
Clinical Nurse Specialist
Cardiovascular Surgical Services
Emory University Hospital
Atlanta, GA

Preface

Postoperative care of the cardiac surgery patient is both challenging and dynamic. Changes in technology, new research findings, the advent of minimally invasive procedures, and the development of off-pump procedures now afford patients of advanced age and with higher levels of acuity the opportunity to undergo procedures for which they were deemed unsuitable candidates not so long ago. Hence, patients with more—and more significant—comorbidities are receiving care in the immediate postoperative period in the intensive care unit.

Patients who undergo cardiac surgery are at risk for several adverse events not only related to their preoperative condition, but also as a result of effects of the surgical procedure and anesthesia. This requires ICU nurses to demonstrate high levels of clinical judgment, clinical inquiry, and caring practices to effectively manage patients and help optimize outcomes. High-level competency as a facilitator of learning is also required as nurses prepare their patients to undergo cardiac surgery. Clearly, ICU nurses, as members of a multidisciplinary team, play a pivotal role in promoting 10-year survival and high quality of life for patients who undergo cardiac surgery.

This book is designed to address the needs of both new and experienced nurses who care for patients in the ICU immediately following cardiac surgery. The purpose of this book is twofold. First, it is designed to prepare the nurse who is first learning to care for patients undergoing cardiac surgery. It addresses significant changes in cardiac surgery and the nursing responsibilities required to meet the needs of these acutely ill patients. Second, the book provides advanced knowledge and a scientific basis for care for nurses who have mastered the essential knowledge and skills necessary to care for this patient population, but who now seek to develop a more in-depth knowledge base about advances in this dynamic field and strategies to optimize patient outcomes. The emphasis throughout the book is providing an evidence-based foundation for care of patients during the vulnerable period immediately following cardiac surgery. A number of chapters in the book will also prove useful to nurses who work in other areas in which there are acute and critically ill patients, as many of the concepts discussed here can be translated into care of patients other than those who have undergone cardiac surgery.

Because this book uses a comprehensive approach to address the needs of patients in the immediate postoperative period following cardiac surgery, it can also be used to help prepare nurses who plan to take the Cardiac Surgery Certification (CSC) subspecialty exam offered by the American Association of Critical-Care Nurses.

Throughout the book, Clinical Inquiry Boxes highlight research findings that have implications for nursing practice. Other features that promote critical thinking and provide application of content are the Case Studies and Critical Thinking Questions that follow the respective chapter content. To further enhance critical thinking and for nurses preparing for the CSC exam, the Self-Assessment Questions found at the end of each chapter can be used as practice questions.

Chapter 1

Clinical Judgment in Critical Care

Susan K. Chase

INTRODUCTION

The critical care unit provides a location for continuous monitoring of unstable patients as well as a context for the use of invasive technology that supports basic life processes for acute and critically ill patients. Learning about technology and mastering its safe use are often the foci of basic critical care education and orientation. Aside from its technology, the more basic value of a critical care unit is the level of clinical judgment that occurs there. The thinking processes of clinicians from a variety of disciplines are essential to safe and effective care. The potential for optimal outcomes is enhanced when clinical judgments occur with the nurse synthesizing and interpreting multiple, often conflicting sources of data (Hardin & Kaplow, 2005).

Working in a critical care area is both exciting and rewarding—but it is also demanding and challenging. Nurses in critical care are central for rapid response to potentially life-threatening conditions and key in humanizing technological care. Since critical care units were first developed, the monitoring of, and early response to changes in, patients' conditions by nurses have revolutionized care. Nurses in critical care areas must make rapid and accurate decisions about diagnostic and treatment approaches in an independent way or based on protocols or standard orders. This chapter describes the processes used by critical care nurses as they make these decisions. It will be useful to new critical care nurses as they learn to provide safe care. At the same time, it will also be useful to experienced critical care nurses who wish to improve their processes of thinking and communicating.

The thinking processes used by critical care nurses (CCNs) differ quite dramatically from the schoolbook description of the "nursing process." The linear process of collecting information, forming a decision, choosing an action, and evaluating that action is rarely used in real-world practice. In critical care, multiple conditions are assessed simultaneously, a variety of actions and interventions are carried out concurrently, and the condition of the patient changes constantly. There is not a single diagnosis or condition that is "resolved." Because the thinking work of CCNs is not a linear process, this chapter is likewise not linear. It deals in general terms with phases of the "thinking work" of nursing, but acknowledges that thinking and acting often overlap in real life.

Clinical judgment is one of the eight nurse competencies of the AACN Synergy Model for Patient Care adopted by the American Association of Critical-Care Nurses (AACN) (Reed, Cline, & Kerfoot, 2007). Clinical judgment is defined as the use of clinical reasoning

including decision making, critical thinking, and achieving a global grasp of a situation, coupled with nursing skills acquired through a process of integrating education, experimental knowledge, and evidence-based guidelines (AACN, 2002).

CLINICAL JUDGMENT PROCESSES

Research has provided a window into how humans think and make decisions. Several models can help clinicians understand their decision-making processes and help them to become more efficient and to reduce errors in judgment. The three models that are useful in critical care are information processing, intuition, and decision analysis (Chase, 2004). Each model contributes a unique perspective to decision making, and clinicians can choose which model to apply based on matters of individual style. The nature of specific problems may also determine which model is useful in a particular situation.

Information Processing

The information processing model uses the analogy of the human brain working like a computer as it processes new information that becomes available. It also relies on the assumption that an "optimal" diagnosis can be made by taking into account the data that are available in the problem situation. The possible diagnoses or problems that might be present for a patient are called "hypotheses" before they are confirmed. There are usually multiple competing hypotheses to explain a particular pattern of data. For example, a nurse may notice that a diabetic patient has a serum glucose level above baseline. This finding might be a result of several causes—a faster than expected glucose infusion, a new infection, or a missed insulin dose, among other possibilities. Each of these possibilities is a hypothesis. Further data collection can help to narrow the options by ruling out certain problems or increasing the likelihood of another explanation. In the example just given, if the nurse notes cloudiness in urine and an elevation of body temperature, then the probability that the hypothesis of infection is correct is increased. This, in turn, directs further action by the nurse. More data can be collected, such as a urinalysis and urine culture, to rule in (confirm) a urinary tract infection.

The information processing model focuses on reevaluating competing hypotheses based on new data (Thompson & Dowding, 2002). In critical care areas, nurses frequently work independently in choosing further data to be collected to support a hypothesis. Units may have protocols that authorize the nurse to proceed with further data collection without obtaining orders from a physician. This relative autonomy increases the necessity for critical care nurses to exercise appropriate judgment. It would not be appropriate judgment for the nurse to run expensive tests if the data do not warrant it. Judgment includes the decision to do things or not to do them. An economy of practice occurs when all appropriate actions—but *only* appropriate actions—are taken. To make the choice of further diagnostic testing, all information present must be considered.

In real life, nurses frequently need to act before all information necessary to confirm a diagnosis is available. If a condition that is suspected is particularly critical, such as impending respiratory failure, actions to support the patient must be taken even before a full understanding of the reason for such failure is obtained. To wait to offer support until the patient is in full respiratory failure is to miss the opportunity to offer timely interventions that support the patient's function. At times, by taking the most appropriate actions for the most likely problem and then noting the patient's response to those measures, the diagnosis is either confirmed or refuted. If the treatment approach does not work, additional reasons for the patient's problems

must be investigated. New data must be considered to help develop a picture that answers the question, "What's going on with this patient?"

In any clinical situation, certain diagnoses or problems are possible, and some are more likely than others. Critical care units are places where monitoring equipment allows for the collection of a wider range of data than in less acute settings. Critical care nurses are the constant collectors and evaluators of clinical data. Early in their careers, nurses new to critical care may focus on the compilation of data through the use of new or unfamiliar equipment such as electrocardiography, monitoring systems that reflect and record hemodynamic parameters through the use of a pulmonary artery catheter or continuous blood pressure through intra-arterial lines. It is appropriate that new nurses focus on perfecting their skills in managing and interpreting data from these systems. The assembly of information is just one small aspect of critical care nursing, however. The data obtained from monitoring systems represent key components to be utilized in understanding the full clinical picture presented by the patient.

Nurses collect and evaluate data to arrive at a diagnosis. Even after an initial medical diagnosis of acute myocardial infarction (AMI) is made, for example, the critical care nurse has many diagnostic options to consider. AMI patients may develop dysrhythmias, cardiogenic shock, pulmonary edema, or anxiety. Early detection of these conditions can lead to early and more effective treatment and better outcomes. As more data are collected, they change the likelihood of each of the possible complications that might occur. A normal respiratory rate and arterial blood gas values within normal limits for the patient's age, for instance, indicate that respiratory failure is not imminent. Even simple data, such as vital signs, offer a view of the wholeness of the patient and change the diagnostic possibilities. A normal respiratory rate might indicate that the patient is not in impending respiratory failure or experiencing anxiety. Standard support and monitoring will likely be sufficient to detect any changes in patient status. A rapid respiratory rate or restlessness in the patient should cause the nurse to set up different levels of support and to collect additional data.

Managing Data

In real life, multiple conditions may occur concurrently, and one finding (e.g., vital sign, hemodynamic parameter, lab value, assessment finding) may provide evidence for a variety of conditions. Because so much information is collected and used to form judgments in acute and critical care settings, flowsheets—either written on paper or assembled electronically—are used to organize and present the many pieces of information. Recognition of any condition depends on seeing patterns in the wide range of data available. Additionally, flowsheets enable healthcare providers to see how data points change over time. Individual values in isolation are not reflective of the whole person, nor are they reflective of the direction that a particular patient's condition is taking. Is the patient becoming more stable or less stable? Is mechanical ventilation providing adequate support of physiologic function, or is the patient so agitated or distressed by being unable to speak that expenditure of unnecessary energy is occurring? Is the patient failing to respond to any treatment approach such that multiple organ dysfunction syndrome is occurring? Seeing the whole of a situation comes with experience. It can lead to intuition, the topic of the next subsection.

Intuition

Once the nurse is oriented to critical care, the patterns of human response to challenges faced in critical situations become more evident and easily recognizable. Eventually, the

nurse is able to see the wholeness of a situation. The pieces of data are not seen discretely, but rather as patterns indicative of the whole. The nurse may simply look at the patient and recognize impending loss of stability or the loss of the will to live. At times, experienced nurses will see a pattern or feel a "gut" response to a clinical situation that allows them to "know" the situation of the patient without spending time processing individual pieces of data. Of course, to provide the data that an interdisciplinary team needs to set up a treatment plan, nurses must generate data and check on those "gut" feelings they have about the patient. What is interesting is that the intuition precedes the action. Nurses can develop their intuitive skills by discussing their "hunches" about patients, by analyzing which indicators led them to their intuitive sense, and by checking their own accuracy. Experienced nurses can do this in unit nursing rounds or in clinical case discussions.

The AACN Synergy Model for Patient Care recognizes that as nurses gain expertise, they move from Level 1, which focuses on data collection, following decision trees, and using standard protocols, to Level 3, where nurses are able to see the wholeness of situations quickly. A sense of understanding of the direction of processes is part of the competency of these nurses. At Level 5, nurses synthesize large amounts of data and help the entire team to recognize the "big picture" of what is happening with the patient (Reed et al., 2007).

Decision Analysis

Decision analysis is an approach to decision making based on mathematical models that take into consideration the likelihood of specific responses given action options. What is the likelihood that a patient who is intubated will develop pneumonia? What is the likelihood that the same intubation will allow for physiologic support during response from trauma or surgery? On a larger scale, if a new closed system suction device is used, what will be the reduced cost of care if the rate of ventilator-associated pneumonia is reduced? Decision analysis uses frequency and cost data to weigh options in care. It can be used for either individuals or groups of patients. Many current guidelines for practice are based on this kind of mathematical analysis.

RELATIONSHIP-CENTERED CARING IN CRITICAL CARE

All nursing is carried out in the setting of relationships. Despite the fact that many critically ill patients are intubated and unable to speak, nurses form relationships with their patients and their families. Such relationships are not just "being nice"; rather, they are central to coming to know patients and how they respond to the challenges of illness. Critical care nurses learn to recognize the patterns of patient responses. How one patient responds to the physical challenge of weaning from mechanical ventilation is different from how another patient does. For example, one patient may become tachypneic in response to the increased work of breathing during weaning, whereas another patient may experience an increased heart rate. Recognizing and communicating patient response patterns is important to excellence in critical care nursing. Recognizing the patterns of how patients respond to challenges can help the nurse decide when in the day is best to provide physical care or to attempt a weaning trial. If a patient did not sleep the previous night, for example, then rest before weaning may result in a better response.

The relationships formed by nurses also extend to patients' families. Family members can provide needed comfort and a quiet presence, or they can spread their own anxiety to the patient. Supporting the family and managing their responses and connection to the

patient are important interventions for optimal outcomes. Family members can assist CCNs in coming to know their patients, thereby helping ensure that the nurses can understand what matters most to the patients.

Now that we have explored the various ways of thinking that can be used in clinical judgment situations, we will see how CCNs can use these models in day-to-day practice.

DAY-TO-DAY PRACTICE

Critical care units are areas where specialized equipment allows for the continuous collection of data related to a patient's status. The quality of the data being collected and recorded is a central issue. If an intra-arterial line is improperly zeroed, the readings will be consistent—but they will be consistently inaccurate, which can lead to improper treatment plans being established. Critical care nurses learn during orientation how to set up monitoring systems in anticipation of patient admission to the unit, and they learn routines of validating systems as they assume responsibility. In many units, technicians are available to set up lines and equipment, but verifying the accuracy of readings is the responsibility of the nurse. In addition, over time, readings can drift for various reasons such as lines moving, patient position changes, or mechanical equipment problems. Experienced nurses learn to constantly assess the reliability of the data they collect. If a data pattern does not match the apparent condition of the patient, the nurse rechecks the source of the data for accuracy. The adage, "Treat the patient, not the numbers," is good to remember regardless of whether the numbers are accurate. Other data that might not be reliable include arterial blood gas values if the sample is not read immediately or if the patient has leukocytosis. Serum chemistry values may also be inaccurate depending on the quality of the sample and the precision of the analysis.

Establishing and verifying the data collection and monitoring system are important first steps in critical care judgment. The next step is establishing regular monitoring routines. Most critical care units have unit-specific routines for data collection, and some establish routines for monitoring particular types of clinical problems. These routines are important because a patient's status may change frequently in critical care, and regular monitoring allows the nurse to detect changes early, when intervention can prevent clinical deterioration. The nurse should consider, however, that each decision about data collection also has its own cost. For example, frequent blood draws over time can result in noticeable blood loss, particularly in pediatric settings. Awakening a patient hourly for days and nights in a row can result in sleep deprivation, which prevents healing and can lead to delirium. Sending samples for lab analysis costs the patient and the entire system financially as well.

The timing of data collection is one of the judgments that nurses should make by considering the entire situation of the patient. Additionally, unit protocols for assessment should be periodically reviewed after considering published reports and patient data. At which phase of recovery from major surgery is the patient most likely to have specific complications? When would data collection be appropriately timed to detect a specific complication? Unit-level practice committees can address questions such as these.

Too often, data collection becomes a mindless routine. The numbers are generated and the flowsheet is filled in, but no one really considers what the data mean. This situation represents a failure of the nurse to exert clinical judgment. It results in wasted energy and resources, and it does not protect the patient. Several ways that the CCN can be thoughtful about the data that are routinely collected are discussed next.

Trending and Knowing the Patient

Flowsheets are developed for specific critical care units to help organize data for processing purposes. By seeing how individual data bits change over time, "trends" can be detected. These trends are more important in determining the status of the patient than any individual piece of data would be. Is the blood pressure making a slow decline over the past two hours? Is this patient's heart rate generally slower than baseline? Identifying such patterns helps to determine the clinical significance of a change in any data reading. For a patient with a normally slow heart rate, a new rate of 80 might be worrisome; for another patient, a rate of 80 would not be a reason for clinical concern. Flowsheets also allow the nurse to see how readings of one parameter change along with other parameters. Blood pressure readings that are gradually decreasing but remain in the acceptable range might not be of concern. However, if the urine output is dropping during the same period, a condition of low cardiac output must be considered. Additional data about recent fluid loss, rates of fluid replacement, and an assessment for crackles in lungs would be needed. Critical care nurses spend much of their time collecting data. This is not the end of task, however, but just the beginning. Taking time to reflect on the "movement" or trend of the data is essential for critical care clinical judgment.

Even in critical care, contextual patient-related factors are important in coming to know the patient. The AACN Synergy Model for Patient Care points out patient characteristics that are part of each encounter. Central to critical care are consideration of patient stability and the predictability of the course of recovery. Other key characteristics include patient resiliency, vulnerability, complexity, and resource availability. The Synergy Model also incorporates a consideration of the patient's ability to participate in decision making and care (Reed et al., 2007). Clearly, coming to know the patient involves more than just gathering physiologic data.

Common Trajectories

Making sense of data requires knowing the individual patient, but it also requires knowing pathophysiology and understanding the workings of the body's compensatory mechanisms for a variety of critical care conditions. Nurses know for their own particular specialty unit—be it cardiovascular surgical, trauma, coronary care, neurosurgical, medical, transplant, or some other unit—the particular problems typically faced by patients in that unit. Critical care judgments are formed through a blend of knowing individual patients and knowing the trajectories that patients are likely to experience in a particular setting. In individual orientation programs or staff meetings, the particularities of units can be discussed and a common understanding developed by nurses or, even more powerfully, in an interdisciplinary perspective.

A trajectory is a predictable path or sequence of events that is commonly seen in a particular setting. For example, following open heart surgery for coronary revascularization with cardiopulmonary bypass, patients commonly require vasopressor administration to maintain blood pressure to support patency of newly implanted vessels. In addition, patients may experience tachycardia that can decrease cardiac output. Patients may be mechanically ventilated and have multiple chest tubes and pacing wires implanted directly in the myocardium. They will have central vascular access to facilitate fluid and medication administration. A common trajectory includes weaning the patient from vasopressors on the first night following surgery, weaning from mechanical ventilation by the morning after surgery (if not extubated before), and a gradual reduction in chest tube drainage. Deviation from this expected trajec-

tory, such as decreased oxygenation when weaning from mechanical ventilation is attempted or continued blood loss from chest tubes, indicates that this particular patient will require an individualized approach to support. Experienced CCNs recognize patients' progress along specific trajectories. A sense of how the patient is progressing down the predictable path of recovery is one way that the CCN sees patterns and senses the wholeness of the situation.

Surveillance

In critical care areas, nurses use a type of thinking that assesses for problems that do not yet exist. This is a different style of thinking than problem identification. It is a continual scanning for signs that a problem is developing. This method of thinking requires several kinds of knowledge, data collection, and processing. CCNs who wait until a problem becomes obvious before they intervene have missed a chance to prevent a cascade of events.

Knowledge that supports effective surveillance includes a deep understanding of the physiologic responses to the critical care setting and to the particular patient problems being addressed. Knowing that tracheal intubation exposes a patient to risk of ventilator associated pneumonia, the CCN with a high level of clinical judgment monitors arterial blood gas results, breath sounds, airway pressures, and vital signs. Waiting until pneumonia is fully evident would result in risk of hemodynamic instability and sepsis, both of which can lead to longer ICU stays or death.

Regular data collection for evidence of stability or signs of problems is essential to the process of surveillance. Most important, though, is the nurse's ability to recognize patterns that indicate deviation from the normal trajectory.

Investigating Problems

Experienced CCNs read their "gut" reactions. When patient responses indicate that things are going as predicted, nurses can alter their vigilance. Conversely, if the patient is not following the predicted trajectory, then the nurse considers appropriately other data sources, and discusses possible meanings of this divergent pattern. The nurse does not "rest" until the picture becomes clearer. Even "hunches" about what is going on can be explored and discussed until the patient's picture becomes clearer and data indicate an appropriate direction for decision.

One practice that critical care nurses use is that of "running possibilities." This process is a form of hypothesis generation, referred to earlier in this chapter. What could be a possible explanation for this finding? Could this person have an unusual presentation of a treatable problem? What if we try a treatment option for a while and see how the patient responds? This sort of thinking frequently happens in conversation with other nurses or with physicians (Chase, 1995).

Communicating Findings

Nurses in critical care have more autonomy than nurses in many other practice settings regarding data collection and treatment decisions such as weaning from various types of support. CCNs do not work in isolation, however, and they contribute to excellence in patient care by working collaboratively with a team of other healthcare providers. One of the skills that CCNs develop is effective communication of their impressions of the status of the patient to other members of the team. Many nurses have had the frustrating experience of believing that the patient needs to be managed in a certain way, but other members of the team do not agree. When the direction of the care and support differs, nurses are obligated to clarify, verify, and question the appropriateness of the treatment plan (if they believe that harm will come to the patient). Learning to communicate data and impressions in ways that allow others to understand

the basis for the CCN's judgment can minimize this source of frustration.

Assembly of data into patterns that have meaning will assist CCNs in communicating their overall impressions. Calling a physician and offering random bits of data will often not result in a positive response. The nurse can better organize this process by coming to know the types of data that individual clinicians value. For example, even if the findings are not abnormal, the amount of chest tube drainage will be important to a cardiac surgeon. When working with new teams of physicians, an anticipatory question can help to establish communication, such as, "Is there any particular parameter that you want us to pay special attention to this evening?" or "I've noticed a downward trend in blood pressure. Is there a level at which you want us to notify you?" Then, should a call be necessary, it has a context. This kind of communication requires "thinking forward."

One method that has been established in healthcare settings to assist with the assembly of data into meaningful patterns is the SBAR (Situation–Background–Assessment–Recommendation) technique. This framework facilitates communication among healthcare providers by providing a focused approach for communicating essential patient information in a usable context so that accurate care decisions can be made (Institute for Healthcare Improvement, 2008).

By understanding the competing hypotheses for the patient's condition, the CCN will be better able to present data in a way that assists the entire team in making good decisions. One kind of data that must be considered is "pertinent negative" data—that is, showing that certain data are normal to reduce the likelihood of one of the diagnostic options. For example, if the blood pressure is trending down, but breath sounds and arterial blood gas results are normal, that combination of findings would decrease the likelihood of left ventricular failure and increase the likelihood that the patient is volume depleted. The breath sounds and arterial blood gas results should be reported even though they are normal because they assist the other clinicians to understand the whole picture: They are "pertinent" even though they are normal.

Mobilizing the Team

Sometimes a CCN may detect that the patient's condition is changing rapidly and must assemble the necessary team members to respond appropriately. To do so, the nurse may need to page respiratory therapy, anesthesia, or other airway management teams, as well as the primary physician or designee. Making the decision to mobilize the team can be a daunting one for new CCNs. Experienced nurses and leaders can assist the new CCN in making this decision in a timely fashion. On the one hand, waiting until the situation becomes obvious would be dangerous for the patient. On the other hand, if the nurse calls the team in unnecessarily, that decision has costs, both financial and personal. It is possible that the CCN's clinical judgment was at a lower level in the AACN Synergy Model and that the call came prematurely or in error.

To deal with such issues, CCNs can discuss the process of mobilizing the team on individual units and reflect on how the process went: Did the nurse assemble sufficient data to generate the calls? Was the potential patient problem severe enough to warrant the call? Was the presentation of findings sufficiently clear? Did other members of the team respond appropriately? In hindsight, would any aspect of the patient's care be managed differently?

Team Decision Making

Ultimately, the critical care process is a team process. Data support the idea that good communication on a unit results in better

patient outcomes (Baggs et al., 1999; Knaus, Draper, Wagner, & Zimmerman, 1986; Kohn, Corrigan, & Donaldson, 2000; Larson, 1999; Page, 2004; Tammelleo, 2001). Units vary widely in how effectively communication occurs. Several possible problems can occur that the CCN should be aware of and try to correct.

The first consideration is nurse-to-nurse communication. Are experienced nurses helpful to new orientees, or do they require the new nurse to "pay their dues"? This kind of hazing should be recognized as such and should be dealt with by unit leadership. Other nurse-to-nurse difficulties can come at change of shift report, where one shift does not help establish the new shift nurses' understanding of patient baselines due to emotionally charged communication.

Other issues that arise may relate to whether the patient unit is orderly, with supplies on hand, and with essential data already assembled. Small things like this can lead to difficult communication and ultimately can result in poor nursing care.

Additional nurse-to-nurse difficulties can happen at the time of patient transfer. It is essential to the clinical judgment process that open and clear communication be established between patient care areas. By sharing with healthcare providers in the new unit what the patient's clinical course or trajectory has been, how this patient is unique, and which approaches have worked best, better clinical judgment is promoted on the new unit.

Choosing Interventional Approaches

Much of our consideration thus far has focused on clinical judgment as it relates to the status of the patient, patient stability, patient movement along a recovery trajectory, or the identification of problems. Judgment is also required regarding how best to respond to the issues that are identified in the assessment process. All management choices should be goal oriented and contextually appropriate. The AACN Synergy Model provides for a way of matching the CCN's competencies to the patient's needs. "Synergy results when the needs and characteristics of a patient, clinical unit, or system are matched with a nurse's competencies" (Hardin & Kaplow, 2005, p. 4). Even given the same medical condition, the CCN's response to the patient should reflect numerous factors, including those described in the Synergy Model. For example, a patient who has high levels of resiliency, as evidenced by return to baseline data after treatments, can be expected to recover more quickly and need less aggressive support than a patient who, because of longstanding concurrent conditions, might not be capable of rallying. A patient with few external resources might require aggressive advocacy on the part of the CCN.

Goal-Oriented Decisions

In line with the concept of trajectory, CCNs should always have a goal in mind when planning specific nursing actions. If the goal is stability, then support of basic physiologic functioning will support that goal. If the goal is to increase participation in care so as to support the patient–family unit, then adjusting visiting times to allow for prolonged contact might be chosen, provided that patient stability is not compromised. The CCN can then reflect on the effectiveness of those interventions in accomplishing the goal.

CCNs can actively support the unit in developing documentation systems that include goals and nursing actions. If a patient is anxious about how the family is responding to critical illness, for example, being able to see and be with a family member can reduce stress and the related catecholamine release that can have negative effects on the cardiovascular system. Nursing actions can have real effects on overall patient status. Promoting comfort and dignity for patients is a requirement for humanistic care and healing.

Supporting the Dying

As discussed earlier, the experienced CCN develops a sense of the big picture of the patient's condition and the direction of the trajectory. Often, critically ill patients have life-threatening conditions that can result in death. Death sometimes happens during aggressive resuscitative efforts. Frequently, however, an impending death is recognized by at least one member of the team. The goals of care may then shift to allow for patient comfort and family communication. The transition to caring for the dying patient can be one that provides the ultimate meaningful contribution on the part of the staff. Too often, however, an impending death is a time of competing goals, shifting direction of care, and difficult communication.

The CCN can assist in the dying process by maintaining a consideration of "Where are we going?" Asking that question during team meetings can assist the entire team in addressing the futility of care. The patient's and family members' goals will also need to be determined as part of this process, and it is often the nurse who assists in clarifying these values (Hiltunen, Medich, Chase, Peterson, & Forrow, 1999).

SUMMARY

A critical care nurse is not a technician. As a professional nurse, the CCN's focus of care is on the whole person and family at a vulnerable time. The focus of care on the physical problems patients face in critical care is obvious. More is known by clinicians about the functioning of the human body of patients in a critical care unit than by providers in almost any other environment of the healthcare system. Critical care nurses learn over time, however, that more is going on in a critical care unit than simply the care of physical bodies. Critically ill patients are whole human beings. Their fear or trust, their will to live, their ability to participate in care, and family support can make a real difference in patient outcomes. Ultimately, the clinical judgments made by CCNs are pivotal to providing care to acute and critically ill patients. Nurses are essential to the process of providing care by virtue of their perspective on meeting the needs of the whole patient. These needs can be based on the eight patient characteristics outlined in the Synergy Model. Nurses' constant presence provides for a way of seeing and knowing the person who is experiencing critical illness. Growing in ability to form exquisitely appropriate clinical judgments is a lifetime challenge—but it is one that is rewarding to both patient and nurse.

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Chapter 2

Cardiovascular Anatomy and Physiology

Susan K. Chase

INTRODUCTION

The heart is a muscular organ located beneath the sternum, between and slightly anterior to the lungs, in a section of the thorax known as the mediastinum. The mediastinum also contains the great blood vessels—the vena cavae, the pulmonary artery, and the aorta—as well as the esophagus and (in children) the thymus gland. Figure 2–1 illustrates the location of the heart.

The heart is surrounded by the pericardium, a dual-layer sac that is minimally elastic. This sac allows for smooth movement of the cardiac muscle within the pericardium. If fluid or blood fills the pericardium, it puts pressure on the heart from the outside and prevents normal filling of heart chambers. The main function of the heart is to pump blood throughout the body, thereby allowing for the delivery of oxygen and nutrients to the body cells and for the transport of waste products to processing or removal organs. Other functions of the cardiovascular system flow from the blood itself: The blood consists of cells that support the body's ability to fight off infection as well as chemicals such as hormones that control processes of bodily systems. In addition, the heart releases hormones that assist in controlling blood flow and pressures.

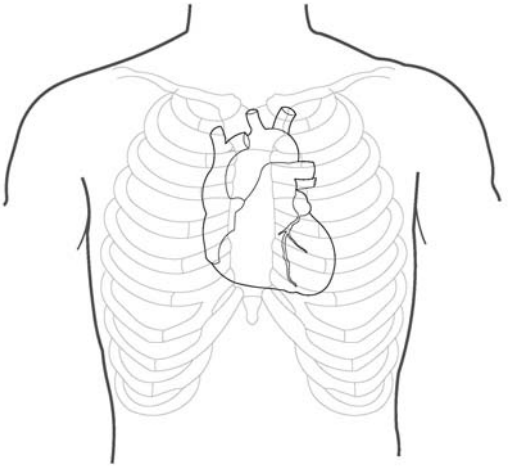

Figure 2–1 The heart and its location in the thoracic cavity.

Source: Illustrated by James R. Perron

CHAMBERS AND VALVES OF THE HEART

The structure of the heart supports its functions. The heart consists of four chambers, each with muscular walls (Figure 2–2). It also has four valves that control the direction of the blood flow through these chambers. The two upper chambers of the heart are the atria; the two lower chambers are the ventricles. Actually, the terminology of "upper" and "lower" refers to a conceptual picture of the heart, with the most anterior chambers of the heart being the right and left ventricles. The muscle walls of the four chambers vary widely in thickness. Because

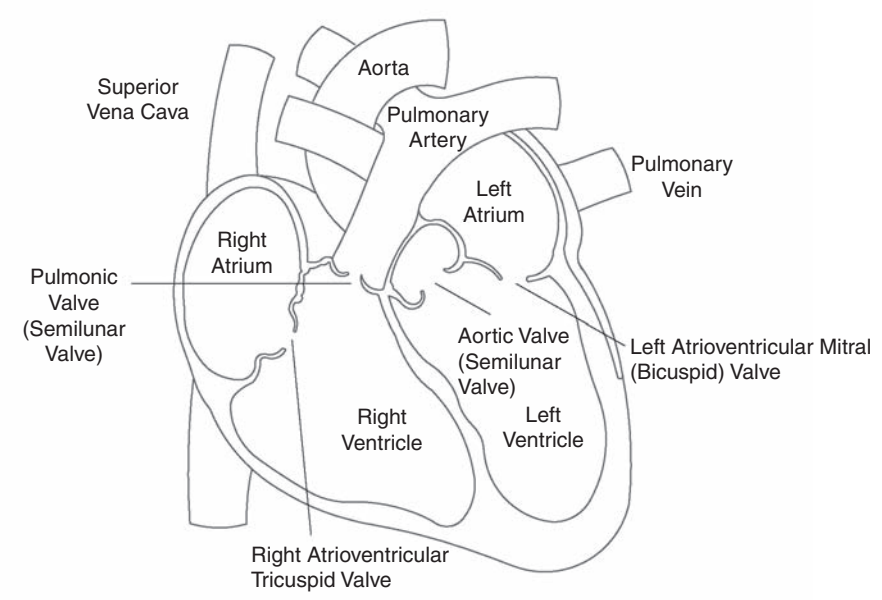

Figure 2–2 Chambers of the heart and valves.

Source: Illustrated by James R. Perron

the left ventricle must pump blood into the systemic circulation, which has relatively higher pressure than the pulmonary system, the wall of the left ventricle is the thickest (13–15 mm). The right ventricle is only 3–5 mm thick. The atria have the thinnest walls (2–5 mm).

POINT OF MAXIMAL IMPULSE

The tip of the left ventricle is positioned anterior and to the left in the mediastinum. When the left ventricle contracts, its tip is forced even more anteriorly toward the chest wall. This movement can be palpated as the "point of maximal impulse" (PMI). The PMI is normally located in the midclavicular line at the fifth intercostal space, but can sometimes vary. Abnormalities in the shape and size of the heart, for example, can alter the position and location of the heart itself. A distended abdomen can flatten and elevate the level of the heart. Hyperextended lungs can depress the level of the heart. Enlargement of the heart can cause the PMI to shift to the left in the chest. Noting the position of the PMI can, therefore, give some indication of the size or position of the heart (Woods, Froelicher, Motzer, & Bridges, 2004).

Although most of the heart tissue is muscle, this organ also has a fibrous band that separates the atria from the ventricles and contains the four cardiac valves, which are themselves made up of connective tissue. The cardiac valves consist of fibrous rings to which valve leaflets are attached. The tricuspid valve contains three flat valve leaflets. The pulmonic and aortic valves each have three leaflets that are termed "semilunar" because of their crescent-like shape. The mitral valve has two flat leaflets that resemble the pointed shape of a bishop's miter. The valves themselves are covered with epithelial tissue. The tricuspid and mitral valves (collectively termed the atrioventricular [AV] valves because of their location) are attached to chordae tendinae, which are connected on their opposite ends to papillary muscles in the ventricles. The muscles prevent the valve leaflets from being pushed backward into the atria when pressure rises in the ventricular chambers during ventricular contraction. Proper functioning of the valves depends on all these features being intact.

The right and left sides of the heart are divided by the septa. The interatrial septum consists of the fossa ovalis (the sealed foramen ovale that normally closes in the postpartum period) and the muscular walls of the right and left atria. The interventricular septum is formed by the ventricular muscle in the lower portions and by the upper membranous section (Woods et al., 2004).

BLOOD FLOW THROUGH THE HEART AND MAJOR BLOOD VESSELS

The right atrium receives blood from the body through the superior and inferior vena cavae as well as from the coronary sinus, which returns the blood that has circulated through the heart muscle itself. Blood enters the right atrium during atrial relaxation (and ventricular systole). When the pressure in the right ventricle decreases during its resting phase (ventricular diastole), the tricuspid valve opens, allowing the blood flow from the right atrium to the right ventricle. After the right ventricle fills with blood, the muscle wall contracts, increasing the pressure in its chamber, which in turn forces the tricuspid valve to close. As pressures continue to increase, blood is forced out of the right ventricle across the pulmonic valve and into the pulmonary artery. The pulmonary artery transports the still unoxygenated blood into the pulmonary vascular system.

In the pulmonary system, the blood circulates through a series of arteries, capillaries, and veins. In the thin-walled capillaries of the pulmonary circuit, red blood cells exchange carbon dioxide for oxygen. The oxygenated blood then returns to the left ventricle, driven by the pressure differential: Pressures in the left ventricle are lower than in the pulmonary vascular system. When the right ventricle relaxes during diastole, the pressure in the right ventricle decreases, which causes the pulmonic valve to close. Blood from the right atrium then refills the right ventricle.

Oxygenated blood returning from the pulmonary vein enters the resting left atrium. When the left atrium pressure rises higher than the pressure in the resting left ventricle, the mitral valve opens. Blood then passes to the left ventricle across the mitral valve. The contraction of the left atrium forces additional blood into the left ventricle. Finally, as the left ventricle contracts, the pressure there increases and forces the mitral valve closed and the aortic valve open. Blood passes from the left ventricle to the systemic circulation across the aortic valve. It flows to the cardiac muscle itself through the right and left coronary arteries, which arise from the lower aorta, just above the aortic valves.

Each of the cardiac chambers has its own range of normal fluid pressures, which depend on the force of contraction of the muscle walls and the position of the cardiac valves in that chamber. Each chamber has a phase when its walls are contracting (systole) and a phase when the muscle is resting (diastole). Most of the time, the words "systole" and "diastole" are used to refer to the phases of the ventricles. Under normal circumstances, due to the electrical control system of the heart, the atria contract together and the ventricles contract together.

It is useful to be able to picture the heart during systole and diastole when interpreting heart sounds (see Box 2–1). During ventricular systole, the AV valves are closed and the semilunar valves are open; blood flows through the latter valves into the pulmonary and systemic circulation. During ventricular diastole, the semilunar valves close and the AV valves open, with blood flowing through the latter valves. Unexpected

Box 2–1 Murmur Differentiation

Valve	Stenosis	Insufficiency
Tricuspid and Mitral	Diastolic	Systolic
Pulmonic and Aortic	Systolic	Diastolic

sounds heard during ventricular systole could result from tight or "stenotic" semilunar valves (aortic or pulmonic) or from incompetent or regurgitant AV valves (mitral or tricuspid). These sounds are best heard between S_1 and S_2. Unexpected sounds heard during ventricular diastole are heard between S_2 and S_1. These sounds can be related to mitral/tricuspid stenosis or aortic/pulmonic insufficiency (regurgitation). Obstruction to forward flow is stenotic, and backward flow of blood is due to an incompetent valve.

Being able to think spatially will assist the nurse in making sense of cardiovascular assessment data. The diagram of the cardiac cycle in Figure 2–3 shows the simultaneous events of cardiac function, including pressure changes in individual vessels and chambers and electrical activity.

CORONARY ARTERIES

Because the pressure in the muscle tissue during ventricular systole is so high, the coronary arteries are perfused during ventricular diastole. The left main coronary artery divides fairly quickly into the left anterior descending (LAD) artery and the circumflex artery (CA) (Figure 2–4). The right coronary artery (RCA) supplies most of the right atrium and ventricle and the SA node (in 60% of people), the AV node (in 80–90% of people), and part of the bundle branches. The left anterior descending artery supplies the left atrium and ventricle, including the ventricular septum. The circumflex artery supplies the posterior portion of the left ventricle and the left atrium. The blood supply to the sinoatrial (SA) node of 40% of the population is received through the left circumflex artery. The venous return of the heart leads to the great coronary vein, which parallels the circumflex artery and eventually returns to the right atrium.

CARDIAC LYMPHATIC SYSTEM

The heart produces a certain amount of lymphatic drainage that flows through the pre-

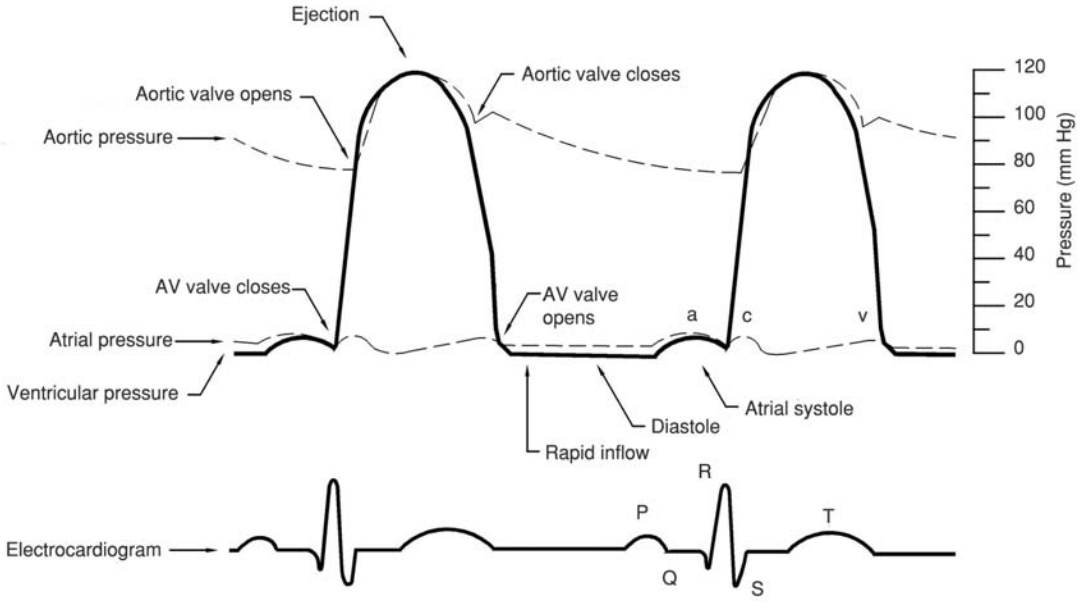

Figure 2–3 The cardiac cycle.

Source: Illustrated by James R. Perron

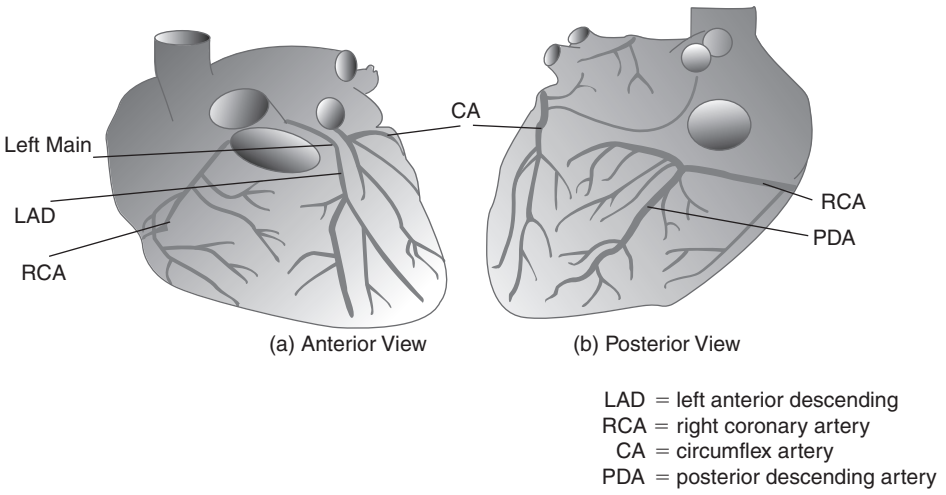

Figure 2–4 Diagram of coronary circulation.

tracheal lymph node and eventually empties into the superior vena cava. Blockage of lymph flow can affect pressures in the heart itself related to venous congestion. The return of lymph to the systemic circulation is critical to prevent interstitial edema. Also of importance is that cardiac lymph fluid contains hormones (atrial natriuretic peptide) and adrenergic neurons (norepinephrine) that can be used as markers for myocardial edema, reperfusion injury, and myocardial damage (Konuralp, Idiz, & Unal, 2001).

People can vary somewhat in terms of the arrangement and area that the coronary arteries supply. Coronary angiography can reveal the individual's unique configuration.

With age, vessels may become narrowed due to plaque and thickening of the arterial walls. Collateral circulation may then develop, as blood is drawn from nearby arterioles to supply areas that might otherwise not be perfused adequately because of blockages to primary blood sources. If collateral circulation is well developed, blockage of a major artery may not cause as much damage as it would for a person with no collateral circulation (Woods et al., 2004).

PRESSURE OF BLOOD IN MAJOR BLOOD VESSELS

Major blood vessels experience variations in their pressures related to cardiac events. Pressure waves in the great veins and the right atrium are given codes (letters) to assist in the interpretation of waveforms (Figure 2–5). For example, the "v" wave represents filling of the atrium from systemic veins. The "x" descent follows the "v" wave and represents change in shape of the atrium as a result of ventricular emptying. The "a" wave represents increased right atrial pressure caused by atrial contraction and is followed by the "y" descent, which represents a decrease in pressure as the tricuspid valve opens. The slight rise in right atrial (RA) pressure, called the "c" wave, represents the increase in pressure coming from the ventricle that causes the tricuspid valve to close.

Arterial pressure increases rapidly with ventricular systole and attains the pressure represented by the systolic blood pressure. The dichrotic notch of the arterial waveform represents the closure of the aortic valve. This closure maintains the pressure of the system circuit at the level represented by the diastolic blood pressure.

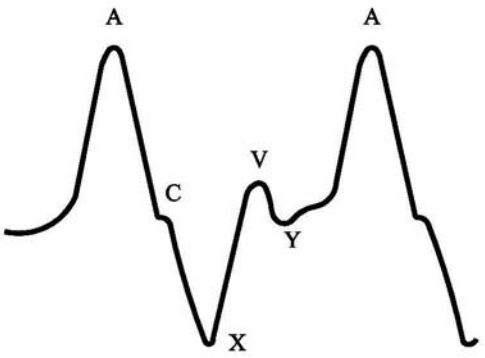

Figure 2–5 Pressure waveforms.
Source: Illustrated by James R. Perron

ELECTRICAL CONTROL OF CARDIAC MUSCLE

Cardiac muscle cells are unique in the body for a number of reasons. First, unlike skeletal muscle cells, they are capable of automaticity. That is, cardiac muscle cells do not require stimulation from an outside force such as a nerve to initiate an action potential, which causes contraction. Second, cardiac muscle cells are interconnected in web-like fashion with separation only by intercalated discs, which allows for impulses to pass through the entire section of the heart like a wave.

Action potentials occur when the polarity (i.e., electrical charges) across the cell membrane changes rapidly. In the resting state, there are more positive ions (sodium ions, which are present in the largest number, but also calcium and magnesium ions) outside the cell membrane as compared with the positive charges inside the cell. Potassium is the chief intracellular cation (positive ion), and there exist relatively more anions (negative ions) inside the cell from proteins and other sources. The cell membrane is therefore "polar"—similar to the scheme used to power a flashlight battery, which has more positive ions on one side than on the other. An action potential spreads to neighboring cardiac cells like a wave.

The atria and ventricles are separated by thick fibrous tissue that supports the four heart valves. This fibrous tissue prevents action potentials from being transmitted between the atria and ventricles. The exception to this barrier occurs in the AV junction, or the AV node. Electrical impulses pass from atria to ventricles across this specialized set of tissues (Figure 2–6). To support simultaneous contraction of the thick ventricular muscle walls, specialized conduction tissue transmits electrical impulses through the bundle branches and the Purkinje fibers; the atria have similar transmission fibers. The electrical impulse is slowed at the AV node, a delay that allows for the atria to contract and empty blood into the ventricles.

Each cell of the heart is capable of initiating an action potential, but different areas of the heart have different basic rates of discharging. Under normal conditions, the SA node, which is located in the right atrium, has the fastest rate; depolarization occurs there approximately 60 to 100 times per minute. Cells of the AV node can depolarize 40 to 60 times per minute unless a more frequent impulse, such as from the SA node, is transmitted through them. Ventricular cells can initiate an action potential 20 to 40 times per minute. This activity is protective to the heart: If something happens to prevent normal action potentials from reaching the ventricle, the heart will still beat. Thus the SA node is normally in control of the heart rate because it has the fastest intrinsic rate.

Box 2–2 provides a closer look at the various phases of action potentials to help

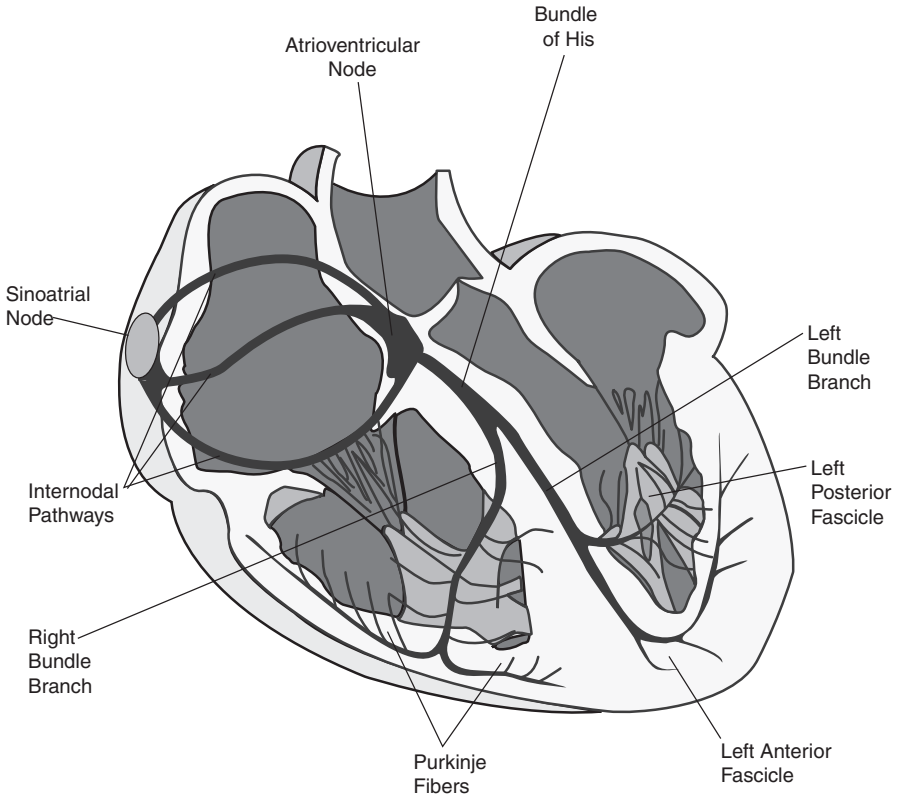

Figure 2–6 Electrical conduction system of heart.

Source: Arrhythmia Recognition: The Art of Interpretation, courtesy of Tomas B. Garcia, MD

explain these events and explain how certain medications can affect them. Action potentials for the SA and AV node are somewhat different from the pattern depicted in Figure 2–7, which allows the SA node to operate more independently. These impulses are more tightly controlled by the slow calcium channels than the sodium channels. Calcium-channel blockers can slow heart rate by slowing the transport of calcium across the cell membrane (McCance & Huether, 2006).

A refractory period occurs after the action potential before the resting concentrations have fully returned to normal. New impulses that reach the tissue during this period will not be transmitted, or potentially can establish abnormal rhythm patterns. Because these activities work against the concentration gradient, they require the expenditure of

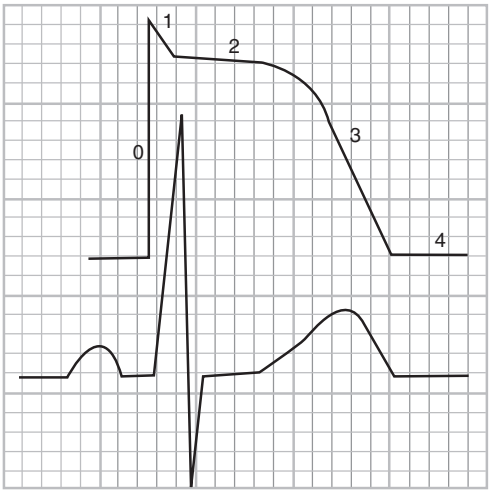

Figure 2–7 Action potential diagram.

Source: Illustrated by James R. Perron

Box 2–2 Action Potential Phases

Phase 0: Depolarization. Sodium ions cross the cell membrane rapidly through sodium channels, causing the polarity of the cell membrane to change rapidly.

Phase 1: Rapid Repolarization. Potassium ions "leak" outside the cell.

Phase 2: Plateau. Opening of slower calcium channels allows cations to enter the cell, balancing the loss of potassium ions.

Phase 3: Rapid Repolarization. At the end of the action potential, the channels close and the cell returns to its resting state by pumping sodium ions out of the cell and bringing potassium ions back inside.

Phase 4. Resting State. This phase is diastole where cells remain resting until an electrical impulse occurs.

energy. As much as two-thirds of the cardiac cell's energy is spent in supporting the sodium–potassium pump. Any loss in energy for the cardiac cell results in interruption of this essential pump fairly quickly. Such a disruption affects the cell's ability to return to normal polarity. Because sodium ions build up in the cell, water (a polar molecule) is attracted to the sodium ion. As a result, internal structures of the cardiac cell swell, resulting in the release of internal cell enzymes. Electrolyte abnormalities can make cardiac cells more prone to develop an action potential and more likely to initiate a transmittable impulse, which causes an abnormal cardiac rhythm.

Electrical events precede mechanical events. When a cardiac cell experiences an action potential, it contracts. As the wave of electrical activity passes through the cardiac muscle wall, the muscle contracts in a wave-like fashion. For this reason, cardiac function is often monitored by electrocardiography. The nurse must be aware that damaged cardiac muscle may not respond with full-force contraction even if the electrocardiogram tracings appear normal.

CARDIAC OUTPUT, PRELOAD, AND AFTERLOAD

As mentioned earlier, the heart functions to pump oxygenated blood to cells, organs, muscles, and tissues and deoxygenated blood back from the systemic circulation. Some concepts have been developed that assist clinicians in understanding overall cardiac function. Cardiac output (CO) is a measure of the amount of blood that is ejected by the heart each minute. Stroke volume (SV) represents the amount of blood that is ejected from the left ventricle with one contraction. Cardiac output, then, is the product of the stroke volume and the heart rate (HR):

$$CO = SV \times HR$$

Stroke volume is influenced by the amount of blood in the ventricle and by the force of contraction of the ventricle. It can also be affected if the aortic valve restricts flow out of the left ventricle. The ejection fraction (EF) is the percentage of the volume of the left ventricle that is ejected with each contraction. A normal ejection fraction is in the range of approximately 65% to 70%, where this value reflects the efficiency of the left ventricle in pumping blood forward into the systemic circulation.

Preload (sometimes referred to as left ventricular end-diastolic pressure) is the pressure found in the left ventricle at the end of diastole. The Frank-Starling law states that stretched muscle fibers produce a more powerful contraction; thus, when the left ventricle is fully

filled, a more powerful contraction becomes possible. Conditions that prevent filling of the ventricle with blood—such as hypovolemia, dehydration, or external pressure on the heart from fluid in the pericardium—will reduce both the pressure in the ventricle and its ability to pump blood forward. Nevertheless, the Frank-Starling law reaches its limit when cardiac chambers are overstretched: The resulting contraction is not as effective as it would have been with slightly less stretch. The overstretching can eventually result in a lower cardiac output.

Afterload is the resistance against which the left ventricle must pump to move blood forward. The pressure of the arterial systemic circulation produces afterload. Smooth muscle tone in arterioles can increase the resistance to blood flow and increase afterload. Medications can also alter the amount of resistance that arteriolar smooth muscle generates. For example, arterial vasodilators decrease afterload, whereas vasoconstrictor agents increase preload, afterload, or both.

EXTERNAL CONTROL OF THE HEART

Nervous system control of the heart comes through the autonomic nervous system and can cause rapid changes in heart activity. The autonomic nervous system consists of sympathetic and parasympathetic nerve fibers. The sympathetic nervous system controls the body's "fight or flight" mechanisms, quickly preparing the total organism to resist an attack.

The parasympathetic system governs the "rest and refresh" responses to stress and has nerves that function more individually. The vagus nerve has the chief parasympathetic influence on the heart by affecting primarily the SA and AV nodes and increasing the conduction block at the AV node. Parasympathetic nerve fibers release acetylcholine, which slows the heart—a function sometimes termed "cholinergic."

Sympathetic stimulation increases heart rate and contractility, affecting all parts of the heart. Sympathetic nerve fibers release norepinephrine, which has profound effect on cardiac contractility and vascular resistance. Additionally, the medulla of the adrenal glands is part of the sympathetic nervous system and can stimulate the release of epinephrine into the systemic circulation—a function sometimes termed "adrenergic." Receptors for adrenergic neurotransmitters can be classified as either alpha (α) or beta (β) receptors. Receptors can be further subclassified as α_1 and α_2, or as β_1 and β_2. Dopamine is another neurotransmitter that affects the cardiovascular system. Table 2–1 summarizes the locations and actions of the various receptors.

The activity of the sympathetic neurotransmitters is determined by the location and type of receptors in various tissues. In this way, the same chemical can have different effects in different locations. The heart is rich in β receptors, so that effect is most prevalent for the heart. The systemic circulation has relatively more α receptors, so that effect is more predominant there. Epinephrine stimulates all types of adrenergic receptors, but norepinephrine has little effect on β_2 receptors. Dilation of α_2 coronary blood vessels is promoted by epinephrine, but not norepinephrine (McCance & Huether, 2006).

SYSTEMIC CIRCULATION

Blood is pumped from the left ventricle into the aorta, the largest artery in the body. The aorta rises from the aortic valve and heads superiorly and to the right, which explains why one listens for aortic valve heart sounds at the first intercostal space on the right sternal border. Arteries branch from the aorta beginning at the aortic arch and continue until the aorta itself branches into the two iliac arteries. Multiple systemic arteries branch off from the aorta as it passes through the body. Arteries then branch into a series of increasingly smaller units until they become

Table 2–1 Autonomic Receptors and Cardiovascular Function

Location	Adrenergic Receptor Type	Adrenergic Effect	Vagus Nerve Cholinergic Effect
Sinoatrial (SA) node	β_1	Increased rate	Decreased rate, arrest
Atrial tissue	β_1	Increased contractility and conduction velocity	Decreased contractility, shorter action potential
Atrioventricular (AV) node	β_1	Increased automaticity and conduction velocity	Decreased conduction velocity and automaticity
Purkinje fibers	β_1	Increased automaticity and conduction velocity	No receptors
Ventricles	β_1	Increased contractility	No receptors
Coronary	α_1, β_1	Constriction, dilation	Dilation
Skin	α_1, β_2	Constriction	Dilation
Skeletal muscle	α_1, β_2	Constriction, dilation	No receptors
Cerebral	α_1	Constriction (slight)	No receptors
Pulmonary	α_1, β_2	Constriction, dilation	
Renal	α_1, β_1, β_2	Constriction, dilation	

the smallest of all blood vessels, the capillaries. The capillaries eventually collect into venules, which combine to form veins, which return blood to the heart through the inferior and superior vena cavae.

The entire circulatory system is lined with endothelial cells that are active in controlling local conditions through the release of chemicals. The types of blood vessels have unique characteristics that affect their function. Arteries have thicker walls containing three layers, including a smooth muscle layer. The constriction of the smooth muscle surrounding the arteries is controlled by the action of chemicals such as epinephrine or norepinephrine. The outer layer of the artery consists of connective tissue. Veins have narrower walls with a thinner muscle layer. Venous blood is squeezed up from the legs through skeletal muscle contraction; the large veins of the leg have internal valves that prevent blood from flowing down by gravity.

The endothelial layer of cells constitutes the capillary wall. Capillaries have incredibly thin walls; the varying spaces between the cells that line them allow for fluid and blood cells to pass through the capillary cell membranes or through the spaces between the cells. In the brain, the extremely tight junctions between the endothelial cells force all fluid to go through these cells. In contrast, capillaries found in other parts of the body have relatively more open spaces between endothelial cells that allow for easier exchange of fluid and dissolved chemicals; this kind of openness is found in the liver, for example. Certain conditions, such as inflammation or sepsis, can result in widened spaces, leading to a condition euphemistically called "leaky" capillaries. Precapillary sphincters—smooth muscle cells that control the smallest arterioles—control blood flow to capillary networks. Under normal circumstances, local acidosis causes opening of a precapillary sphincter to an area, thereby increasing its blood supply.

Pressures in the arterial system can be measured by use of a sphygmomanometer or by direct arterial cannulation. Pressures in the capillary network are lower than arteriolar pressures; venous pressures are even lower, supporting venous return. Blood flow is determined by control of pressures and resistance to flow. Pressure inside the circulatory system (indeed, in any system) favors flow to an area of lower pressure. Resistance to blood flow comes in the form of pressure in vessels, which is partly determined by smooth muscle tone and length of the blood vessels. In general, the key principle governing blood flow can be summarized as "the greater the resistance, the lower the flow." Resistance to flow can also be increased if the blood is more viscous, such as occurs with polycythemia. Blood flow can also be affected by the shape and internal smoothness of the blood vessels.

SYSTEMIC CONTROL OF BLOOD PRESSURE

In addition to acting through the sympathetic nervous system, which can increase the blood pressure by increasing vascular resistance, the body can control blood pressure through other chemical pathways. The kidneys autoregulate their blood flow so that pressures at the glomerulus are sufficiently high to maintain filtration. If the kidneys detect decreased blood flow, they also release renin. Renin leads to a production of angiotensin I, which is later converted to angiotensin II. Angiotensin II increases systemic vascular resistance by causing vasoconstriction. It also stimulates the release of aldosterone from the adrenal glands. Aldosterone, a mineralocorticoid, causes sodium—and therefore water—retention. If the cause of renal blood flow decrease is blood loss, these compensatory mechanisms are helpful. Conversely, if low cardiac output is caused by pump failure, then these mechanisms actually work against cardiac function by increasing afterload and water retention. Angiotensin-converting enzyme (ACE) inhibitors block this pathway, and guidelines now recommend their use both for heart failure and following myocardial infarction.

The body's water levels are regulated by antidiuretic hormone (ADH), a chemical released from the posterior pituitary gland. When diuresis slows, the amount of free body water increases. This effect then reduces the concentration (osmolality) of dissolved substances in the body.

Natriuretic peptides are released by parts of the body in response to plasma volume changes. Atrial natriuretic protein (ANP) is released by atrial monocytes if they detect increased pressure in the right atrium. ANP inhibits ADH, thereby causing a loss of body water, and ultimately reducing the pressure in the right atrium. Brain natriuretic protein (BNP) was first discovered in the brain, but is also released from the heart cells; the level of this protein can reflect overall ventricular function (McCance & Huether, 2006).

DISORDERS OF MAJOR BLOOD VESSELS

Sometimes, as a result of longstanding pressure inside arteries or because of turbulent flow caused by irregularities in the internal shape of the artery, weaknesses may develop in the arterial wall. An aneurysm is a widening in an artery that can completely surround the artery or that can consist of an outpouching at one part of the circumference of the artery. When aneurysms rupture, they cause rapid blood loss from the artery into surrounding tissues and a reduction in blood flow to areas normally supplied by the artery.

The aorta is prone to aneurysm development because it sustains the highest pressures in the vascular system. When an aneurysm involves all three levels of the arterial wall, it is termed a "true aneurysm." This type of aneurysm typically involves the entire circumference of the

vessel. Other aneurysms form between the layers of the artery, particularly following vascular surgery. In this case, blood leaks through the endothelial and tunica media layers and collects under the adventitia.

Aneurysms are usually undetectable until they threaten to rupture or actually do rupture. Symptoms depend on the location of the aneurysm. A widening aneurysm can result in decreased blood flow to small arteries in the area. A ruptured aneurysm causes pain, which can often be referred, meaning the pain is perceived in an area of the body different from where the actual injury is located. Thoracic aneurysms can cause dyspnea or dysphagia due to pressure on the esophagus and lung tissue. An abdominal aortic aneurysm can result in ischemia to tissues normally supplied by blood from the area below the aneurysm.

Diagnosis of aneurysms may be made through ultrasonography and by using imaging technologies such as computed tomography (CT) or magnetic resonance imaging (MRI). The goals of treatment are to reduce blood loss by reducing blood pressure until surgical repair can be accomplished. Asymptomatic aneurysms are sometimes detected on chest radiograph or by abdominal palpation of an aortic aneurysm.

The decision to make a surgical repair depends on the relative risk of the repair itself compared with the risk of the aneurysm's rupture. If, for example, the renal arteries are compromised by the location of the aneurysm, then the intravascular repair may not be possible. The age of the patient and the size of the aneurysm are factored into this decision. Intravascular approaches to supporting the integrity of the artery have been developed that allow for much quicker recovery by patients. The location of the aneurysm is key to understanding the symptoms produced by the arterial defect.

SUMMARY

By understanding the anatomy and physiology of the cardiovascular system, the nurse is able to make reasoned responses to patient problems. Concepts such as cardiac output and electrophysiology will be daily concerns of the critical care nurse. Accurate assessment of cardiovascular function and early detection of problems are essential to providing high-quality care. Cardiovascular conditions remain the leading cause of death in the United States. Nurses caring for acute and critically ill patients will be needed who are expert in the care of patients with these conditions.

SELF-ASSESSMENT QUESTIONS

1. You have a patient who is scheduled to have a coronary artery bypass graft. On physical exam, you note that the PMI is located at the left axilla. This assessment finding more than likely indicates
   a. cardiac tamponade.
   b. axis deviation.
   c. tension pneumothorax.
   d. hypertrophy.
2. Blood flowing through the pulmonic valve is leaving the
   a. right ventricle.
   b. left ventricle.
   c. right atrium.
   d. left atrium.
3. The Frank-Starling law illustrates the relationship between
   a. vasoactive agent administration and hemodynamic response.
   b. sympathetic response and parasympathetic response.
   c. fluid status and cardiac function.
   d. acetylcholine and degree of bradycardia.

4. In the presence of increased afterload, which agents are useful in lowering resistance to cardiac ejection?
   a. Vasodilator agents
   b. Anticholinergic agents
   c. Vasoconstrictor agents
   d. Beta blockers
5. Decreased left ventricular preload can occur in the setting of
   a. cardiac tamponade.
   b. hypovolemia.
   c. cardiogenic shock.
   d. hypoxemia.
6. Angiotensin II increases systemic vascular resistance by causing
   a. vasoconstriction.
   b. parasympathetic stimulation.
   c. aldosterone release.
   d. production of renin.
7. Common signs of thoracic aneurysms include
   a. hypertension and cough.
   b. dyspnea and dysphagia.
   c. pleuritic chest pain and shortness of breath.
   d. bradycardia and jugular venous distention.
8. Atrial natriuretic protein is released when pressure is detected in the
   a. left atrium.
   b. left ventricle.
   c. right atrium.
   d. right ventricle.
9. Which neurotransmitter is released by the sympathetic nerve fibers, resulting in a profound effect on cardiac contractility and on vascular resistance?
   a. Angiotensin
   b. Acetylcholine
   c. Norepinephrine
   d. Dobutamine
10. Administration of a beta blocker will result in which of the following outcomes?
    a. Increased cardiac output
    b. Vasoconstriction
    c. Decreased contractility
    d. Compensation by dopaminergic receptors

Answers to Self-Assessment Questions

1. d
2. a
3. c
4. a
5. b
6. a
7. b
8. c
9. c
10. c

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Woods, S. L., Froelicher, E. S., Motzer, S. A., & Bridges, E. (Eds.). (2004). *Cardiac nursing* (5th ed.). Philadelphia: Lippincott Williams & Wilkins.

WEB RESOURCES

St. Jude Medical presents an informational video about the anatomy of the human heart and how it pumps blood throughout your body: www.heartlibrary.com/heart-library-heart-anatomy.aspx?flashmov=heart-anatomy&currPage=HD

Heart Anatomy, Interior View, is an interactive photograph that allows the learner to point to any structure for highlighted identification on the picture. GateWay Community College, Phoenix, AZ: www.gwc.maricopa.edu/class/bio202/cyberheart/hartint0.htm

Heart Anatomy, Posterior View, is an interactive photograph that allows the learner to point to any structure for highlighted identification on the picture. GateWay Community College, Phoenix, AZ: www.gwc.maricopa.edu/class/bio202/cyberheart/hartbak.htm

Assessment with a diaphragm of the stethoscope: www.youtube.com/watch?v=T2Hs5fqcflg

Assessment with a bell of the stethoscope: www.youtube.com/watch?v=vSg-AU1EUaM

The Auscultation Assistant provides heart sounds, heart murmurs, and breath sounds to help medical students and others improve their physical diagnosis skills: www.med.ucla.edu/wilkes/intro.html

Basic cardiac assessment: www.youtube.com/watch?v=dp5m2tXHDmA

Second Life heart murmur simulator: www.youtube.com/watch?v=xJY2Iwbzop4

Chapter 3

Indications for Cardiac Surgery

Kristine J. Peterson

INTRODUCTION

The heart has fascinated human beings for centuries as the seat of life and emotions. Although the idea of operating on the heart is not new, it was only in the mid-twentieth century that such surgery became practical. With the development of cardiopulmonary bypass came the ability to create a bloodless surgical field and a motionless target for surgical therapies. Since that time, multiple advances in surgical techniques, patient management, technology, and pharmacotherapy have resulted in the emergence of cardiac surgery as a dynamic and very successful medical and nursing specialty.

The ability to operate on the heart saves thousands of lives each year. These surgeries present exciting and varied challenges for the critical care nurse and interdisciplinary team. The purpose of this chapter is to review the types of cardiac surgery and their indications.

SURGERY FOR ISCHEMIC HEART DISEASE

An estimated 79.4 million adult Americans have some form of cardiovascular disease, with approximately 15,800,000 having coronary heart disease (CHD), according to the American Heart Association (AHA, 2007). Once significant coronary artery stenosis is established, the three major treatment strategies used to prevent further ischemic damage are medical therapy, percutaneous revascularization, and surgical revascularization. Revascularization is the process of restoring blood flow and oxygen delivery to the myocardium. Its dual purposes are to alleviate angina symptoms and to prolong life. Percutaneous transluminal coronary angioplasty (PTCA) uses an arterial catheter and various mechanical means to increase the diameter of diseased coronary arteries, thereby improving blood flow. Surgical revascularization, also known as coronary artery bypass grafting (CABG), uses arterial or venous vessels to create a new pathway for blood to reach the coronary arteries, thus "bypassing" the stenosis. In 2004, 427,000 coronary artery bypass graft procedures were performed on 249,000 patients in the United States (AHA, 2007). With advances in percutaneous strategies such as drug-eluting stents (DES), the number of percutaneous revascularizations has increased while the number of surgical revascularizations has declined (AHA, 2007). Decisions regarding whether coronary revascularization is indicated for an individual patient now include which type of revascularization is indicated.

Medical Therapy versus Surgical Revascularization

Several trials comparing medical therapy with CABG were conducted between 1972 and 1984 (Hueb et al., 1995; Kloster et al, 1979;

Varnauskas, 1988; Yusuf et al., 1994). High-risk patients were defined based on the severity of their angina or ischemia, the number of diseased vessels, and the presence of left ventricular (LV) dysfunction (Brown, Sundt, & Gersh, 2008). The studies consistently indicated the greatest benefits of CABG over medical therapy in those patients at highest risk. CABG has not been shown to be of more benefit than medical therapy for patients with single-vessel disease (Brown et al., 2008). Yusuf et al. (1994), in a meta-analysis, demonstrated significantly higher survival at 5, 7, and 10 years after CABG for patients at high and moderate risk, but not for low-risk patients.

Percutaneous versus Surgical Revascularization

A large number of clinical trials have compared short- and long-term outcomes from percutaneous coronary intervention (PCI) and surgical revascularization (Goy et al., 1999; Goy et al., 2000; Hannan et al., 2005; Hoffman et al., 2003; Hueb et al., 1995; Serruys et al., 2001). In the Arterial Revascularization Therapy Study (ARTS—the largest of these trials), 1- and 5-year follow-up data indicated no differences in major adverse cardiac and cerebrovascular events (MACCE) between CABG and stent placement (Serruys et al., 2001). CABG resulted in fewer revascularizations, but PCI provided substantial cost savings (Gruberg, 2005). Other studies have found that CABG resulted in reduced 5-year mortality, less angina, and fewer revascularization procedures than PCI (Hoffman et al., 2003). Adding stents to PCI reduced revascularizations, but the need for such follow-up procedures remained significantly lower in CABG patients (Hannon et al., 2005; Hoffman et al., 2003). Mercado et al. (2005) demonstrated that PCI and CABG resulted in no differences in 1-year death, myocardial infarction (MI), and stroke rates, but higher revascularization rates than found with PCI. It should be noted that these studies were all conducted prior to 2003, when DES were first introduced.

The ARTS II registry compared outcomes with a sirolimus-eluting stent and PCI to those in the PCI and CABG arms of the ARTS I study (Gruberg, 2005). ARTS II registry patients had a higher incidence of diabetes, hypertension, and hypercholesterolemia than did members of the ARTS I group, but included fewer smokers than in the original study. Rates of stable versus unstable angina were similar in the two groups, although the patients enrolled in ARTS II had more complex lesions than those in the first registry. MACCE-free survival at one year was not significantly different between the two groups, probably due to the higher revascularization rate in ARTS II patients.

In a meta-analysis comparing DES with bare metal stents, Babapulle, Joseph, Belisle, Brophy, and Eisenberg (2004) found no differences in survival or MI. DES placement resulted in fewer restenoses and major cardiac events. DES technology continues to evolve, and DES are implanted frequently. The impact of these stents on long-term outcomes compared with CABG procedures remains to be seen (Brown et al., 2008).

INDICATIONS FOR CORONARY ARTERY BYPASS GRAFTING

The American College of Cardiology (ACC)/American Heart Association (AHA) Task Force on Practice Guidelines has established recommendations for CABG in certain subsets of patients. These recommendations reflect the severity of angina, the presence of ST-segment elevation MI (STEMI), the presence of unstable angina or non-ST-segment elevation MI (NSTEMI), left ventricular function, presence or absence of life-threatening ventricular arrhythmias, previous PCI, or previous CABG (Eagle et al., 2004). Table 3–1 lists the level of recommendations for each patient population.

Table 3–1 ACC/AHA Indications for CABG

Asymptomatic or Mild Angina

Class I: Evidence and/or general agreement that the intervention is effective.

1. Left main coronary artery stenosis
2. Proximal LAD and proximal left circumflex stenosis
3. Triple-vessel disease
4. Proximal LAD and one- or two-vessel disease with LVEF less than 50% or a large amount of myocardium at risk on noninvasive studies
5. One- or two-vessel disease not involving proximal LAD with LVEF less than 50% or a large amount of myocardium at risk on noninvasive studies

Class IIa: Evidence/opinion is in favor of efficacy.

1. Proximal LAD stenosis and one- or two-vessel disease

Class IIb: Efficacy is less well established by evidence/opinion.

1. One- or two-vessel disease not involving proximal LAD

Stable Angina

Class I: Evidence and/or general agreement that the intervention is effective.

1. Left main stenosis
2. Proximal LAD and proximal left circumflex
3. Triple-vessel disease
4. Two-vessel disease with proximal LAD stenosis and LVEF less than 50% or demonstrable ischemia
5. One- or two-vessel disease without proximal LAD stenosis but with a large amount of myocardium at risk and high-risk criteria on noninvasive testing
6. Disabling angina refractory to medical therapy
7. Proximal LAD stenosis with one-vessel disease and meets high-risk criteria or large amount of myocardium at risk
8. One- or two-vessel disease without proximal LAD stenosis but meets high-risk criteria or large amount of myocardium at risk

Class IIa: Evidence/opinion is in favor of efficacy.

1. Proximal LAD stenosis with one-vessel disease
2. One- or two-vessel disease without proximal LAD stenosis, but with a moderate amount of myocardium at risk and demonstrable ischemia

Unstable Angina/NSTEMI

Class I: Evidence and/or general agreement that the intervention is effective.

1. Left main stenosis
2. Proximal LAD and proximal left circumflex stenosis
3. Ongoing ischemia not responsive to maximal nonsurgical therapy
4. One- or two-vessel disease without proximal LAD stenosis when PCI not possible and meets high-risk criteria on noninvasive testing

Class IIa: Evidence/opinion is in favor of efficacy.

1. Proximal LAD stenosis with one- or two-vessel disease

Class IIb: Efficacy is less well established by evidence/opinion.

1. One- or two-vessel disease without proximal LAD stenosis when PCI not possible

STEMI

Class I: Evidence and/or general agreement that the intervention is effective.

1. Failed PCI with persistent pain or hemodynamic instability and anatomically feasible
2. Persistent or recurrent ischemia refractory to medical treatment with acceptable anatomy who have a significant amount of myocardium at risk and not a candidate for PCI

continues

Table 3–1 ACC/AHA Indications for CABG (continued)

3. Requires surgical repair of post-infarction ventricular septal rupture or mitral valve insufficiency
4. Cardiogenic shock in patients younger than 75 years of age who have ST-segment elevation, left bundle branch block (LBBB), or a posterior MI within 18 hours of onset
5. Life-threatening ventricular arrhythmias in the presence of 50% or more left main stenosis or triple-vessel disease

Class IIa: Evidence/opinion is in favor of efficacy.

1. Primary reperfusion in patients who have failed fibrinolytics or PCI and are in the early stages (6–12 hours) of an evolving STEMI
2. Mortality for CABG is elevated in the first 3–7 days after STEMI/NSTEMI; after 7 days, the criteria for revascularization in previous sections apply

Presence of Poor LV Function

Class I: Evidence and/or general agreement that the intervention is effective.

1. Left main stenosis
2. Proximal LAD and proximal left circumflex stenosis
3. Proximal LAD stenosis and two- to three-vessel disease

Class IIa: Evidence/opinion is in favor of efficacy.

1. Significant amount of viable myocardium and noncontractile myocardium

Presence of Life-Threatening Ventricular Arrhythmias

Class I: Evidence and/or general agreement that the intervention is effective.

1. Left main disease
2. Three-vessel disease
3. Bypassable one- or two-vessel disease if resuscitated cardiac arrest or sustained ventricular tachycardia
4. Proximal LAD disease and one- or two-vessel disease if resuscitated cardiac arrest or sustained ventricular tachycardia

Class IIa: Evidence/opinion is in favor of efficacy.

1. Bypassable one- or two-vessel disease
2. Proximal LAD disease and one- or two-vessel disease

Failed PCI

Class I: Evidence and/or general agreement that the intervention is effective.

1. Ongoing ischemia with a significant amount of myocardium at risk
2. Hemodynamic instability

Class IIa: Evidence/opinion is in favor of efficacy.

1. Foreign body in critical position
2. Hemodynamic instability with coagulopathy and no previous sternotomy

Class IIb: Efficacy is less well established by evidence/opinion.

1. Hemodynamic instability with coagulopathy and previous sternotomy

Previous CABG

Class I: Evidence and/or general agreement that the intervention is effective.

1. Disabling angina refractory to medical therapy
2. Nonpatent previous bypass grafts, but with Class I indications for native CABG

Class IIa: Evidence/opinion is in favor of efficacy.

1. Large amount of myocardium at risk
2. Vein grafts supplying LAD or large amount of myocardium are greater than 50% stenosed

LAD = left anterior descending coronary artery; LV = left ventricle; LVEF = left ventricular ejection fraction; LBBB = left bundle branch block; STEMI = ST-segment elevation myocardial infarction; PCI = percutaneous coronary intervention; NSTEMI = non-ST-segment elevation myocardial infarction.

Source: Brown, Sundt, & Gersh, 2008. Used with permission.

The recommendations are categorized into three classes: I, II, and III. Class I indicates that evidence and/or general agreement exists that the intervention is effective. Class II indicates that conflicting evidence and/or a divergence of opinion exists about the efficacy of the intervention. Class II is further divided into Class IIa and Class IIb: Class IIa indicates that evidence/opinion is in favor of the intervention's efficacy, whereas Class IIb interventions have less efficacy as established by evidence/opinion. For Class III interventions, evidence and/or general opinion suggests that the intervention is not effective.

The strength of the level of evidence for specific interventions is also identified according to the type or presence of research. For example, Level of Evidence A indicates that findings from multiple randomized clinical trials or meta-analyses supported use of an intervention. Level of Evidence B indicates that a single randomized trial or a series of nonrandomized trials supported an intervention. Level of Evidence C is assigned to those interventions supported by consensus opinion of experts, case studies, or standard of care (Eagle et al., 2004).

CABG in Diabetics

The connection between diabetes mellitus and CHD is well established. Diabetes produces a prothrombotic, inflammatory, and proliferative state in the vascular endothelium and carries a higher risk for restenosis. Individuals with diabetes tend to have more diffuse coronary disease—a state that favors use of a procedure that can produce complete revascularization. Regardless of the revascularization procedure used, diabetics have a higher risk of adverse outcomes (BARI Investigators, 2000). For persons with diabetes, there is a trend toward lower mortality and fewer revascularizations with CABG than with PCI. As yet, no studies have compared the outcomes from PCI with DES with the outcomes from CABG in patients with diabetes. Currently, these patients should be evaluated with revascularization standard criteria (Brown et al., 2008).

CABG in Patients with Concomitant Carotid Disease

Perioperative stroke risk ranges from 2% to 12% in patients with unilateral carotid occlusion to 5% in patients with significant bilateral stenosis (Naylor, Cuffe, Rothwell, Loftus, & Bell, 2003). Accordingly, carotid endarterectomy is recommended before or concurrently with CABG in patients with symptomatic carotid stenosis and for patients with 80% unilateral or bilateral carotid stenosis (Eagle et al., 2004).

Minimally Invasive Myocardial Revascularization

Off-pump CABG (OPCAB) has resulted in patency rates and survival rates equivalent to those seen with traditional CABG (Puskas et al., 2003; Sharony et al., 2004). In addition, emerging evidence suggests that neurological-related risks are lower when off-pump techniques are used to treat patients (Stamou et al., 2002). Currently, OPCAB is used chiefly for low-risk patients with single-vessel disease (Brown et al., 2008). New surgical techniques such as total endoscopic CABG (TECAB) will continue to change the landscape of CABG. OPCAB is discussed in more detail in Chapter 7.

TRANSMYOCARDIAL LASER REVASCULARIZATION

Despite advances in prevention, medical therapy, PCI, and CABG, CHD remains the single greatest cause of death for Americans (AHA, 2007). A substantial number of patients with diffuse CHD have refractory angina despite maximal medical and interventional therapy (Horvath & Zhou, 2008). Severe, diffuse CHD

is often not amenable to complete revascularization, leaving myocardium at risk even after such a procedure is attempted. Incomplete revascularization is a predictor of adverse events (Horvath & Zhou, 2008).

Options to treat refractory CHD include angiogenesis, genetic therapies, and transmyocardial revascularization. These strategies involve various means of creating new pathways for blood to reach the myocardium. Transmyocardial laser revascularization (TMR) is a procedure whereby transmyocardial channels are created from the epicardium into the ventricle via a laser. The new channels then allow blood from the ventricle to reach the myocardium directly. Results of trials comparing TMR with medical therapy have been mixed, with studies showing inconsistent sustained symptom relief with the former technique (Horvath et al., 2001).

Early clinical trials of various techniques in the 1980s demonstrated limited benefit, with patients experiencing only short-lived relief of angina and little improvement in exercise tolerance (Horvath & Zhou, 2008; Morrow, Gersh, & Braunwald, 2005). Advances in laser technology improved these results (Frazier, March, & Horvath, 1999). Since that time, more than 25,000 patients have been treated worldwide (Horvath & Zhou, 2008). Two trials failed to show any benefit from this approach (Morrow et al., 2005; Saririan & Eisenberg, 2003). Several follow-up reports of patients at 43 months and 5 years, however, demonstrated significantly improved angina symptoms and reduced hospitalizations; some patients were actually angina free at 5 years (Aaberge et al., 2002; Horvath et al., 2001).

Perioperative mortality has improved from the earliest studies to approximately 1% to 5% (Frazier et al., 1999). Four-year follow-up data indicate a nonsignificant increase in survival for TMR compared with an approach relying on continued medical therapy (Aaberge et al., 2002). The Society of Thoracic Surgeons' (STS) National Cardiac Database data from 1998 to 2001 indicate perioperative mortality of 6.4% and 4.2% when TMR is performed with CABG (Peterson et al., 2003).

There remains divergence of opinion as to the role of TMR (Brown et al., 2008; Horvath & Zhou, 2008). Nevertheless, the STS has issued guidelines for the use of TMR in stable patients with medically refractory angina (see Table 3–2).

SURGERY FOR VALVE DISEASE

Valve disease can occur due to congenital or acquired factors. Decisions regarding medical or surgical management (repair or replacement) are made by weighing the risk and benefits of each treatment modality. Valve surgery is discussed in detail in Chapter 5.

SURGICAL MANAGEMENT OF ARRHYTHMIAS

Durrer and colleagues (1967) first reported the successful initiation and termination of a tachycardia in 1967, when they induced and successfully terminated atrioventricular reentrant tachycardia (AVRT). Advancements in understanding of arrhythmia initiation and propagation as well as surgical techniques created a role for surgical ablation for tachyarrhythmias (Eckart & Epstein, 2008). The need for open heart surgery limited this role and spurred the development of catheter ablation techniques. Many arrhythmias—including atrioventricular nodal reentrant tachycardia (AVNRT), AVRT, atrial tachycardia, atrial flutter, atrial fibrillation (AF), and ventricular tachycardia (VT)—have been mapped and ablated using catheter techniques with varying degrees of success (Eckart & Epstein, 2008). For persistent or recurring dysrhythmias, surgical management remains an option.

Table 3–2 Indications for Transmyocardial Laser Revascularization as Sole Therapy

1. LVEF greater than 30% with CCS Class III or IV angina refractory to medical therapy, CABG, or PCI, reversible ischemia of LV free wall and CHD corresponding to the regions of ischemia.
2. Criteria in number 1 with either LVEF less than 30%, angina refractory to medical therapy or surgical revascularization (e.g., PTCA, stent), history of MI, cardiogenic shock and heart failure, uncontrolled ventricular dysrhythmia, or an area of ischemia that will not respond to CABG.
3. End-stage coronary artery disease for which medical therapy or surgical revascularization is no longer feasible and patient does not have indications for cardiac transplantation.
4. Refractory severe intractable angina. NYHA Class III or IV angina with symptoms refractory to medical therapy at endurable or highest safe dose, viable myocardium not amenable to surgical revascularization (e.g., PTCA, stent, coronary atherectomy, CABG), and the patient has made maximal attempts to stabilize acute conditions (e.g., severe ventricular dysrhythmia, decompensated heart failure, or acute MI).

CABG = coronary artery bypass graft; CCS = Canadian Cardiovascular Society; CHD = coronary heart disease; LVEF = left ventricular ejection fraction; MI = myocardial infarction; NYHA = New York Heart Association; PCI = percutaneous coronary intervention.

CCS Class III = Apparent restriction of ordinary activity.

CCS Class IV = Angina at rest; patient cannot perform any physical activity without discomfort.

NYHA Class III = Patient is comfortable at rest; less than ordinary activity causes fatigue, palpitation, dyspnea, or angina.

NYHA Class IV = Inability to carry on any physical activity without discomfort. Symptoms of heart failure or angina may be present even at rest. Decreased comfort with any physical activity.

Sources: Aetna, 2008; Bridges et al., 2004; Campeau, 1976; Eckstein et al., 2001.

Atrial Fibrillation

The most important goals of therapy for AF are symptom alleviation and reducing the risk of stroke (Cheng & Arnsdorf, 2007). Much research attention has been given to determining the efficacy of rhythm control versus rate control in AF. Currently, pharmacological rate control with anticoagulation is the most common treatment strategy. Rate control may be advantageous in that a less aggressive dosing regimen is associated with a lower risk of side effects and there are no proven survival benefits for a rhythm control strategy (Voeller, Schuessler, & Damiano, 2008; Wyse et al., 2002). However, pharmacological therapy is limited in this indication by its inconsistent efficacy and side effects. For this reason, strong interest remains in nonpharmacological approaches to AF, including catheter ablation, surgery, pacing, and implantable atrial defibrillators.

The most important indication for surgical treatment of AF is intolerance to the dysrhythmia in patients who have failed pharmacological therapy. Other indications include paroxysmal atrial flutter or AF unresponsive to medical therapy, tachycardia-induced cardiomyopathy, patients who are intolerant of antiarrhythmic agents, patients who have a contraindication to long-term anticoagulation, and patients with chronic AF who have a stroke despite adequate anticoagulation (Voeller et al., 2008).

Surgical techniques have included left atrial isolation, the Corridor procedure, pulmonary vein isolation, atrial transection, and the Maze and Cox/Maze procedures (Cheng & Arnsdorf, 2007; Voeller et al., 2008). Procedures that

involve both atria have been most effective in controlling AF (Barnett & Ad, 2006). The majority of patients have a surgical procedure for AF in conjunction with another cardiac surgery such as a valve surgery (Cheng & Arnsdorf, 2007). The most common surgical approaches to AF are the Cox/Maze III procedure and a modification of this technique that uses radiofrequency (RF) ablation instead of surgical incisions (Barnett & Ad, 2006; Cheng & Arnsdorf, 2007; Chiappini, et al., 2004; Doty et al., 2007; Gaynor et al., 2005; Voeller et al., 2008). The Maze procedure interrupts the reentrant pathways required for AF using surgical incisions. The original procedure has been modified several times to the current version Cox/Maze III procedure. In addition, the Cox/Maze IV procedure (Voeller et al., 2008), which involves the use of RF ablation, is now in use. A number of studies have attempted to determine whether RF or surgical incision ablation is more effective; to date, the results have been mixed.

In a review of 276 patients who underwent the Cox/Maze I, II, III, and IV procedures, no difference in survival was found between the procedures at a mean follow-up of 5.8 years (Gaynor et al., 2005). There was a significantly greater freedom from AF recurrence with the Cox/Maze III and IV procedures compared with the Cox/Maze I and II procedures. The results indicated that the Cox/Maze IV procedure further increased freedom from AF at 6 months; however, the follow-up time was too short to compare this procedure with other versions (Gaynor et al., 2005). Another study indicated that the RF procedure results in high efficacy and shortened operative time (Mokadam et al., 2004). A number of studies have reported that the Cox/Maze IV RF ablation procedure is at least as safe and effective as the Cox/Maze III procedure (Chiappini et al., 2004; Topkara et al., 2006).

The tissue around the pulmonary veins can produce ectopic beats that trigger AF. In one study, pulmonary vein isolation reliably produced a return to sinus rhythm (de Lima et al., 2004). Other studies have not confirmed these results, however, and lone pulmonary vein isolation is not currently recommended (Doty et al., 2007; Eckart & Epstein, 2008). The Cox/Maze III procedure remains the standard surgical therapy for AF, although energy ablation is emerging as an effective alternative.

Surgical Management of Ventricular Tachycardia

Ventricular arrhythmias—particularly sustained VT—are not uncommon in patients who survive an acute myocardial infarction (AMI); the incidence in this patient population is approximately 3% (Pinto & Josephson, 2007). Surgery to eradicate ventricular tachyarrhythmias began in the 1970s with a technique called endocardial resection (Pagé, 2004). Over time, the initial enthusiasm for the development of surgical therapies for VT waned as medical therapies such as mapping, catheter ablation, and implantable devices advanced. The success of various surgical procedures in preventing VT is counterbalanced by the myocardial damage and high intraoperative mortality associated with these procedures (Pagé, 2004; Pinto & Josephson, 2007). Any surgical therapy for VT seems to be most successful if the origin of the arrhythmia is in irreversibly damaged myocardium, which most commonly occurs after AMI. When patients have diffuse myocardial damage, such as cardiomyopathy, surgical therapies are not successful (Pagé, 2004).

The development of multiple-site endocardial and epicardial mapping systems has contributed greatly to the efficacy of surgical management of VT. Localization of the site of earliest activation during VT on both the epicardial and endocardial surfaces allows for precise targeting of therapies. Surgical man-

agement approaches to VT now include mapping-guided energy ablation, subendocardial resection, and ventricular endoaneurysmorrhaphy. Subendocardial resection (SER) involves surgical removal of scar tissue, portions of an aneurysm, or other sites generating abnormal electrograms. Endoaneurysmorrhaphy involves incising the aneurysm and using a Dacron patch to control the shape and size of the ventricle (Pagé, 2004). These techniques have achieved mortality and sudden cardiac death rates that are comparable to those achieved with implantable cardioverter defibrillators (ICDs) (Pagé, 2004).

Indications for surgery for VT are ill defined and largely dependent on the surgeon's judgment. Consensus does exist that VT caused by ischemic heart disease is most amenable to surgical therapy. Other factors that should be considered when deciding on the best course of action include the patient's response to antiarrhythmic agents, history of cardiac arrest, inducibility of the rhythm, need for aneurysm resection, LV end-diastolic volume, NYHA functional class, and presence of mitral valve regurgitation (Pagé, 2004).

SURGICAL THERAPIES FOR HEART FAILURE

Following a myocardial infarction, both the infarcted and non-infarcted areas undergo pathological changes such as thinning and fibrous replacement, which alter the size and shape of the ventricle and ultimately lead to heart failure. Medical therapy can improve symptoms and increase lifespan, but it cannot cure the condition. Mechanical support, such as a ventricular assist device (VAD), has been used as a bridge to transplant and occasionally as a bridge to recovery, but is not generally regarded as a feasible long-term therapy for heart failure. The treatment of choice for end-stage heart failure is cardiac transplantation, although the shortage of donor hearts renders this option available to only a few patients. Accordingly, other surgical options have been developed. Cardiac transplantation is discussed in detail in Chapter 19.

Coronary Revascularization as Treatment for Heart Failure

Coronary revascularization via CABG or PCI is known to improve ejection fraction (EF) when viable myocardium exists. Revascularization can be used even for patients with an EF as low as 10%. CABG for ischemic cardiomyopathy results in improved quality of life and fewer hospitalizations compared with medical therapy alone, and in survival rates superior to transplant for the first 2 years. Revascularization for heart failure is indicated for those patients with documented viable myocardium and no evidence of right ventricular dysfunction. Revascularization is not a good option for heart failure patients with right ventricular dysfunction, signs of right-sided heart failure, or pulmonary hypertension (Spoor & Bolling, 2008).

MITRAL VALVE REPAIR IN DILATED CARDIOMYOPATHY

Ventricular failure and dilatation will eventually result in mitral regurgitation (MR). MR is often a pre-terminal event and is associated with a survival time of 6–24 months (Spoor & Bolling, 2008). Early attempts at mitral valve replacement in these cases produced high mortality rates, and the practice was discouraged. Mitral valve repair is discussed in more detail in Chapter 5.

VENTRICULAR RECONSTRUCTION TECHNIQUES

Ventricular reconstruction techniques are based on the principle that the ventricular wall tension is proportional to the left ventricular radius and pressure and inversely

proportional to the wall thickness (Law of Laplace) (Spoor & Bolling, 2008). By changing the size and shape of the ventricle, these techniques seek to reduce wall tension and improve LV function. Specifically, surgical reconstruction techniques attempt to remove or isolate dysfunctional myocardium, reduce the diameter of the ventricle, and restore a more elliptical ventricular shape (Fang, 2007). Additional goals are to relieve ischemia by revascularization if possible, and to further reduce ventricular size and volume via mitral valve repair (Menicanti & Di Donato, 2002).

ENDOANEURYSMORRHAPHY

One of the earliest approaches to treating post-infarction aneurysms sought to excise the aneurysmal area and reapproximate the wall edges, a procedure known as endoaneurysmorrhaphy. Although this approach attempts to restore more normal ventricular geography, study data indicate that it does not improve LV function (Fang, 2007).

PARTIAL LEFT VENTRICULECTOMY

Batista and colleagues (1996) described a procedure to restore the proper mass-to-diameter ratio for the left ventricle. To do so, a section of the left ventricular wall from the apex to the mitral annulus was removed, and the edges were reapproximated. The mitral valve was repaired or replaced as necessary. Improvements in signs of heart failure and EF have been achieved in more than 150 patients who have undergone a left ventriculectomy. The procedure produces a reduction in LV volume and, therefore, a reduction in LV wall stress (Fang, 2007). In a series of North American patients who underwent this procedure, researchers identified a short-term improvement in EF, reduced heart size, and symptom improvement, but a high incidence of long-term failure (McCarthy et al., 2004). Other centers have reported similar results (Batista et al., 1997; Franco-Cereceda et al., 2001; Starling et al., 2000).

Given that mitral valve repair has been shown to improve heart failure, it is unclear whether the results of the Batista procedure are attributable to the surgical reduction in ventricular size or to the effects of the mitral valve repair. Other surgical procedures have achieved results superior to the partial left ventriculectomy. The Batista procedure is no longer preformed in most of North America, though it is sometimes used in other areas where cardiac transplantation is less available (Spoor & Bolling, 2008).

GEOMETRIC VENTRICULAR RECONSTRUCTION

The Dor procedure, also known as endoventricular circular patch plasty repair, is a procedure whereby the left ventricle is reconstructed using a purse-string suture to isolate nonfunctional segments of myocardium (rather than excising them) and a circular patch to control the shape of the ventricle. The Dor procedure is usually performed concomitantly with a CABG (Di Donato et al., 2001). The first case series report demonstrated a significant improvement in LVEF, which was maintained at 1-year follow-up. In addition, in those patients for whom data were available, 92% had improved NYHA functional class, and 91% of patients with ventricular tachycardia were free of VT at 1-year follow-up (Dor, Saab, Coste, Sabatier, & Montiglio, 1998). Mickleborough, Merchant, Inanov, Rao, and Carson (2004) reported that a similar left ventricular reconstruction procedure improved patients' LVEF and NYHA functional class and had low perioperative mortality.

Another modification of the original Dor concept—the surgical anterior ventricular endocardial restoration (SAVER) procedure—

has produced similar results. Studies have shown that SAVER leads to a significant reduction of left ventricular volume and a significant increase in EF as well as significant reductions in hospitalizations for heart failure. Survival at 18 months was 89%. A majority of patients had concomitant CABG, and 23% had mitral valve replacement at the time of the procedure (Athanasuleas et al., 2004; Hernandez et al., 2006). Studies have reported consistently favorable results with ventricular reconstruction (Athanasuleas et al., 2004; Mickleborough et al., 2004).

DYNAMIC CARDIOMYOPLASTY

Dynamic cardiomyoplasty (DCMP) is an innovative technique whereby the latissimus dorsi muscle is wrapped around the heart. An implanted stimulator is then used to stimulate the muscle to contract in synchrony with ventricular contraction. A clinical trial of DCMP was terminated early due to limited enrollment and borderline clinical improvement (Fang, 2007). Dynamic cardiomyoplasty is rarely performed in the United States, but it remains in use in other areas.

The concept of DCMP is spurring research into other techniques that may produce favorable results by "girdling" the ventricle. For example, the ACORN Cardiac Support Device (ACSD; Acorn Medical, Minneapolis, Minnesota) is a polyester mesh fabric that is wrapped snugly around the ventricles. This device provides passive support to the ventricles, which should reduce wall stress and prevent further remodeling. Another device used to decrease wall stress is the Myocor Myosplint (Myocor Medical, St. Paul, Minnesota). With this device, which is currently in clinical trials, transventricular tension bands are placed through the right and left ventricular walls. The tension on these bands can be tailored to the patient by tightening them individually to achieve a 20% reduction in wall stress.

Yet another novel device for heart failure is the mitral valve annuloplasty ring. This new three-dimensional ring, which improves mitral valve function and left ventricular shape (Maisano et al., 2005), is approved for clinical use in the United States. The mitral valve annuloplasty ring is discussed in more detail in Chapter 5.

When combined with optimal medical therapy, revascularization via CABG, geometric mitral reconstruction, and left ventricular reconstruction have achieved results comparable to transplantation. Spoor and Bolling (2008) list these interventions as first-line surgical options for heart failure. Ventricular reconstruction is indicated for patients with anteroseptal MI with a dilated left ventricle, EF less than 20%, left ventricular dyskinesis, and symptoms of angina, heart failure, or arrhythmias, or for asymptomatic patients with inducible ischemia on provocative testing (Fang, 2007). By contrast, this procedure is not indicated for patients who have pulmonary artery systolic pressures greater than 60 mm Hg, severe right ventricular dysfunction, or regional dyskinesis or akinesis without ventricular dilation (Fang, 2007).

CARDIAC TRANSPLANTATION

Transplantation is considered the definitive therapy for end-stage heart failure. The principal limitation to heart transplantation is the growing gap between the number of potential recipients and the number of available donor organs. Given this mismatch in supply and demand, selection of recipients becomes an ethical dilemma as well as a clinical issue (Colucci & Peña, 2007). A centralized organ allocation system has been established in the United States, known as the United Network for Organ Sharing (UNOS); it works alongside local committees to assure that the scarce donor organs are allocated in an objective, equitable, and medically justified way to recipients in the greatest need. Evaluation of

potential candidates is complex and is directed at selecting those patients with refractory end-stage heart disease who have the potential to maintain compliance with a complex post-transplant regimen (Nwakanma, Shah, Conte, & Baumgartner, 2008). Factors considered in allocation include the amount of waiting time, the patient's clinical status, and the geographic distance between the donor hospital and the transplant center (Colucci & Peña, 2007).

The most common refractory heart diseases leading to transplantation are nonischemic cardiomyopathy (45%) and coronary artery disease (38%) (Taylor et al., 2007). The highest mortality rate associated with these conditions is seen in the first 6 months after diagnosis. There is an increase in mortality rate by 3.4% each year. Patient survival had a projected half-life (the time at which 50% of those transplanted remain alive) of 11 years in 2002 (Taylor et al., 2007).

The goals of transplantation are to improve survival and quality of life. As medical therapy has improved both of these outcomes, the role of transplant has become less clear. Most candidates for transplantation must have failed aggressive medical therapy.

Efforts to determine who will benefit most from transplantation have centered on identifying predictors of mortality from heart failure. The primary indicator currently is severely impaired exercise capacity as demonstrated by peak VO_2 of 10 mL/kg/min or less (Colucci & Peña, 2007; Nwakanma et al., 2008). An additional tool is a prognosis score, such as the Heart Failure Survival Score (HFSS) or the Seattle Heart Failure Model (SHFM). These scores use clinical indicators to stratify patients into risk categories or survival rate categories. Unlike the HFSS, the Seattle model incorporates current medical therapy regimens, so it may be more reflective of current practice (Colucci & Peña, 2007). Although these tools may be useful, they are merely one of many factors considered when evaluating a patient for transplantation.

Table 3–3 lists the currently accepted indications and contraindications for cardiac transplantation. Cardiac transplantation is discussed in more detail in Chapter 19.

Heart–Lung Transplantation

Introduced in 1982, heart–lung transplantation was used for patients with end-stage cardiopulmonary and septic lung disease (Sheikh, Pelletier, & Robbins, 2008). The procedure reached its peak in the 1990s. Since then, however, due to improvements in single- and double-lung transplant techniques as well as donor allocation to critically ill heart recipients, the number of procedures has declined substantially (Sheikh et al., 2008). Today, the most common indication for heart–lung transplantation is congenital heart disease with secondary pulmonary hypertension (Eisenmenger syndrome). Congenital conditions that may lead to Eisenmenger syndrome include atrial and ventricular septal defects, patent ductus arteriosus, and truncus arteriosus. Other pediatric and neonatal complex congenital heart anomalies have been successfully treated with heart–lung transplant as well (Sheikh et al., 2008). Additional indications for adult heart–lung transplantation include primary pulmonary hypertension with secondary right heart failure, cystic fibrosis, septic lung disease, severe coronary artery disease with end-stage lung disease, and parenchymal lung diseases with severe right-sided failure such as idiopathic pulmonary fibrosis, lymphangioleiomyomatosis, sarcoidosis, and desquamative interstitial pneumonitis (Sheikh et al., 2008; Taylor et al., 2007).

Recipients are selected on the basis of progressively disabling symptoms and ability to withstand full rehabilitation after the transplant (Sheikh et al., 2008). Table 3–4 outlines the generally accepted transplant candidate

Table 3–3 Indications and Contraindications for Cardiac Transplantation

Indications

- Severe heart failure refractory to medical therapy
- Severely limiting ischemia not amenable to revascularization
- Recurrent symptomatic ventricular tachyarrhythmias refractory to medical therapy, devices, or surgery
- Cardiac tumors (rare)

Diseases

Systolic heart failure (EF less than 35%)

- Ischemic
- Dilated
- Valvular
- Hypertensive
- Other

Ischemic heart disease with intractable angina

- Refractory to maximal tolerated medical therapy
- Not amenable to revascularization (CABG, PCI, or TMR)
- Unsuccessful revascularization

Intractable arrhythmia

- Uncontrolled with implantable cardioverter defibrillator
- Not amenable to electrophysiologic guided therapy
- Not a candidate for ablative therapy

Hypertrophic cardiomyopathy: Class IV heart failure symptoms persist despite maximal medical therapy

- Alcohol injection
- Myomectomy
- Mitral valve replacement
- Maximal medical therapy
- Pacemaker therapy

Congenital heart disease in which fixed pulmonary hypertension is not a complication

- Cardiac tumor
- Confined to myocardium
- No evidence of distant disease via extensive metastatic workup

Contraindications and Excluded Etiologies

Systolic heart failure as a result of

- Amyloidosis
- HIV infection
- Cardiac sarcoma

continues

Table 3–3 Indications and Contraindications for Cardiac Transplantation (continued)

Absolute contraindications

- Age greater than 70 years (depending on facility)
- Fixed pulmonary hypertension (unresponsive to pharmacologic therapy)
  - Pulmonary vascular resistance greater than 5 Wood units or U/m^2
  - Transpulmonary gradient greater than 15 mm Hg
- Systemic illness that will limit survival despite transplantation
  - Neoplasm other than skin cancer (less than 5 years' disease-free survival)
  - HIV/AIDS (CD4 count less than 200 cells/mm^3)
  - SLE or sarcoidosis that has multisystem involvement or is still active
  - Any systemic process with a high probability of recurrence in the transplanted heart
  - Irreversible renal or hepatic dysfunction

Relative contraindications

- Recent malignancy
- Chronic obstructive pulmonary disease (COPD)
- Recent and unresolved pulmonary infarction and pulmonary embolism
- Diabetes mellitus with end-organ damage
  - Nephropathy
  - Neuropathy
  - Retinopathy
- Peripheral vascular or cerebrovascular disease
  - Not amenable to surgical or percutaneous therapy
  - Asymptomatic carotid stenosis greater than 75% or symptomatic carotid stenosis of less severity
- Active peptic ulcer disease
- Current or recent diverticulitis
- Other systemic illness likely to limit survival or rehabilitation
- Severe obesity or cachexia
- Severe osteoporosis
- Active alcohol or drug abuse
- History of noncompliance or psychiatric illness likely to interfere with long-term compliance
- Psychosocial impairment that jeopardizes the transplanted heart
  - Antisocial personality disorder
  - Medication noncompliance
  - Cigarette smoking
  - Inability to rely on alternative caregivers in the event of patient impairment
  - Absence of psychosocial support

AIDS = acquired immune deficiency syndrome; CABG = coronary artery bypass grafting; CD4 = cluster of differentiation, 4 (tests for T_4 helper cells); EF = ejection fraction; HIV = human immunodeficiency virus; PCI = percutaneous coronary intervention; SLE = systemic lupus erythematosus; TMR = transmyocardial laser revascularization.

Sources: Colucci, & Peña, 2007; Nwakanma, Shah, Conte, & Baumgartner, 2008; Steinman et al., 2001.

Table 3–4 Heart–Lung Transplant Recipient Criteria and Contraindications

Criteria

Life expectancy of less than 18–24 months

Disabling symptoms
- Dyspnea, cyanosis
- Syncope
- Hemoptysis
- NYHA Class III or IV

Age less than 50 years

End-stage disease of heart and lungs

Untreatable end-stage pulmonary, organ, or vascular disease, or any combination of these

Disease processes (not all-inclusive)
- Irreversible primary PH with HF
- COPD with HF
- Nonspecific pulmonary fibrosis
- Emphysema with severe HF
- Cystic fibrosis with severe HF or end-stage bronchiectasis with compromised cardiac function
- Eisenmenger syndrome with irreversible PH and HF
- Severe CAD or cardiomyopathy with irreversible PH
- Noncomplex congenital heart disease associated with PH not amenable to lung transplantation and repair by standard cardiac surgery
- Pulmonary fibrosis with unmanageable PH or HF
- Congenital heart defect not amenable to standard repair
- Irreversible right-heart failure from pulmonary hypertension

Contraindications

Absolute Contraindications

Multiple organ system dysfunction

Current substance abuse (alcohol, drug, or both)

Bone marrow failure

Active malignancy

HIV infection

Progressive neuromuscular disease

Clinically severe obesity

Current tobacco use/smoking (must be abstinent for at least one year)

Noncompliance with other regimens

Other major organ system disease or infection (e.g., major vascular disease)

Collagen vascular disease (scleroderma, SLE, sarcoidosis) if not limited to the lung

Psychosocial history that limits ability to adhere to strict pre- and post-transplant regimens

Morbid obesity (BMI greater than 40 kg/m^2 or greater than 35 kg/m^2) with presence of comorbidities

Liver or kidney dysfunction (bilirubin greater than 2.5 mg/dL and creatinine clearance less than 50 mL/minute, respectively)

Inadequate functional status

Relative Contraindications

Age (greater than 55–65 years)

Eating disorder (anorexia, obesity)

Peripheral and coronary vascular disease

Ventilator support or other conditions associated with limitations in mobility

Steroid dependency

Chest wall deformity

Resistant infection (bacterial or fungal)

Lack of social support

CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; HF = heart failure; NYHA = New York Heart Association; PH = pulmonary hypertension; SLE = systemic lupus erythematosus.

NYHA Class III = Patient is comfortable at rest; less than ordinary activity causes fatigue, palpitation, dyspnea, or angina.

NYHA Class IV = Inability to carry on any physical activity without discomfort. Symptoms of heart failure or angina may be present even at rest. Decreased comfort with any physical activity.

Sources: Aetna, 2008; BlueCross BlueShield of North Carolina, 2008; Mancini, 2008; North Carolina Department of Health and Human Services, Division of Medical Assistance, 2008; Sheikh, Pelletier, & Robbins, 2008; Spiwak, 2008.

selection criteria. Contraindications for heart–lung transplantation are similar to those for heart transplantation (Sheikh et al., 2008).

SURGERY FOR ADULT CONGENITAL HEART DISEASE

The congenital heart defects most commonly seen in adults are atrial septal defect (ASD) and bicuspid aortic valve (Wiegers & St. John Sutton, 2007). As increasing numbers of children with congenital heart defects survive to adulthood, estimates are that approximately 760,000 adults will be living with congenital heart disease by 2020 (Laks, Marelli, Plunkett, & Myers, 2008). Most of these patients will have had corrective or palliative surgery in infancy to minimize the long-term consequences of congenital defects. For the purposes of this chapter, this section focuses on management of ASD.

Most ASDs of less than 8 mm close spontaneously. For those patients whose defects do not close, the primary cause of symptoms is left-to-right shunting. Data indicate that an open ASD will increase in diameter over time, leading to increased shunting with age (McMahon et al., 2002). An ASD is most often asymptomatic until the individual reaches adulthood; by age 30 to 40 years, however, AF and reduced exercise tolerance as a result of the defect are usually evident. Chronic left-to-right shunting may cause right ventricular failure, tricuspid regurgitation, atrial arrhythmias, paradoxical embolization, and cerebral abscesses. Ultimately, irreversible pulmonary hypertension and right-to-left shunting and hypoxia will develop (Laks et al., 2008; Wiegers & St. John Sutton, 2007). Surgery is usually recommended before these adverse sequelae occur. Surgical repair should be considered for patients with an ASD when the ratio of pulmonary flow to systemic flow is greater than 1.5:1, and pulmonary vascular resistance is less than 6–8 U/m^2 (Laks et al., 2008). ASDs may be closed by patch under cardiopulmonary bypass or via percutaneous closure using a closure device.

Outcomes from surgical closure are good. Adverse events and death are more frequent with medical therapy than with surgical closure, regardless of the patient's age (Attenhofer et al., 2005; Laks et al., 2008). Although long-term follow-up has been limited to date, data indicate that for those patients whose ASDs are suitable for percutaneous closure, outcomes are also favorable, showing good closure, improved functional capacity, and improved right and left ventricular performance (Laks et al., 2008; Wiegers & St. John Sutton, 2007). Two studies comparing outcomes with surgical closure to those with percutaneous closure indicated complete closure rates were higher with surgery but significantly fewer complications occurred with percutaneous closure (Berger, Vogel, Alexi-Meskishvili, & Lange, 1999; Du et al., 2002).

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy (HCM) is a common genetic cardiovascular disease characterized by abnormal myocytes leading to hypertrophy without dilatation and preserved systolic function (Maron et al., 2003; McKenna, 2007; Padera & Schoen, 2008). Hypertrophy is most severe in the ventricular septum. This asymmetrical growth usually arises at the level of the LV outflow tract, leading to subaortic stenosis or asymmetrical HCM. In addition, abnormal systolic anterior motion of the mitral valve contributes to the outflow obstruction (Maron et al., 2003; McKenna, 2007; Padera & Schoen, 2008; van der Lee et al., 2005).

HCM has a variable course, although two presentations commonly lead to treatment. Impaired diastolic filling due to massive hypertrophy is usually responsible for signs

and symptoms of heart failure, although obstruction of the LV outflow tract contributes as well (Maron, 2005; Maron et al., 2003; McKenna, 2007; Padera & Schoen, 2008). In addition, HCM is the most common cause of sudden cardiac death in young adults (Padera & Schoen, 2008).

Four approaches are employed for treatment of HCM: pharmacological therapy, dual-chamber pacing, surgery, and chemical ablation (McKenna, 2007; Padera & Schoen, 2008; van der Lee et al., 2005; Woo et al., 2005). Pharmacological therapy is usually the first approach to signs and symptoms of heart failure; indeed, it is often the only treatment needed in patients without outflow obstruction (Maron et al., 2003). Beta blockade is the standard first-line therapy, with verapamil and disopyramide often being added to the treatment regimen to take advantage of their negative inotropic properties (Maron et al., 2003). For a majority of patients, this course of treatment is effective; a few patients develop systolic heart failure, however, and become candidates for heart transplantation.

A second treatment approach, dual-chamber pacing, was introduced in the 1990s. Pacing was thought to change the geometry of ventricular contraction and lead to reduced LV outflow tract obstruction and symptomatic improvement (McKenna, 2007). This procedure is currently recommended only for HCM patients with sinus or AV node dysfunction (Gregoratos et al., 2002).

The mainstay of treatment for patients with HCM who develop significant drug-refractory LV outflow tract obstruction has been surgical LV myectomy and/or mitral valve replacement (Maron, 2005; Maron et al., 2004; Maron et al., 2003; McKenna, 2007; van der Lee et al., 2005; Woo et al., 2005). The myectomy procedure involves excision of a section of subaortic septal muscle that is approximately 3–7 cm long and 3–12 grams in weight, with or without mitral valve replacement (Maron et al., 2004). The LV myectomy reduces anterior mitral systolic motion and MR and reduces the LV outflow tract obstruction (Woo et al., 2005). The procedure is performed in conjunction with perioperative transesophageal echocardiography to precisely determine the amount of tissue to be removed. This procedure has a long history and experience and is well documented to be safe, provide excellent hemodynamic results, and improve quality of life (Maron, 2005; Maron et al., 2004; Maron et al., 2003; McKenna, 2007; Padera & Schoen, 2008; van der Lee et al., 2005; Woo et al., 2005; Yacoub, 2005).

Systolic anterior motion (SAM) of the mitral valve has been shown to be a major determinant of the amount of outflow obstruction. Mitral valve replacement (MVR) is often added to the myectomy procedure, depending on the degree of SAM and obstruction.

Although successful, surgery is recommended for only some 5% of all patients with HCM (Maron et al., 2003). Left ventricular myectomy is recommended for those patients with drug-refractory symptomatic outflow obstruction (peak gradients greater than 50 mm Hg as measured by echocardiography under resting conditions and/or gradients greater than 50 mm Hg as measured under physiologic exercise). Surgery may also be considered in symptomatic patients with documented outflow obstruction under physiologic exercise but in whom resting obstruction is either absent or very mild (Maron et al., 2003). One additional subset of patients may benefit from LV myectomy—young, asymptomatic patients with documented severe outflow tract obstruction (gradient 75–100 mm Hg) (Maron et al., 2003). Surgery is usually not recommended for asymptomatic or mildly symptomatic patients (Maron et al., 2003). MVR is indicated when MR is present in addition to outflow tract obstruction (McKenna, 2007).

Finally, a percutaneous alternative for relieving outflow obstruction is available.

Ethanol septal ablation is accomplished by infusing ethanol into the first septal branch of the left anterior descending coronary artery via an angioplasty catheter (McKenna, 2007). Studies of this approach indicate that ethanol ablation reduces outflow tract obstruction, increases exercise capacity, and improves symptoms (McKenna, 2007; Padera & Schoen, 2008; van der Lee et al., 2005; Yacoub, 2005). Available data indicate that ethanol ablation markedly reduces symptoms but results in more complications and requires more repeated interventions than myectomy. Hemodynamic results appear to be better with surgery. The effects of ethanol septal ablation on sudden cardiac death are as yet unknown (Maron et al., 2003; van der Lee et al., 2005; Yacoub, 2005).

Although the number of ethanol ablation procedures is clearly increasing, more research into this treatment alternative is warranted. Indications for ethanol ablation are similar to those for surgical intervention: Patients should have severe heart failure symptoms refractory to pharmacological therapy as well as a gradient of greater than 50 mm Hg either at rest or under physiologic exercise (Maron et al., 2003). Left ventricular septal myectomy remains the gold standard for treatment of outflow tract obstruction (Maron et al., 2004; van der Lee et al., 2005; Yacoub, 2005).

OTHER CARDIAC SURGERIES: PERICARDIAL SURGERY

Bleeding and cardiac tamponade are known complications after cardiac surgery. Typically, the surgeon leaves the pericardium open and places mediastinal chest tubes to manage any postoperative bleeding. Despite these precautions, it is still possible for blood and clots to accumulate in the mediastinal space and impair ventricular filling (cardiac tamponade).

Reexploration of the mediastinum is indicated for signs of tamponade, including a sudden decrease or cessation of chest tube output, tachycardia, narrowing pulse pressure, and decreased cardiac index. Partial resection of the pericardium is known as a pericardial window. In such a procedure, a portion of the pericardium is excised to allow fluid to drain into the pleural or peritoneal space and prevent reaccumulation of pericardial fluid.

CARDIAC TUMORS

A benign tumor, myxoma accounts for half of all benign cardiac tumors. Approximately 75% of these masses arise in the left atrium, most often occurring in individuals between ages 20 and 50 years. Myxomas are more common in women. These tumors cause obstruction of blood flow, which in turn leads to the clinical presentation of heart failure, signs of central nervous system (CNS) embolization, and constitutional symptoms such as fever, weight loss, fatigue, weakness, arthralgia, and myalgia. Resection of the myxoma is the only effective therapy and should be performed early to prevent embolization (Walkes, Smythe, & Reardon, 2008).

Other benign cardiac tumors in adults include lipoma and fibroelastoma. Large lipomas are resected when they are symptomatic because of their potential for obstruction. Papillary fibroelastomas occur on the heart valves and may cause obstruction or CNS embolization; these tumors should also be resected. Primary cardiac malignancies are extremely rare (Walkes et al., 2008).

CASE STUDY

P.S. is a 73-year-old man with a history of angina pectoris and atherosclerotic heart disease. He underwent angioplasty and stenting to the left PDA and an obtuse marginal 2 years ago. He has known aortic stenosis and has been followed with echocardiogram for this condition. P.S. had been doing well until the last several months, when he began noticing substernal burning and pain with exercise that was relieved by rest. The patient noted that the pain occurred with any exercise after a meal. The latest echo by his primary care physician reveals an aortic gradient of 80 mm Hg, a jet velocity of 6.5 m/sec (meters per second), and an aortic valve area of 0.5 cm^2. Additional history includes hyperlipidemia, hypertension, and a sedentary lifestyle. P.S. is mildly overweight, is not diabetic, does not smoke, and drinks only occasionally. His father was hypertensive; there is no history of aortic valve disease. P.S. takes atorvastatin 10 mg daily, atenolol 25 mg twice daily, aspirin (ASA) 81 mg daily, and a daily vitamin. He has been retired from his mail-carrying position since 1993 and lives at home with his wife. He was referred to a cardiac surgeon for aortic valve replacement.

P.S. underwent coronary angiography, which revealed an 85% stenosis in the proximal LAD and another 90% in the first diagonal. A left dominant system with a previously stented PDA was noted, with heavy atherosclerosis that was deemed not bypassable. Ventricular function was normal. Preoperative lab studies were normal. The surgeon estimated that P.S. had a risk of less than 2% for major complications such as heart attack, stroke, and death, and a 4–5% risk of other complications such as pneumonia, bleeding, reoperation, and pacemaker. His surgery was scheduled for the next day.

The operative report noted a tricuspid aortic valve, severe aortic stenosis, and advanced calcifications of the aortic valve leaflets. He underwent CABG with a left internal mammary artery graft to the left anterior descending and a radial artery graft to the first diagonal. The aortic valve was replaced. The operation was uneventful, and the patient was transferred to the intensive care unit in stable condition.

P.S. had a smooth recovery. He was extubated within 4 hours and transferred to the progressive care unit the next morning. P.S. was discharged to home on postoperative day 6. His discharge instructions included consumption of a low-fat, low-cholesterol diet and outpatient cardiac rehabilitation. P.S. was given an appointment for the hospital's "Living with Heart Disease" class and was discharged on ASA 325 mg PO once daily, lisinopril 2.5 mg PO once daily at bedtime, metoprolol 50 mg PO twice daily, and atorvastatin 10 mg PO once daily. He was to follow up with his surgeon in 2 weeks and his primary care physician in 4 weeks.

SELF-ASSESSMENT QUESTIONS

1. What is the primary reason for surgery in the Case Study patient?
   a. STEMI
   b. Severe aortic stenosis
   c. Coronary artery disease
   d. Aortic dissection
2. What are the indications for CABG in this patient?
   a. One- or two-vessel disease not involving proximal LAD with LVEF less than 50% or a large amount of myocardium at risk on noninvasive studies
   b. Proximal LAD stenosis with one- or two-vessel disease

   c. Failed PCI with persistent pain or hemodynamic instability and anatomically feasible
   d. Asymptomatic or mild angina and proximal LAD stenosis and one- or two-vessel disease
3. What are the indications for aortic valve replacement?
   a. Patients undergoing CABG with mild AS when there is evidence that progression may be rapid
   b. Severe AS and LV systolic dysfunction (ejection fraction less than 50%)
   c. Symptomatic patients with severe AS
   d. Moderate AS undergoing CABG or surgery on aorta or other heart valves
4. Which classification is the indication for aortic valve replacement?
   a. Class III
   b. Class IIa
   c. Class IIb
   d. Class I
5. P.S. would be at high risk for which specific postoperative complications?
   a. Bleeding, ileus, AMI
   b. Ileus, pacemaker, prolonged ventilation
   c. Pacemaker, bleeding, CVA
   d. Pneumonia, ileus, prolonged ventilation
6. When P.S. asks you why he is being discharged on lisinopril, what do you tell him?
   a. Lisinopril's antiplatelet actions will help prevent clotting.
   b. Lisinopril is an antianginal agent.
   c. Lisinopril is prescribed because P.S. has decreased myocardial function.
   d. Lisinopril provides plaque stabilization and antihypertensive therapy.
7. Which characteristics would indicate severe AS?
   a. Mean gradient of 80 mm Hg, jet velocity of 6.5 m/sec, and valve area of 0.5 cm^2
   b. Mean gradient of 20 mm Hg, jet velocity of 2 m/sec, and valve area of 1.8 cm^2
   c. Mean gradient of 35 mm Hg, jet velocity of 3 m/sec, and valve area of 1.2 cm^2
   d. Mean gradient of 45 mm Hg, jet velocity of 3 m/sec, and valve area of 1.0 cm^2
8. Which symptoms might indicate the presence of worsening AS?
   a. Sudden cardiac death, AMI, and a diastolic murmur
   b. Systolic murmur, angina, and syncope
   c. Angina, syncope, and CHF
   d. Syncope, sudden cardiac death, and elevated C-reactive protein
9. What are effective therapies for severe AS other than aortic valve replacement?
   a. Antihypertensives, digoxin, and diuretics
   b. Diuretics, amiodarone, and lisinopril
   c. Lisinopril, amiodarone, and an ICD
   d. The only effective long-term therapy for AS is valve repair or replacement.
10. What is the most probable etiology of P.S.'s aortic stenosis?
    a. Rheumatic heart disease
    b. Degenerative calcification
    c. Bicuspid aortic valve
    d. Papillary muscle dysfunction

Answers to Self-Assessment Questions

1. b
2. d
3. c
4. d
5. c
6. d
7. a
8. c
9. d
10. b

Clinical Inquiry Box

Question: Is ablation of atrial fibrillation in octogenarians during cardiac surgery safe?

Reference: Grubitzsch, H., Beholz, S., Dohmen, P. M., Dushe, S., & Konertz, W. (2008). Concomitant ablation of atrial fibrillation in octogenarians: An observational study. *Journal of Cardiothoracic Surgery, 3*(1), 21.

Objective: To evaluate the outcomes of octogenarians undergoing atrial fibrillation ablation.

Method: Twenty-eight patients aged 80 years or older were evaluated at 3 months, 6 months, 12 months, and annually thereafter for atrial fibrillation duration, prevalence, aortic valve disease, ICU length of stay, and 30-day mortality.

Results: Octogenarians were similar to controls regarding AF duration and left atrial diameter. The prevalence of paroxysmal AF, aortic valve disease, ICU stay, hospital stay, and 30-day mortality were increased in octogenarians. After 12 months, 14 octogenarians (82%) and 101 controls (68%, nonsignificant) were in sinus rhythm; 59% who were not taking antiarrhythmic drugs in either group were in sinus rhythm (nonsignificant).

Conclusion: Even in advanced age, ablation for atrial fibrillation should be considered for octogenarians. The implication of this study for cardiac surgical nurses is to enhance awareness that the "oldest of old" will continue to be candidates for cardiac surgery and should be considered for surgical strategies such as ablation regardless of age.

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Chapter 4

Preoperative Cardiac Surgery Nursing Evaluation

Roberta Kaplow, Sonya R. Hardin, Brenda Hardin-Wike, Timothy E. McMurry

INTRODUCTION

Preoperative evaluation and preparation of the patient for cardiac surgery affects postoperative outcomes and progress. The primary goal of a presurgical assessment is evaluation of perioperative risk. An in-depth assessment assists in minimizing surgical risk and potential morbidity and mortality. The literature supports the preoperative optimization of a patient's cardiovascular status as part of the effort to improve patient outcomes (Halaszynski, Juda, & Silverman, 2004). An evaluative screening identifies special needs that may require modification of the patient's course of treatment before, during, and after surgery.

RISK FACTORS OF MORBIDITY AND MORTALITY FOLLOWING CARDIAC SURGERY

The major risk factors for adverse outcomes of cardiac surgery include advanced age, emergency surgery, previous cardiac surgery, dialysis dependency, and creatinine level of 2 mg/dL or higher. Preoperative renal insufficiency is an independent risk factor for morbidity and mortality (Mageed & El-Ghoniemy, 2007).

Research has demonstrated a decreased incidence of physical and psychological problems that adversely affect recovery when preoperative education of patients is completed. Evidence further indicates that preoperative patient education results in increased patient compliance, resulting in decreased length of hospital stay (Cupples, 1991; Shuldham, 2001).

Numerous risk assessment tools have been developed to predict mortality in patients undergoing heart surgery. Some of these scoring tools include the Parsonnet, Cleveland Clinic, French, Euro, Pons, and Ontario Province Risk scores (Geissler et al., 2000). The Parsonnet score has been found to be predictive in the oldest of old individuals who require cardiac surgery (Chaturvedi, deVarennes, & Lachapelle, 2007). Prolonged hospital stays and increased mortality are associated with higher scores. Table 4–1 presents the criteria utilized in the Parsonnet score.

NURSING ASSESSMENT

Nursing assessment prior to cardiac surgery typically begins during an outpatient visit, but may occur during an acute inpatient admission. The latter situation may occur in patients with conditions that increase their operative risk. The preoperative nursing assessment provides baseline information for the postoperative period, along with an opportunity to develop a relationship with the patient. Components of the preoperative

Table 4–1 Parsonnet Score

Factor	Weight
Female gender	1
Comorbidities	
Morbid obesity	3
Diabetes	3
Hypertension	3
Preoperative IABP	2
Dialysis dependency	2
Pacemaker dependency	5
Left ventricular aneurysm	5
Emergency surgery following PTCA	10
Acute renal failure	10
Cardiogenic shock	30
Ejection Fraction	
Good (≥ 50%)	0
Fair (30–49%)	2
Poor (< 30%)	4
Age	
< 70 years	0
70–74 years	7
75–79 years	12
>80 years	20
Reoperation	
First	5
Second	10

Risk Scores: Good (0-4); Fair (5-9); Poor (10-14); High (15-19); Extremely High (> 20).

IABP = intra-aortic balloon pump; PTCA = percutaneous transluminal coronary angioplasty.

Source: Parsonnet, Dean, & Bernstein, 1989. Used with permission.

assessment include information from the patient, family, and medical records, and the physical exam.

Preoperative Patient Interview

The purpose of a patient interview is to review past medical and surgical histories and to conduct a systems evaluation to identify processes that may affect the outcome of a patient's cardiac surgery. The interview helps the nurse evaluate patient and family knowledge as well as determine educational needs related to the planned procedure. Understanding of the underlying illness, planned surgical course, and willingness and ability to adhere to the surgical regimen are also evaluated. Put simply, the nurse is responsible for the overall assessment of the patient's physical and psychological readiness for surgery. Data suggest that cardiac surgery patients who receive preoperative education with or without coping strategies as opposed to routine preoperative preparation experience less emotional distress, have better physical and psychological recovery, and experience fewer hypertensive episodes postoperatively (Anderson, 1987).

Baseline information is obtained about the patient's clinical history, including the type of heart disease, associated symptoms, resource availability, stability, and ability to participate in care and decision making. The level of resilience will be determined when the nurse ascertains the degree of compensation the patient has developed.

During the patient interview, the nurse should seek to discover any information that can affect perioperative risk and postoperative management. Several risk factors have been identified in the literature as influencing the mortality of cardiac surgery patients. Table 4–2 lists many of these comorbid conditions.

The nurse should also inquire if the patient has any history of gastrointestinal bleeding, peptic ulcer disease, or bleeding diathesis. Any of these conditions may affect the antiplatelet regimen following revascularization or the choice of a valvular prosthesis. Likewise, the nursing evaluation should gather information on the presence of cardiac risk factors as well as presence of associated medical diseases, such as COPD, cerebrovascular or other peripheral arterial occlusive disease, and hypertension.

The patient's baseline sleep patterns should be determined. Patients who undergo coronary artery bypass grafting (CABG) proce-

Table 4–2 Factors That May Affect Cardiac Surgery Patient Mortality

Alcohol use	Tobacco use
Diabetes	Elevated serum creatinine (2 mg/dL or higher)
Chronic airway disease	Previous cardiac surgery
Recent myocardial infarction	Low left ventricular ejection fraction
Chronic heart failure	Pulmonary hypertension
Unstable angina	Depression
Obesity	Hypoalbuminemia
Active endocarditis	Procedure urgency
Ventricular septal rupture	Critical preoperative condition
Dialysis	Advanced age

Sources: Albert & Antman, 2003; Blumenthal et al., 2003; Engelman et al., 1999; Roques et al., 1999.

dures are at risk of developing sleep disturbances postoperatively. The presence of anxiety and depression should be assessed as well, as these psychosocial conditions may develop in the postoperative cardiac surgery patient (Hedges & Redeker, 2008).

Nutrition Evaluation

The preoperative evaluation should also look for indicators of nutritional deficiency. In particular, malnutrition is a risk factor associated with significant morbidity and mortality in surgical patients. During the nursing evaluation, it is essential that all cardiac surgery patients undergo nutritional screening to identify malnourished or at-risk patients so as to ensure an adequate nutritional plan is included as part of the patient's care.

In addition to the nutritional assessment screens available, unintentional weight loss, protein-calorie malnutrition, laboratory findings (e.g., anemia, hypoalbuminemia, prealbumin, vitamin B_{12} deficiency), and low body mass index are among the variables suggesting nutritional deficiency (Hengstermann, Nieczaj, Steinhagen-Thiessen, & Schulz, 2008; Meyyazhagan & Palmer, 2002). Patients with hypoalbuminemia (< 2.5 g/dL) should have their nutritional status optimized 1 to 4 weeks prior to cardiac surgery, as they are at great risk for sepsis and respiratory failure. Enhanced nutrition is also essential to promote wound healing and meet postoperative metabolic demands (Albert & Antman, 2003). This can be accomplished with dietary enhancement or enteral feeding if no contraindications are present. Patients who are undergoing cardiac surgery and who have low body mass index (< 20 kg/m^2) and hypoalbuminemia (< 2.5 g/dL) are at increased risk of postoperative morbidity and mortality (Engelman et al., 1999). Further, patients with hypoalbuminemia are at increased risk for bleeding, renal failure, and prolonged ventilatory support. Conversely, patients with a high percentage of body fat have a greater risk for sternal wound and saphenous vein harvest site infections and atrial arrhythmias (Albert & Antman, 2003).

Discharge Planning

To begin proactive discharge planning, the patient's living arrangements are assessed. Many patients need assistance at discharge owing to limited social and financial resources. Early discharge planning alleviates stressors and anxiety for both the patient and family (Carroll & Dowling, 2007).

Physical Assessment

Cardiac Assessment

For patients undergoing cardiac surgery, the assessment of the cardiovascular system will likely be more extensive than the assessment of the other body systems. Blood pressure, temperature, assessment of peripheral pulses, and weight are recorded. Blood pressure readings should be obtained from both arms. Blood pressure difference between arms is associated with increased morbidity and mortality (Clark & Powell, 2002) and subclavian artery stenosis (Osborn, Vernon, Reynolds, Timm, & Allen, 2002). This condition may eliminate the possibility of using the internal mammary artery for grafting (Fortier, Demaria, & Perrault, 2002). It has been suggested that stenting the subclavian artery will make the vessel suitable for CABG (Rogers & Calhoun, 2007).

Auscultation of the heart and carotid arteries will provide essential baseline information. Heart sounds should be evaluated in terms of their rate, rhythm, and presence of extra sounds, murmurs, gallops, or rubs. If the patient is noted to have a slow heart rate and a Mobitz II or complete heart block is noted, insertion of a temporary transvenous pacemaker wire should be considered (Albert & Antman, 2003).

Identification of aortic regurgitation (AR) is a significant finding, as this condition may be exacerbated during cardiopulmonary bypass and lead to acute left ventricular distention (Albert & Antman, 2003). AR is identified with the presence of an early diastolic murmur that can be heard at the second and third intercostal spaces (ICSs) at the right-sternal border and at the second and fourth ICSs at the left-sternal border. The murmur of AR usually decreases in intensity (decrescendo) and disappears before the S_1 heart sound (Choudhry & Etchells, 1999).

A carotid bruit is a sound associated with turbulent flow and may indicate arterial stenosis. Auscultation of the carotid arteries is performed from the base of the neck to the angle of the jaw while breath holding. A bruit is usually most audible in the upper third of the carotid near the bifurcation (Cassar, Fingernet, & Woodcome, 1997).

As noted in Chapter 3, perioperative stroke risk ranges from 2% to 12% with unilateral carotid occlusion to 5% with significant bilateral stenoses (Naylor, Cuffe, Rothwell, Loftus, & Bell, 2003). Accordingly, carotid endarterectomy is recommended before or concurrently with CABG in patients who have symptomatic carotid stenosis and for patients who have 80% unilateral or bilateral carotid stenosis (Eagle et al., 2004).

Peripheral vascular assessment is performed to help determine the extent of peripheral perfusion. Components of this evaluation include determining the presence and strength of pulses in all extremities, capillary refill time, extremity and nail bed color, and temperature. Calculating the ankle-brachial index helps evaluate the arterial blood flow to the lower extremities; steps to determine this index appear in Chapter 10. The results of this calculation are then used to rate degree of peripheral artery disease and will help determine if the saphenous vein is suitable for use during cardiac surgery (Creager & Libby, 2004).

A cardiac assessment further entails determining presence of varicose veins. Presence of significant numbers of lower-extremity varicosities may indicate the need to use upper-extremity vessels (e.g., radial artery) as conduits during CABG. Using arm veins for conduits necessitates avoiding placing intravenous lines in the affected arm (Albert & Antman, 2003).

Pulmonary Assessment

Postoperative pulmonary complications contribute significantly to morbidity and mortality. A thorough pulmonary assessment,

including identification of associated risk factors, is pivotal so that implementation of strategies to mitigate complications can begin in a timely fashion (Khan & Hussain, 2005).

Lung auscultation provides information about respiratory rate and breath sounds, and the presence of crackles or wheezing. Presence of crackles indicates fluid in the alveoli, which may require diuresis prior to surgery. Presence of decreased breath sounds or adventitious sounds may be related to an undiagnosed condition that may increase the risk of postoperative pulmonary complications or to underlying heart or lung disease. In either case, optimizing the patient's clinical condition preoperatively is indicated (Khan & Hussain, 2005).

A patient's smoking history should be determined. Studies in which patients undergoing CABG were evaluated suggest that current smokers are more likely to develop pulmonary complications, require mechanical ventilation longer, and have higher mortality rates than patients who stopped smoking either 2 or 6 months prior to surgery (Khan & Hussain, 2005). Further, the incidence of complications in patients who quit smoking more than 6 months prior to cardiac surgery is similar to the rate in patients who never smoked.

Preoperative cardiac surgery patients should be assessed for preexisting pulmonary disease to help anticipate potential postoperative conditions. Specifically, a history of pulmonary hypertension and COPD are two predictors of extubation failure in cardiac surgery patients (Rady & Ryan, 1999). Patients with COPD, bronchitis, poor control of asthma symptoms, productive cough, or poor exercise tolerance are also more likely to develop postoperative complications (Albert & Antman, 2003; Hulzebos, Van Meeteren, De Bie, Dagnelie, & Helders, 2003; Jensen & Yang, 2007; Khan & Hussain, 2005). The American College of Chest Physicians recommends that patients with COPD who are undergoing cardiac surgery have preoperative pulmonary function testing (American Thoracic Society & American College of Chest Physicians, 2003).

Abdominal Assessment

A preoperative abdominal assessment is important to determine the presence of an abdominal aortic aneurysm (AAA), which is a potential contraindication of the use of an intra-aortic balloon pump (IABP—discussed in detail in Chapter 10) (Albert & Antman, 2003). Abdominal palpation to detect abnormal widening of an aortic pulsation is suggested to be the most effective method to determine presence of an AAA. The width is compared with the intensity of aortic pulsation to establish the presence of an AAA. Findings from palpation, however, are limited in patients with abdominal obesity. Diagnostic evaluation with ultrasound may also be performed (Lederle & Simel, 1999).

To palpate for presence of an AAA, the patient should be positioned supine with knees raised. The abdomen should be relaxed. To locate aortic pulsation, the abdomen is palpated a few centimeters from the umbilicus toward the patient's head and just left of the midline (the umbilicus is the indicator of the level of the aortic bifurcation). Next, both hands are positioned on the abdomen with palms down, and an index finger is placed on either side of the pulsating area to confirm that it is the aorta (each systole should move the two index fingers apart) and to measure the aortic width (Lederle & Simel, 1999).

Neurologic Assessment

A patient who is undergoing cardiac surgery may develop neurologic impairment during the intraoperative or postoperative period. A baseline assessment will help facilitate identification of changes in neurologic status (Albert & Antman, 2003). Baseline data can

help prevent unnecessary testing that might otherwise be performed to evaluate postoperative neurologic symptoms, which, in fact, might have been present preoperatively. The risk for postoperative delirium has been reported to be 11.5% in cardiac surgery patients. Risk factors include cognitive impairment, atrial fibrillation, a history of peripheral vascular disease, major depression, and advanced age (Kazmierski et al., 2006).

PREOPERATIVE ASSESSMENT OF HEART DISEASE

Typically, patients undergoing cardiac surgery have coronary artery disease (CAD). In fact, increasing numbers of patients who are undergoing cardiac surgery have decreased cardiac function and several comorbid conditions, and many have undergone interventional procedures for their cardiac condition in the past (Albert & Antman, 2003). The risk of surgery in terms of associated morbidity and mortality is logically higher in these patients.

A baseline assessment of underlying heart function is essential to help identify those patients who are at risk during the intraoperative period. Data specific to heart function as well as the presence and extent of comorbidities such as diabetes and hypertension should be collected. The patient history should include determination of when cardiac comorbidities (e.g., myocardial infarction [MI]) occurred and whether associated complications are present (e.g., heart failure, ischemia, dysrhythmias) (Beattie & Hurtado, 2002; Moonesinghe & Kelleher, 2006).

The relationship between CAD and valvular disease is discussed in Chapter 5. Patients with valvular heart disease are vulnerable to additional intraoperative and postoperative risk. A preoperative cardiac assessment for these patients should evaluate the impact of valvular disease on ventricular function and should be partly based on the patient's hemodynamic status (Albert & Antman, 2003).

Cardiac History

Nursing evaluation includes assessment of the current level of symptoms. During the patient interview, any increase in intensity or frequency of symptoms should be relatively easy to uncover. The interview is used to identify the degree of the patient's associated functional impairment and to observe for indications that heart function is inadequate during exertion. Several classification systems can be used to assess the functional status of patients with heart disease; these systems evaluate angina, heart failure, and other aspects of heart disease. For example, the Canadian Cardiovascular Society's (CCS) functional classification system is used for the evaluation of angina; the New York Heart Association's (NYHA) classification is used to evaluate heart failure (Campeau, 2002; Hurst, 2007).

Preoperative evaluation of a patient's current medical status should include a cardiac history. Specifically, the presence and severity of symptoms of CAD should be determined. In addition to assessing presence of risk factors for CAD (e.g., tobacco, hypertension, diabetes, hyperlipidemia), obtaining a list of the patient's current medications and their usage will provide essential information. Severity of pain should be rated on a zero to ten scale. Characteristics of angina patterns should be described in terms of onset; location; duration; character; precipitating, aggravating, and alleviating factors; and frequency. From this information, healthcare providers can decide whether the patient has stable or unstable angina. Existence of a previous or recent MI and presence of dysrhythmias or palpitations are also essential pieces of information. Signs of pulmonary edema or pulmonary hypertension or other associated cardiovascular, peripheral vascular, or valvular heart disease should be identified as well

(Rupert, 2007). The surgeon should be notified of significant findings and if possible preoperative hospital admission is anticipated.

A committee for the American College of Cardiology/American Heart Association (ACC/AHA) Task Force on Practice Guidelines revised these organizations' guidelines for conducting a preoperative cardiovascular evaluation. Although, strictly speaking, these recommendations are designed for patients undergoing noncardiac surgery, many of the factors may be used as a guideline for patients undergoing cardiac surgery. Three of the major clinical predictors of cardiac risk in the guidelines are the presence of unstable angina, the presence of significant dysrhythmias, and recent MI (Eagle et al., 2002).

Dyspnea is another symptom of heart disease to be evaluated in the preoperative cardiac surgery patient; it usually results from inadequate tissue oxygen delivery. Patients may report difficult, labored, or uncomfortable breathing. Some of the more common causes of dyspnea include heart failure, cardiac ischemia, asthma, COPD, and pneumonia. If dyspnea is noted, determination of whether it has a cardiac or pulmonary etiology is vital. Indices of a cardiac etiology include a history of dyspnea on exertion, paroxysmal nocturnal dyspnea (PND), orthopnea, and chest pain. Physical findings may include jugular venous distention, S_3 gallop, ascites, and peripheral edema. Radiologic studies may reveal pleural effusion or cardiomegaly (Boyars, Karnath, & Mercado, 2004). Dyspnea is commonly observed in patients with valvular disease; it may also be experienced by patients with ventricular dysfunction.

Orthopnea is the sensation of breathlessness when the patient is lying in a position of rest. It is relieved by sitting or standing. With worsening cardiac disease, orthopnea often develops such that the patient needs to elevate the head of the bed with more than one pillow to breathe comfortably while recumbent.

PND is a feeling of shortness of breath that awakens the patient. It is usually relieved when the patient assumes an upright position (Mukerji, 1990).

Serological Testing

In addition to patient history, preoperative testing with serological and other diagnostic methods should be performed. Data from these tests will help determine surgical and postoperative risk, and define the presence or extent of any new or known comorbid conditions.

Laboratory data that may be collected preoperatively include complete blood count (CBC); coagulation profile; liver, renal, and thyroid function; electrolytes; and albumin level. Identifying the presence of anemia or infection is an important consideration when evaluating cardiac patients, as there are always risks of intraoperative bleeding and dilutional effects with bypass procedures. Attaining and maintaining a hematocrit greater than 35% is recommended. In addition, CBC data will help suggest presence of an infection from an elevated white blood cell count. Preoperative treatment of infection should be implemented (Albert & Antman, 2003). If CBC results reveal thrombocytopenia, a decision as to whether the patient should receive heparin should be made, as thrombocytopenia may be an indication of heparin-induced thrombocytopenia (HIT). Further testing must be done to confirm HIT. If a patient tests positive for HIT, an alternative anticoagulation method should be considered for cardiopulmonary bypass. For example, bivalirudin (Angiomax®), a direct thrombin inhibitor, has been used in cardiac surgery patients requiring bypass (Pappalardo et. al., 2007).

Patients will be heparinized during bypass procedures. Any coagulopathies should be corrected (e.g., with fresh frozen plasma or platelet

transfusion, administration of vitamin K) prior to surgery to minimize risk of postoperative bleeding (Albert & Antman, 2003).

Assessment of liver function should be conducted to help predict how medications, including anesthetic agents, will be metabolized (Albert & Antman, 2003). The value in optimizing a patient's nutritional status preoperatively was discussed earlier; albumin level is one component of that assessment.

Sometimes patients develop acute renal failure (ARF) following cardiac surgery. One of the risk factors of this complication is preexisting renal dysfunction. Further, the mortality rate of patients who develop postoperative ARF and require hemodialysis is reported to be approximately 64%. Patients with normal renal function have a mortality rate of slightly more than 4% (Albert & Antman, 2003). These data speak to the essential nature of evaluating a patient's preoperative renal function.

Although thyroid function tests are not part of the usual preoperative assessment, it has been suggested that such evaluation may be warranted in patients with dysrhythmias such as AF. The risks of hyperthyroidism (e.g., ischemia, heart failure, rapid ventricular rate associated with the AF) and hypothyroidism (e.g., hypometabolism, decreased clearance of anesthesia, prolonged mechanical ventilation) provide justification of preoperative thyroid function assessment in patients undergoing cardiac surgery (Albert & Antman, 2003).

Preoperative serum electrolytes should be evaluated, particularly potassium and magnesium levels. Notably, the presence of hypokalemia, hypomagnesemia, or both can predispose the patient to develop dysrhythmias. Imbalances should be corrected before surgery to prevent intraoperative complications (Albert & Antman, 2003).

Maintaining serum glucose levels within a normal range decreases the rate of cardiac surgery complications. Effective treatment and monitoring of these data for the cardiac surgery patient should begin in the preoperative setting. Data suggest that presence of hyperglycemia in cardiac surgery patients increases mortality, length of stay, and infection rates (Furnary & Wu, 2006).

Diagnostic Studies

In addition to laboratory tests, a number of diagnostic procedures may potentially be performed for the preoperative cardiac surgery patient. Results of these tests will provide information about cardiac anatomical and physiologic issues and pulmonary status, help identify those patients who may be at higher risk (and the degree of risk) with surgery, alert the surgeon that preoperative "fine-tuning" may be necessary, or suggest that modifications of fluids or medications, or both, intraoperatively may be anticipated. Some of the diagnostic procedures that may be performed for these purposes include echocardiography, computed tomography (CT), magnetic resonance imaging (MRI), radionuclide scanning, cardiopulmonary exercise testing, cardiac catheterization, and pulmonary function tests (Albert & Antman, 2003; Moonesinghe & Kelleher, 2006).

Echocardiography may be performed to discover any cardiac anatomical irregularities that might affect surgery (Albert & Antman, 2003). Results of an echocardiogram (ECG) may reveal conditions such as decreased ejection fraction or RV function, presence of aortic stenosis or insufficiency, or mitral insufficiency. These data may be used to reevaluate the surgical plan, identify intraoperative risk, or devise a plan to optimize the patient's clinical status as much as possible prior to surgery (Albert & Antman, 2003). Echocardiography may be used to evaluate wall motion and to estimate the pressure drop (gradient—a measure of heart valve efficiency) associated with valvular disease (Moonesinghe & Kelleher, 2006).

CT may be performed to identify any cardiac anatomical irregularities that might affect the surgery outcome (Albert & Antman, 2003). Although traditional two-dimensional CT provides some detail of the heart, the heart is beating—and therefore moving—during the procedure. By contrast, the newer technology of multislice CT angiography provides three-dimensional images of the heart, which allows for better visualization of the beating heart. Presence of arterial blockages, heart function, and wall motion may all be assessed with this technique (Moonesinghe & Kelleher, 2006; Sun, 2007).

As part of the preoperative evaluation, MRI may be performed to identify any cardiac anatomical irregularities, assess cardiac function and perfusion, and evaluate valves and blood vessels (Albert & Antman, 2003; American Heart Association, 2008a). Cardiac MRI (CMR) creates cardiac images while the heart is beating, thereby providing both still and moving images of the heart and major blood vessels. Chamber size and damage from MI may be determined through use of this technology as well (Moonesinghe & Kelleher, 2006; National Heart Lung and Blood Institute, 2007).

Radionuclide scanning is performed to help evaluate blood supply to the myocardium, and to identify the extent of damage from any previous MI. Intravenous administration of a radionuclide (e.g., thallium, technetium) can highlight those areas of the myocardium that are hypoperfused from partial or complete arterial occlusion (American Heart Association, 2008b).

Cardiopulmonary exercise testing may be performed to assess the heart's functional reserve—that is, the amount of work the heart is able to do in extraordinary conditions (Albert & Antman, 2003). During this evaluation, concomitant cardiac and ventilatory effects of exercise are assessed. Gas exchange, heart rate, and blood pressure measurements, along with ECG evaluation, provide information on actual energy expenditure and stroke volume during exercise. The oxygen extraction from each beat is also measured at varying work intensities (Wasserman, Hansen, Sue, Stringer, & Whipp, 2004).

Cardiac catheterization is considered the gold standard for the diagnosis of CAD. In the case of the cardiac surgery patient, it is performed to evaluate coronary anatomy and efficacy of cardiac contractility (American Heart Association, 2008c). Data such as baseline right atrial, pulmonary artery systolic, diastolic, and occlusive pressures, as well as pulmonary vascular resistance, ejection fraction, and cardiac output, will help determine LV and RV function, augment valve function data, and assist with intraoperative and postoperative hemodynamic management. Administration of fluids and vasoactive agents will be guided by these data, as will the choice of the operative procedure itself. A cardiac catheterization may sometimes reveal the presence of a LV mural thrombus, which places the patient at risk for a stroke in the intraoperative or postoperative period. In patients with valvular heart disease, cardiac catheterization may be used to estimate the degree of regurgitation. Unlike echocardiography, which provides an indirect measurement of the pressure gradient, cardiac catheterization provides for a direct measurement of this parameter (Albert & Antman, 2003; LeBoutillier & DiSesa, 2003; Moonesinghe & Kelleher, 2006).

Pulmonary function tests may be performed on patients who have preexisting lung disease (e.g., COPD). As noted earlier in this chapter, patients with a history of COPD are at greater risk for developing postoperative complications and requiring prolonged intubation. Data from a preoperative arterial blood gas sample can help guide postoperative weaning (Albert & Antman, 2003).

Given that carotid artery stenosis is a risk factor for stroke following CABG, a preoperative carotid ultrasound should be considered. Patients with a carotid bruit or a history of

cerebrovascular accident are at greater risk for developing this complication. Assessment of the carotid arteries preoperatively may decrease the postoperative risk of stroke (Durland et al., 2004; Tarzamni, Afrasyabi, Farhoodi, Karimi, & Farhang, 2007).

Patients who are undergoing cardiac surgery should receive a preoperative dental examination. In one study, gingivitis and lymphadenopathy were observed in 64% and 42% of patients, respectively, and oral hygiene was unsatisfactory in the majority of patients evaluated (Jegier, Smalc, Ciesielski, Jander, & Jegier, 2006). If patients have not had a dental evaluation for several years, an undetected oral infection may potentially be present; such a condition predisposes the patient to adverse postoperative outcomes (Yasny & Silvay, 2007).

Medications

A comprehensive review of the patient's current medication profile and a concomitant medical and surgical history are essential to assist with preoperative planning and prevent intraoperative and postoperative complications. Although some medications may be withheld before cardiac surgery, many others are continued or adjusted during the preoperative period, particularly those used to manage hypertension or heart disease (Moonesinghe & Kelleher, 2006).

Nitrates

Nitrates should be continued up to the time of surgery to avoid an ischemic event. Further, preoperative intravenous administration of a nitrate or other vasodilator (e.g., prostacyclin, nitric oxide) may be indicated to decrease pulmonary vascular resistance (PVR) and enhance RV function in patients with RV dysfunction (Albert & Antman, 2003).

Inotropes/Vasodilators

If patients have a history of PVR that results in RV dysfunction, preoperative administration of an inotropic agent may be indicated. Agents such as dobutamine (Dobutrex®) or milrinone (Primacor®) may be used because of their vasodilator effects (Albert & Antman, 2003).

Beta Blockers

Discontinuing beta-adrenergic-blocking agents can result in a hypersympathetic state that could precipitate myocardial ischemia, infarction, rebound hypertension, tachycardia, or dysrhythmias (Wiesbauer et al., 2007). As noted in Chapter 15, the incidence of AF following cardiac surgery varies with the procedure performed. As many as 65% of patients who undergo combined CABG/valve surgery and 10% to 40% of patients who undergo CABG alone develop postoperative AF. Identified risk factors include greater age, history of hypertension or AF, and heart failure. Preoperative prophylactic administration of beta blockers has reportedly decreased the incidence of AF by 70% to 80% in patients who undergo CABG. Some researchers suggest that sympathetic tone, which is augmented during cardiac surgery, is diminished when beta blockers are taken. Results from other studies suggest that administration of amiodarone (Cordorone®) or sotalol (Betapace®) may decrease the incidence of AF following CABG, whereas administration of a calcium channel blocker or digoxin (Lanoxin®) does not decrease incidence of AF (Albert & Antman, 2003). It has been further suggested that beta blockers be tapered or changed to short-acting agents to help patients avoid potential intraoperative myocardial depression (Wiesbauer et al., 2007).

Afterload Reducers

It is recommended that patients with mitral regurgitation and heart failure receive an angiotensin-converting enzyme (ACE) inhibitor or sodium nitroprusside (Nipride®) preoperatively to help reduce afterload. These medications should be titrated to achieve a

systolic blood pressure of 90–100 mm Hg (Albert & Antman, 2003).

ACE Inhibitors

While most cardiac medications are not held in the preoperative cardiac surgery patient, ACE inhibitors are typically discontinued 24 to 48 hours before surgery. Continuation of ACE inhibitors may result in intraoperative hypotension, most notably in patients undergoing CABG procedures. The key risk associated with ACE inhibitor discontinuation is arterial graft spasm and increased requirements for vasodilator therapy (Bertrand et al., 2001; Moonesinghe & Kelleher, 2006).

Calcium Channel Blockers

Some preclinical studies suggest that calcium channel blockers interact with inhalation anesthetic agents and some neuromuscular blocking agents. The clinical significance of these data is low, however. While it is agreed that calcium channel blockers should be held in patients undergoing cardiac surgery, the optimal timing for stopping these agents remains a point of contention (Murphy & Wechsler, 2007).

Anticoagulants

Medications affecting hemostasis or bleeding are discontinued in preparation for cardiac surgery. Specifically, warfarin (Coumadin®) is held 2–4 days prior to surgery so that the international normalized ratio (INR) reaches a level less than 2.0. Patients who are at risk for developing thrombosis should receive intravenous heparin when the INR reaches subtherapeutic levels (Dunning et al., 2008). Clopidogrel (Plavix®) is held 7 days before surgery to decrease the risk of excessive intraoperative bleeding and transfusion requirements (Moonesinghe & Kelleher, 2006). Clopidogrel is also associated with a two- to five-fold increase in risk for surgical reexploration and a 30% to 100% increase in blood loss from the chest tube (Dunning et al., 2008). Aspirin irreversibly inhibits platelet function and has been shown to increase perioperative blood loss. Data suggest that a preoperative aspirin-free interval of 2–10 days improves platelet function and reduces blood loss and transfusion requirements. However, if the patient is undergoing urgent cardiac surgery and has acute coronary syndrome, aspirin therapy should continue until the day of surgery (Dunning et al., 2008; Weightman et al., 2002).

Aprotinin (Trasylol®), an anti-fibrinolytic agent that works by inhibiting activation of plasminogen to plasmin, is not recommended for routine use in cardiac surgery because of data indicating a correlation with postoperative renal dysfunction and a likely increase in mortality. This agent may be prescribed for patients at particularly high risk of bleeding in an effort to decrease blood loss and the need for blood transfusions during cardiac surgery. As this issue is currently being reviewed by the Food and Drug Administration, these recommendations may soon change (Dunning et al., 2008).

Current guidelines established by the American College of Chest Physicians recommend heparin prophylaxis for high-risk groups. Other data suggest use of low-molecular-weight heparin (LMWH) as a bridge for patients receiving chronic anticoagulation who are undergoing cardiac surgery (Douketis, Johnson, & Turpie, 2004; Dunning et al., 2008). Use of LMWH is also associated with a low risk for thromboembolic and major bleeding complications (Douketis et al., 2004). Usual practice entails discontinuing unfractionated heparin 4–5 hours before surgery or LMWH 12–24 hours before surgery (Hartsell & Will, 2004).

Heparinization during CABG procedures is performed to prevent intraoperative thrombosis. Complicating the preoperative anticoagulation management issue is the fact that extended preoperative use of LMWH or unfractionated heparin results in a decrease in intraoperative response to

heparin—a phenomenon known as heparin resistance or altered heparin responsiveness. Aside from preoperative heparin infusion, other identified associated factors of heparin resistance include infection and use of IABP therapy (Bar-Yosef, Cozart, Phillips-Bute, Mathew, & Grocott, 2007).

Hypoglycemics

Patients who are taking oral hypoglycemic agents for type II diabetes should have these agents withheld preoperatively for several days. The combination of metformin (Glucaphage®) and sulfonylureas is known to contribute significantly to postoperative morbidity and mortality (Evans, Ogston, Emslie-Smith, & Morris, 2006). Long-acting insulin is usually discontinued preoperatively as well. In contrast, insulin glargine (Lantus®), a long-acting basal insulin, may be continued during the surgical period (Marks, 2003). Other patients who receive insulin therapy may have their dose withheld on the day of surgery, with medication levels being regulated based on blood glucose monitoring.

Statins

Data suggest that statin therapy should be continued through the day of surgery. A reduction in morbidity and mortality has been reported with ongoing use of such medication (Collard, Body, Shernan, Wang, & Mangano, 2006; Durazzo et al., 2004).

Herbal Remedies

Use of herbal remedies can cause increased risk of bleeding and drug interactions. While the medication profile obtained during the preoperative evaluation should include information about the use of herbal remedies, more is being learned about potential interactions between these supplements and other medications every day. As this growing knowledgebase has significant clinical implications for the cardiac surgery patient, no herbal remedies should be taken for at least 2 weeks prior to surgery. Garlic, ginseng, echinacea, ginkgo biloba, St. John's wort, valerian, kava, flavonoids, and grapefruit juice are all known to decrease platelet activity (Hodges & Kam, 2002). Ginseng may also cause hypoglycemia. Kava and valerian may cause an enhanced sedative effect of anesthetic agents. St. John's wort may cause increased metabolism of many of the drugs used in the perioperative period (Ang-Lee, Moss, & Yuan, 2001).

MANAGEMENT OF HIGH-RISK PATIENTS

Ventricular Dysfunction

Preoperative cardiac surgery patients with heart failure and a history of hypertension, ischemia, hypertrophic cardiomyopathy, or acute valvular dysfunction are at risk for, and should be assessed for, ventricular dysfunction (De Marco & McGlothlin, 2005). High morbidity and mortality rates are associated with cardiac surgery in patients who have severe LV dysfunction and clinically significant heart failure secondary to ischemic or valvular heart disease (Kotlyar et al., 2001). Patients with LV dysfunction and valvular disease (e.g., mitral regurgitation, aortic stenosis) require preoperative management of their hemodynamic status. Measures required may include administration of nitroprusside or an ACE inhibitor or use of IABP therapy to stabilize these patients' condition prior to surgery (Albert & Antman, 2003). Nitroprusside has been demonstrated to rapidly stabilize the patient with decompensated heart failure due to severe LV dysfunction and aortic stenosis (Khot et al., 2003).

Patients with acute tricuspid regurgitation secondary to infective endocarditis are at

increased risk for right ventricular dysfunction (Nauser & Stites, 2001). The presence of right ventricular dysfunction also increases patients' perioperative risk (Mathew, Anand, Addai, & Freels, 2001). Patients should be evaluated for evidence of pulmonary hypertension, and those with a pulmonary artery systolic pressure greater than 60 mm Hg should be treated with agents for lowering pulmonary vascular resistance.

Severe chronic ventricular dysfunction occurs in patients with chronic hypertension, or mitral or aortic valve disease, and in those with prior left ventricular MI. These disorders affect left ventricular function, leading to left-sided—and ultimately right-sided—heart failure (De Marco & McGlothlin, 2005).

The presence of heart failure may cause surgery to be delayed while healthcare providers attempt to improve the patient's cardiac function and decrease surgical risk. Therapy focuses on maintaining adequate preload and afterload. Medication or the IABP may be used to augment afterload reduction. In such a case, the nursing evaluation focuses on identifying and optimizing the patient's unstable hemodynamic status.

Infective Endocarditis

Patients with infective endocarditis (IE) require close monitoring for hemodynamic instability and development of multiple organ dysfunction. Given that heart failure influences the prognosis of an individual with IE most significantly, early surgery should be considered in those patients with IE, acute mitral or aortic regurgitation, and signs of heart failure. Other indications for urgent surgery include, but are not limited to, unstable valve prosthesis, gram-negative or fungal endocarditis, major embolism, persistent mobile and large vegetation during the first 2 weeks of antimicrobial therapy, enlarged vegetations or persistent bacteremia or fever after 7–10 days of organism-specific antimicrobial therapy, and valve perforation (Vikram, 2007).

Severe Aortic Stenosis

Patients with severe aortic stenosis who develop LV dysfunction have greater intraoperative and postoperative risk. Data suggest, however, that performing surgery on these patients is safe (Borowski, Ghodsizad, Vchivkov, & Gams, 2007). The patient with aortic stenosis who develops heart failure and decreased ejection fraction likely has symptoms related to increased afterload and alterations in contractility. If immediate surgery is not required, management may include interventions to augment cardiac output with a positive inotrope infusion (e.g., dobutamine) or to decrease peripheral resistance through administration of a vasodilator (Carabello, 2002).

SUMMARY

Patients who present for cardiac surgery have higher levels of complexity than in the past. Often, because of comorbid or concomitant conditions, surgical procedures are combined, creating potentially higher levels of vulnerability and instability. An in-depth preoperative evaluation of the patient's history and cardiac status, along with collection of laboratory data and possibly invasive and noninvasive procedures, is critical to prevent poor outcomes postoperatively. Early detection of potential complications can improve outcomes and help ensure a successful recovery. Critical care nurses are in a unique position to utilize clinical inquiry techniques and critical thinking skills to uncover those risk factors and data that can redirect interventions to become more individual specific.

CASE STUDY

M.J. is a 60-year-old frail female who has four-vessel disease and is scheduled for coronary artery bypass grafting. She is admitted preoperatively for evaluation and to control her blood pressure, as she admits to being forgetful about taking her antihypertensive medications. Her medication profile includes a beta blocker, ACE inhibitor, proton pump inhibitor, Glucaphage® (metformin), and an antidepressant. M.J. has an allergy to cephalosporins. Her medical history includes type II diabetes for 5 years, atherosclerosis, and alcohol abuse. The patient is very nervous and is concerned about the pain she will experience postoperatively.

Critical Thinking Questions

1. Given the history of this patient, which potential postoperative problems might the nurse expect.
2. Should sleeping medication be given to this patient the night before surgery?
3. Does this patient have a higher risk of bleeding?
4. Which type of antibiotic should be given to this patient prior to surgery to prevent infection given her history?

Answers to Critical Thinking Questions

1. Given that she is frail, M.J. might have nutritional deficiencies that could affect her recovery. She is also noncompliant with medication, requiring intervention prior to her cardiac surgery. Diabetes is another risk factor associated with morbidity.
2. Patients can have ischemia with stress. Medication to facilitate sleep the night before surgery can help to decrease stress. At least 40% of patients become ischemic preoperatively if good premedication is not provided (Adams & Antman, 2001).
3. More than likely given her history of alcohol abuse. Laboratory findings would be useful in identifying M.J.'s precise level of risk.
4. Vancomycin is the agent of choice for patients with a penicillin or cephalosporin allergy and to allay concerns about the potential for infection with methicillin-resistant *Staphylococcus aureus* and *S. epidermis* (Lemmer, Richenbacher, & Viahakes, 2003).

SELF-ASSESSMENT QUESTIONS

1. During the first preoperative meetings, the nurse evaluates the patient and family for all the following *except*
   a. understanding of the underlying illness.
   b. planned course of cardiac surgery.
   c. ability to comply with surgical regimen.
   d. satisfaction with the facility.
2. The nurse is interested in any aspects of the cardiac surgery patient's history that may affect the antiplatelet regimen after revascularization. Which of the following histories would be a cause for concern?
   a. Peptic ulcer disease
   b. Overactive bladder disease
   c. Migraines
   d. Fractured tibia

3. During the preoperative nutritional assessment, the nurse should question the patient on weight, diet, and food preferences. Which of the following data should be reported as being a source of concern?
   a. Unintentional weight loss
   b. BUN 26 mg/dL
   c. High-protein diet to lose weight
   d. Six caffeinated drinks daily
4. A carotid bruit is
   a. common in individuals older than age 70 years.
   b. loudest in the upper third of the carotid artery.
   c. heard best during normal breathing.
   d. an indication of cerebral dementia.
5. Which condition would prevent the use of an internal thoracic artery?
   a. Asthma
   b. Dental infection
   c. Mastectomy
   d. Diabetes
6. Atrial fibrillation occurs frequently in patients, especially in those with
   a. tricuspid valve disease.
   b. aortic valve disease.
   c. mitral valve disease.
   d. pulmonic valve disease.
7. Which of the following medications would you consider holding on the day of surgery?
   a. Proton pump inhibitor
   b. ACE inhibitor
   c. Statin
   d. Antidepressant
8. A patient scheduled for a CABG will have blood sent for type and crossmatch. Which factor increases the probability that the patient will have antibodies?
   a. Previous transfusions
   b. Null parity
   c. Steroid utilization
   d. MRSA carrier
9. The onset of accelerating angina warrants assessment and diagnostic evaluation in the pre-cardiac-surgery patient to rule out a myocardial infarction. As a nurse, you know that the best outcome is expected if cardiac surgery occurs within _____ hours of the MI.
   a. 4 b. 8
   c. 12 d. 24
10. Which of the following patients is at highest risk for right ventricular dysfunction?
    a. A patient with four-vessel disease
    b. A patient with a septal aneurysm
    c. A patient with mitral valve stenosis
    d. A patient with acute tricuspid regurgitation

Answers to Self-Assessment Questions

1. d
2. a
3. a
4. b
5. c
6. c
7. b
8. a
9. a
10. d

Clinical Inquiry Box

Question: How does obesity affect mortality after cardiac surgery?

Reference: Rockx, M. A., Fox, S. A., Stitt, L. W., Lehnhardt, K. R., McKenzie, F. N., Quantz, M. A., et al. (2004). Is obesity a predictor of mortality, morbidity and readmission after cardiac surgery? *Canadian Journal of Surgery*, *47*(1), 34–38.

Objective: To determine whether obesity is a predictor of mortality, morbidity, or early readmission to hospital.

Methods: A retrospective study was undertaken that included 1310 patients who had cardiac surgery between 1999 and 2002 in an academic hospital. Outcome variables such as stroke, reoperation for bleeding, life-threatening cardiac arrest or arrhythmia, new renal failure requiring dialysis, septicemia, mediastinitis, sternal dehiscence, respiratory failure, postoperative myocardial infarction, and low cardiac output necessitating intra-aortic balloon pump use were correlated with body mass index (BMI).

Results: An increased BMI was associated with a higher likelihood of readmission to hospital within 30 days of discharge and sternal wound dehiscence. However, an increased BMI did not increase the risk of early postoperative death, stroke, reoperation for bleeding, life-threatening cardiac arrest or arrhythmia, new renal failure requiring dialysis, septicemia, mediastinitis, respiratory failure, postoperative MI, or low cardiac output necessitating IABP use.

Conclusion: Obesity was not associated with adverse outcomes after cardiac operations, except for an increased risk of sternal dehiscence and early hospital readmission. Although obesity is often perceived as a major risk factor for cardiac surgery, this study did not find as high a correlation as one might expect. Nurses providing care to obese patients must be aware of the increased risk of sternal dehiscence and the potential for hospital readmission, however, and prevention strategies should be utilized to mitigate these risks.

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Chapter 5

Heart Valve Surgery

Catherine Drumm, Kristine J. Peterson

INTRODUCTION

Heart valve surgery is performed to either repair or replace a failing valve. Cardiac valves allow for one-way, low-resistance blood flow. The opening and closing of a valve occur according to pressure gradients between each side of the valve. The valves must open widely to allow for rapid blood movement and minimal cardiac work; conversely, they must remain tightly closed to prevent backward flow of blood. Proper functioning of cardiac valves depends on normal fibroelastic tissue of the valve leaflets, proper number of cusps of the valve, ability to open and close rapidly, normal-sized ring or annulus, and proper function of chordae tendinae and papillary muscles (mitral and tricuspid) (Fann, Ingels, & Miller, 2008; Mihaljevic, Sayeed, Stamou, & Paul, 2008). This chapter describes the various valve surgery procedures and their associated care implications.

VALVULAR HEART DISEASE

Valvular heart disease (VHD) is defined according to the valve or valves affected and the type of functional alteration. Abnormality of the valve is identified as either stenosis (narrowing or constriction that creates a pressure gradient) or regurgitation (incomplete closure of the valve leaflets resulting in a backflow of blood).

Valvular heart disease may be caused by either congenital or acquired factors. Congenital factors include a bicuspid rather than tricuspid valve, and other congenital malformations. For example, a congenital condition found in adults is Marfan syndrome, a connective tissue disorder. In Marfan syndrome, the chemical makeup of the connective tissue supporting the heart valves is abnormal. As a consequence, the valve leaflets may not remain tightly closed, and the result is a backward flow of blood. This dysfunction increases myocardial workload and may ultimately lead to an enlarged ventricle. Marfan syndrome is most clinically significant when the mitral or aortic valve is affected (American Heart Association, 2008).

Acquired causes of valve disease include ischemic coronary artery disease (CAD), degenerative changes associated with aging, rheumatic changes, infective endocarditis from a bacterial infection, neoplasm, or thrombus (Fann et al., 2008; Hill, 2007; Mihaljevic et al., 2008).

The relationship between ischemic CAD and VHD is bidirectional. On the one hand, myocardial infarction due to CAD can result in ventricular remodeling (a pathological change in the shape and size of the ventricle). Chordae tendinae, papillary muscle, and the valve annulus may be affected by ischemia as well, leading to impaired valve function. On

the other hand, a malfunctioning valve will cause an increase in myocardial workload and can eventually lead to ischemia as a symptom of valve disease.

Stenosis or regurgitation can occur with any valve when rheumatic changes are the underlying etiology. These changes may be present for many years before the patient manifests symptoms. Degeneration of valves due to aging occurs when fibrous material and calcium are deposited in and around leaflets, resulting in their malfunction. Data suggest that the etiology of aortic valve disease is becoming less frequently related to rheumatic disease (RD) and more frequently attributable to age-related degenerative changes. In one study, while RD remained the most common cause of aortic stenosis, its incidence was found to be decreasing. Conversely, the incidence of valve malfunction as a result of degenerative changes was found to be increasing. Correspondingly, the frequency of RD, which was the leading cause of aortic regurgitation (AR) in this study, was found to be decreasing. The incidence of aortic regurgitation related to degenerative changes did not change in this study (Matsumura et al., 2002).

Infective endocarditis occurs when bacteria attach to and destroy the surface of a valve leaflet or chordae. If a valve is damaged, immune cells, platelets, and fibrin migrate to the site to initiate healing of the valve. If bacteria become trapped under layers of these cells, "clumps" of tissue (vegetations) can develop on the valves and within the heart muscle, leading to endocarditis. Vegetations may also break off and become emboli (Homma & Grahame-Clarke, 2003).

Decisions about whether to pursue medical or surgical management and which type of surgical management to use, if necessary, are based on the goals of maximizing the life of the valve and minimizing complications of treatment (Shemin, 2008). Often the decision to repair or replace a valve is made once the surgeon has an opportunity to visualize the valve. Nevertheless, patients are educated on both repair and replacement procedures. Pros and cons exist for both mechanical and biological valves. Therefore, the surgeon and the patient together decide which type of valve will be used for replacement.

Mechanical valves are believed to be more durable than bioprosthetic valves, but require that patients receive lifelong anticoagulation therapy. The introduction of International Normalized Ratio (INR) self-testing, however, should reduce the incidence of thromboembolic and hemorrhagic complications related to the use of mechanical valves (Thompson et al., 2008).

AORTIC STENOSIS

Aortic stenosis is the most common adult valve lesion in the United States. Its usual causes are a bicuspid valve and degenerative calcification (Gaasch, 2007a), with the latter etiology being the most frequent cause (Carabello, 2004; Mihaljevic et al., 2008). Given that the U.S. population is aging, the incidence of aortic stenosis is increasing. Long thought to be a disease of stress and degenerative changes, the calcification of aortic stenosis is now regarded as a proliferative and inflammatory process, similar to atherosclerosis (Carabello, 2004; Mihaljevic et al., 2008).

Aortic stenosis, in which the aortic valve does not open completely, creates a left ventricular outflow tract obstruction and increases workload and afterload of the left ventricle (LV). The increase in afterload is the etiology of the signs and symptoms associated with aortic stenosis (LeBoutillier & DiSesa, 2003).

Factors involved in grading the severity of aortic stenosis include the mean systolic gradient across the valve, blood velocity, valve area, LV function, and severity of symptoms (Bonow et al., 2006; Carabello, 2004; Mihaljevic et al., 2008). Normally the pressures in the

LV and the aorta are virtually equal during systole, meaning there is no aortic systolic gradient. As the valve opening narrows, however, the pressure required to eject blood—and therefore the pressure in the LV—increases, creating a gradient. The normal aortic valve area is 2.6–3.5 cm^2 (Mihaljevic et al., 2008). As the valve area narrows and the gradient increases, blood velocity increases. Mild aortic stenosis is associated with a mean gradient of less than 25 mm Hg, valve area of greater than 1.5 cm^2, and jet velocity of less than 3 m/sec. Severe aortic stenosis is associated with jet velocity of more than 4 m/sec, mean systolic gradient greater than 40 mm Hg, and a valve area of less than 1.0 cm^2 (Bonow et al., 2006; Mihaljevic et al., 2008).

Classic signs and symptoms of aortic stenosis include angina, syncope, sudden cardiac death, and heart failure. Typically, these conditions appear only after a prolonged latent period, when the disease is already severe. Most patients have left ventricular hypertrophy. Initially, myocardial contractility and ejection fraction are not affected. Ultimately, however, the patient becomes symptomatic when the LV begins to fail. A systolic murmur may be audible in the aortic area. Symptoms of heart failure (e.g., crackles, edema) may be present (Gaasch, 2007a; LeBoutillier & DiSesa, 2003).

Once patients become symptomatic, if jet velocity is greater than 4 m/sec, there is a reported event-free survival of 21% (Bonow et al., 2006). In patients with heart failure, time from onset of symptoms to death is 2 years; in those with angina, time from onset of symptoms to death is 5 years (Mihaljevic et al., 2008). Medical therapy may improve symptoms of heart failure but is not effective long-term therapy for aortic stenosis (Bonow et al., 2006; Mihaljevic et al., 2008). Table 5–1 outlines the American College of Cardiology/

Table 5–1 Indications for Aortic Valve Replacement in Aortic Stenosis

Class I

1. Symptomatic patients with severe AS
2. Patients with severe AS undergoing CABG
3. Patients with severe AS undergoing surgery on aorta or other heart valves
4. Severe AS and LV systolic dysfunction (EF < 50%)

Class IIa

1. Patients with moderate AS undergoing CABG or surgery on aorta or other heart valves

Class IIb

1. Asymptomatic patients with severe AS and abnormal response to exercise (symptoms or fall in blood pressure with exercise)
2. Adults with severe asymptomatic AS if there is a high likelihood of rapid progression (age, calcification, and CAD) or if surgery might be delayed at the time of symptom onset
3. Patients undergoing CABG with mild AS when there is evidence that progression may be rapid
4. Asymptomatic patients with extremely severe AS when expected mortality is 1% or less

Class III

1. Not recommended for prevention of sudden death in asymptomatic patients with AS who have none of the Class IIa/IIb indications

AS = aortic stenosis; CABG = coronary artery bypass grafting; CAD = coronary artery disease; EF = ejection fraction; LV = left ventricle.

Sources: Bonow et al., 2006; Mihaljevic, Sayeed, Stamou, & Paul, 2008; Vahanian et al., 2007.

American Heart Association (ACC/AHA) recommendations for aortic valve replacement (AVR) in the presence of aortic stenosis.

As discussed in Chapter 3, the AHA classifies recommendations based on the degree of agreement and type and/or amount of available evidence. The level of recommendation is classified as Class I, II, or III. Class I indicates that evidence and/or general agreement exists that the intervention is effective. Class II refers to conflicting evidence and/or a divergence of opinion about the efficacy. Class II is further subdivided into Class IIa and Class IIb: Class IIa indicates that evidence/opinion is in favor of efficacy, whereas Class IIb recommendations have less efficacy as established by evidence/opinion. Class III refers to evidence and/or general opinion that an intervention is not effective.

The strength of the level of evidence is also identified according to the type and/or presence of research. For example, Level of Evidence A indicates that findings from multiple randomized clinical trials or meta-analyses supported the use of an intervention. Level of Evidence B indicates that a single randomized trial or nonrandomized trials supported an intervention. Level of Evidence C refers to consensus opinion of experts, case studies, or standard of care (Eagle et al., 2004).

A new option for the treatment of severe aortic stenosis in patients who are considered to be at high risk and inoperable is percutaneous aortic valve replacement. First performed in 2002, this investigational procedure involves insertion of a tri-leaflet bioprosthesis made from equine pericardium (Edwards Lifesciences, Irvine, California). The device is mounted on a balloon catheter and delivered through the arterial system via a guidewire. After predilatation of the native valve, the device is inserted into the midpoint of the native valve. Placement of the prosthetic valve takes place in the cardiac catheterization lab under fluoroscopy. A femoral arterial retrograde or femoral vein antegrade/transseptal approach is used for placement. The patient typically remains hospitalized 2 to 4 days post-procedure (Grube et al., 2007; Lauck, Mackay, Galte, & Wilson, 2008).

Postoperatively, the ICU nurse must be vigilant with blood pressure monitoring. Aortic stenosis may result in left ventricular hypertrophy, increased afterload, and a noncompliant left ventricle. Following valve replacement for aortic stenosis, the left ventricle may not anticipate the reduction in afterload and continue to pump hard. Avoiding hypertension is essential to avoid disrupting suture lines (Khalpey, Ganim, & Rawn, 2008).

In other patients, the LV hypertrophy that is present may result in outflow obstruction, and postoperative hemodynamic instability may subsequently occur as a result of preload reduction or if the patient develops bradycardia or a heart block. Treatment in this case entails volume repletion, administration of beta blockers, and increasing afterload. Connecting intraoperatively placed pacing wires to an external generator may be anticipated (Khalpey et al., 2008).

AORTIC REGURGITATION

When aortic regurgitation is present, there is a reflux of blood from the aorta into the LV during diastole because the valve leaflets fail to close completely and to remain tightly closed during diastole. Acute aortic regurgitation imposes a large volume load that a normal LV cannot accommodate. The sudden increase in end-diastolic volume (preload) will result in increased left ventricular end-diastolic pressure (LVEDP) and decreased cardiac output. Patients with concomitant CAD may develop left ventricular dilation and cardiac failure. Such patients often present with heart failure. Symptoms of aortic regurgitation depend on the acuity of onset, severity of regurgitation, and left ventricular function. As LV dilation occurs over time, patients may be asymptomatic for long periods. Most patients will have an audible diastolic blowing murmur (LeBoutillier & DiSesa, 2003).

Causes of aortic regurgitation include conditions that prevent the valve cusps from aligning properly and conditions that dilate the aortic annular ring. Such conditions include idiopathic degeneration, calcific aortic disease, rheumatic disease, endocarditis, bicuspid valve, and dissection of the ascending aorta. Other causes of aortic regurgitation include trauma, chronic systemic hypertension, aortitis of various etiologies, and connective tissue disease such as Marfan syndrome, Reiter disease, Ehlers-Danlos syndrome, and rheumatoid arthritis (Bonow et al., 2006; Mihaljevic et al., 2008). Most commonly, aortic regurgitation is seen concomitantly with aortic stenosis (e.g., aortic disease, rheumatoid disease, or degenerative disease) (Mihaljevic et al., 2008).

Indications for Aortic Valve Replacement in Aortic Regurgitation

Patients with acute aortic regurgitation will develop hemodynamic instability and LV failure. Even small regurgitant volumes will cause a large increase in LVEDP. The result is low cardiac output, high LV end-diastolic volumes (LVEDV), and increased heart rate. Any changes in diastolic filling or heart rate can disturb the balance and result in early LV failure (Mihaljevic et al., 2008).

Like chronic aortic stenosis, chronic aortic regurgitation has a slow, insidious onset and progression. Aortic regurgitation may be well tolerated for years. Because it develops slowly, the LV compensates with hypertrophy and an increase in sympathetic tone to keep the LVEDP relatively low and maintain cardiac output. This change results in a characteristic sign of aortic regurgitation, a widened pulse pressure (Mihaljevic et al., 2008). If left untreated, this process will lead eventually to myofibril slippage, ventricular remodeling, and irreversible changes in LV function.

While acute aortic regurgitation should be treated with early valve replacement, valve replacement is not recommended for asymptomatic patients with chronic aortic regurgitation and good LV function (Mihaljevic et al., 2008). A ratio of chamber volume to wall thickness and measurements of LV end-systolic volume are used to guide surgery decisions in chronic aortic regurgitation. Deteriorating LV function, as indicated by an ejection fraction (EF) less than 50–55% and an end-diastolic dimension greater than 70 mm or an end-systolic dimension greater than 50 mm, would indicate need for surgery (Mihaljevic et al., 2008). Table 5–2 lists the indications for surgery for aortic regurgitation.

Postoperatively, because patients have dilated ventricles from the aortic regurgitation and its associated aortic insufficiency, they may require administration of intravenous vasodilators. Agents such as milrinone (Primacor®) and dobutamine (Dobutrex®) may be indicated for inotropic support and to promote ventricular emptying. The intra-aortic balloon pump (IABP) also may be used (LeBoutillier & DiSesa, 2003); it is discussed in detail in Chapter 10. It is often challenging to optimize a patient's hemodynamic status following surgery to correct aortic regurgitation (Khalpey et al., 2008).

MITRAL STENOSIS

Mitral stenosis, like aortic stenosis, is a condition where the valve leaflets do not open completely, creating resistance to the forward flow of blood into the LV during diastole. Mitral stenosis is predominantly caused by rheumatic heart disease (Bonow et al., 2006; Fann et al., 2008). Other causes, which are less common, include left atrial myxoma, thrombus, annular calcification, endocarditic vegetation, malignant carcinoid syndrome, and metabolic disorders (Bonow et al., 2006; Fann et al., 2008).

Most commonly, rheumatic disease is acquired in childhood; however, mitral stenosis does not usually become symptomatic

Table 5–2 Indications for Aortic Valve Replacement in Aortic Regurgitation

Class I

1. Symptomatic patients with severe AR irrespective of LV function (Level B)
2. Asymptomatic patients with chronic, severe AR and LV systolic dysfunction (EF < 50%) (Level B)
3. Chronic pure, severe AR while undergoing CABG or surgery on the aorta or other heart valves (Level C)

Class IIa

1. Asymptomatic patients with pure, severe AR and normal LV systolic function but with severe LV dilatation (end-diastolic dimension > 75 mm or end-systolic dimension > 55 mm) (Level B)

Class IIb

1. Patients with moderate AR while undergoing surgery on the ascending aorta (Level C)
2. Patients with moderate AR while undergoing CABG (Level C)
3. Asymptomatic patients with severe AR and normal LV function at rest (EF > 50%) when end-diastolic dimension > 70 mm or end-systolic dimension = 50 mm and evidence of progressive LV dilatation, decreasing exercise tolerance, or abnormal hemodynamic response to exercise (Level C)

Class III

1. Not indicated for asymptomatic patients with normal LV function at rest (EF > 50%) when dilatation is not moderate or severe based on end-systolic and end-diastolic dimensions (Level B)

AR = aortic regurgitation; CABG = coronary artery bypass grafting; EF = ejection fraction; LV = left ventricle.
Sources: Bonow et al., 2006; Vahanian et al., 2007.

until decades later. The valve leaflets gradually become thickened and calcified. Often, the chordae and commissures fuse (Bonow et al., 2006; Fann et al., 2008; Todd & Higgin, 2005). Left atrial pressure (LAP) rises as the disease worsens and a progressively higher gradient develops across the mitral valve. Pulmonary artery systolic pressure increases as the valve area narrows. Defining characteristics of severe mitral stenosis include a gradient of greater than 10 mm Hg, LAP greater than 15 mm Hg, valve area less than 1.0 cm^2, and pulmonary artery systolic pressure greater than 50 mm Hg (Bonow et al., 2006; Fann et al., 2008).

Because of the resistance to the forward flow of blood, patients with mitral stenosis will not develop volume overload in the LV and will likely have satisfactory LV function. These individuals, however, have pulmonary hypertension, right ventricular failure, and tricuspid insufficiency. Symptoms of low cardiac output and pulmonary venous congestion develop as left atrial and pulmonary pressures rise. At first, symptoms may occur only on exertion. As the valve area narrows, symptoms occur with less exertion, emotional stress, or atrial fibrillation (AF). Dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, and fatigue are the first symptoms to occur. Once pulmonary hypertension develops, right-sided heart failure with edema, hepatomegaly, ascites, and tricuspid regurgitation are seen. The ECG may reveal right ventricular hypertrophy (LeBoutillier & DiSesa, 2003).

Intervention is warranted for symptomatic patients with moderate or severe mitral stenosis, and it may be indicated for asymptomatic patients with new-onset AF; in contrast, intervention is not usually considered for patients with mild mitral stenosis (Bonow et al., 2006; Fann et al., 2008; Sorrentino, 2007). The presence of pulmonary hypertension, low cardiac output, and right-sided heart failure warrants further evaluation (Fann et al., 2008). Individuals with the latter symptoms and concomitant calculated valve area of less than 1 cm^2 should undergo surgical correction of the mitral valve (LeBoutillier & DiSesa, 2003).

Mitral Commissurotomy versus Mitral Valve Replacement

Percutaneous mitral balloon valvotomy (PMBV), also known as commissurotomy, has very successfully reduced left atrial gradient, increased mitral valve area, and improved symptoms in patients with mitral stenosis (Bonow et al., 2006; Carabello, 2004; Farhat et al., 1998; Sorrentino, 2007). Percutaneous intervention is recommended for patients with pliable, noncalcified valves; minimal chordae fusion; no atrial thrombus; and no mitral regurgitation (Bonow et al., 2006; Farhat et al., 1998; Sorrentino, 2007). If the patient is a high-risk surgical candidate, PMBV may be a viable alternative, even if the valve anatomy is not ideal (Sorrentino, 2007). Percutaneous intervention has been shown to result in more favorable outcomes than closed surgical commissurotomy (Farhat et al., 1998). Open or surgical commissurotomy is associated with a significantly higher reoperation rate at 10 years than mitral valve replacement (Fann et al., 2008; Wiegand, 2003).

Mitral Valve Replacement and Repair

Once the decision is reached for surgical intervention, many factors must be considered when deciding which technique is optimal. These factors include patient age at time of replacement, presence of sinus rhythm, desire to avoid anticoagulation, contraindications to anticoagulation, high-risk occupation or lifestyle, size and condition of the annulus, comorbidities, and desire to become pregnant (Gudbjartsson, Absi, & Aranki, 2008). In general, mitral valve repair is better suited to treating mitral regurgitation than as a therapy for mitral stenosis (Bonow et al., 2006; Gudbjartsson et al., 2008). Table 5–3 lists the indications for mitral valve replacement and repair in mitral stenosis.

Postoperatively, the ICU nurse should assess the patient's level of pulmonary hypertension by comparing it with the preoperative level. The more significant the pulmonary hypertension, the greater the likelihood for postoperative right ventricular (RV) failure. Increased central venous pressure is an indication of possible RV decompensation (LeBoutillier & DiSesa, 2003).

Performing a transesophageal echocardiogram postoperatively will assist in the assessment of right and left ventricular function. Administration of dobutamine or milrinone in combination with norepinephrine (Levophed®) may be indicated to enhance contractility of the right ventricle and decrease pulmonary vascular resistance (right-sided afterload). Prudent fluid administration in combination with inotropic support should augment cardiac output in these patients. As an IABP does not affect right ventricular function, its postoperative use in patients who have undergone mitral valve repair is usually not indicated. Use of a right ventricular assist device may be indicated in the immediate postoperative period (Khalpey et al., 2008; LeBoutillier & DiSesa, 2003).

MITRAL REGURGITATION

In mitral regurgitation, the valve leaflets do not close tightly, resulting in a backward jet

Table 5–3 Indications for Mitral Valve Repair or Replacement in Mitral Stenosis

Class I

Consider mitral valve repair if possible over replacement.

1. Symptomatic (NYHA functional Class III–IV) patients with moderate or severe MS when PMBV is not available or contraindicated due to left atrial thrombus or valve morphology is not favorable for PMBV (Level B).
2. Symptomatic patients with moderate to severe MS and moderate to severe mitral regurgitation should have MVR unless valve repair is possible (Level C).

Class IIa

1. MVR is indicated for severe MS and severe pulmonary hypertension (PASP > 60 mm Hg) with NYHA Class I–II symptoms when patients are not candidates for PMBV or repair (Level C).

Class IIb

1. Repair may be considered for asymptomatic patients with moderate or severe MS who have had recurrent embolic events while on adequate anticoagulation and who have valve anatomy favorable for repair (Level C).

Class III

1. Not indicated for mild MS.
2. Closed commissurotomy should not be done in patients receiving mitral valve repair (open commissurotomy preferred) (Level C).

MS = mitral stenosis; MVR = mitral valve repair; NYHA = New York Heart Association; PASP = pulmonary artery systolic pressure; PMBV = percutaneous mitral balloon valvotomy.

Source: Adapted from Bonow et al., 2006.

of blood into the left atrium during ventricular systole. Proper function of the mitral valve depends on a complicated interaction between the mitral leaflets, annulus, chordae tendinae, papillary muscles, and the left atrium and ventricle (Fann et al., 2008).

The most common causes of mitral regurgitation include ischemic CAD, mitral valve prolapse syndrome, infective endocarditis, rheumatic heart disease, mitral annular calcification, dilated cardiomyopathy, congenital anomalies, and collagen vascular disease (Bonow et al., 2006; Fann et al., 2008). As with other valvular lesions, a sudden cause—such as ruptured papillary muscle or chordae tendinae—will result in acute and severe mitral regurgitation. As noted in Chapter 3, ventricular failure and dilation will result in mitral regurgitation. This is often a preterminal event and carries a survival time of 6 to 24 months (Spoor & Bolling, 2008). Alternatively, mitral regurgitation may progress slowly over time, with symptoms appearing only when the disease is very advanced.

Mitral regurgitation causes an increase in LVEDP and a decrease in afterload. An enlarged left atrium is likely as well. If mitral regurgitation develops suddenly, cardiac output will decrease. Patients may present with signs of severe heart failure, and the ECG may reveal findings consistent with ischemia. Patients may also have AF and associated decrease in cardiac output related to the enlarged left atrium (LeBoutillier & DiSesa, 2003).

The decision to perform corrective surgery is based on a number of factors, such as the

degree of mitral regurgitation, severity of symptoms, left ventricular function, feasibility of valve repair, presence of AF, presence and degree of pulmonary hypertension, and patient expectations (Gaasch, 2007b). Severe mitral regurgitation is characterized by the following findings:

- Jet area ≥ 7 mm
- Regurgitant volume ≥ 60 mL/beat
- Regurgitant fraction ≥ 50%
- Regurgitant orifice area ≥ 0.4 cm^2
- Enlarged left atrium and ventricle (Bonow et al., 2006; Gaasch, 2007b)

Asymptomatic patients with severe mitral regurgitation can be safely followed for some time (Gaasch, 2007b; Rosenhek et al., 2006).

Care in the immediate postoperative period may be challenging. Upon repair of the mitral valve for mitral regurgitation, the left atrium will no longer be receiving regurgitant blood from the LV and the patient will experience an immediate increase in afterload (systemic vascular resistance [SVR]). Further compounding the potential for cardiac dysfunction postoperatively are pulmonary hypertension and effects of myocardial hibernation (discussed in Chapter 13) that take time to be reversed. Patients, therefore, are at risk for the development of right ventricular failure (LeBoutillier & DiSesa, 2003).

Patients may require administration of inotropes (e.g., milrinone, dobutamine) or IABP therapy to reduce afterload. Combination therapy of milrinone and epinephrine may also be used to accomplish afterload reduction, provide an inotropic effect, and decrease pulmonary hypertension (Khalpey et al., 2008).

Patients should be monitored for right ventricular failure. If they develop decreased blood pressure, cardiac output, pulmonary artery pressures, and pulmonary artery occlusive pressure, or elevated central venous pressure, right ventricular failure should be suspected (LeBoutillier & DiSesa, 2003).

A number of methods are used in mitral valve repair, including annuloplasty, open commissurotomy, and primary, anterior, and posterior leaflet repair techniques. Selection of the technique is based on the type, extent, and location of the defect of the mitral valve. A preoperative echocardiogram provides the surgeon with these data (Savage & Bolling, 2005).

Because of the relationships among the mitral valve apparatus, ventricular geometry, and ventricular function, mitral valve restoration can improve ventricular function. In a procedure known as geometric mitral reconstruction (GMR), an annuloplasty ring is used to restore a more normal mitral valve anatomy; this technique has achieved favorable outcomes. Specifically, GMR has consistently resulted in significant improvements in mean EF and a reduction in NYHA symptomatology (Radovanovic et al., 2002; Spoor & Bolling, 2008; Spoor, Geltz, & Bolling, 2006). GMR is indicated for patients with cardiomyopathy and mitral regurgitation.

The efficacy of medical therapy for asymptomatic mitral regurgitation is the topic of ongoing debate; however, diuretics, digoxin, and arterial vasodilators may be used to decrease ventricular size, regurgitant orifice size, and regurgitant volume (Bonow et al., 2006; Fann et al., 2008). The presence of left ventricular enlargement, LV dysfunction, pulmonary hypertension, or recurrent AF indicates the need for surgery (Rosenhek et al., 2006). Table 5–4 outlines the indications for surgical correction of mitral regurgitation.

Increased mortality after mitral valve surgery has been found among perimenopausal women. The higher mortality rate is thought to be associated with a state of estrogen withdrawal that may trigger inflammatory responses; these responses may, in turn, potentiate ischemia-reperfusion injury (Song et al., 2008).

Table 5–4 Indications for Surgery for Mitral Regurgitation

Class I

1. Symptomatic patients with severe MR (Level B)
2. Chronic, severe MR and NYHA functional Class II–IV symptoms in the absence of severe LV dysfunction (EF < 30% and/or LV end-systolic dimension > 55 mm) (Level B)
3. Asymptomatic patients with chronic, severe MR and mild to moderate LV dysfunction (EF = 30–60% and/or LV end-systolic dimension ≥ 40 mm) (Level B)
4. MV repair is preferred over MVR for the majority of patients (Level C).

Class IIa

1. MV repair for asymptomatic patients with chronic, severe MR and preserved LV function, and when the likelihood of successful repair without residual MR is > 90% (Level B)
2. Asymptomatic patients with chronic, severe MR, preserved LV function, and new-onset atrial fibrillation (Level C)
3. Asymptomatic patients with chronic, severe MR, preserved LV function, and pulmonary hypertension (PASP > 50 mm Hg at rest or > 60 mm Hg with exercise) (Level C)
4. Patients with chronic, severe MR due to primary abnormality of the mitral valve apparatus, NYHA functional Class III–IV symptoms, severe LV dysfunction, and in whom mitral valve repair is highly likely (Level C)

Class IIb

1. Patients with chronic, severe MR due to severe LV dysfunction with NYHA functional Class III–IV symptoms despite optimal medical therapy for heart failure (Level C)

Class III

1. Not indicated for patients with asymptomatic MR with preserved LV function and doubtful feasibility of repair
2. Not indicated for patients with mild or moderate MR (Level C)

EF = ejection fraction; LV = left ventricular; MR = mitral regurgitation; MVR = mitral valve replacement; NYHA = New York Heart Association; PASP = pulmonary artery systolic pressure.

Source: Adapted from Bonow et al., 2006.

Mitral Valve Repair versus Mitral Valve Replacement

Prosthetic valves are categorized as mechanical or biologic (tissue) valves. Mechanical valves are manufactured from man-made materials such as metal alloys, pyrolite carbon, and Dacron. Biologic valves are constructed from bovine, porcine, and human cardiac tissue, although they may contain some man-made materials. Mechanical prosthetic valves are more durable and last longer than biologic valves, but they carry an increased risk of venous thrombotic events, necessitating long-term anticoagulation therapy. Biologic valves do not require anticoagulation therapy, but they are less durable due to their tendency toward early calcification, tissue degeneration, and stiffening of the leaflets.

Advantages and disadvantages of mitral valve replacement with either a prosthetic or mechanical valve must be carefully weighed by the patient. Indications for either type of valve vary by patient characteristics and surgeon preference (Gudbjartsson et al., 2008).

Because of the disadvantages associated with prosthetic valves, surgeons have been interested in valve repair for some time. The

decision to repair a valve as opposed to replacing it depends on the degree of regurgitation, pathophysiology of the regurgitation, LV function, and ability of the surgeon (Chen & Cohn, 2008). Good evidence suggests that mitral valve repair, if feasible, results in decreased operative mortality, greater increase in LV ejection fraction, improved survival, enhanced preservation of LV function, durability of repair, and decreased incidence of venous thrombotic events (Chen & Cohn, 2008; Flameng, Herijgers, & Bogaerts, 2003; Gaasch, 2007b). It is postulated that the improved outcomes are attributable to retention of the mitral valve apparatus (leaflets, annulus, chordae, and papillary muscles), which helps to preserve LV function (Chen & Cohn, 2008; Flameng et al., 2003). The benefit may be less in older patients or patients undergoing concomitant CABG (Gaasch, 2007b). Valve repair is currently the treatment of choice for mitral regurgitation (Chen & Cohn, 2008).

TRICUSPID VALVE DISEASE

The tricuspid valve has an annular ring and three leaflets connected via chordae tendinae to papillary muscles that are integrated with the right ventricle. It is located between the right atrium and ventricle, near the atrioventricular (AV) node, right coronary artery, and coronary sinus. Its function is to maintain forward flow of blood.

Tricuspid Regurgitation

The functional defects that are seen in tricuspid disease are classified as either primary or secondary. Primary valve disease is caused by conditions that affect valve anatomy—for example, congenital abnormalities, rheumatic disease, infective endocarditis, toxicities, tumor, and blunt trauma. Secondary tricuspid disease can result from right ventricular pathology, pulmonary hypertension, increased right ventricular systolic pressure (especially if greater than 55 mm Hg), mitral or aortic valve disease (that results in elevated LAP and LVEDP), left-sided heart failure, dilated cardiomyopathy, tricuspid annular dilatation, or pulmonary embolism (Bonow et al., 2006; Phillips, 2005; Shah & Raney, 2008). Occasionally, wires inserted through the valve such as an automatic implantable cardioverter defibrillator (AICD) or pacemaker may cause tricuspid regurgitation (Bonow et al., 2006; Shemin, 2008). Following the initial classification, tricuspid valve disease is addressed according to the pathology—either tricuspid stenosis or tricuspid regurgitation (Phillips, 2005).

Symptoms of tricuspid regurgitation include fatigue, weakness, signs of right-sided heart failure, abnormal venous pulsations, and often AF. Tricuspid stenosis most often presents with some degree of tricuspid regurgitation as well. It is characterized by signs of peripheral venous distention, abnormal venous pulsations, fatigue, malaise, and signs of low cardiac output (Shemin, 2008).

Tricuspid Stenosis

Patients with tricuspid stenosis have an obstruction to blood flow from the right atrium to the right ventricle. The most common etiology for tricuspid stenosis is rheumatic heart disease. Other conditions associated with tricuspid stenosis include carcinoid syndrome, endocarditis, and intracardiac tumors. The clinical presentation of tricuspid stenosis is logically consistent with right-sided heart failure—that is, it consists of decreased cardiac output, fatigue, anasarca, hepatomegaly, and ascites (Bashore, 2006; Phillips, 2005).

Surgery for Tricuspid Valve Disease

Surgery for Tricuspid Regurgitation

The most common cause of tricuspid regurgitation is mitral valve disease; therefore,

indications for tricuspid surgery will often be influenced by the extent of mitral valve disease (Shemin, 2008). The clinical picture and severity of disease play a major role in determining the appropriate management. The primary indication for tricuspid valve repair is severe tricuspid regurgitation in patients requiring surgery for mitral valve disease. Data favor tricuspid replacement or annuloplasty for severe, symptomatic primary tricuspid regurgitation. Tricuspid replacement is also indicated for severe tricuspid regurgitation when the valvular disease is not amenable to repair. Less well-established evidence suggests that tricuspid annuloplasty may be performed in patients with less than severe tricuspid regurgitation, when there is pulmonary hypertension or tricuspid annular dilatation, and when patients are undergoing mitral valve surgery. Tricuspid valve replacement or annuloplasty is not indicated for asymptomatic patients with pulmonary artery (PA) pressures less than 60 mm Hg and a normal mitral valve. Surgery is also not indicated for mild primary tricuspid regurgitation (Bonow et al., 2006).

Other types of tricuspid repair, including many annuloplasty techniques to repair a dilated annulus, have been more successful. In such procedures, the tissue that supports the tricuspid leaflets is sutured so the leaflets adjoin correctly. The net effect is a smaller opening, thereby allowing the valve to close completely. Research indicates that tricuspid dilatation may be a better predictor of the need for corrective surgery than the degree of tricuspid regurgitation (Matsunaga & Duran, 2005).

Repair of mitral valve disease may reduce the extent of tricuspid disease. As many as 20% of patients having surgery for mitral valve disease will also receive a tricuspid annuloplasty, but only 2% will require a valve replacement (Shemin, 2008). The surgeon typically considers the size and function of the right ventricle, degree of pulmonary hypertension, size of the right atrium, and clinical picture when making decisions about which procedure to select. Advances in echocardiography have improved the effectiveness of preoperative assessment, intraoperative evaluation of valve function, and outcomes. Nevertheless, the timing and choice of techniques for tricuspid surgery remain controversial and largely depend on the surgeon's experience and judgment (Bonow et al., 2006; Shemin, 2008).

Surgery for Tricuspid Stenosis

Surgical procedures for tricuspid stenosis include closed commissurotomy, open commissurotomy, and open valvuloplasty. A commissurotomy entails opening the commissures (the contact area for the valve leaflets) that have developed scarring and no longer open to allow blood to flow. Valvuloplasty entails insertion of a balloon to stretch or enlarge the valve opening; this procedure is used infrequently due to the concomitant presence of tricuspid regurgitation (Bashore, 2006) and inconsistent evidence supporting its efficacy (Phillips, 2005). All of tricuspid stenosis procedures are associated with poor long-term outcomes, as progressive tricuspid regurgitation often develops. Ultimately, valve replacement may be indicated for sustained symptom relief (Phillips, 2005). Tricuspid balloon valvotomy for tricuspid stenosis is no longer recommended because a significant degree of tricuspid regurgitation usually develops following this procedure as well (Bonow et al., 2006).

INFECTIVE VALVE ENDOCARDITIS

Infective endocarditis is a microbial infection of a blood-contacting structure in the heart or great vessels. It produces fever, heart murmur, signs of heart failure, and bacteremia and can produce rapid hemodynamic deterioration (Bonow et al., 2006). The characteristic sign is vegetation, although ulceration, abscesses, and destruction of heart structures may occur as well. Infective endocarditis can be difficult to diagnose because positive cultures may not be obtained. The Duke Criteria

are used to diagnose infective endocarditis in these cases (Stamou, Petterson, & Gillinov, 2008). Medical therapy is usually the first-line treatment.

Indications for surgery differ; however, mortality has been reduced since antimicrobial therapy has been supplemented with earlier surgical intervention (Schick, Gaasch, & Sexton, 2007). When surgery is indicated, valve repair has been successful in mitral valve disease, just as it has in other valve disease. Early surgery is more likely to result in a successful repair and reduces the risk of infection of the prosthesis (Bonow et al., 2006).

Zegdi et al. (2005) reported good long-term results from valve repair in a review of patients who underwent mitral valve repair for infective endocarditis between 1989 and 1994. These researchers found an 80% 10-year survival rate, 9% reoperation rate, 95% rate of NYHA Class I or II function, and very low rates of minor mitral regurgitation. Other surgeons have reported good outcomes with mitral valve repair for infective endocarditis when tissue is acceptable for repair (Ruttman et al., 2005). There is agreement that surgery is indicated for those patients with acute infective valve endocarditis and life-threatening heart failure or cardiogenic shock (Bonow et al., 2006; Schick et al., 2007; Yee, 2005). Tables 5–5 and 5–6 outline the indications for surgical treatment of native and prosthetic valve endocarditis, respectively. In general, surgery for native valve infective endocarditis

Table 5–5 Indications for Surgery in Native Valve Infective Endocarditis

Class I

1. Acute IE with valve dysfunction resulting in heart failure (Level B)
2. Valve destruction resulting in elevated LVEDP or LAP even if asymptomatic
3. IE caused by fungal or drug-resistant organism
4. Presence of heart block, annular abscess, aortic abscess, destructive penetrating lesions such as a fistula, or local spread of infection (Level B)
5. *Staphylococcus aureus* as the infectious organism

Class IIa

1. Recurrent emboli or persistent vegetations despite appropriate antimicrobial therapy (Level C)

Class IIb

1. Mobile vegetations larger than 10 mm on the aortic or mitral valve

IE = infective endocarditis; LAP = left atrial pressure; LVEDP = left ventricular end-diastolic pressure.

Sources: Bonow et al., 2006; Horstkotte et al., 2004; Schick, Gaasch, & Sexton, 2007; Stamou, Petterson, & Gillinov, 2008.

Table 5–6 Indications for Surgery in Prosthetic Valve Infective Endocarditis

Class I

1. Presence of heart failure (Level B)
2. Dehiscence on fluoroscopy or echocardiography (Level B)
3. Presence of increasing obstruction or regurgitation (Level C)
4. Presence of complications such as abscesses (Level C)
5. IE occurring less than 12 months after valve replacement

Class IIa

1. Persistent bacteremia or recurrent emboli despite appropriate antimicrobial therapy (Level C)
2. Relapsing infection (Level C)

Class III

1. Not indicated in uncomplicated IE at first infection with a sensitive organism (Level C)

IE = infective endocarditis.

Sources: Bonow et al., 2006; Horstkotte et al., 2004; Karchmer, 2007.

carries a better prognosis than surgery for prosthetic valve infective endocarditis (Stamou et al., 2008).

NURSING MANAGEMENT OF THE PATIENT FOLLOWING VALVE SURGERY

The immediate postoperative period is the most critical for the patient who has undergone valve surgery. A patent airway must be maintained. The patient's vital signs, hemodynamic status, and level of consciousness should be assessed frequently. In addition, the possibility that the patient may need IV fluids, blood transfusion, titration of vasoactive agents, pain management, and chest tube maintenance requires vigilance on the part of the nursing staff. Table 5-7 identifies common nursing goals and interventions. Care of the immediate postoperative cardiac surgery patient and monitoring for postoperative complications are discussed in detail in Chapters 8 and 13, respectively.

Table 5–7 Postoperative Goals and Nursing Interventions

1. Achieve and maintain normal body temperature. Monitor and optimize vital signs and hemodynamic status (stroke volume index, cardiac index, central venous pressure, pulmonary artery occlusive pressure, systemic vascular resistance, SvO_2 [if available]).
2. Monitor for presence of dysrhythmias.
3. Monitor drainage from the chest tube.
4. Reposition patient every 2 hours and increase activity level when stable.
5. Monitor the patient's respiratory status, and assure adequate deep breathing and coughing to prevent atelectasis.
6. Monitor for and report any neurologic changes from baseline.
7. Maintain adequate renal perfusion. Document daily weight and fluid intake and output.
8. Monitor serum electrolytes.
9. Maintain adequate fluid volume; avoid preload reduction.
10. Maintain optimal pain management (less than 4 or according to reasonable patient expectations).

COMPLICATIONS OF HEART VALVE SURGERY

While complications of heart valve surgery are rare, the ICU nurse should be aware of the possibility of their development and implement measures to try to prevent their development. Complications of heart valve surgery reported in the literature include the following conditions:

- Venous thrombotic events (Shuhaiber & Anderson, 2007)
- Atrial dysrhythmias (Bossone et al., 2007; Kolh, Kerzmann, Honore, Comte, & Limet, 2007; Ngaage, Cowen, Griffin, Guvendik, & Cale, 2008)
- Renal insufficiency (Bossone et al., 2007; Kolh et al., 2007; Ngaage et al., 2008)
- Heart failure (Bossone et al., 2007)
- Neurological complications, stroke, or transient ischemic attack (Bossone et al., 2007; Filsoufi, Rahmanian, Castillo, Bronster, & Adams, 2008; Kolh et al., 2007; Mihaljevic et al., 2004; Ngaage et al., 2008)
- Respiratory insufficiency (Kolh et al., 2007; Ngaage et al., 2008; Tabata et al., 2008)
- AV block (Kolh et al., 2007; Mihaljevic et al., 2004)
- Myocardial infarction (Kolh et al., 2007; Ngaage et al., 2008)
- Sternal wound infection (Mihaljevic et al., 2004; Rahmanian et al., 2007; Tabata et al., 2008)

- Bleeding—requiring reexploration in most cases (David, Armstrong, Maganti, & Ihlberg, 2008; Mihaljevic et al., 2004; Ngaage et al., 2008; Rahmanian et al., 2007; Tabata et al., 2008)
- Circulatory failure (Haddad et al., 2007)
- Low cardiac output state (Ngaage et al., 2008)
- Gastrointestinal complications (Ngaage et al., 2008)

Two case reports of uncommon complications—left ventricular–right atrial communication (Frigg, Cassina, Siclari, & Mauri, 2008) and an immobilized prosthetic mitral valve (Murugesan, Banakal, & Muralidhar, 2008)—following valve surgery have also been described.

Ways to prevent complications and strategies to improve mortality continue to be examined by researchers. For example, statin therapy has been found to improve morbidity and 30-day mortality in valvular surgery patients (Fedoruk, Wang, Conaway, Kron, & Johnston, 2008).

The possibility of either a deep sternal wound infection or development of an infected valve has been reported as well. Because of the significant impact on morbidity and mortality following cardiac surgery, prophylactic antibiotic administration both preoperatively and for as long as 48 hours postoperatively is part of the standard of care. If an infection is suspected, cultures should be obtained and empiric antibiotic therapy started. Modifications are subsequently made based on culture and sensitivity reports (Tang, Maganti, Weisel, & Borger, 2004). As with all patients, meticulous hand hygiene is essential to help prevent development of postoperative infection.

The potential for postoperative dysrhythmias (most notably, atrial fibrillation) has been reported. The ICU nurse plays a pivotal role in promptly recognizing dysrhythmia development, determining the clinical significance and hemodynamic response to them, and implementing treatment measures. Management of postoperative dysrhythmias is discussed in detail in Chapter 15.

Development of venous thrombotic events is likely following valvular surgery, particularly if the patient received a mechanical valve. Preventive measures include use of sequential compression devices and administration of an anticoagulant in the immediate postoperative period. When feasible, early ambulation should be initiated (Thompson et al., 2008).

SUMMARY

Selection of a mechanical or biological prosthetic valve has lifelong implications. These patients may require lifestyle modification and medication therapy for the rest of their lives. Advances in technology in the area of heart valve surgery offer more options, facilitate less invasive techniques, and potentially may improve outcomes. Vigilant postoperative nursing care is critical to help ensure a good outcome for the patient who undergoes valve surgery.

CASE STUDY

A patient who had childhood rheumatic heart disease is diagnosed with mitral valve prolapse (MVP). The patient has a history of type II diabetes and increased body mass index. She experiences pain during periods of emotional stress and presents with mitral regurgitation (MR) and a murmur that gets more intense during systole.

Critical Thinking Questions

1. Which medications should this patient take to manage the mitral valve prolapse?
2. Which factors should contribute to the decision to have surgery for repair of the valve defect?

Answers to Critical Thinking Questions

1. Most people with mitral valve prolapse who experience symptoms will be prescribed medication to treat chest pain, heart rhythm abnormalities, and/or transient ischemic attack. Medications may include beta blockers for heart rhythm, aspirin, or warfarin (Coumadin®).
2. The severity of the symptoms would significantly factor into the decision as well as the need to perform early repair to prevent valve replacement.

SELF-ASSESSMENT QUESTIONS

1. A patient with newly diagnosed aortic regurgitation will have which of the following?
   a. Increased pulmonary artery occlusive pressure
   b. Decreased sympathetic tone
   c. Decreased cardiac output
   d. Widening pulse pressure
2. Which type of prosthetic valve requires long-term anticoagulation therapy?
   a. Mechanical
   b. Biologic
   c. Bovine
   d. Porcine
3. Patients who are postoperative aortic regurgitation repair may require administration of which of the following?
   a. Dopamine (Intropin®)
   b. Neosynephrine (Phenylephrine®)
   c. Milrinone (Primacor®)
   d. Judicious intravenous fluids
4. The primary symptom of mitral stenosis is
   a. chest pain.
   b. hypertension.
   c. exertional dyspnea.
   d. syncope.
5. Patients with mitral stenosis will likely present with which of the following signs?
   a. Decreased ejection fraction
   b. Elevated left ventricular end-diastolic pressure
   c. Elevated pulmonary artery pressure
   d. Decreased left atrial pressure
6. The nurse caring for a patient immediately following surgical repair of mitral stenosis should observe for which of the following?
   a. Elevated central venous pressure
   b. Pulmonary hypertension
   c. Decreased pulmonary vascular resistance
   d. Increased cardiac output
7. The nurse caring for a patient immediately following surgical repair of mitral regurgitation should observe for which of the following?
   a. Increased left atrial pressure
   b. Increased systemic vascular resistance
   c. Decreased pulmonary artery systolic pressure
   d. Decreased ejection fraction
8. An open surgical procedure that repairs the valve by suturing the torn leaflets, chordae tendinae, or papillary muscles is called
   a. angioplasty.
   b. valve replacement.
   c. valvuloplasty.
   d. coronary artery bypass grafting (CABG).

9. Your patient requires mitral valve replacement and is contemplating which type of valve to select. Which of the following statements would help the patient in the decision-making process?
   a. "If you receive a mechanical valve, it will not last as long as a biologic valve."
   b. "If you receive a mechanical valve, you will need to take a blood thinner over the long term."
   c. "You should probably also consider valve repair, as many surgeons have been interested in that procedure."
   d. "Both types of valves are equally good, because the papillary muscles and annulus are retained when either valve is used."
10. Which of the following acute valve disorders is most likely to constitute a medical emergency?
    a. Mitral valve prolapse
    b. Aortic valve stenosis
    c. Aortic valve regurgitation
    d. Mitral valve stenosis

Answers to Self-Assessment Questions

1. d
2. a
3. c
4. c
5. c
6. a
7. b
8. c
9. b
10. c

Clinical Inquiry Box

Question: Does valve replacement in the elderly increase quality of life?

Reference: Filsoufi, F., Rahmanian, P. B., Castillo, J. G., Chikwe, J., Silvay, G., & Adams, D. H. (2008). Excellent early and late outcomes of aortic valve replacement in people aged 80 and older. *Journal of the American Geriatrics Society*, *56*(2), 255–261.

Objective: To investigate early and late outcomes of aortic valve replacement (AVR) in a cohort of patients aged 80 and older.

Method: This retrospective study of consecutive patients undergoing AVR was performed. The study included 1308 patients undergoing AVR; 231 subjects were aged 80 years or older, and 1077 subjects were younger than 80 years of age. Patient characteristics, hospital mortality, morbidity, length of stay, and long-term survival were analyzed.

Results: Subjects aged 80 and older were more likely to be female, had a lower body mass index, and presented significantly with more comorbidities, such as heart failure, renal failure, and extensive aortic calcification. Hospital mortality was slightly higher in subjects age 80 and older (5.2%) as compared with 4.5% in those younger than aged 80. Respiratory failure occurred more frequently in those aged 80 and older. Age of 80 and older was not a predictor of hospital mortality. The median length of stay was significantly higher in those aged 80 and older than in those younger than 80 (10 days versus 7 days). Five-year survival was essentially the same for those aged 80 and older within the general U.S. population.

Conclusion: AVR will result in good outcomes in patients aged 80 and older, with minimal increase in postoperative mortality and acceptable postoperative morbidity. Respiratory failure is the main postoperative complication in patients aged 80 and older. Implications for nurses are that cardiac surgery is an appropriate intervention for individuals older than age 80. Therefore, the need to provide care to this population will continue to increase over the next 15 years as the U.S. population ages.

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WEB RESOURCES

American Association of Cardiovascular and Pulmonary Rehabilitation: www.aacvpr.org

American College of Cardiology: www.acc.org

American Heart Association: www.americanheart.org

Mended Hearts: www.mendedhearts.org

National Heart, Lung, and Blood Institute: www.nhlbi.nih.gov

The Society of Thoracic Surgeons: www.sts.org

Chapter 6

Minimally Invasive Cardiac Surgery

Jenniffer Hughes, Kirsten Platt

INTRODUCTION

Since 1967, coronary artery bypass grafting (CABG) surgery has entailed creating a median sternotomy incision, creating cardiac standstill with a cardioplegia solution, and being connected to a bypass machine to maintain oxygenation and perfusion during cardiac standstill (Ley, 2006). Cardiopulmonary bypass (CPB) refers to the temporary rerouting of blood from the right atrium to the aorta via an oxygenator (bypass machine), such that blood flow is circumvented around the heart and lungs during the surgical procedure. During a CABG, an anastomosis of the left internal mammary artery (LIMA) to the left anterior descending artery (LAD) is made (Chen-Scarabelli, 2002). While data attest to the efficacy of overall medical therapy in those patients with left main coronary disease and in those with three-vessel disease with decreased left ventricular function, CABG procedures are not without the risk of complications (Duhaylongsod, 2000).

Traditional CABG surgery is associated with a prolonged ventilation time (initially days; now 8–12 hours), prolonged intensive care unit (ICU) stay (initially 1 week; now 24 hours if the case is uncomplicated), prolonged hospitalization (initially several weeks; now 1 week), a prolonged rehabilitation phase (now 8–12 weeks), and potential sternal wound infection (SWI). Other complications associated with traditional CABG are listed in Table 6–1. These and other complications associated with CABG provided some of the needed motivation to develop procedures to perform CABG on a beating heart (Chikwe, Donaldson, & Wood, 2006; Duhaylongsod, 2000).

Modern technology made for the advancement of surgical instrumentation, and surgical intervention has followed much the same path. After almost a decade of laparoscopic procedures being performed in the late 1980s, cardiac surgeons began to accept minimally invasive cardiac surgery (MICS) in the mid-1990s (Mack, 2006). Patients undergoing MICS either have a small incision to access the operative site or undergo the surgical procedure without the use of a bypass machine, or both. Goals of MICS that have been identified include attaining a patent graft with equal or better efficacy than is achievable with traditional CABG procedures, returning to baseline activity level faster, and decreasing pain, morbidity and mortality, length of stay (LOS), and cost (Chikwe et al., 2006; Duhaylongsod, 2000). Data suggest that MICS decreases the need for blood transfusions, LOS, and risk of infection and other complications associated with traditional cardiac

Table 6–1 Pathophysiologic Changes Associated with a Traditional CABG Procedure

Pathophysiologic Change	Etiology
Bleeding and thrombotic complications: disseminated intravascular coagulation (DIC), heparin-induced thrombocytopenia (HIT), and thrombosis (HITT)	• Activation of platelets and plasma proteins • Patients are heparinized and given supplementary doses during bypass, titrated against clotting studies • Bleeding times after full reversal of heparin do not become normalized for as long as 12 hours after bypass
Considerable interstitial fluid shifts	• Increased systemic venous pressure • Volume loading • Decreased plasma protein concentration (secondary to dilution and absorption onto the bypass circuit, and the inflammatory response increasing capillary permeability)
Increased levels of cortisol, epinephrine, and norepinephrine (remain elevated for at least 24 hours)	• Stress of surgery • Hypothermia • Cardiopulmonary bypass • Nonpulsatile flow
Hyperglycemia	• Stress of surgery • Hypothermia • Cardiopulmonary bypass • Nonpulsatile flow
Decreased circulating triiodothyronine (T_3)	• Stress of surgery • Hypothermia • Cardiopulmonary bypass • Nonpulsatile flow
Decreased myocardial compliance and contractility	• Myocardial stunning • Ischemia • Edema
Decreased myocardial function (for 6 to 8 hours postoperatively)	• Ischemia-reperfusion injury
Progressive need for volume resuscitation	• Vasodilation • Capillary leak
Pulmonary edema	• Activation of complement system • Sequestration of neutrophils in pulmonary vasculature (can mediate increase in capillary permeability, which is compounded by fluid shifts)
Pulmonary dysfunction	• Cardiopulmonary bypass decreases the effect of surfactant • General anesthesia • Median sternotomy • Cardiopulmonary bypass increases shunts, decreases compliance and functional residual volume, and can cause acute lung injury

continues

Table 6–1 Pathophysiologic Changes Associated with a Traditional CABG Procedure (continued)

Pathophysiologic Change	Etiology
Ischemic stroke	• Emboli released during the cannulation and clamping of the aorta
Hemorrhagic stroke	• Anticoagulation necessary for bypass
Impaired renal function	• Hemodilution • Microemboli • Catecholamines • Low perfusion pressure • Diuretics • Hypothermia • Hemolysis
Peptic ulceration	• Stress response
Endotoxin translocation, adding to the inflammatory response	• Greater permeability of gut mucosa

Sources: Chikwe, Donaldson, & Wood, 2006; Duhaylongsod, 2000; Glenville, 1999; Ley, 2006.

surgery (Ley, 2006). There is also reportedly less trauma and faster recovery with MICS than with traditional surgical methods (Sun et al., 2006). Data regarding reduction of complications of cardiac surgery are somewhat inconsistent, however. Scherer and colleagues (2006) reported no difference in the development of postoperative atrial fibrillation (AF) with MICS and traditional CABG procedures.

MINIMALLY INVASIVE CARDIAC SURGERY

While no official definition of MICS has been established, it is often defined as cardiac surgery without the use of cardiopulmonary bypass (CPB) or sternotomy; rather, smaller incisions are made (Chikwe et al., 2006; Mayfield, 2007). MICS also refers to a variety of procedures used to bypass blocked coronary arteries (Cowles, 2008).

With the advent of new surgical instrumentation, including robotics, MICS is now becoming the standard in certain patient populations. Specifically, MICS can be performed for multiple-vessel coronary artery disease (CAD); valve repair/replacement (mitral, aortic, or tricuspid); limited-access and totally endoscopic pulmonary vein isolation and the Maze procedure to treat AF; congenital cardiac defects (e.g., patent ductus arteriosis); and thoracic endografting for aortic aneurysm disease treatment (Mack, 2006). Any procedure that is done on the surface of the heart, with the exception of surgery performed to correct atrial septal defects (ASD) and ventricular septal defects (VSD), can be done without the use of CPB. The advantages of this approach are many, while the risks are few. There is not only greater patient satisfaction because of the decreased level of pain associated with MICS, but also decreased hospital LOS and resumption of activities of daily living (ADLs).

Types of MICS Procedures

Several different MICS procedures have been developed, each of which has its own criteria. One of two approaches is used in these surgeries: (1) a mini-thoracotomy incision without use of CPB or (2) an endoscopic approach

with use of CPB. The major types of MICS procedures that are currently performed include minimally invasive direct coronary artery bypass (MIDCAB), off-pump coronary artery bypass (OPCAB, also known as beating-heart surgery), robot-assisted coronary artery bypass (RACAB), minimally invasive direct view, and keyhole heart surgery (Cowles, 2008). A number of alternative names for MICS appear in the literature.

Minimally Invasive Direct Coronary Artery Bypass

MIDCAB, which is an alternative approach to traditional CABG, has been performed since 1996 (Chen-Scarabelli, 2002). Differences between the two approaches are threefold. First, the incision size is much smaller for MIDCAB; several 3-inch to 5-inch incisions are made between the ribs as compared to a 10-inch to 12-inch median sternotomy incision in conventional CABG procedures. Second, because MIDCAB is a beating heart procedure, no cardioplegia is instilled to stop the heart. Third, because MIDCAB is a beating heart surgery and no cardioplegia is instilled, CBP is not required for MIDCAB procedures.

MIDCAB procedures are performed on patients with one or two blockages to the left anterior descending (LAD) coronary artery or its branches on the front of the heart. Blockage of the right coronary artery (RCA) may be bypassed as well. MIDCAB surgery is performed without the need for a median sternotomy. Instead, a smaller mini-thoracotomy incision is made on the left chest to expose the heart, and part of the costal cartilage is removed. Once the field is exposed, the affected artery is temporarily closed off and freed at the lower end. An opening is made in the pericardium to expose part of the LAD. The heart's movement is limited during this part of the procedure owing to the device attached to it. The artery is connected to the LAD or branch below the area of the blockage, and then the artery is reopened (Cowles, 2008).

The use of MICS as a treatment modality has decreased for patients with single-vessel disease. This is related to the efficacy of percutaneous coronary intervention and drug eluting stents for treatment of this condition (Mack, 2006).

The right internal mammary artery (RIMA) is used to bypass the RCA, and the LIMA is used to bypass the LAD. The internal mammary artery (IMA) conduits are patent for an average of 20 years as opposed to the 6-vein conduits, which last for only 6 to 10 years (Edgar, Ebersole, & Mayfield, 1999). The MIDCAB is performed without CPB; however, a perfusionist is on standby in case there is need for conversion to a traditional sternotomy.

Three approaches may potentially be used for the MIDCAB; the specific approach chosen depends on the surgeon's preference and the patient's anatomy. In all of these procedures, the patient is intubated with a double-lumen endotracheal tube, thereby allowing for ventilation of the right lung and deflation of the left lung, providing more room to manipulate the heart.

The first approach is performed through a left thoracotomy incision approximately 8 to 12 cm in length between the fourth and fifth ribs. Rib spreaders are used to spread and elevate the rib cage to provide ample space to dissect the IMA. The MIDCAB is performed on a beating heart, so it requires the use of a cardiac stabilization device. The stabilizer (see Chapter 7) provides a direct view, dampens the movement of the epicardium, and permits a nontraumatic grip on the beating heart (Edgar et al., 1999). The device helps the surgeon isolate the diseased vessel and stabilize the localized region of epicardium for anastomosis.

In the second approach, a 5- to 8-cm vertical incision is made on either side of the ster-

num, depending on the vessel being bypassed. Intercostal cartilage is removed from the third and fourth ribs to provide a better view of the operative site and dissection of the IMA. This approach can easily be converted to a full sternotomy should the patient require CPB (Edgar et al., 1999).

The third approach is strictly used for LIMA-to-LAD bypass and involves an 8- to 10-cm horizontal incision along the fourth intercostal space. The procedure then follows the steps in the first approach—the incision placement is the only variation.

A left mini-thoracotomy approach provides direct visualization of the LAD, with the LIMA being the graft vessel of choice. The right mini-thoracotomy approach provides direct visualization of the RCA, with the RIMA being the graft vessel of choice (Edgar et al., 1999; Sharony et al., 2006). Despite the LIMA and the RIMA being the vessels of choice, traditional saphenous vein graft via endoscopic approach can be used as well.

The advantages of MIDCAB include decreased pain secondary to a less invasive surgical approach; earlier mobilization secondary to decreased pain, shorter LOS in the ICU and hospital, greater cost-effectiveness, low morbidity and mortality (Chen-Scarabelli, 2002), no CPB, and decreased infection rates, especially with sternal wound infections. Disadvantages include the potential change in strategy in the operating room (OR) if more extensive surgical intervention is required, including a sternotomy and limited patient and vessel choices. Table 6–2 lists additional advantages and disadvantages of the MIDCAB approach.

Table 6–2 Advantages and Disadvantages of the MIDCAB Approach

Advantages	Disadvantages
Faster recovery/return to routine ADLs	Limited access and exposure to the operative area
Reduced morbidity/mortality	Technical difficulty with beating heart
No risk of sternal wound infection	Need experienced surgeon
LIMA/RIMA more resistant to atherosclerosis/increased longevity of patency	Increased risk of incomplete revascularization
No adverse effects related to CPB	No data on long-term patency
Lower cost	Unable to access/visualize posterior heart for revascularization
Shorter hospital stay	Procedure limits target vessels
Decreased blood loss	No circulatory support
No aortic manipulation	Acute graft occlusion and incomplete revascularization risk increased
Lower intraoperative morbidity/mortality in patients with cardiogenic shock, acute MI, or LV dysfunction	No cardioplegia
Capable of revascularization of multiple-vessel lesions	

ADLs = activities of daily living; CPB = cardiopulmonary bypass; LIMA = left internal mammary artery; LV = left ventricular; MI = myocardial infarction; RIMA = right internal mammary artery.

Sources: Chen-Scarabelli, 2002; Edgar, Ebersole, & Mayfield, 1999.

With aortic and mitral valve surgery—whether repair or replacement is required—CPB and cardioplegia will be used, but the approach is still minimally invasive. The advantages of minimally invasive valve replacement are essentially the same as with MICS, with the exception of the complications of CPB and cardioplegia. These issues are discussed in detail in Chapter 7.

Off-Pump Coronary Artery Bypass

OPCAB (beating heart) procedures may be performed on four or five vessels, as compared with MIDCAB, where only one or two can be repaired. This procedure entails a median sternotomy incision; no bypass machine is required. With OPCAB, an artery or vein from the lower extremities is used to make the bypass, and a device restricts heart movement, as the heart continues beating during this procedure (Cowles, 2008). Reported benefits of OPCAB include reductions in blood loss, need for transfusions, inflammatory response, renal insufficiency, ventilatory time, incidence of AF, and LOS in ICU and hospital. It is suggested that elderly patients at high risk for surgery might benefit the most from the OPCAB approach (Mack, 2006).

OPCAB procedures can be adapted so that the IMA harvesting and bypass can be performed either using a MIDCAB approach with a small thoracotomy incision or endoscopically (totally endoscopic coronary artery bypass grafting [TECAB]). An alternative approach is endoscopic atraumatic coronary artery bypass grafting (endoACAB). In this combination of both of the aforementioned approaches, the IMA is harvested using an endoscopic approach and the anastomosis is performed with direct visualization through a small thoracotomy incision (Chikwe et al., 2006).

OPCAB is discussed in more detail in Chapter 7.

Robot-Assisted Coronary Artery Bypass

Robotic surgery was first envisioned by the military in the 1980s as an answer to war-time injuries by providing a surgeon in every foxhole, thus allowing a surgeon to perform life-saving surgery from a central location on a soldier located in a distant Mobile Army Surgical Hospital (Berlinger, 2006). By 1985, a robot, along with computed tomography (CT) guidance, was used to place a needle for a brain biopsy. In 1988, a robot was used to perform prostate surgery.

By the 1990s, robotic surgery was becoming more finely tuned. The use of minimally invasive laparoscopic surgery was finding its limits; laparoscopic instruments were rigid tools that could only move along two axes—up and down, clockwise and counterclockwise. The need for a more manipulative surgical intervention was realized with the introduction of the ROBODOC® (Integrated Surgical Systems) and the more developed da Vinci® Surgical System (see Figure 6–1) and Computer Motion AESOP and ZEUS systems (Intuitive Surgical).

The surgical robot consists of a collection of wristed tools called manipulators. The manipulators receive digital instructions from an interfaced computer. The (real-life) surgeon stays seated at a computer console with a three-dimensional display and acts as the "driver" of the computer. The surgeon initiates the digital instructions by controlling the hand grips. By using the hand grips, the surgeon's hand movements at the console are then duplicated by the robot, with software filtering out physiologic hand tremors.

In 1998, the first robotically assisted CABG was performed using the da Vinci surgical robot. Unaccommodating places are what robot-assisted surgery is all about—the size of the human surgeon hand is not optimal for maneuvering in tiny spaces (Berlinger, 2006).

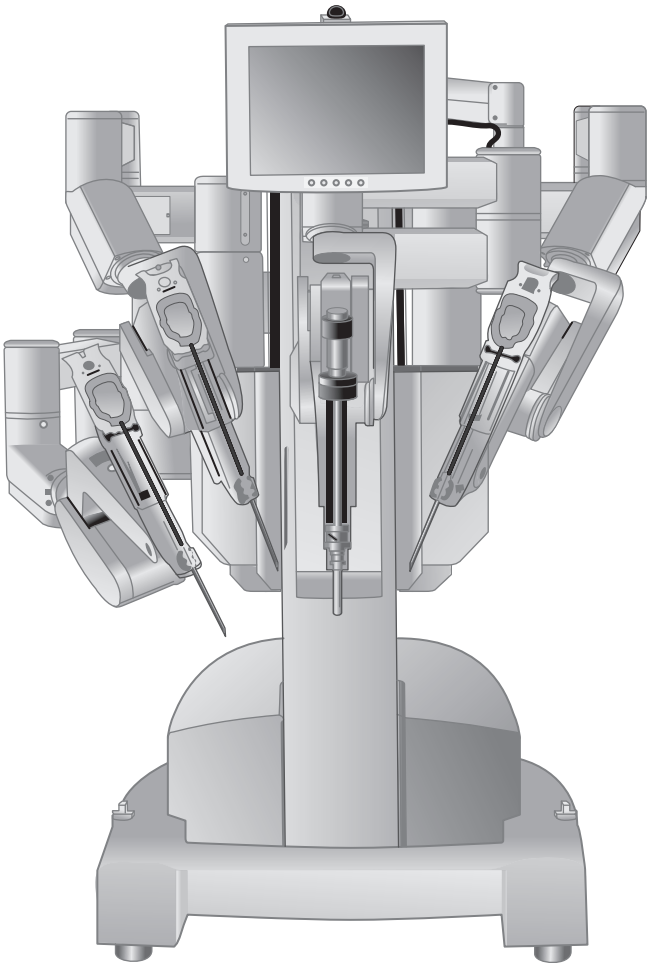

Figure 6–1 da Vinci® Surgical System.
Source: Courtesy of Intuitive Surgical System.

Several surgeons have reported performing successful robotic-assisted anastomoses of the left internal thoracic artery to the LAD.

Robotics has been used successfully for repair of ASD and for mitral valve repair. Widespread acceptance of the use of robotics for these procedures is limited, however, as performance of these procedures without robotic assistance is feasible in many facilities (Mack, 2006). In addition, a few prohibitive factors have constrained the adoption of the robotic approach. First, only a limited number of facilities in the United States have the capability to perform robotic cardiac surgery. Second, surgeons must undergo an extensive training program before they can perform this type of surgery. Third, this approach is expensive: A robotic system can cost more than $1 million, not including the cost of the disposable tools required for each surgery.

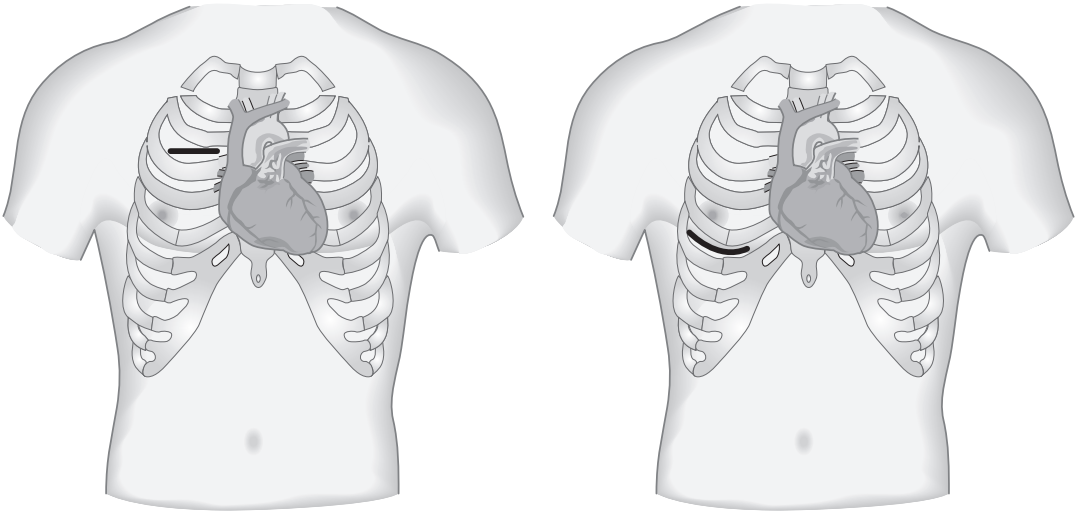

Figure 6–2 (Left) Minimally invasive AV surgery incision. **(Right)** minimally invasive MV surgery incision.

Source: Courtesy of the University of Southern California.

Minimally Invasive Direct View

Minimally invasive techniques have also been developed for the repair or replacement of the mitral valve (MVR) and repair or replacement of the aortic valve (AVR). The incision for an AVR is located on the right side of the chest below the right clavicle and above the right nipple. The incision for the MVR is also located on the right chest but below the right nipple (Figure 6–2). The main benefit of minimally invasive direct-view valve surgery is the avoidance of a median sternotomy. An 8-cm incision is made and cartilage is removed to allow for direct visualization of the valves.

Unlike with the MIDCAB procedure, CPB and cardioplegia are required because the valve surgery takes place inside the heart rather than in front of it. MICS for valve repair will use smaller incisions than the traditional CABG approach. As with all minimally invasive surgeries, LOS, cost, and recovery time are reduced with the direct-view procedures. Minimally invasive procedures are also preferred in patients who have undergone a sternotomy in the past (Mayfield, 2007). One study compared outcomes of patients who underwent valve surgery with either redo-sternotomies or a minimally invasive approach. The overall hospital mortality in the minimally invasive group was 5.6% versus 11.3% in the sternotomy group. In addition, LOS was shorter and the 5-year survival rate was slightly more favorable in the minimally invasive surgery group (Sharony et al., 2006).

Endovascular/"Keyhole" Procedures

Subsumed under the heading of "minimally invasive procedures" are the endovascular techniques known as "keyhole" procedures. Traditional endoscopes used for gynecological and general surgery are 10 mm in width—too large to access the heart through the intercostal space. Advances in technology have now made endoscopic heart surgery possible, however, through the development of 5-mm and 3-mm endoscopes. The 5-mm endoscope makes it easy to maneuver between the ribs, thereby increasing visibility. This latter type of endoscope has been used to close a patent ductus arteriosus, eliminating the need for a thoracotomy. In addition, a newly engineered digital camera and processing

make pictures taken from the 5-mm scope better than those taken with the 10-mm scope. The 3-mm endoscopes are designed to feel and work like the standard instruments used by cardiac surgeons (Mayfield, 2007).

Some concerns have arisen regarding keyhole procedures—including, inaccurate depth perception, less tactile feedback, need for experienced assistance, and decreased degrees of motion of the surgeons' hands. As a result, robotics has been applied to counteract the problems of keyhole procedures (Deeba & Darzi, 2006).

Along with treatment for vessels and valves, the keyhole technique known as the Maze procedure has been used as a treatment for AF. The "gold standard" treatment for AF is the Cox/Maze III procedure. This latter procedure entails a number of incisions being made on the right and left atria, where the term "Maze" refers to the pattern of incisions made in the atrium. The incisions cause scarring, which does not conduct electricity, stops irregular electrical activity, and eradicates AF. The scarring also prevents future irregular electrical signals from developing. The Cox/Maze III procedure is performed during a CABG procedure, which requires a sternotomy and CPB. Maze procedures are discussed in detail in Chapter 3.

A cutting-edge technique for the Maze procedure has recently been developed. The Ex-Maze is performed endoscopically on the outside of a beating heart. An ablation device uses unipolar radiofrequency energy with vacuum-maintained contact and suction-controlled saline perfusion to ensure uniform energy transmission and transmural lesion development (Kiser, Wimmer-Greinecker, & Chitwood, 2007). Because the procedure is performed on a beating heart, atrial function can be monitored during treatment. Patients can convert to normal sinus rhythm during the procedure or within 6 weeks. The Ex-Maze procedure is safer than the original techniques, is associated with less postoperative pain, and causes fewer complications because it is less invasive and CPB is not required. Using this approach, congenital heart defects can be repaired through minimally invasive endoscopic techniques. For example, atrial and ventricular septal defects (ASD and VSD) can be closed without stopping the heart by using specialized endovascular catheters and transesophageal echocardiography to repair the holes. In some cases, robots are used to assist with the procedure.

Endoscopic procedures have another benefit: They assist in making reentry into the sternum safer. Using this approach, the surgeon can readily visualize structures behind the sternum. Adhesions can form between the heart and the sternum, which can cause damage to the heart if reentry is required. Now the adhesions can be cut with the assistance of the scope prior to a second sternotomy, thereby reducing the risk of damaging the heart (Mayfield, 2007).

Endoscopic technology can also be used to harvest the saphenous vein. Instead of a long incision spanning from the inner thigh to the lower leg, the vein can be dissected out using endoscopy. This technique requires smaller incisions and, therefore, produces less pain (Mayfield, 2007).

Endoscopic procedures have made more complex cardiac procedures possible, especially with the introduction of robotics. Surgical treatment of multiple-vessel disease through the smaller incisions had proven difficult. Robotics was implemented to facilitate endoscopic cardiac surgery in these circumstances. Harvesting of the IMAs was successful with robotics; vascular anastomosis still presents a challenge, however, and robotics is rarely used for this purpose (Mack, 2006).

When endoscopy is combined with robotic-assisted surgery, the robotic arms and small camera are advanced through small incisions made in the intercostal spaces. Motion sensors are attached to the robotic wrists to control the instruments. The surgeon sits at the console and looks through two lenses (like a microscope) that display the image from the

Table 6–3 Robotic Surgery Procedures

- Single- and multiple-vessel CABG
- Mitral valve repair and replacement
- Aortic valve repair and replacement
- ASD repair
- VSD repair
- Removal of cardiac tumors
- Ablation for treatment of atrial fibrillation (Maze procedure)

ASD = atrial septal defect; CABG = coronary artery bypass grafting; VSD = ventricular septal defect.

camera. The computer generates a three-dimensional image of the surgical site, with foot pedals controlling the camera. As the surgeon moves, the robotic arms mimic movements and may even be more precise than the surgeon's natural hand movement (Pike & Grundy, 2003). The use of robotics has made multiple-vessel, minimally invasive, beating heart CABG possible. This technology can be used on beating heart as well as during arrested heart procedures. Table 6–3 outlines procedures that can be performed with robotic assistance.

Inclusion Criteria for MICS

A select group of patients fit the MIDCAB criteria. Those included would be individuals with a proximal LAD that is at least 1.5 mm in diameter and not calcified (Caimmi et al., 2004). Other patient eligibility criteria are related to the small incision size. If blockage of one or two coronary arteries on the exterior of the heart exists, whether the patient is healthy or is at too great a risk for a traditional bypass, MIDCAB may be performed. Patients who are considered viable candidates for OPCAB include those with very low ejection fraction, COPD or emphysema, kidney disease, or high risk for stroke (Cowles, 2008). Other sources suggest MIDCAB surgery specifically is indicated for patients who are at high risk for percutaneous coronary intervention, stent restenosis, or redo-CABG with an occluded LAD graft (Calafiore et al., 1997; Subramanian, 1997).

Exclusion Criteria for MICS

A number of exclusion criteria for MIDCAB are listed in the literature. These contraindications are essentially related to the patient's anatomy and the degree of difficulty anticipated in locating the LAD. If patients have an LAD with a diameter of less than 1.5 mm, a coronary artery with a calcified score greater than 2, or an intramyocardial position of the coronary artery requiring surgery, they are not eligible for MIDCAB (Caimmi et al., 2004). While not listed as an exclusion criterion, it has been suggested that patients with small coronary arteries who need several bypass procedures should undergo traditional CABG procedure (Cowles, 2008). Morbid obesity is regarded as an exclusion criterion by some surgeons because of the difficulty of performing surgery through the small incisions used in minimally invasive surgery (Chikwe et al., 2006; Schell, Gundry, & Grichnik, 2001). Contraindications for MIDCAB specifically include a lesioned vessel located endomyocardially, concomitant surgical intervention to treat other cardiac disorders, inadequate flow, diameter or length of the IMA, and stenosis of the subclavian artery (Calafiore et al., 1997; Subramanian, 1997). Finally, presence of AF or COPD is suggested to make surgical care more challenging, such that these conditions are considered contraindications to MICS by some (Schell et al., 2001).

A number of exclusion criteria have been reported for robotic mitral surgery as well. These contraindications include presence of renal failure, liver dysfunction, bleeding diathesis, severe pulmonary hypertension, significant aortic or tricuspid valve disease, recent ischemia or stroke (less than 30 days),

and history of right thoracotomy (Kypson & Chitwood, 2004).

PREOPERATIVE NURSING CARE

Preoperative cardiac surgery teaching should be employed, with the patient and family being educated about the possibility of the MIDCAB procedure evolving into a standard CABG intervention. Teaching should include the participation of not only the patient, but also any caregivers. Preoperative teaching for the intended procedure should include a review of the potential complications and the standard of care employed by the facility.

Emphasis should be placed on the decreased amount of postoperative pain experienced, but patients should be encouraged to report pain levels honestly to help avoid complications. Specifically, patients should be encouraged to volunteer information regarding pain level and efficacy of treatment. Such is not always the case, however, and pain management may be inadequate as a result (Watt-Watson, Stevens, Garfinkel, Streiner, & Gallop, 2004).

The value of aggressive pulmonary toileting and early ambulation cannot be over-emphasized. The technique of coughing and deep breathing as well as the use of incentive spirometry should be taught in the preoperative period.

POSTOPERATIVE NURSING CARE

Immediate postoperative care of patients who have undergone MICS will follow the same path as care for those who required a sternotomy. Approximately 1 hour prior to the patient's arrival to the ICU, the OR nurse usually calls in a report to the admitting ICU nurse. After receiving the initial brief report, the family should be updated. Early contact establishes a rapport with family and provides time to obtain information for the admission assessment and emergency contact names and numbers. The family should be notified where they will be contacted and the anticipated time until visitation after the patient arrives in the ICU. If the patient was not in the hospital prior to the surgery and did not receive preoperative education, the family should be prepared about what to expect with the ICU environment and visitation guidelines to help reduce their stress level. Questions should be addressed, and any anticipated resources (e.g., pastoral care) may be provided at this time.

Admission to the ICU: The First 15 Minutes

Patients who are intubated will be sedated and possibly chemically paralyzed. Depending on the facility, the anesthesia provider will start a sedation infusion to promote comfort, decrease myocardial oxygen consumption, and enhance tolerance to the ventilator until weaning commences. The anesthesia provider will provide a more in-depth report including, but not limited to, the patient's past medical history, allergies, intraoperative course, last set of pertinent lab results, volume of crystalloid and colloids given, antibiotics administered and times, urinary output, and, if CPB was required, the length of time on bypass and the length of time the aorta was cross-clamped.

While settling the patient after surgery, maintaining hemodynamic stability is essential. Baseline vital signs should be obtained and all pressure lines zeroed and leveled to the phlebostatic axis (see Figure 6–3). In addition to vital signs, an initial hemodynamic assessment should include a review of current medications, cardiac rhythm, and central venous pressure (CVP). If the patient has a pulmonary artery catheter in place, pulmonary artery pressure (PAP) and pulmonary artery occlusive pressure (PAOP) should be measured. Data should be obtained to allow for calculation of cardiac output (CO), SvO_2, and systemic vascular resistance (SVR) (Khalpey, Ganim, & Rawn, 2008). PAP readings and waveforms

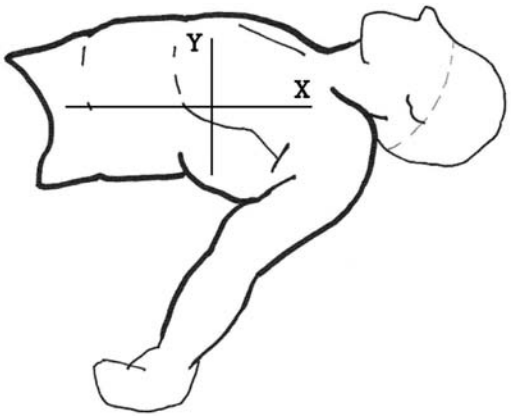

Figure 6–3 The phlebostatic axis (intersection of the X and Y reference lines).
Source: Illustrated by James R. Perron

should be confirmed with the anesthesia provider to assess any changes and the PA catheter location should be noted. Fluid and medication infusion rates should be titrated to maintain hemodynamic stability.

Among patients who did not have CPB, hypothermia is uncommon. A temperature less than 35 °C (95 °F) is considered hypothermic; in such a case, warming techniques should be implemented. Patients who have undergone MICS are routinely hypovolemic and have labile blood pressure, requiring volume repletion to achieve hemodynamic stability. The frequency with which vital signs and a hemodynamic profile are obtained depends on the facility guidelines and the patient's condition.

The patient's height and weight should be entered into the monitoring system database to assure accurate calculations based on body surface area (e.g., cardiac index). If the patient is hypothermic, cardiac index values will be skewed and reflect "cold numbers." A true hemodynamic picture will not be reflected until the patient is normothermic.

Lab specimens should be collected as prescribed by unit protocol or as indicated by the patient's clinical status. Baseline postoperative labs will likely include an arterial blood gas, ionized calcium, serum chemistries, coagulation profile, and a complete blood count (CBC). If the patient is bleeding, a fibrinogen level may be obtained as well. The patient should also have a portable chest radiograph and an ECG performed.

An ECG after a beating heart, minimally invasive procedure is imperative because graft closure secondary to thrombosis, arterial graft spasm, and a kinked conduit are all potential complications. Early graft closure may be manifested as ST-segment elevation, T-wave inversion, and Q waves present in the leads reflective of the revascularized myocardium (Edgar et al., 1999).

Chest tubes are connected to –20 cm suction and should be assessed for amount and type of drainage, patency, and presence of clots. If a patient's blood pressure permits, the head of the bed should be elevated to facilitate chest tube drainage.

Secondary Assessment: The Next 15 Minutes

After initial stabilization of the MICS patient is achieved and a preliminary patient assessment for clinically significant issues (e.g., bleeding, hypotension, hypertension, agitation, dysrhythmias) is performed, a more focused head-to-toe assessment is completed. The neurologic assessment is ongoing and more complete as the patient emerges from anesthesia. Skin is assessed for temperature, color, and location of incisions (procedure based). Pain should be anticipated, and its level should be assessed with a scale appropriate for the cognitive status of the patient. If a mini-thoracotomy incision was made, pain remains an expected finding.

Measures to relieve the thoracotomy incision pain should be implemented. In the initial postoperative period, patients will receive opioid analgesics. They should also be premedicated prior to potentially painful procedures (e.g., chest tube removal), although studies suggest that this care is not

Table 6–4 Hemodynamic Parameters

Parameters	Normal Values
Systolic and diastolic blood pressure	100–130/60–90 mm Hg
Mean arterial pressure	70–105 mm Hg
Right atrial pressure (central venous pressure)	0–8 mm Hg
Right ventricular pressure	25–30/0–8 mm Hg
Pulmonary artery pressure	15–30/6–12 mm Hg
Pulmonary artery occlusive pressure	4–12 mm Hg
Derived Hemodynamic Parameters	
Cardiac output/cardiac index	4–8 L/min; 2.5–4.2 L/min/m^2
Systemic vascular resistance	770–1500 dyne/sec/cm^{-5}
Pulmonary vascular resistance	20–120 dyne/sec/cm^{-5}
Systemic vascular resistance index	1680–2580 dyne/sec/cm^{-5}
Pulmonary vascular resistance index	69–177 dyne/sec/cm^{-5}
Stroke volume/index	60–130 mL/beat; 30–65 mL/beat/m^2
Right ventricular stroke work	8–16 g-m/beat
Right ventricular stroke work index	5–10 g-m-m^2/beat
Left ventricular stroke work	58–104 g-m/beat
Left ventricular stroke work index	50–62 g-m-m^2/beat
Oxygenation Parameters	
Arterial oxygen saturation	95–100%
Mixed venous oxygen saturation	60–80%
Arterial oxygen content	17–20 mL/dL
Venous oxygen content	12–15 mL/dL
Oxygen delivery	900–1150 mL/min
Oxygen consumption	200–290 mL/min
Oxygen extraction ratio	22–30%

Sources: Blount, 2007; Khalpey, Ganim, & Rawn, 2008; LiDCO, 2008.

provided consistently (Puntillo, 1994). As discussed in Chapter 14, inadequate analgesia can result in tachycardia, increased peripheral vascular resistance, imbalance between oxygen supply and demand, hypoxemia, pneumonia, and atelectasis. Lower levels of pain are typically encountered with a minimally invasive procedure. Chest tubes, however, can be a source of pain.

HEMODYNAMIC MONITORING

Successful hemodynamic monitoring begins with knowing the normal range for hemodynamic values. "Normal," in this case, is a relative term, as normal values are based on healthy individuals with healthy hearts. The values most commonly monitored in cardiac surgery patients are listed in Table 6–4 and covered in more detail in Chapter 9.

Managing a patient's hemodynamic profile entails evaluating the patient's clinical condition and past medical and surgical histories so the correct decision is made about how to optimize preload, afterload, and contractility. Depending on comorbidities, hemodynamic values may be skewed. For example, a patient with pulmonary hypertension may have elevated pulmonary artery pressures and CVP secondary to lung disease. A valuable source for a patient's baseline hemodynamic values

Table 6–5 Commonly Used Cardiac Surgery Postoperative Medications

Medication	Dosage Range
Vasodilator Medications	
Nitroglycerin	0.25–3 mcg/kg/min *or* 10–200 mcg/min
Nitroprusside	0.5–10 mcg/kg/min
Vasoactive Medications	
Dobutamine	2–20 mcg/kg/min
Dopamine	2–20 mcg/kg/min
Epinephrine	1–30 mcg/min
Milrinone	Loading dose: 50 mcg/kg, then 0.1–0.75 mcg/kg/min
Norepinephrine	1–40 mcg/min
Phenylephrine	10–500 mcg/min
Vasopressin	0.01–0.1 unit/min
Antiarrhythmic Medications	
Amiodarone	Loading dose: 150–300 mg (depending on presence or absence of pulse) Maintenance infusion: 1 mg/min × 6 hours; then 0.5 mg/min for the next 18 hours
Diltiazem	5–15 mg/hr
Lidocaine	1–4 mg/hr

Sources: Albright, Zimmerman, & Selzman, 2002; Bojar & Warner, 1999; Kayser & Schell, 2006; Levy, Bailey, & Deeb, 2002.

is the cardiac catheterization lab report. Trying to maintain a patient with underlying disease within the standard norms is unrealistic and can even be detrimental to the patient. For example, a patient with hypertension may not have adequate kidney perfusion with a MAP of 80 mm Hg, but instead may need a slightly higher MAP of 90–95 mm Hg to maintain end-organ perfusion.

The key to hemodynamic stability starts with maintaining and normalizing heart rate and stroke volume (SV). This goal may be accomplished through the administration and titration of fluids and medications. SV is affected by preload, afterload, and contractility (Zellinger, 2007); these variables are described further in Chapter 9.

Some of the more common etiologies of hemodynamic compromise are myocardial ischemia, hypothermia, and postoperative dysrhythmias (Khalpey et al., 2008). These are discussed in detail in Chapters 13 and 15. Table 6–5 lists some of the medications most commonly used to optimize hemodynamic status. The goal of therapy is optimal end-organ perfusion with hemodynamic stability. Although hemodynamic parameter goals should be individualized, suggested minimum values for most patients will likely include an SvO_2 near 60%, a MAP greater than 65 mm Hg, and cardiac index (CI) greater than 2 L/min/m^2 (Khalpey et al., 2008).

POSTOPERATIVE COMPLICATIONS

Postoperative complications of cardiac surgery in general are discussed in detail in Chapter 13. It is essential for the ICU nurse to monitor patients who have undergone MICS procedures for development of complications, inter-

vene to prevent them from occurring, and promptly recognize and treat any complications that develop following cardiac surgery.

MICS-Specific Complications

Some of the complications related to MICS specifically are felt to be related to the more technically challenging nature of these procedures and to procedure-related stress on the heart. Complications reported in the literature include dysrhythmias, hypotension, MI, bleeding, brain injury (if intraoperative blood flow is decreased), infection, pulmonary complications, and bone and muscle surgical site injury (Cowles, 2008). Lung herniation, while rare, has also been reported (Athanassiadi, Bagaev, Simon, & Haverich, 2008).

Dysrhythmias

Dysrhythmias are common following cardiac surgery. AF can occur in patients with no prior history from electrolyte imbalances, volume overload, surgical manipulation, or acid–base imbalance. Without the atria contracting (atrial kick), there is a 25–30% loss of cardiac output. Management with an AV nodal blocker such as diltiazem (Cardizem®) or metoprolol (Lopressor®) for rate control, or amiodarone for rhythm conversion, is recommended. If AF is accompanied by hemodynamic instability, or if the patient experiences a rapid ventricular response that does not respond to pharmacologic therapy, synchronized cardioversion is recommended (Fuster et al., 2006).

The patient who underwent valve surgery or a Maze procedure may develop bradycardia or heart block as a result of intraoperative manipulation around the conduction system (Ishikawa et al., 2007). Such a patient may have epicardial pacing wires in place from surgery, so the heart can be temporarily controlled. If pacing wires were not placed during the procedure, transcutaneous pacing pads or transvenous pacing wires may be placed (Lemmer, Richenbacher, & Vlahakes, 2003). Another option is a pulmonary artery catheter with a pacing port, which can pace the ventricle. Institution-specific equipment will dictate which pacing options are available.

Lethal arrhythmias, such as ventricular tachycardia (VT) or ventricular fibrillation (VF), can also occur in the immediate postoperative period. Electrolyte imbalance, cardiac irritability from the surgery, and acidosis may be contributing factors to their development. Resuscitation of these dysrhythmias should follow the American Heart Association (AHA) recommendations. Development of VF may require opening the patient's chest at the bedside. Postoperative dysrhythmias are discussed in more detail in Chapter 15.

Hypothermia

Hypothermia can present a variety of problems postoperatively. For example, it can cause bleeding, platelet dysfunction, and generalized impairment of the coagulation cascade. It may also stimulate the sympathetic nervous system, leading to hemodynamic instability, dysrhythmias, vasoconstriction, hypertension, and increased SVR, thereby making the heart work harder to pump. These effects can increase myocardial oxygen demand and produce myocardial ischemia (Frank, 2001; Khalpey et al., 2008). Controlled rewarming with any available method (e.g., forced warm-air device, fluid warmer) is an essential nursing intervention.

Shivering is another problem associated with hypothermia. If a patient is shivering, there is a twofold to threefold increase in oxygen consumption (seen as a decrease in SvO_2) and CO_2 production. Shivering also causes adrenergic stimulation and discomfort (Bhattacharya, Bhattacharya, Jain, & Agarwal, 2003). For all these reasons, controlling shivering is important. One of the few effective agents to treat postoperative shivering is meperidine (Demerol®) (Kranke, Eberhart, Roewer, & Tramèr, 2002).

Rewarming the patient causes vasodilation, resulting in decreasing blood pressure and

filling pressures. Administration of volume and vasopressors may be indicated. Postoperative cognitive impairment due to cerebral hyperthermia has been reported in patients who are warmed too quickly following cardiac surgery (Borger & Rao, 2002). Hypothermia is more prevalent in patients who have undergone on-pump procedures.

Bleeding

Postoperative bleeding is a risk with any cardiac surgery, including a minimally invasive approach. The risk increases if the procedure is performed on CBP, as higher doses of heparin are administered. Hemodilution, fibrinolysis, and hypothermia are also risk factors for postoperative bleeding.

When assessing chest tube drainage, patency as well as the consistency and color of drainage should be noted. Dark blood will usually indicate venous or older blood; bright red blood is usually arterial or fresh blood. If an off-pump patient is bleeding, it is generally surgical in nature. Postoperative bleeding is discussed in more detail in Chapter 13.

POSTOPERATIVE VENTILATORY SUPPORT

Patients who undergo MICS may be extubated in the operating room. If not, they can generally be weaned from the ventilator relatively quickly (3–6 hours). Patients may be weaned from mechanical ventilation when certain criteria, which may vary among facilities, are met. In general, these conditions may include the patient being awake and cooperative, dissipation of neuromuscular blocking agent effects (usually manifested with a sustained head lift), hemodynamic stability, absence of dysrhythmias, ABG values within the physiological range, normal chest radiograph findings, normothermia, no evidence of bleeding once the sternum has been closed, chest tube drainage less than 100 mL/hr, and urine output more than 1 mL/kg/hr (Chikwe et al., 2006; Kapoor et al., 2008). In addition, other factors should be considered while weaning the patient off ventilatory support, such as surgical technique used (on-pump versus off-pump; CPB may lead to capillary leak), patient age, comorbidities, length of time in the operating room, intraoperative course, and presence of any postoperative complications. Weaning from mechanical ventilation following cardiac surgery is discussed in detail in Chapter 11.

RECOVERY FROM MICS

Patients who undergo MICS procedures traditionally have a quicker and less complicated recovery than patients who undergo conventional CABG procedures. Table 6–6 summarizes the recovery associated with these procedures.

SUMMARY

Minimally invasive cardiac surgery, with all of its benefits, provides a viable option for the patient who meets the criteria established for this type of procedure. The major benefit is the decreased level of pain associated with MICS, which leads to early ambulation and better pulmonary toileting. Decreased incidence of postoperative complications related to CPB and aortic cross-clamping, intraoperative anticoagulation, cardioplegia, and sternal wound infections have all been documented.

The cardiac surgery ICU nurse takes on many roles when caring for this type of patient: educator, advocate for the patient and family, and collaborator with the multidisciplinary team.

Robotic surgery, while still in its infancy, continues to evolve. Indeed, with continued training on the part of the surgeon and the development of new equipment, a new era of cardiac surgery is yet to unfold. While MICS is not an innovative new treatment for cardiac disease, it is certainly an attractive new approach to a traditional procedure.

Table 6–6 Recovery from Cardiac Surgery

	LOS in ICU	LOS in Hospital	Time to Return to Normal Activities
MIDCAB	1 day	3 days	2 weeks
OPCAB	1 day	5–7 days	2–3 months
Traditional CABG	1–3 days*	5–10 days	2–3 months

*Data vary regarding definition of prolonged ICU stay, ranging from ≥ 2 or 3 days.

CABG = coronary artery bypass grafting; ICU = intensive care unit; LOS = length of stay; MIDCAB = minimally invasive direct coronary artery bypass; OPCAB = off-pump coronary artery bypass.

Sources: Abrahamyan, Demirchyan, Thompson, & Hovaguimian, 2006; Atoui, Ma, Langlois, & Morin, 2008; Cowles, 2008.

CASE STUDY

A 70-year-old patient came to the emergency department with chest pain, shortness of breath, and nausea. He reported having multiple similar episodes over the last month, with the symptoms normally resolving with rest after 30 minutes. Today, the symptoms continued for more than 2 hours, and his wife forced the patient to seek treatment. He rated his pain as an 8 on a 0 to 10 scale. He also reported a history of COPD, 50-pack-year smoking history, chronic renal insufficiency, diet-controlled diabetes, and transient ischemic attack.

The patient was immediately evaluated. Oxygen was applied, an IV established, and labs obtained, including a CBC, chemistry panel, PT/PTT, CK-MB, troponin, and arterial blood gas. Sublingual nitroglycerin was administered 3 times with no relief of pain; 2 mg of IV morphine was given, which decreased the patient's pain from an 8 to a 6. A 12-lead ECG revealed ST elevation in leads I, aVL, V_3, V_4, V_5, and V_6, indicating an anterior lateral wall MI.

The patient was taken to the catheterization lab. Cardiac catheterization revealed a proximal occlusion of the LAD with good collateral circulation. The right coronary and the circumflex arteries had 10% occlusion, and the patient's ejection fraction was 40%. He was deemed not to be a candidate for angioplasty or stent placement because of a tortuous arterial anatomy.

A MIDCAB procedure was scheduled owing to the patient's comorbidities. Preoperative education was performed by the ICU nurse on the evening before surgery was scheduled.

Early the next morning, the patient underwent a MIDCAB procedure with a minithoracotomy approach under general anesthesia. The surgeon anastomosed the LIMA to the LAD; no cardiopulmonary bypass was required.

The patient was transported to the ICU, hooked to hard-wire monitoring, and placed on a ventilator. He had an intra-arterial catheter, left pleural chest tube to a drainage collection device at –20 cm suction, and urinary catheter. Initial labs were obtained and the assessment completed. Hemodynamic data were as follows: heart rate 90; sinus rhythm with no ectopy; BP 112/64 (80); PA 32/20 (24); CVP 13; CO 3.5; CI 1.9; SVR 1554; SvO_2 70; SpO_2 100%; temperature 36.0 °C; urinary output. 180 mL; and CT drainage 50 mL. A portable chest radiograph and ECG were obtained. The patient was extubated within 2 hours after his arrival to the ICU.

A postoperative angiography revealed a patent graft. The patient's postoperative course was uneventful, and he was discharged home on postoperative day 5.

Critical Thinking Questions

1. Which postoperative test will provide the necessary information about this patient's graft status, including the patency of the graft?
2. If graft occlusion has occurred, which ECG findings will be present in this case?
3. Why is diltiazem ordered postoperatively?

Answers to Critical Thinking Questions

1. 12-lead ECG
2. The patient will likely manifest ST-segment elevation, T-wave inversion, and the presence of Q waves in leads V_1–V_5, indicating changes in the anterior wall—that is, the area of the heart that was revascularized.
3. One of the causes of graft closure is IMA graft vasospasm. Administration of a calcium channel blocker helps to prevent postoperative spasm of the graft vessel.

SELF-ASSESSMENT QUESTIONS

1. Which of the following is *not* an advantage of MICS over traditional CABG?
   a. Shorter operative time
   b. Breastbone not retracted
   c. Less blood loss
   d. Better cosmetic results
2. A patient who has undergone a MIDCAB procedure asks you when he should be able to return to work. Which of the following time frames should you give him?
   a. 4–5 days after discharge from hospital
   b. 2 weeks
   c. 2–3 months
   d. 6–10 months
3. Which of the following statements is true regarding minimally invasive valve surgery?
   a. It doesn't require being placed on a bypass machine.
   b. There is a decreased chance of infection.
   c. Patients who are obese may be good candidates for this approach.
   d. It is the ideal approach for patients with multiple forms of valve disease.
4. An advantage of robotic surgery over other forms of MICS may include:
   a. experience is not as essential since the surgeon is not actively doing the procedure
   b. there is a decreased chance of blood loss
   c. cosmetic results
   d. lower risk of infection
5. Which of the following statements is true regarding MICS procedures?
   a. No bone cartilage is removed.
   b. The ascending aorta is manipulated.
   c. Use of a cardiac stabilizer is required.
   d. The heart temporarily loses pericardial support.
6. Valve procedures require cardiopulmonary bypass because
   a. of the anatomic location involved.
   b. of surgeon preference.
   c. there is less risk of postoperative bleeding.
   d. comorbidities associated with valve disease.

7. Which of the following is a potential manifestation of a patient experiencing postoperative shivering following MICS?
   a. Increased SvO_2
   b. Decreased CVP
   c. Increased pCO_2
   d. Decreased sympathetic stimulation
8. One advantage associated with endovascular "keyhole" procedures is
   a. increased accuracy of depth perception.
   b. less experience is required because the procedure is less invasive.
   c. increased degree of motion of the surgeon's hands.
   d. enhanced visibility.
9. Which of the following patients is a good candidate for MICS?
   a. Male, age 80, 70% circumflex occlusion, inpatient, cardiogenic shock
   b. Female, age 50, morbid obesity, day 2 s/p inferior lateral MI, IABP
   c. Male, age 63, mitral valve regurgitation, EF 60%, plays golf 3 times a week
   d. Female, age 40, s/p bilateral mastectomy, 3-vessel CAD
10. A benefit of the Cox/Maze III procedure for atrial fibrillation is
    a. this procedure uses ablation so return of AF is unlikely.
    b. this procedure does not require the patient to go on cardiopulmonary bypass.
    c. incisions are made on both atria to stop irregular electrical activity.
    d. no sternotomy incision is required.

Answers to Self-Assessment Questions

1. a
2. b
3. b
4. d
5. c
6. a
7. c
8. d
9. c
10. c

Clinical Inquiry Box

Question: Do patients who undergo minimally invasive cardiac surgery have better outcomes or quality of life?

References: Yamada, T., Ochiai, R., Takeda, J., Shin, H., & Yozu, R. (2004). Comparison of early postoperative quality of life in minimally invasive versus conventional valve surgery. *Journal of Anesthesia*, *17*(3), 171–176.

Objective: To evaluate the quality of life in patients who undergo minimally invasive cardiac surgery.

Method: Two groups of patients were included in the study—66 who underwent MICS and 50 who underwent conventional cardiac surgery (CCS). Patients had either aortic or mitral valve surgery.

Results: Shorter hospitalization, less pain medication use, and a lower incidence of delirium were found with the MICS group as compared with the CCS group. Members of the MICS group were able to take food earlier and have a urinary catheter removed before members of the CCS group.

Conclusion: Earlier recovery from MICS procedure as compared to CCS allows for improved quality of life through the early reinstitution of daily activities.

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WEB RESOURCES

Minimally invasive valve surgery: www.youtube.com/watch?v=y4pHVQvBhx0

Minimally invasive aortic valve surgery: www.youtube.com/watch?v=wT23obEeVlM

Minimally invasive robotic cardiac surgery: www.youtube.com/watch?v=phs8CG0iTyI&feature=related

Minimally invasive direct coronary artery bypass (MIDCAB): www.youtube.com/watch?v=V4vvSAwaBtU

Hybrid Maze procedure: www.youtube.com/watch?v=ubxVAqoENwI

Chapter 7

Cardiopulmonary Bypass and Off-Pump Coronary Artery Bypass

Julie Miller

INTRODUCTION

For years, nurses have cared for patients who have undergone traditional coronary artery bypass grafting (CABG) surgery, in which the patient is placed on a cardiopulmonary bypass circuit. Since 1990, however, nurses have seen an increase in the number of patients undergoing off-pump coronary artery bypass (OPCAB) surgery, in which the surgeon sews the grafts onto the beating heart. Nursing care of patients who have received the CABG and OPCAB procedures has a number of similarities and differences.

As recently as 15 years ago, coronary bypass surgery patients spent 2 to 3 days on a ventilator, sedated, with a pulmonary artery catheter (PAC) in place and multiple vasoactive drips infusing to maintain optimal hemodynamic status. Today, a patient undergoing CABG or OPCAB may be discharged from the operating room extubated, without a PAC, and be transferred from the ICU to a progressive care unit within 12 hours of surgery. Regardless of the short stay, patients remain critically ill when transferred from the ICU. Nurses are often faced with the challenge of patients and families who are anxious over the potential for death throughout the course of hospitalization.

Anxiety during the preoperative and postoperative periods has been correlated with poor outcomes such as increased pain levels (Nelson, Zimmerman, Barnason, Nieveen, & Schmaderer, 1998), more readmissions (Duits, Boeke, Taams, Passchier, & Erdman, 1997), poor psychological outcomes (Boudrez & De Backer, 2001), and worse quality of life (Duits et al., 1997). Factors predictive of increased anxiety include being female, having to wait for surgery, pain prior to surgery, concerns over returning to work, prior anxiolytic or antidepressant use, and difficulty sleeping (Gallagher & McKinley, 2007). Nurses must assess patients' anxiety levels throughout the hospitalization and seek to understand the best patient-specific approach in countering their stress. The provision of realistic information about what to expect through every step of the care delivered and effective pain management are crucial in decreasing anxiety levels.

Despite the need to address anxiety levels, the hemodynamic challenges, constant observation for potential complications and need for the astute critical care nurse remain the same. This chapter will explore the similarities and differences in the care of the traditional on-pump coronary artery bypass grafting (CABG or ONCAB) patient compared to the patient who undergoes off-pump bypass (OPCAB).

POTENTIAL COMPLICATIONS OF BYPASS SURGERY

Stroke, infection, bleeding, dysrhythmias, myocardial infarction, gastrointestinal dysfunction, renal failure, and death are all potential

complications for the bypass surgery patient, whether the procedure is performed with the on- or off-pump technique. The risk for atrioventricular heart block is present in both types of bypass procedures, and both types of patients will have epicardial pacing wires placed. Nursing challenges for bypass surgery patients include ensuring hemodynamic stability, monitoring for and treating cardiac dysrhythmias, balancing the need to adequately medicate for pain while guarding against oversedation and respiratory complications, and monitoring for and intervening to prevent the myriad of potential postoperative complications.

ONCAB patients undergo surgery while their heart is not beating. In this procedure, through a median sternotomy incision, the heart is stopped using cardioplegia solution. Oxygen needs are met by cannulating the aorta and placing the patient on the cardiopulmonary bypass (CPB) circuit. ONCAB carries a higher risk of aortic dissection and embolization because of the cannulation of the aorta for bypass procedures (Wijeysundera et al., 2005).

Heparin is utilized to maintain patency of the CPB circuit and to reduce the risk of microemboli formation. Heparin-induced thrombocytopenia (HIT) and bleeding are potential complications for all patients receiving heparin. In addition, the CPB circuit can contribute to the development of systemic inflammatory response syndrome (SIRS) and microemboli (Bruins et al., 1997). Moderate hypothermia is utilized during the ONCAB procedure to decrease myocardial oxygen demand. The postoperative rewarming process contributes to vasodilation and can worsen the effects of SIRS.

As part of the ONCAB procedure, the bypass grafts are sewn onto the heart and aorta while the heart is not beating. When the surgery is completed, the heart is restarted and the CPB circuit withdrawn. There is a risk that the patient will not be able to be weaned from CPB and may require an intra-aortic balloon pump (IABP) or pacemaker postoperatively. Intra-aortic balloon pump therapy is discussed in detail in Chapter 10. On rare occasions, a patient's heart does not restart following CPB.

OFF-PUMP CORONARY ARTERY BYPASS

OPCAB is performed either through a median sternotomy incision or via a thoracotomy incision, also known as minimally invasive direct coronary artery bypass (MIDCAB). Robotic-assisted coronary artery bypass (ROBOCAB) surgery is another type of off-pump procedure that is done through a minimally invasive approach. Minimally invasive surgery is discussed in detail in Chapter 6.

In OPCAB, the surgeon sews the grafts onto the beating heart using specialized instruments to stabilize the myocardial tissue where the surgeon is sewing the graft (St. Andre & DelRossi, 2005). These instruments, known as stabilizers, are similar in shape to the sewing foot for a sewing machine (see Figure 7–1).

Studies have noted that patients undergoing OPCAB receive fewer grafts than those undergoing ONCAB. This pattern may lead to a higher reintervention rate for OPCAB patients (Sedrakyan, Wu, Parashar, Bass, & Treasure, 2006). The risk for aortic dissection

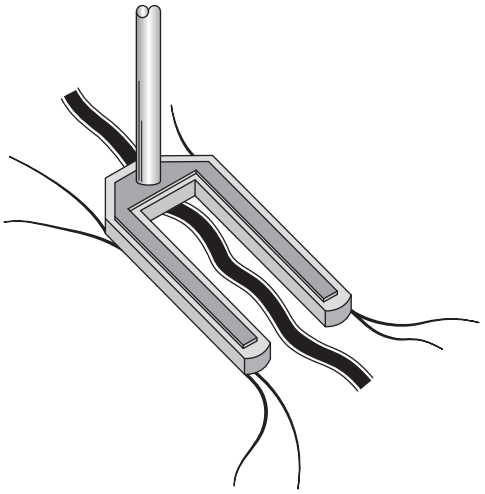

Figure 7–1 Stabilizer used in OPCAB.
Source: Illustrated by James R. Perron

with OPCAB is less than traditional CABG, however (Shekar, 2006). Approximately 50 of 1000 (5%) patients undergoing off-pump bypass procedures may need to be converted to on-pump procedures (Sedrakyan et al., 2006). This possibility should be discussed with the patient and family during preoperative teaching.

OPCAB is performed on a patient with either mild hypothermia or normothermia. Hypothermia contributes to postoperative bleeding by causing impairment in the clotting cascade. It is theorized that less bleeding occurs with mild hypothermia as compared to the moderate hypothermia (30–34 °C) utilized in the ONCAB procedure. Mild hypothermia does help reduce myocardial oxygen demand and may be beneficial to both ONCAB and OPCAB patients (St. Andre & DelRossi, 2005).

COMPLICATIONS OF ON-PUMP SURGERY VERSUS OFF-PUMP SURGERY

Off-pump coronary artery bypass grafting, also known as a beating heart procedure, was developed partly to offset the risk of postoperative alterations associated with on-pump procedures. Specifically, patients who undergo OPCAB are felt to be less likely to develop cerebral hypoperfusion, embolization, and inflammatory response associated with on-pump procedures (Abu-Omar, Balacumaraswami, Pigott, Matthews, & Taggart, 2004; Demaria et al., 2002; Fearn et al., 2001). Recent work has found that the cytokine and chemokine production is similar in ONCAB and OPCAB, but biomarkers such as ecotaxin, macrophage inflammatory protein-1 beta (MIP-1β), and interleukin-12 (IL-12) were found more prevalent in the setting of ONCAB. Although more research needs to be conducted on the inflammatory response most often seen in ONCAB, OPCAB does appear to produce less of an inflammatory response, which can improve cardiopulmonary outcomes (Castellheim et al., 2008).

Assessment for postoperative bleeding is essential, especially given that mediastinal reexploration rates for bypass surgery patients are as high as 5% (Raja, 2005). Bleeding in these patients can be attributed to CPB, hypothermia, fibrinolytic agents administered during the procedure, heparin reversal, and loose anastomoses. As OPCAB was developed, concern was voiced that these patients would have more bleeding due to the risk of sewing onto the beating heart. In fact, data from randomized controlled trials suggest that OPCAB patients experience less bleeding postoperatively than ONCAB patients (Raja, 2005).

In all post-bypass patients, assessment for bleeding is necessary. The mediastinal and pleural tubes must be monitored hourly for amount and quality of drainage, including assessment for clots. Monitoring for narrowing of pulse pressure is performed, as this finding could indicate cardiac tamponade in the post-bypass patient.

Heparin is utilized to maintain vessel patency and prevent thrombus formation during OPCAB, but the amount is about one-third to one-half the dose used in traditional CABG (Zenati, 2005). Because heparin is utilized in both on- and off-pump procedures, it is imperative that the nurse assess all post-bypass patients for bleeding, check lab data for presence of a coagulopathy, and assess for HIT.

Protamine is a polypeptide isolated from salmon sperm (Arslan, Tarhan & Yilmaz, 2005). It is utilized in both on- and off-pump procedures to bind heparin and reverse its anticoagulant effect (Stafford-Smith et al., 2005). In one study, researchers estimated that protamine caused adverse events in approximately 2.6% of cardiac surgery patients (Kimmel, Sekeres, Berlin, Goldberg, & Strom, 1998). Risk factors for protamine reactions include being a diabetic patient who uses protamine-containing insulin, previous drug reaction, and allergy to protamine or fish. An estimated 39% of bypass surgery patients have these risk factors (Kimmel et al., 1998).

A minor protamine reaction may result in hypotension and an increase in pulmonary artery pressures (St. Andre & DelRossi, 2005). This effect is more common in patients who have diabetes, perhaps related to their use of protamine-containing insulin. Anaphylaxis has been associated with administration of protamine, and the affected patient may suffer cardiac arrest. Any adverse reaction to protamine increases the risk of mortality for both ONCAB and OPCAB patients (Welsby et al, 2005).

The critical care nurse must be vigilant in monitoring for protamine reactions, including assessing the patient for different presentations of these reactions. Massive systemic vasodilation is manifested by hypotension, decreased systemic vascular resistance (the amount of work the heart must do to eject blood), and increased cardiac output (the amount of blood ejected by the heart every minute). Acute pulmonary vasoconstriction will lead to an increase in pulmonary artery pressures (PAP) with subsequent right ventricular failure. The hemodynamic profile in this type of reaction will reveal bradycardia, decreased cardiac output, elevated PAP, and elevated systemic and pulmonary vascular resistance.

In recent years, studies have tested new drugs suggested as candidates to replace protamine for reversing heparin and improve the safety of the bypass procedure for all CABG patients. Unfortunately, none of these drugs has demonstrated a superior safety profile as compared to protamine during clinical trials (Stafford-Smith et al., 2005). As a consequence, heparin–protamine remains the only drug combination approved for use in the CPB circuit.

Recent studies have evaluated a direct thrombin inhibitor, bivalirudin (Angiomax®), as a replacement for heparin anticoagulation for CPB and OPCAB. These studies indicate that bivalirudin can be used safely in patients with heparin allergy or increased risk for HIT (Koster et al., 2006; Koster et al., 2007). Nursing care for a patient receiving bivalirudin includes astute monitoring of lab data and for bleeding.

Patients who undergo bypass procedures may develop postoperative temporary metabolic, hemodynamic, and neurohormonal changes (Hedges & Redeker, 2008). For example, in one study, on- and off-pump cardiac surgery patients were evaluated at 24-hour intervals. Both groups of patients had elevated cardiac markers and white blood cell, neutrophil, and monocyte counts postoperatively; the levels were consistently and significantly higher in the on-pump group. In addition, the hematologic abnormalities persisted longer in the on-pump group. Patients who underwent OPCAB had less of a rise in serum lactate levels. Those whose peak lactate level was greater than 4.0 mmol/L were more likely to develop postoperative morbidities, including those hemodynamic, pulmonary, and renal in nature as well as myocardial infarction. The same group of patients had a greater tendency toward hypoxic episodes, were intubated longer, had a higher length of stay, and consumed more hospital resources. Three patients in the on-pump group required postoperative use of the IABP (Warang et al., 2007).

Hemodynamic alterations may occur after cardiac surgery. In one study, patients who underwent OPCAB developed a postoperative increase in pulmonary artery pressure and a concomitant drop in cardiac output (Do et al., 2002). In another study, patients receiving on-pump cardiac surgery developed a decrease in cardiac output and increase in SVR. These hemodynamic effects were attributed to the transient decline in triiodothyronine concentrations that is associated with CABG. Patients who received thyroid hormone perioperatively demonstrated improvement in cardiac output and SVR (Klemperer et al., 1995).

HEMODYNAMIC MONITORING

In the initial postoperative period for both ONCAB and OPCAB patients, the primary focus is hemodynamic stability. The first 6 hours postoperatively tends to be when the patient is the most vulnerable and unstable.

Cardiac dysfunction tends to manifest as decreased compliance and contractility from the pressure-overloaded myocardial tissue. A pressure-overloaded ventricle will have reduced compliance and be stiff, which will result in a decreased ejection fraction, cardiac output, and contractility.

Preoperative ischemia and duration of the operative procedure contribute to instability in patients who undergo either on- or off-pump procedures. In the ONCAB patient, hemodynamic instability is related to effects from the CPB circuit and the cold potassium cardioplegia used to reduce myocardial oxygen demand. In contrast, manipulation of the beating heart for OPCAB leads to decreased compliance and contractility (St. Andre & DelRossi, 2005).

A patient who has had valve replacement is typically volume overloaded (St. Andre & DelRossi, 2005). In both ONCAB and OPCAB surgeries, fluid needs may be higher than expected; thus the critical care nurse will need to assess all interventions for their effect on hemodynamics to ensure adequate preload. Hemodynamic profiles of cardiac surgery patients are discussed in detail in Chapter 9.

RISKS OF ON-PUMP SURGERY VERSUS OFF-PUMP SURGERY

A number of risks are associated with coronary artery bypass surgery, whether it is performed on an on- or off-pump basis. Specifically, stroke, atrial fibrillation, acute renal failure, acute liver failure, bleeding, infection, and death have all been associated with on- and off-pump surgery.

The ONCAB procedure and the CPB circuit have shown to increase the risk for development of acute renal failure, stroke, liver failure, atrial fibrillation, and bleeding (Sedrakyan et al., 2006). Use of the CPB circuit has also been associated with the development of microemboli and SIRS. SIRS occurs in OPCAB patients, albeit to a lesser degree than in ONCAB patients (Raja, 2005). The risk of death for both OPCAB and ONCAB is approximately 1% to 2% (Shekar, 2006). Nursing interventions for both OPCAB and ONCAB patients include assessment for and prevention of these adverse events and preoperative teaching that includes a discussion of these potential risks.

OPCAB was developed to try to minimize the risks of the cardiopulmonary bypass circuit (Verma et al., 2004). A meta-analysis revealed a reduced incidence of stroke, atrial fibrillation, and infections with OPCAB as compared to ONCAB (Sedrakyan et al., 2006). Women undergoing bypass surgery are at a higher risk for complications. Recent studies have also shown that OPCAB benefits women by reducing their intraoperative and postoperative morbidity and mortality rates (Puskas et al., 2007). The off-pump bypass is technically more challenging than ONCAB, and critics cite this difference as a factor that complicates the process of setting up randomized controlled studies and comparing outcomes for on- and off-pump procedures. Many studies that focus on OPCAB are observational, making the ability to generalize the results difficult (Wijeysundera et al., 2005).

Cognitive Decline

Cognitive decline has been noted in patients who have undergone coronary artery bypass. It had been theorized that this decline in function was related to the CPB circuit. In a recent study comparing ONCAB, OPCAB, and healthy patients, however, researchers determined that the rate of cognitive decline in both types of surgery was the same. In this study, OPCAB proponents had theorized there would be less cognitive decline without CPB. Demographic data revealed that cognitive decline was present prior to surgery in both the ONCAB and OPCAB groups at a higher level than in the healthy patients (Selnes et al., 2007). At this time, the decline in cognitive function does not appear to be related to CPB. Cognitive decline in patients undergoing coronary artery bypass surgery will require more study to determine the contributing factors.

Table 7–1 Steps for Performing the Allen Test

Step 1: Simultaneously locate the radial and ulnar artery; palpate and compress them with three digits.
Step 2: Maintaining compression on the radial and ulnar arteries, ask the patient to clench and unclench the hand 10 times.
Step 3: Release pressure from the ulnar artery and monitor the time it takes for flushing to return to the palm, thumb, and nail beds.
Step 4: If the amount of time it takes for flushing to return is greater than 6 seconds, this means that collateral flow is impaired. The radial artery should not be used as a graft.

Source: Asif & Sarkar, 2007.

Graft Occlusion

Both on- and off-pump procedures utilize the saphenous vein and arterial conduits for grafts. Saphenous vein harvesting is accomplished endoscopically, which reduces the pain and scarring associated with the historical harvest approach of an inner thigh to ankle incision (O'Hanlon, 2000). Vein grafts are implanted in a reverse direction relative to their valves and have a higher occlusion rate when compared to the left internal thoracic artery grafts (Desai et al., 2007).

Arterial grafts include the left internal thoracic artery, radial artery, and, less commonly, the right internal thoracic artery. The intrathoracic arteries, also known as mammary arteries, are used to bypass the anterior coronary circulation and require only one anastomosis. The elimination of anastomosis to the ascending aorta may reduce emboli, which might otherwise cause stroke (St. Andre & DelRossi, 2005). Arterial grafts have been shown to decrease the need for revascularization and reduce short- and long-term mortality; approximately 80% of these grafts are still patent 8 years after implantation (St. Andre & DelRossi, 2005).

The radial artery, which was first utilized as a graft in the 1970s, has regained popularity as a graft in recent years due to its long patency duration (Hayward, Hare, Gordon, Matalanis, & Buxton, 2007). Improved harvest techniques for radial artery grafts and the use of calcium channel blockers intraoperatively and postoperatively (e.g., diltiazem [Cardizem®]) for 6 months have produced patency rates similar to those for other arterial grafts at 5 years (Desai et al., 2007; Hayward et al., 2007; Sajja, Mannam, & Sompalli, 2005).

Patient Assessment

Ongoing preoperative and ongoing postoperative assessments are crucial for patients undergoing radial artery harvest. In the preoperative phase, the nurse performs a detailed assessment of the patient's history, activity level, and collateral ulnar blood flow to the affected hand(s). Collateral blood flow to the hand is most commonly assessed by using the Allen test. Specifically, the Allen test is used to assess the adequacy of blood supply to the hand through the ulnar artery. Table 7–1 outlines the performance and evaluation criteria included on the Allen test. The literature varies in interpretation of an Allen test, with 5 to 9 seconds being considered a positive result (Desai et al., 2007; Gurbuz, Findik, Cui, & Aytac, 2007; Hayward et al., 2007). The recommended contraindication for radial graft harvest is a positive Allen test (the red color of the palm returns) in greater than 6 seconds (Asif & Sarkar, 2007). A positive Allen test has been reported to have a predictive value of 53%, which means there is a need to investigate collateral flow further.

Techniques to more closely examine collateral flow include the use of Doppler flow measurements, thumb systolic pressure, finger-pulse plethysmography, and pulse oximetry (Asif & Sarkar, 2007). Some sources suggest that the Allen test could give a false-negative result and

that, regardless of the results, it is always mandatory to have a preoperative ultrasound study if radial artery harvesting is being considered (Agrifoglio et al., 2005).

Patients who perform manual labor, are physically active with their hands, have suffered a stroke with upper limb involvement, have peripheral vascular disease, Raynaud's disease, or experienced a traumatic injury to the affected side should not be considered candidates for radial artery harvest (Desai et al., 2007; Hayward et al., 2007; Serricchio et al., 1999; Shah et al., 2007). Additionally, smoking, diabetes, hypertension, and hyperlipidemia have been associated with diminished radial artery graft patency rates. Recent data suggest that patients with peripheral vascular disease are more likely to have early occlusion of a radial artery graft (Desai et al., 2007).

Data from one small prospective study suggest that radial artery graft patency rates are decreased in the OPCAB population (Gurbuz et al., 2007). Desai and colleagues (2007) report that women are more likely to have longer graft patency with radial artery grafts when compared to saphenous vein grafts for non-left anterior descending bypasses. Although more studies on this topic are necessary, the current evidence points to women benefiting from complete arterial revascularization instead of vein grafting and to patients with peripheral vascular disease benefiting from vein grafts.

Radial Artery Harvesting

In the early development of radial artery harvesting, it was recommended that the nondominant hand be the site of harvest owing to fear of hand ischemia. Shah and colleagues (2007) suggest that harvesting of radial arteries from the dominant hand can be accomplished safely with minimal adverse effects for the patient, as hand ischemia is actually a rare occurrence. Depending on surgeon preference, the radial artery donor site may or may not have a drain placed. If a drain is placed, it is usually removed when drainage is less than 20 mL for 8 hours. The incision will be covered loosely with a gauze dressing and wrapped with a compressive wrap for 24 hours (Schouchoff & Belhumeir, 2000).

Postoperative assessment of the affected extremity includes the amount and quality of drainage, signs and symptoms of infection, and the six P's for diminished arterial blood flow (i.e., pain, pulselessness, pallor, paresthesia, paralysis, and polar [cold]). Patients should be made aware that they may experience loss of motor strength and numbness on the affected extremity. These symptoms usually resolve in most patients 6 months postoperatively. Patients who smoke report higher levels of sensory loss but no difference in motor function compared to nonsmokers (Shah et al., 2007).

Compartment Syndrome

The literature reports a rare occurrence of compartment syndrome in the vein donor limbs for coronary artery bypass (James, Friedman, Scher, & Hall, 2002). Nursing assessment of the donor limb should include assessment for diminished blood flow. Like their counterparts undergoing radial artery harvesting, vein graft donors should have the six P's assessed. Early symptoms of compartment syndrome include severe pain and tenderness on passive stretch. This assessment may be masked by the use of sedation and narcotic analgesia in the early postoperative period.

The demonstrated decrease in complications with OPCAB has had the benefit of reducing the cost of performing CABG (Raja, 2005). Other factors contributing to the reduction in cost for OPCAB are shorter lengths of stay in the ICU, shorter intubation times, and decreased use of blood products due to diminished blood loss (Bayrak et al., 2007). Despite the mounting evidence that OPCAB has some advantages over ONCAB, the majority of bypass surgeries performed in the United States remain on-pump procedures.

SUMMARY

Nursing care of both on- and off-pump coronary artery bypass patients continues to

advance as evidence mounts regarding the risks and advantages of each procedure. On- and off-pump patients remain at risk for a myriad of complications. Patients who undergo off-pump procedures tend to experience a lower incidence of stroke, infection, and atrial fibrillation; a notable cost savings with the use of off-pump procedures has also been documented. As the techniques and utilization of off-pump surgery continue to evolve, so will the skill and practice of the expert cardiac surgery nurse. Care of these patients will continue to be highly challenging and rewarding.

CASE STUDY

A 49-year-old female is scheduled for off-pump bypass surgery utilizing the left internal mammary artery, radial artery, and saphenous veins for grafts. She is 4 days post inferior wall myocardial infarction. Her coronary angiogram showed three-vessel disease with multiple distal stenoses. The patient has a history of hypertension and type II diabetes. She smoked in her twenties but has been smoke free for 20 years.

Critical Thinking Questions

1. Identify three teaching points important for this patient.
2. Discuss the use of radial artery grafts in women.
3. Compare and contrast the advantages/disadvantages of on- and off-pump coronary artery bypass surgery.

Answers to Critical Thinking Questions

1. a. Discuss the risks and possible complications of CABG.
   b. Discuss lifestyle changes and medications needed postoperatively to help ensure long graft survival. Education should be provided on medications, dietary modifications, management of diabetes and high blood pressure, and stress reduction.
   c. Discuss the graft site locations and the postoperative care.
2. Arterial grafts are typically recommended given that they have been found to have longer patency durations than saphenous veins. Given its superior quality, the length of functionality of the radial artery is thought to decrease the need for repeat operations. Studies report that 5-year survival among women who received a radial artery graft was significantly better than among women who did not receive such a graft (Lawton et al., 2005).
3. a. Off-pump CABG does not require that the patient's heart be stopped and that a heart–lung machine be used. Instead, the surgeon uses a stabilizer to hold the tissue in place while the heart is still beating.
   b. Off-pump CABG usually reduces the need for blood transfusion during the procedure and results in shorter hospital stays.
   c. A lower rate of complications is seen with off-pump CABG. Patients are less likely to experience stroke, memory impairment, or decreased ability to concentrate.

SELF-ASSESSMENT QUESTIONS

1. Off-pump bypass has been associated with reduced rates for which of the following complications?
   a. Renal failure, sepsis, and death
   b. Liver failure, bleeding, and decreased cognitive function
   c. Stroke, infection, and atrial fibrillation
   d. SIRS, renal failure, and microemboli formation

2. Patients with which of the following characteristics are more likely to have a protamine reaction?
   a. Allergy to fish
   b. Renal failure, on dialysis
   c. Type II diabetes, taking Glucophage
   d. Bleeding disorder
3. Contraindications to radial artery graft harvest include which of the following?
   a. Allen test of 18 seconds in the nondominant hand
   b. Male patient who lays tile for a living
   c. Female patient with a history of Raynaud's disease
   d. All of the above
4. Surgeons may opt to use which of the following agents for anticoagulation in the CPB circuit for patients with a previous history of heparin-induced thrombocytopenia?
   a. Enoxaparin
   b. Bivalirudin
   c. Heparin
   d. Protamine sulfate
5. A patient has been admitted for OPCAB. Your preoperative assessment reveals cool and pale fingertips. The patient reports weakness from a previous stroke in the donor arm. The surgeon is at the bedside requesting that the patient sign the consent form. What should you do?
   a. Encourage the patient to sign and witness the signature.
   b. Perform the Allen test to assess for collateral flow from the ulnar artery.
   c. Ask to speak to the surgeon in private regarding your assessment findings.
   d. Explain the procedure to the patient, including its potential risks and benefits.
6. Protamine reactions in coronary artery bypass patients present with which of the following signs and symptoms?
   a. Decreased SVR, hypotension, and increased cardiac output
   b. Increased SVR, decreased pulmonary artery pressures, and hypertension
   c. Hypotension and increased SVR and cardiac output
   d. Increased pulmonary artery pressures and decreased SVR and cardiac output
7. Narrowing pulse pressures and clots in the mediastinal tubes could indicate _____ in both on- and off-pump coronary artery bypass patients.
   a. bleeding
   b. cardiac tamponade
   c. protamine reaction
   d. systemic inflammatory response syndrome
8. The cost reductions noted with OPCAB have been linked to
   a. increased use of blood products.
   b. endoscopic harvest of saphenous vein grafts.
   c. use of cardioplegia.
   d. decreased complication rates.
9. Off-pump patients have a reduced incidence of AV blocks and do not have epicardial pacing wires placed intraoperatively.
   a. True
   b. False
10. SIRS has been identified in both on- and off-pump bypass patients. A factor that could worsen the systemic vasodilation associated with SIRS is
    a. rewarming the patient.
    b. prolonged hypothermia.
    c. fluid resuscitation.
    d. administration of vasopressors.

Answers to Self-Assessment Questions

1. c
2. a
3. d
4. b
5. c
6. a
7. b
8. d
9. b
10. a

Clinical Inquiry Box

Question: Is there a difference in sleep patterns postoperatively between on-pump and off-pump CABG?

Reference: Hedges, C., & Redeker, N. S. (2008). Comparison of sleep and mood in patients after on-pump and off-pump coronary artery bypass surgery. *American Journal of Critical Care, 17*(2), 133–139.

Objective: To examine sleep and mood of patients having cardiac surgery with the on- and off-pump techniques.

Methods: After the second postoperative night while staying on a cardiac step-down unit, 129 cardiac surgery patients were asked to complete the Pittsburg Sleep Quality Index, a sleep diary, and the Profile of Mood States tool. Subjects wore wrist actigraphs for measuring movement at night. The sample consisted of 48 patients who underwent on-pump surgery and 81 patients who underwent off-pump surgery.

Results: There was no significant difference between groups on subjective sleep characteristics, mood disturbance, or preoperative sleep quality. Off-pump CABG was associated with less movement as measured by the actigraphs, indicating fewer awakenings. There was no length of sleep difference between the groups. No gender-related difference in outcomes was noted in either the on-pump or off-pump group.

Conclusion: Given that this study was limited by the small sample size, further studies are needed to examine the differences in sleep between on- and off-pump patients. Relationships among sleep and mood should be examined in relation to other variables, such as acuity level, comorbidities, type of cardiac surgery, history of sleep apnea, and characteristics of the hospital environment such as staffing ratios. Cardiac nurses should be aware of the importance of sleep in all cardiac surgery patients. Regardless of whether an on-pump or off-pump surgical approach is used, they should provide interventions that promote sleep in postoperative patients.

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WEB RESOURCES

Off-Pump Video: In a Webcast from Memorial Hermann Heart and Vascular Institute in Houston, TX, cardiovascular and thoracic surgeons Miguel Gomez, M.D., and Donald Gibson, M.D., give the general public a rare glimpse into the operating room to view surgery "off pump," on a beating heart. URL: http://video.google.com/videoplay?docid=9014695099760440284&q=on+pump+and+off

CABG Video for Patient and Families: http://www.brightcove.tv/title.jsp?title=627018303&channel=537078573

Chapter 8

Recovery from Anesthesia

Toni Patrice Johnson

INTRODUCTION

According to the American Heart Association (AHA, 2008), more than 6.9 million cardiac procedures were performed in the United States in 2005. Of these procedures performed, 699,000 total open-heart procedures were reported (AHA, 2009). Thousands of cardiac surgery patients have enjoyed speedy recovery times, thanks to advances in surgical techniques, anesthetic agents, and postoperative medications. More importantly, improvements in anesthetic techniques have allowed patients to transition quickly from the intensive care unit (ICU) for the immediate postoperative period to a general unit and then home in increasingly shorter periods of time. Given these trends, ICU nurses must be assiduous in the care of these patients. This chapter focuses on care in the immediate postoperative period.

HAND-OFF COMMUNICATION

Postoperative care begins immediately after the patient is transferred from the operating room (OR). Following cardiac surgery, patients are typically transferred to the ICU for monitoring, hemodynamic stabilization, assessment for complications, and possibly extubation. Vital information is exchanged in the hand-off communication between the anesthesia provider and the ICU nurse. Data should include pertinent information regarding the surgical procedure, any intraoperative complications or events, hemodynamic and ventilatory status, cardiopulmonary bypass (CPB) time, recent laboratory data, type and amount of intravenous fluids and blood products administered, reversal of anticoagulants, pertinent medical and surgical history, preoperative status, location of intravenous lines and invasive catheters, vasopressor and inotropic agents used, and current infusion rates. Additional information includes use of mechanical cardiac assist devices, presence of pacing wires, length of surgery, estimated blood loss, intraoperative intake and output, patient position on the OR table, location of drains and dressings, and anesthetic and reversal agents administered. Table 8–1 lists the more common anesthetic agents used.

In addition to the information provided by the anesthesia provider, an extensive preoperative evaluation is conducted prior to cardiac surgery. Details of this evaluation are described in Chapter 4. Information about existing comorbidities, cardiac disease, tobacco use, nutritional status, medication history, preoperative cardiac status, and any optimizing that might have taken place prior to surgery will help the ICU nurse anticipate the patient's immediate postoperative course and potentially required interventions. By way

Table 8–1 Anesthetic Agents/ Adjuncts Commonly Used in Cardiac Surgery

Intravenous Induction Agents

Propofol (Diprivan®)
Etomidate (Amidate®)
Thiopental sodium (Pentothal®)
Methohexital (Brevital®)

Neuromuscular Blocking Agents

Rocuronium (Zemuron®)
Vecuronium (Norcuron®)
Succinylcholine (Anectine®)
Atracurium besylate (Tracrium®)
Mivacurium chloride (Mivacron®)
cis-Atracurium (Nimbex®)
Doxacurium (Raplon®)
Pancuronium (Pavulon®)
Tubocurarine
Metocurine

Analgesics/Sedatives

Fentanyl (Sublimaze®)
Sufentanil (Sufenta®)
Alfentanil (Alfenta®)
Remifentanil (Ultiva®)
Morphine sulfate
Midazolam (Versed®)
Lorazepam (Ativan®)

Inhalation Agents

Isoflurane (Forane®)
Sevoflurane (Ultane®)
Enflurane (Ethrane®)
Halothane (Fluothane®)

Sources: Dozier, 2007; Savino & Cheung, 2008.

of illustration, as discussed in Chapter 5, the hemodynamic profile and resultant interventions indicated for patients who undergo cardiac surgery for valvular disease will vary with the pathophysiology of each of the respective disorders (e.g., aortic insufficiency versus stenosis) (Khalpey, Ganim, & Rawn, 2008).

A patient's comorbidities may also help the ICU nurse anticipate problems in the immediate postoperative period. For example, patients with a history of conditions such as valvular disease, recent myocardial infarction (MI), arterial hypertension, diabetes, previous cardiac surgery, chronic peripheral vascular disease, involvement of three or more vessels, elevated serum creatinine, ejection fraction less than 40%, or COPD are more likely to require prolonged mechanical ventilation (Suematsu et al., 2000).

If the patient underwent CPB, the potential for a systemic inflammatory response with associated hemodynamic effects should be anticipated. As described in Chapter 13, the inflammatory response may be related to the surface of the CPB circuit being in contact with blood or reperfusion injury associated with aortic cross-clamping (Laffey, Boylan, & Cheng, 2002).

IMMEDIATE POSTOPERATIVE CARE

The foremost objectives when caring for a cardiac surgery patient in the immediate postoperative period are maintenance of cardiac perfusion and maximization of tissue perfusion (Baltimore, 2001; Smartt, 2004). Goals of the first hour of care include stabilization of hemodynamic, oxygenation, and thermoregulatory status. Postoperative care requires assessment of physiologic parameters and hemodynamic monitoring, as well as assessment, prompt recognition, and treatment of potential complications that are related to either patient comorbidities, effects of anesthesia, or the surgical procedure itself.

Control of the cardiac surgery patient's blood pressure in the immediate postoperative period is important. The ICU nurse should monitor for hypertension to avoid associated complications such as bleeding, myocardial ischemia, dysrhythmias, stroke, or graft dehiscence. Initial management of

hypertension may entail administration of opioids, sedatives, or both. However, infusion of a vasodilator may be required if initial therapies are not effective in controlling hypertension (Karski et al., 2001).

■ ASSESSMENT

The nurse performs a detailed physical assessment. ECG monitoring of heart rate and rhythm is performed. The patient's hemodynamic profile (e.g., blood pressure, pulmonary artery pressures, pulmonary artery occlusive pressure [PAOP], central venous pressure [CVP], cardiac output/index, and systemic vascular resistance [SVR]); temperature; and pulse oximetry are evaluated. Additional invasive monitoring (e.g., mixed venous saturation) may be monitored as well. The ICU nurse can then correlate these findings with an assessment of peripheral perfusion. If temporary pacing wires are present, they should be checked to ensure proper function for emergent temporary pacing. A baseline postoperative ECG should be attained to determine presence of ischemia, infarction, conduction abnormalities, or graft spasm (Khalpey et al., 2008).

Assessment of neurologic status typically includes level of consciousness, degree of orientation, pupil size and reaction, and ability to move extremities. A more in-depth neurologic assessment may follow later in the postoperative period. Inherent in a neurologic assessment is an initial and ongoing assessment of pain. If the patient is able to self-report the level of pain, that is the most reliable indicator. The ICU nurse should differentiate incisional pain from anginal pain. If the patient is cognitively impaired and cannot self-report, use of a valid and reliable behavioral pain rating scale should be used. Management of pain in the postoperative cardiac surgery patient is discussed in detail in Chapter 14.

An initial respiratory assessment typically includes auscultation of breath sounds, oxygen delivery mode, presence of symmetrical chest expansion, and respiratory rate, depth, effort, and rhythm. If the patient remains on mechanical ventilation, assessment of tube placement by the markings on the endotracheal tube should be noted, and ventilator settings (e.g., mode, fractional inspired oxygen [FiO_2], rate, tidal volume, positive end-expiratory pressure [PEEP], pressure support, alarm settings) should be verified as applicable. Typical ventilator settings in the immediate postoperative period following cardiac surgery are discussed in Chapter 11. Once these data are obtained, the patient's respiratory status can be correlated with pulse oximetry and ABG results. A baseline chest radiograph should be obtained to verify placement of the endotracheal tube (2 to 3 cm above the carina), catheters, wires, or any other devices that were inserted in the OR. The presence of any postoperative atelectasis, pneumothorax, or other common respiratory complication following cardiac surgery can also be determined (Khalpey et al., 2008).

Types and number of drainage catheters will vary based on the operative procedure and approach used. If a minimally invasive approach is used, a small-diameter catheter will be noted. If the patient had a sternotomy but the pleural space is not opened, the patient will have a mediastinal chest tube, or a chest tube in the mediastinal and pleural spaces will be present (Baltimore, 2001).

Tubes are connected to –20 cm of wall suction. The ICU nurse should assess the amount, color, and viscosity of initial operative and subsequent drainage. Patency of the catheters must be maintained at all times. If the patient is experiencing bleeding, then volume repletion, treatment of the underlying cause (if possible), and monitoring of the patient's coagulation profile are indicated (Baltimore, 2001). Surgical reexploration may be indicated if blood loss exceeds 200 mL/hr for 4 hours, 300 mL/hr for 3 hours, 400 mL/hr for 2 hours, or 500 mL/hr for 1 hour (Khalpey et al., 2008; St. Andre & DelRossi, 2005).

If a mediastinal chest tube becomes clotted, cardiac tamponade may ensue. Signs and symptoms may include sudden decrease or cessation of mediastinal bleeding, dyspnea, decreased cardiac output (CO) and hypotension, tachycardia, low-voltage QRS on ECG, increased CVP, altered mental status, cyanosis or pallor, anxiety, and restlessness (Talmor & Lisbon, 2005). Other signs and symptoms are described in Chapter 13.

Preventive measures include positioning the patient on the side with the head of the bed elevated 30 degrees, to facilitate drainage of the catheters. Until the condition is treated, the ICU nurse should administer volume to help counteract the decrease in preload from the associated decrease in diastolic filling pressures of the tamponade. Administration of afterload reducers (i.e., vasodilator) may help promote contractility (Baltimore, 2001). Cardiac tamponade management is discussed in detail in Chapter 13.

An initial assessment of the patient's fluid and electrolyte status should be performed upon admission to the ICU. In addition to the output from drains, a correlation between the patient's hemodynamic status and the intraoperative intake and output of fluids should be made. The ICU nurse should anticipate third spacing of fluid in the immediate postoperative period (Khalpey et al., 2008). Evaluation of serum electrolytes should be included in the initial assessment, as imbalances may be anticipated. Anticipated alterations and management of fluid and electrolytes in the postoperative cardiac surgery patient are discussed in detail in Chapter 17.

ANESTHETIC AGENTS

"Balanced" anesthesia or "fast-tracking" is generally employed to facilitate early extubation of the cardiac surgery patient while concomitantly decreasing anxiety, pain, length of ICU stay, and complications; minimizing mechanical ventilation time; and promoting a quicker, uneventful recovery from anesthesia. Typically, shorter-acting agents—although more costly—result in earlier extubation and reduced postoperative stays (Myles & McIlroy, 2005). It is important for the ICU nurse to recognize signs and symptoms, multisystem effects, and postoperative nursing implications of commonly used anesthetic agents administered during surgery.

Induction Agents

Combinations of intravenous agents are administered to augment the effects of inhalation agents. Classifications of these agents include barbiturates (e.g., thiopental sodium, methohexital), nonbarbiturates (e.g., etomidate, propofol), and tranquilizers (e.g., midazolam, lorazepam) (Dozier, 2007; Savino & Cheung, 2008).

Barbiturates depress the central nervous system (CNS). Thiopental sodium, for example, causes cardiovascular depression and negative inotropy, resulting in hypotension, decreased CO, and peripheral vascular resistance. Barbiturates also cause respiratory depression, which puts the patient at risk for apnea, airway obstruction, and, at higher doses, loss of laryngeal reflexes; the latter effect puts the patient at risk for aspiration (Dozier, 2007; Savino & Cheung, 2008). Other side effects may include headache, emergence delirium, prolonged somnolence, and nausea. Nursing considerations include monitoring for the prolonged effects of thiopental, which could persist for as long as 36 hours (Schick, 2004).

Etomidate is a hypnotic agent with no analgesic effects. It is considered the agent of choice in patients with cardiovascular instability. When this agent is used, it is less likely to cause hypotension. Heart rate, contractility, and CO remain stable, and negative inotropic effects are negligible with etomidate (Savino & Cheung, 2008; Schick, 2004). Some

patients may develop postoperative nausea and vomiting (PONV), hiccoughs, involuntary tremors, or suppressed adrenal function following administration of etomidate (Dozier, 2007).

Propofol (Diprivan) is a sedative that is primarily used as an induction agent. Compared with barbiturates, it causes less myocardial depression. Hypotension seen following propofol administration is felt to be related to arterial and venous dilation (Savino & Cheung, 2008). An infusion of propofol is generally initiated en route to the ICU and discontinued 10 to 15 minutes prior to ventilator weaning. The maintenance infusion rate is 50 to 150 mcg/kg/min. Propofol has a low incidence of postoperative side effects and is less likely to cause PONV than etomidate. It allows the patient to quickly regain consciousness with minimal residual CNS effects, allowing for early extubation. As propofol has no analgesic properties, postoperative analgesics will be required (Drain, 2003).

Benzodiazepines are used as adjuncts to induction agents prior to cardiac surgery. Midazolam (Versed®) may also be used postoperatively for sedation in the patient who remains intubated. This agent can cause respiratory depression and mild vasodilation, but it minimizes PONV (Couture, May, O'Brien, & Smith, 2006). Nursing considerations include monitoring of vital signs and oxygen saturation. If severe, respiratory depression may be reversed by administering flumazenil (Romazicon®) (Dozier, 2007).

Inhalation Agents

Inhalation agents cause circulatory depression and hypotension as a result of vasodilation and decreased contractility (Savino & Cheung, 2008). They may be administered either alone or in combination with intravenous anesthetics (Schick, 2004). Nursing considerations include monitoring for ventricular ectopy, fibrillation, and tachycardia, which generally manifest during the immediate postoperative period. Inhalation agents typically do not possess analgesic properties. They are eliminated through the lungs; the amount of time it takes depends on the patient's CO. Patients will require oxygen therapy and encouragement to cough and deep breathe (Dozier, 2007). Hemodynamic monitoring is essential given the sensitization to catecholamines associated with many of these inhalation agents.

Because some of the inhalation agents are fat soluble and are absorbed into adipose tissue, elimination and recovery times are longer when these agents are given. Further, patients with higher percentages of body fat will have a longer recovery time when administered fat soluble inhalation agents. Prompt management of pain and PONV are other vital ICU nursing responsibilities at this time (Dozier, 2007).

Sevoflurane and halothane have depressant effects on the respiratory system. Additionally, smooth bronchial muscles, laryngeal, and pharyngeal reflexes are blunted by these agents, placing the patient at risk for aspiration. Sevoflurane and halothane side effects may include decreased responsiveness to oxygenation and ventilation and elevated carbon dioxide levels. Halothane decreases mucociliary function for as long as 6 hours, which increases the patient's risk for atelectasis and pneumonia. Its cardiovascular effects include myocardial depression and peripheral vasodilation. Two benefits of halothane are the associated low incidence of PONV and its bronchodilator properties, making this agent useful in patients with pulmonary disease (Dozier, 2007). Sevoflurane does not appear to irritate the respiratory system or to sensitize the heart to catecholamines, although it may cause hypotension by decreasing afterload (Dozier, 2007).

Enflurane and isoflurane may cause laryngospasm, coughing, and breath holding. These side effects predispose the patient to non-cardiogenic pulmonary edema. Attributes of isoflurane include that it is not

associated with increased cardiac sensitization to catecholamines, stabilizes the cardiovascular system, and has the least related increase in cerebral blood flow (Dozier, 2007).

Enflurane has residual CNS depressant effects, which manifest during the postoperative period. Other effects include decreased blood pressure, stroke volume, and SVR, and increased heart rate; this medication also sensitizes the heart to catecholamines. Enflurane causes mild coronary vasodilation and puts the patient at increased risk for development of junctional rhythms (Savino & Cheung, 2008). A benefit of enflurane is the low associated incidence of PONV (Dozier, 2007). Nursing considerations include anticipation of delayed awakening and extubation.

Isoflurane augments the effects of nondepolarizing muscle relaxants. It is a coronary artery vasodilator that is associated with increased coronary perfusion (Savino & Cheung, 2008). Isoflurane and halothane can cause postoperative shivering, with an associated increase in myocardial oxygen demand (Weinbroum & Geller, 2001).

Neuromuscular Blocking Agents

Neuromuscular blocking agents (NMBAs) are used as adjuncts to inhalation agents to provide relaxation of skeletal muscles, facilitate intubation, and decrease shivering (Savino & Cheung, 2008). These agents are classified as either depolarizing or nondepolarizing agents. NMBAs that are commonly administered during cardiac surgery include rocuronium, vecuronium, and succinylcholine. Succinylcholine is an example of a depolarizing NMBA; rocuronium and vercuronium are examples of nondepolarizing agents and are short- to medium-acting agents, respectively. Rocuronium, *cis*-atracurium, doxacurium, and vecuronium have no cardiovascular side effects and, therefore, are useful in cardiac surgery. These agents are eliminated by the hepatic system (as opposed to the renal system) (Savino & Cheung, 2008); as a consequence, their effects will be prolonged in patients with severe liver disease.

Return paralysis can occur during the early postoperative period. The ICU nurse should observe for a descending trend in minute ventilation, which can be caused by inadequate reversal of the NMBA. Nondepolarizing agents are reversed with anticholinesterase drugs (e.g., neostigmine [Prostigmin®]). Depolarizing NMBAs cannot be pharmacologically reversed because they are metabolized by pseudocholinesterase, an endogenous enzyme.

It is essential that the ICU nurse realize that NMBAs have no amnestic or analgesic properties, nor do they cause a loss of consciousness. Analgesics must be administered to the postoperative cardiac surgery patient despite the patient's inability to quantify pain levels (Dozier, 2007). Medications to achieve decreased level of consciousness or amnesia must similarly be administered if those effects are desired in the postoperative cardiac surgery patient.

Opioids

Intravenous opioids are used as analgesics or as induction agents. When administered, these medications decrease the response and perception to pain. The most frequently used opioid in cardiac surgery is fentanyl. Nursing considerations include monitoring for bradycardia, which may be treated with atropine or glycopyrrolate (Robinul®). PONV is a common side effect of opioids and is of clinical concern.

POSTOPERATIVE CARE

Hemodynamic Management

The primary goal of care for the cardiac surgery patient in the immediate postoperative period is optimization of hemodynamic status to help achieve a balance between oxygen supply and demand. This goal can best be accomplished by maintaining an adequate CO.

As described in Chapter 9, CO is affected by a patient's preload, afterload, and contractility. Preload refers to the heart's filling pressures, reflected as the amount of volume returning to the right and left sides of the heart. It is evaluated by measuring central venous pressure (CVP) and PAOP, respectively. Afterload refers to the amount of work the heart must do to eject blood. Typically, left-sided afterload (SVR) is evaluated most often. These two parameters can be evaluated and manipulated by the ICU nurse to optimize a patient's hemodynamic profile.

Causes of alterations in preload in the postoperative cardiac surgery patient include vasodilation from a systemic inflammatory response associated with CPB procedures, medications, vasodilation from rewarming, bleeding, third spacing, and urinary output. Volume repletion is indicated for patients with decreased preload. The decision of whether to use crystalloids or colloids for fluid resuscitation remains unresolved given the pros and cons of each option. If volume resuscitation alone is inadequate to maintain filling pressures and CO in a patient who has adequate pump function and vasodilation, consideration should be given to adding an infusion of a vasopressor (e.g., neosynephrine, vasopressin, methylene blue) (Khalpey et al., 2008). Chapter 12 discusses vasopressor therapy in more detail.

An increase in afterload may be related to postoperative hypertension, use of medications that cause vasoconstriction, hypothermia, pain, anxiety, hypovolemia, or postoperative pump failure. Infusion of a vasodilator (e.g., nitroprusside, nitroglycerin, nicardipine) is indicated for patients who are hypertensive or who have inadequate pump function but with individual-specific normal blood pressure (Khalpey et al., 2008). Vasodilator therapy is discussed in more detail in Chapter 12.

A decrease in afterload may be caused by vasodilation from the CPB-associated systemic inflammatory response, administration of medications that cause vasodilation, or fever (Khalpey et al., 2008).

Once a patient's preload and afterload have been optimized, if CO is inadequate, administration of an inotropic agent to augment contractility may be considered. Agents such as milrinone or dobutamine increase CO by augmenting contractility and decrease afterload by causing vasodilation (Khalpey et al., 2008). Chapter 12 discusses inotropic agents in more detail.

As described in Chapter 13, although acceptable postoperative hemodynamic values will vary with the patient's cardiac history, optimal hemodynamic parameters in a postoperative cardiac surgery patient include a CI of more than 2 $L/min/m^2$, PAOP of approximately 15 mm Hg, CVP less than 15 mm Hg, mean arterial pressure (MAP) more than 65 mm Hg, systolic blood pressure (SBP) in the range of 90–140 mm Hg, and systemic vascular resistance index in the range of 1400–2800 $dyne/sec/cm^{-5}/m^2$ (Khalpey et al., 2008).

Alterations in Heart Rate and Rhythm

Postoperative dysrhythmias can be anticipated in the postoperative cardiac surgery patient. The most common dysrhythmias are atrial in origin; ventricular dysrhythmias and bradycardic rhythms are possible as well. Dysrhythmias may or may not manifest in the initial postoperative period. If present, however, dysrhythmias may cause hemodynamic instability. If the patient has a clinically significant dysrhythmia, then pharmacologic control of rate, rhythm, or both, may be indicated. Management of alterations in heart rate and rhythm is discussed in detail in Chapters 12 and 15.

Postoperative Nausea and Vomiting

Postoperative nausea and vomiting is a common occurrence in the immediate postoperative period, primarily due to the medications

Table 8–2 Multimodal Management of Postoperative Nausea and Vomiting

Dexamethasone
5-HT_3 receptor antagonists
H_1 blockers
Scopolamine patch
Droperidol
NK_1 antagonists
Hydration
Pain and comfort management

Sources: Ali, Taguchi, Holtmann, & Kurz, 2003; ASPAN, 2006.

administered intraoperatively. PONV increases the risk of pulmonary aspiration, disrupts surgical repairs secondary to retching, increases postoperative bleeding, and causes electrolyte disturbances (e.g., hypokalemia, hyponatremia, hypochloremia), dehydration, and esophageal rupture and tears (Couture et al., 2006). PONV can be minimized by assessing for risk factors (e.g., age, gender, history of PONV or motion sickness, and use of volatile anesthetics and opioids) in the preoperative phase and by implementing preventive strategies utilizing a multimodal approach (see Table 8–2).

Administering prophylactic antiemetics that affect different receptor sites in the brain has been shown to decrease the incidence of PONV. Medications that may be used to treat PONV include ondansetron (Zofran®), promethazine (Phenergan®), and prochlorperazine (Compazine®). If PONV is not relieved following two doses of antiemetics, it should be reported to the anesthesia provider (Dozier, 2007). If it is not contraindicated or if the cause of PONV is hypotension, hydration may also be effective in reducing the occurrence of PONV (Ali, Taguchi, Holtmann, & Kurz, 2003). Patients who are vomiting should be positioned to prevent aspiration (Dozier, 2007).

Thermoregulation (Hypothermia)

According to American Society of PeriAnesthesia Nursing (ASPAN, 2006) standards, postoperative nursing considerations include the identification of patients at risk for hypothermia and application of passive and active warming devices (e.g., bonnet, cotton blankets, socks, forced air warming device). Patients are considered hypothermic if they have a temperature of less than 96.8 °F (36 °C) (ASPAN, 2006). Others define postoperative hypothermia as a temperature less than 95 °F (35 °C) (Khalpey et al., 2008). Factors affecting the development of hypothermia include patient age, health status, surgical procedure, exposed body areas, duration of anesthesia or surgery, ambient room temperature, prepping and irrigation solutions, administration of cool IV fluids, and peripheral vascular disease (Dozier, 2007).

The postoperative cardiac surgery patient should be monitored every 30 minutes until normothermic. Adjusting ambient room temperature or warming oxygen may also be beneficial (Bräuer et al., 2004; Frank, 2000).

Attaining and maintaining postoperative normothermia is vital, as inadvertent postoperative hypothermia has been linked to adverse effects. Overall, postoperative patients admitted from the OR with a core temperature less than 36 °C have prolonged mechanical ventilation, shivering, and increasing oxygen consumption. Hemodynamic effects of hypothermia include increased SVR and greater likelihood of developing dysrhythmias, hypertension, tachycardia, decreased preload, impaired contractility, or coronary graft spasm (Khalpey et al., 2008; Lemmer, Richenbacher, & Vlahakes, 2003).

Hypothermia alters drug metabolism, causing delays in patients' emergence from anesthesia. It also causes a disruption of the coagulation pathway, increasing the need for blood transfusions. Hypothermia leads to delays in wound healing, which increases sus-

ceptibility to surgical site infections, and shivering, which increases myocardial oxygen demand and consumption (Silvestry, 2008; St. Andre & DelRossi, 2005; Talmor & Lisbon, 2005).

Postoperative Respiratory Management

In addition to managing a patient's hemodynamic status, respiratory management is another pivotal role of the ICU nurse in the immediate postoperative cardiac surgery period. Unless the patient was "fast-tracked" and extubated in the OR, short-term mechanical ventilation is employed until anesthetic agents have been eliminated. Early extubation should be a goal for all patients.

Weaning and extubation protocols vary among facilities. Nevertheless, these processes are generally based on adequate muscle strength, pulmonary function, and hemodynamic stability (Baltimore, 2001; Smartt, 2004). As discussed in Chapter 11, extubation criteria typically include presence of a heart rate less than 140, respiratory rate less than 25, normothermia, and absence of ischemia and infusion of vasoactive agents. The patient should be alert and cooperative (i.e., able to respond to commands). Presence of a cough and gag reflex are important, as the patient must be able to maintain a patent airway following extubation. The patient must also demonstrate adequate muscle strength by sustaining a head lift for at least 5 seconds. Other weaning criteria include ability to breathe spontaneously and adequately while maintaining adequate oxygen saturation and arterial blood gas values. Physiologic parameters that may be measured to assess potential readiness for extubation include a negative inspiratory force (NIF) of at least 20–25 cm H_2O, minute volume no greater than 10 L/min, and vital capacity 10–15 mL/kg (Hemant, Chacko, & Singh, 2006; Khalpey et al., 2008).

Typically, cardiac surgery patients are extubated within 4 to 12 hours after their arrival in the ICU from the OR (Baltimore, 2001). Upon determination that the patient is ready for extubation, the patient's mouth should be suctioned and the tube-securing device is removed. The cuff on the endotracheal tube is deflated with a syringe. The presence of an air leak must then be ascertained; such a leak may be either heard or felt. The patient is instructed to take a deep breath and cough, with the tube being removed toward the end of the cough. Supplemental humidified oxygen is applied (Dozier, 2007). Placement on low-flow oxygen such as nasal cannula is common practice.

Stir-up Regime

Cardiac surgery patients require the "stir-up regime" in the immediate postoperative period if they received an inhalation agent as part of their anesthesia, as these agents cause respiratory depression and are eliminated with ventilation. The stir-up regime is accomplished by elevating the head of the bed and encouraging deep breathing and coughing at regular intervals. This practice facilitates movement of the inhalation agent from an area of higher concentration (the patient's lungs) to an area of lower concentration (room air), which is how the agent will be eliminated (Dozier, 2007).

Complications Related to Extubation

Complications following extubation are fairly uncommon but may include laryngospasm, noncardiogenic pulmonary edema, bronchospasm, hypoventilation, and hypoxia.

Laryngospasm and Noncardiogenic Pulmonary Edema

Laryngospasm is a partial or complete blockage of air flow into and out of the lungs owing to spasms of the vocal cord (Fodale et al., 2004). Causes include aspiration, suctioning, and histamine release associated with

some medications. Signs of laryngospasm include "rocking" respirations, wheezing, stridor, dyspnea, use of accessory muscles, and tachypnea. The patient should be encouraged to cough, as this action may be effective in eradicating a partial obstruction (Dozier, 2007).

Patients can have laryngospasm during extubation, which can trigger noncardiogenic pulmonary edema. Noncardiogenic pulmonary edema occurs following an acute airway obstruction, such as when the patient forcefully inspires against a closed glottis, thereby creating an increase in intrathoracic pressure and resulting in pulmonary edema (Van Kooy & Gargiulo, 2000). Protein and fluid accumulate and extravasate into the alveoli without an associated increase in PAOP (Colucci, 2008). Symptoms of this condition, which typically have a rapid onset, include agitation, tachypnea, tachycardia, decreased oxygen saturation, and pink, frothy sputum. Crackles will be audible.

Prompt recognition and treatment of both laryngospasm and noncardiogenic pulmonary edema are crucial; indeed, the patient may require reintubation until these problems resolve. Treatment of laryngospasm generally involves positive-pressure breathing with a bag-valve-mask device with 100% oxygen and mandibular support. If these measures prove ineffective, succinylcholine can be administered intravenously. Lidocaine may be effective in preventing a laryngospasm.

Noncardiogenic pulmonary edema management involves maintenance of a patent airway, supplemental oxygen, and administration of a diuretic. Mechanical ventilation with PEEP may be required in severe cases (Marley & Riess, 2004). Chest radiograph may reveal findings consistent with pulmonary edema. Treatment of noncardiogenic pulmonary edema includes supplemental oxygen, respiratory support, and diuretics (Dozier, 2007).

Bronchospasm

Bronchospasm can occur as a result of constriction of bronchial smooth muscles after extubation. It resolves quickly after airway irritants are eliminated. Symptoms include wheezing, dyspnea, and tachypnea. Treatment involves administration of a bronchodilator and humidified oxygen. In severe cases, muscle relaxants, lidocaine, epinephrine, or hydrocortisone may be administered to relax the airway (Carlson, 2004).

Hypoventilation and Hypoxia

Hypoventilation is common in the immediate postoperative period. It may result from the anesthetic agents administered or the surgical procedure itself. Treatment entails eradicating the underlying cause. If the underlying cause is related to opioid administration, then treatment may include administration of naloxone (Narcan®) for patients with shallow or slow respirations. Institutional policy varies regarding use of opioid antagonists (Dozier, 2007).

Hypoxemia is defined as oxygen saturation less than 90%. Hypoxemia can have numerous undesired sequelae, including cardiac dysrhythmias and myocardial ischemia. Signs and symptoms may include cyanosis, agitation, somnolence, tachycardia, bradycardia, hypertension, and hypotension. Depending on the severity of the symptoms or hypoxemia, reintubation and mechanical ventilation may be required (Dozier, 2007).

Inadequate reversal of NMBAs' effects can cause hypoventilation and hypoxia after extubation. Extubation of a patient who is partially paralyzed increases the individual's risk of developing postoperative complications. Residual respiratory muscle weakness can cause airway obstruction, hypoventilation, and an impaired response to hypoxia. Cardiac surgery patients are at increased risk if they receive a long-acting NMBA whose action is

inadequately reversed with anticholinesterase agents. Re-paralysis can occur when an NMBA has a longer half-life than the reversal agents. If this problem occurs, the patient will demonstrate weak, shallow respirations and poor chest rise; anxiety and restless may become apparent as well. Treatment involves administration of additional doses of a reversal agent, respiratory support, and temporary reintubation until muscle strength is regained.

During weaning and extubation, opioids should be used judiciously. Opioids decrease respiratory effort, oxygen saturation, and respiratory rate and depth. Pain management is of concern; however, small doses of short-acting analgesics (e.g., dexmedetomidine [Precedex™]) may be recommended (Khalpey et al., 2008). Complications that arise after extubation can be minimized by recognizing and treating respiratory emergencies and by adhering to weaning and extubation criteria (Haghenbeck & Keeler, 2003).

POTENTIAL POSTOPERATIVE COMPLICATIONS

The ICU nurse plays a pivotal role in preventing or promptly identifying and treating postoperative complications. Among the more common complications seen in the immediate postoperative period are hemodynamic compromise, respiratory insufficiency, neurologic issues, and hematological problems. Some complications are related to patient comorbidities; others are related to the surgical procedure itself. These complications and the associated ICU nursing responsibilities are discussed in detail in Chapters 13 and 16. Potential complications related to effects of anesthesia are addressed in this section. One unique complication related to the surgical procedure is covered here as well.

Malignant Hyperthermia

Malignant hyperthermia (MH) is a genetic, life-threatening disorder that is triggered by certain anesthetic agents, depolarizing skeletal muscle relaxants, and stress. With this condition, a defect in the sarcoplasmic reticulum leads to a buildup of excess calcium in the mycoplasm. This results in sustained skeletal muscle contraction that is intense and prolonged, leading to a hypermetabolic state of heat production.

The onset of MH usually occurs during induction of anesthetic agents. Halothane, enflurane, isoflurane, desflurane, and succinylcholine are the most common triggering agents. The triggering of events is characterized by muscle rigidity of the jaw (masseter rigidity), tachypnea, tachycardia, elevated CO_2 level, cyanosis, respiratory and metabolic acidosis, elevated serum creatine phosphokinase (CPK), and hyperkalemia. Late signs include temperature elevation, bleeding from venipuncture sites, and rhabdomyolysis. MH typically manifests in the OR but it can develop within 24 hours postoperatively (Litman & Rosenberg, 2005).

Treatment of MH includes discontinuance of triggering agents and immediate intravenous administration of dantrolene sodium (Dantrium®) 2.5 mg/kg (up to a maximum dose of 10 mg/kg). Dantrolene inhibits the release of calcium. Once the loading dose is administered, dantrolene is infused at a dose of 1 mg/kg every 4 hours for at least 48 hours (Dozier, 2007).

Hyperventilation, administration of 100% oxygen, body surface area cooling, administration of sodium bicarbonate, maintenance of fluid and electrolyte balance, and treatment of associated conditions (e.g., hypertension, dysrhythmias) are also essential interventions in the setting of MH. Lab data that may be obtained include arterial glood gas, serum electrolytes, liver enzymes, renal function studies, blood counts, and coagulation profile

(Dozier, 2007). Effective management involves prompt recognition, guidance of the multidisciplinary team, and expert direction from the Malignant Hyperthermia Association of the United States (MHAUS).

Pseudocholinesterase Deficiency

Prolonged mechanical ventilation after cardiac surgery may be caused by a deficiency in pseudocholinesterase. A small percentage of patients lack this enzyme, which is responsible for metabolizing medications such as succinylcholine. Patients with pseudocholinesterase deficiency who receive these medications exhibit prolonged responses to these medications, can have sustained skeletal muscle paralysis, and remain apneic for as long as 48 hours after administration. Management involves emotional support and mechanical ventilation until the effects of the medication are completely eliminated (Dozier, 2007).

Protamine Sulfate Allergic Reactions

Protamine sulfate is administered as a reversal agent for heparin. If it is given too rapidly, severe hypotension and anaphylactic reactions may result. Consequently, caution should be used when administering protamine sulfate to patients who may be at increased risk of allergic reaction—specifically, individuals who have previously undergone procedures such as coronary angioplasty or CPB, diabetics who have been treated with protamine insulin, patients who are allergic to fish, and men who have had a vasectomy or are infertile and may have antibodies to protamine. Patients undergoing prolonged procedures involving repeated doses of protamine should be subject to careful monitoring of clotting parameters. A rebound bleeding effect may occur as long as 18 hours postoperatively (Hepner & Castells, 2003).

SUMMARY

Although much progress has been made with respect to the postoperative care of the cardiac surgery patient, critical thinking and caring practices of the ICU nurse are primary determinants of positive outcomes. The initial hours following cardiac surgery are tenuous. The patient's preoperative status, the intraoperative course, and the effects of anesthesia all contribute to the complexity of the patient's profile.

CASE STUDY

A 52-year-old patient with a history of coronary artery disease, hypertension, hyperlipidemia, and mitral regurgitation underwent coronary artery bypass grafting with a left internal mammary artery graft and mitral valve repair. Her intraoperative course was unremarkable. Postoperatively, the patient was admitted to the ICU extubated with a mediastinal tube and chest tube that were draining bloody fluid, and on a nitroglycerin infusion. Her admission vital signs were BP 132/74; HR 92; RR 24; temperature 97.5 °F; CVP 11; PAP 32/16; PAOP 10 mm Hg; cardiac output 4.1 L/min; and cardiac index 2.7 L/min/m^2.

The patient's initial postoperative course was uneventful. Two hours after admission, the ICU nurse recorded the following vital signs and hemodynamic data: BP 86/48; HR 120; RR 28; CVP 22; PAP 36/22; PAOP 23; cardiac output 3.2 L/min; and cardiac index 2.5 L/min/m^2. The ICU nurse also noted a decrease in the drainage from the mediastinal and

chest tubes. The patient reported feeling short of breath and that "something was just not right." She appeared anxious and pale, as compared to her baseline postoperative status.

The ICU nurse initiated a fluid bolus and alerted the cardiac surgeon. A bedside echocardiogram was performed, and a large pericardial effusion and a clot were noted. The patient returned to the OR for clot evacuation and relief of the cardiac tamponade. Subsequently, the patient returned to the ICU in stable condition. The remainder of her initial postoperative course was uneventful.

Critical Thinking Questions

1. Why was this patient at risk for the development of cardiac tamponade?
2. Why is diagnosis of cardiac tamponade challenging in the postoperative cardiac surgery patient?
3. Why was a fluid bolus indicated with the patient's hemodynamic profile?

Answers to Critical Thinking Questions

1. During cardiac surgery, the pericardial sac is entered and is usually not sutured back together before chest closure. This leaves a communication between the heart and the mediastinum, which can lead to the potential accumulation of blood or fluid (Lemmer et al., 2003; St. Andre & DelRossi, 2005). The accumulation compresses the atria, restricts venous return to the heart and ventricular filling, and results in a decrease or cessation of preload, causing a potential precipitous fall in CO (Massé & Antonacci, 2005). Early tamponade is usually a result of persistent mediastinal bleeding not being evacuated by chest tubes, as is what occurred in this case.
2. Diagnosis may be difficult because hypotension, tachycardia, and elevated filling pressures are common in cardiac surgery patients in the most immediate postoperative period. In addition, some of the other characteristic symptoms of cardiac tamponade—for example, muffled heart sounds, pulsus paradoxus, and neck vein distention—are not helpful in making the diagnosis in the cardiac surgery patient.
3. The pathophysiology of cardiac tamponade entails fluid accumulation around the cardiac chambers, which causes a restriction of diastolic filling, with an impact on cardiac output. This patient manifested clinically significant hemodynamic instability as a result of the cardiac compression caused by pericardial fluid. While preparation is being made to correct the underlying cause, supportive management includes augmenting preload and minimizing the hypotensive episode.

SELF-ASSESSMENT QUESTIONS

1. Which of the following patients is *most* likely to require prolonged ventilatory support? A patient with:
   a. an elevated BUN
   b. two-vessel disease
   c. an ejection fraction of 45%
   d. arterial hypertension
2. Your postoperative cardiac surgery patient has the following vital signs: BP 152/96; HR 104; RR 18; temperature 97.2 °F. Which of the following medications is *initially* indicated?
   a. Morphine
   b. Nitroglycerin
   c. Labetalol
   d. Nicardipine

3. Your postoperative cardiac surgery patient has decreased drainage from the mediastinal chest tube. Vital signs are as follows: BP 88/50; HR 106; RR 24, CVP 12; cardiac output 3.1 L/min. Which of the following actions is indicated *initially*?
   a. Milk the chest tube
   b. Raise the head of bed to 45 degrees
   c. Administer a fluid bolus
   d. Prepare for echocardiogram
4. Your postoperative cardiac surgery patient received an inhalation agent as part of general anesthesia. Which of the following developments should the ICU nurse anticipate?
   a. Hyperventilation
   b. An initial reduced need for analgesics
   c. Peripheral vasoconstriction
   d. Ventricular ectopy
5. You are caring for a postoperative cardiac surgery patient who has been in the ICU for the past hour. The patient was not extubated in the OR. You note a decrease in the patient's minute ventilation since admission. You suspect
   a. development of postoperative atelectasis.
   b. presence of pseudocholinesterase deficiency.
   c. inadequate reversal of the neuromuscular blocker.
   d. hepatic dysfunction.
6. Which of the following patients would benefit from an infusion of a vasodilator in the immediate postoperative period following cardiac surgery? A patient with:
   a. normotension and inadequate pump function
   b. inadequate volume repletion and adequate pump function
   c. cardiopulmonary bypass-related systemic inflammatory response
   d. inadequate cardiac output and optimized preload and afterload
7. Which of the following is an expected hemodynamic effect of hypothermia?
   a. SVR 1700 dyne/sec/cm^{-5}
   b. Heart rate 58
   c. SVO_2 76%
   d. CVP 12 mm Hg
8. Which of the following postoperative cardiac surgery patients has criteria suggesting readiness to wean from mechanical ventilation? A patient with:
   a. BP 104/60; HR 144; temperature 97.5 °F
   b. a positive cough and gag reflex, NIF 25 cm H_2O, receiving low-dose norepinephrine
   c. new ST-segment elevation but no Q waves, sustained head lift for 10 seconds, vital capacity 10 mL/kg
   d. minute ventilation 8 L/min, RR 24, HR 136
9. Your postoperative cardiac surgery patient was recently extubated and develops "rocking respirations," wheezing, tachypnea, and dyspnea. Which of the following is *initially* indicated?
   a. Bag-valve-mask ventilation
   b. Administration of succinylcholine
   c. Administration of a nebulized bronchodilator
   d. Immediate reintubation
10. Which of the following ABG results should the ICU nurse anticipate in a patient with malignant hyperthermia?
    a. pH 7.30; pCO_2 50; pO_2 60; SaO_2 90; HCO_3 25
    b. pH 7.50; pCO_2 30; pO_2 50; SaO_2 85; HCO_3 18
    c. pH 7.55; pCO_2 45; pO_2 71; SaO_2 93; HCO_3 34
    d. pH 7.29; pCO_2 38; pO_2 68; SaO_2 92; HCO_3 19

Answers to Self-Assessment Questions

1. d
2. a
3. c
4. d
5. c
6. a
7. a
8. d
9. a
10. a

Clinical Inquiry Box

Question: Can postoperative cardiac surgery patients be admitted to the ICU and have acceptable outcomes?

Reference: Sino, C. A., & Martich, G. D. (1999). Who goes to the ICU postoperatively? *Chest, 115*(5 suppl), 125S–129S.

Objective: To evaluate the outcomes of postoperative admission to the ICU following cardiac surgery.

Method: This study included an evidence-based review of the available clinical literature, direct observation of facilities utilizing a rapid recovery program following cardiac surgery, and informal collaboration with colleagues who use the ICU for immediate postoperative management of cardiac surgery patients.

Results: Most of the evaluations that were performed involved care of patients following coronary artery revascularization procedures. Efficacy of ICU admission was evaluated based on time to extubation, length of stay in the ICU and hospital, incidence of postoperative complications including reintubation and ICU readmission, patient satisfaction, and resource savings.

Conclusion: Only limited data are available from controlled clinical trials. Those data suggest that postoperative admission of cardiac surgery patients can result in rapid recovery and decreased resource utilization and costs, while maintaining high levels of quality of care and patient satisfaction. These outcomes are not apparently related to the patient's preoperative risk. A multidisciplinary approach can result in shortened postoperative stays in the ICU following cardiac surgery.

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WEB RESOURCES

Dr. Bernadine Healy takes a tour of the hospital. The operating room is a virtual beehive during heart surgery and afterward in the intensive care unit. URL: http://video.google.com/videosearch?hl=en&q=postoperative%20cardiac%20surgery&um=1&ie=UTF-8&sa=N&tab=wv#q=Navigating%20the%20hospital%20part%203&hl=en&emb=0

Cardiac tamponade: While not taken on a postoperative cardiac surgery patient, this video demonstrates a swinging heart due to fluid in high tension within the pericardial space and subsequent echocardiogram following treatment. URL: http://video.google.com/videosearch?hl=en&q=postoperative%20cardiac%20surgery&um=1&ie=UTF-8&sa=N&tab=wv#q=cardiac%20tamponade%20&hl=en&emb=0&start=0

Chapter 9

Hemodynamic Monitoring

Mary Zellinger

INTRODUCTION

Hemodynamic monitoring of the patient after cardiac surgery is a routine part of the immediate postoperative care. Data obtained during this period guide the clinician in initiating the optimal intervention to ensure a smooth recovery. Hemodynamics, or the study of the dynamics of blood circulation, can be assessed through both invasive and noninvasive mechanisms; the ultimate goal is to determine the adequacy of cardiac output (the amount of blood ejected by the heart each minute). This chapter reviews the essentials of hemodynamic monitoring in the patient who has undergone cardiac surgery. Both basic and newer technologies are discussed.

ESSENTIALS OF HEMODYNAMIC MONITORING

Monitoring assists in determining changes in fluid status and cardiac performance at the earliest possible time so that treatment fluctuations in three factors that affect cardiac output—preload, afterload, and contractility (see Box 9–1)—can be quickly addressed.

New monitoring devices and techniques are introduced annually to the critical care arena, each of which has the goal of increasing accuracy and decreasing invasiveness of monitoring. It is imperative for the clinician to incorporate data from a variety of sources when assessing the hemodynamic picture so as not to rely on a single—and potentially misleading—parameter.

Box 9–1 Hemodynamic Monitoring Terms and Definitions

Preload: the volume of blood either in the right atrium or in the left ventricle at the end of diastole or the beginning of systole. Preload is quantified with central venous pressure (CVP) and pulmonary artery occlusive pressure (PAOP), respectively; these parameters reflect a patient's volume status. The end-diastolic volume (EDV) is related to the amount of stretch of the sarcomeres. Preload is a reflection of all of the elements that affect tension of the chamber wall at the end of filling (diastole).

Afterload: the amount of work the heart must do to eject blood; the impedance or resistance to ventricular contraction. Afterload reflects all of the elements that affect tension of the myocardial wall during systole.

Contractility: the ability of the myocardial muscle to shorten itself or the amount of strength produced by the myocardium when it ejects blood. It is influenced by neural factors and certain metabolic states (e.g., hypoxia, hypercarbia, or decrease in pH).

Cardiac output: the amount of blood ejected by the heart each minute.

Sources: Norton, 2001; Rothe, 2003.

INITIAL POSTOPERATIVE ASSESSMENT

Following cardiac surgery, the ICU nurse will connect the patient to the bedside monitor upon receipt from the operating room. The ECG leads are connected to the bedside monitor from the transport monitor, and heart rate and rhythm are assessed. The pulse oximetry probe is connected to the finger, earlobe, or forehead. Pulse oximetry is a simple, noninvasive method of monitoring the percentage of hemoglobin that is saturated with oxygen. The target oxygen saturation (SpO_2) is 95% or greater in a patient without a history of COPD.

Preparing Hemodynamic Equipment

After elevating the head of the bed, the transducers are leveled at the phlebostatic axis, which is located at the fourth intercostal space, midpoint of the anterior–posterior diameter (see Figure 6–3 in Chapter 6). The transducers are then zero-balanced, establishing atmospheric pressure as zero. Leveling at the phlebostatic axis is performed to eradicate the effects of hydrostatic forces on the hemodynamic pressures (AACN, 2004). A square wave test is performed to ensure responsiveness (see Box 9–2 and Figure 9–1a). Proper setup and functioning of the monitoring system itself are essential to obtain accurate values, regardless of the specific parameter being measured. A number of variables—such as the number of stopcocks, the length of the tubing, the responsiveness of the tubing, and the presence of air bubbles—can influence the accuracy of the readings.

Vital Signs and Hemodynamic Assessment

An initial assessment of vital signs and hemodynamic parameters (see Box 9–3) is obtained, ensuring that the latter are assessed at end-

Box 9–2 Square Wave Test

A square wave test (also referred to as a fast flush or dynamic response test) is performed to assure that the waveforms that appear on the monitoring screen accurately reflect pulmonary artery pressures (AACN, 2004). It is accomplished by pulling and releasing the pigtail or squeezing the button of the flush device so that the flow through the tubing is increased (from 3 mL/hr obtained with a pressure bag inflated to 300 mm Hg). This causes a sudden rise in pressure in the system, such that a square wave is generated on the monitor oscilloscope. An acceptable response is the pressure waveform reverting to baseline within one to two oscillations. If the response is lacking in shape, amplitude, or time to return to baseline, the ICU nurse should troubleshoot the system until an acceptable response is achieved (McGhee & Bridges, 2002). If an underdamped or overdamped waveform is present, hemodynamic measurements will not be accurate. It is recommended that a square waveform test be performed when the system is being initially set up, at least once a shift, after opening the catheter system (e.g., for rezeroing, blood sampling, or changing tubing), and whenever the pressure waveform appears to be damped or distorted (AACN, 2004).

An overdamped waveform is sluggish and has an exaggerated or falsely widened and blunt tracing. It will cause the patient's systolic blood pressure (SBP) to be recorded as falsely low and the diastolic blood pressure (DBP) to be recorded as falsely high. Causes of an overdamped waveform include the presence of large bubbles in the system, loose connections, no or low fluid in the flush bag, low pressure of the flush solution pressure bag, or a kink in the catheter (AACN, 2004) (see Figure 9–1b).

An underdamped waveform consists of an over-response, which is seen as an exaggerated, narrow, artificially peaked tracing. In this case, the waveform overestimates the patient's SBP and underestimates the DBP. Causes of an underdamped waveform include the presence of small bubbles in the system, the pressure tubing being too long, or a defective transducer (AACN, 2004) (see Figure 9–1c).

expiration. Readings are obtained at this point in the respiratory cycle to eliminate the effects of changes in intrathoracic pressure that occur with breathing (McGhee & Bridges, 2002). The frequency of obtaining subsequent sets of vital signs and hemodynamic parameters varies by facility and according to the patient's clinical status.

Depending on unit-specific protocols and, perhaps, physician order, in addition to baseline hemodynamic values, cardiac output/index may be measured. From cardiac output/index and invasive pressure data, several hemodynamic calculations can be performed, yielding valuable information about cardiac performance.

The ICU nurse will check the vasoactive drips and other fluids infusing to verify their type, infusion status, and dosages. The relationships among the amount of volume infused and lost in the operating room, baseline postoperative vital signs, and hemodynamic status are assessed. Setting monitor alarm limits specific to the patient's baseline profile, and ensuring these alarms are activated, are crucial at this stage.

Patient Assessment

A complete baseline physical assessment is then completed. While the primary nurse is performing the baseline assessment, a number of concomitant essential activities related to the patient's hemodynamic status are performed. These activities are listed in Box 9–4.

A comprehensive head-to-toe assessment will enable the nurse to evaluate several indices to determine the overall adequacy of perfusion. A complete neurological assessment may

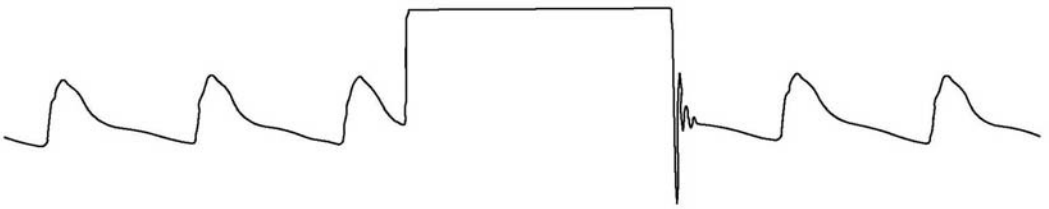

Figure 9–1a Square wave test.

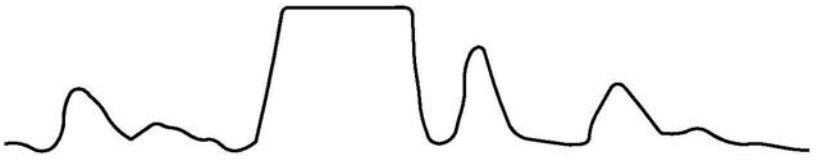

Figure 9–1b Overdamped waveform.

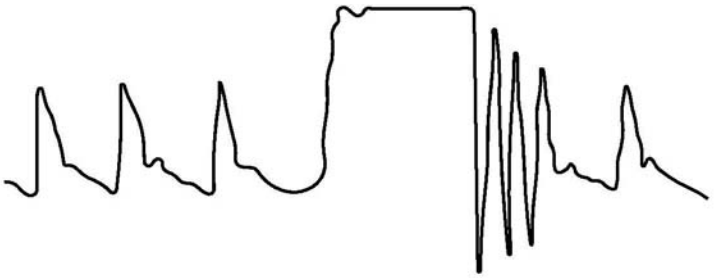

Figure 9–1c Underdamped waveform.

Source: Illustrations by James R. Perron

Box 9–3 Hemodynamic Parameters and Normal Values

Parameters	Normal Values
Systolic and diastolic blood pressure	100–130/60–90 mm Hg
Mean arterial pressure	70–105 mm Hg
Right atrial pressure (central venous pressure)	0–8 mm Hg
Right ventricular pressure	25–30/0–8 mm Hg
Pulmonary artery pressure	15–30/6–12 mm Hg
Pulmonary artery occlusive pressure	4–12 mm Hg
Derived Hemodynamic Parameters	
Cardiac output/cardiac index	4–8 L/min / 2.5–4.2 L/min/m^2
Systemic vascular resistance	770–1500 dyne/sec/cm^{-5}
Pulmonary vascular resistance	20–120 dyne/sec/cm^{-5}
Systemic vascular resistance index	1680–2580 dyne/sec/cm^{-5}
Pulmonary vascular resistance index	69–177 dyne/sec/cm^{-5}
Stroke volume/index	60–130 mL /beat/ 30–65 mL/beat/m^2
Right ventricular stroke work	8–16 g-m/beat
Right ventricular stroke work index	5–10 g-m-m^2/beat
Left ventricular stroke work	58–104 g-m/beat
Left ventricular stroke work index	50–62 g-m-m^2/beat
Oxygenation Parameters	
Arterial oxygen saturation	95–100%
Mixed venous oxygen saturation	60–80%
Arterial oxygen content	17–20 mL/dL
Venous oxygen content	12–15 mL/dL
Oxygen delivery	900–1150 mL/min
Oxygen consumption	200–290 mL/min
Oxygen extraction ratio	22–30%

Sources: Blount, 2007; Khalpey, Ganim, & Rawn, 2008; LiDCO, 2008.

Box 9–4 Postoperative Cardiac Surgery Initial Admission Responsibilities

Assessment of Chest Drainage System

- Connect chest drainage system to suction.
- Note and record the amount of drainage from the OR. Correlate these findings with the patient's baseline hemodynamic profile.

Assessment of Fluid Status

- Compare intraoperative intake and output with baseline hemodynamic profile and vital signs to help determine fluid volume status.

Diagnostics

- Obtain lab samples per protocol (e.g., electrolytes, ABG, CBC, coagulation profile) and other diagnostic procedures (e.g., 12-lead ECG to check for potential intraoperative or postoperative ischemia, chest radiograph to verify endotracheal tube placement and assess for presence/degree of pneumothorax).

prove challenging if the patient has not been reversed from general anesthesia or is receiving a continuous infusion of an anesthetic agent or sedation. Some hospital protocols require the anesthetic agent or sedation infusion be weaned and temporarily discontinued in the immediate postoperative period so that appropriate neurological function can be confirmed. The infusion can then be restarted until the ventilator weaning process begins. An awake and alert patient is one indicator of adequacy of cardiac output.

Extremity movement, warm skin, and palpable pulses indicate acceptable perfusion, unless obstructive peripheral vascular disease is present and limits perfusion to the distal extremities. Assessment of heart sounds will provide additional information about cardiac function and any valve dysfunction. The presence of extra heart sounds, although normal in certain situations, warrants further investigation. An S_3 or S_4 heart sound may be a sign of decreased ventricular compliance. The presence or sudden absence of murmurs may indicate changes in native or prosthetic valve function. Placing the head of the bed between 30 and 45 degrees and observing for jugular vein distention will reinforce other findings of right-sided heart failure or fluid overload.

Breath sounds should be auscultated in all fields, noting any areas that are diminished or abnormal. Pulmonary congestion may be indicative of pulmonary dysfunction from the surgical process, be the effect of complications from mechanical ventilation, or occur as a result of cardiac dysfunction.

Urinary output is another indication of adequacy of cardiac output, although it may sometimes misrepresent the adequacy of perfusion to the kidneys. Postoperatively, cardiac surgery patients should be evaluated for renal insufficiency if urinary output is less than 0.5 mL/kg/hr for 2 to 3 consecutive hours and serum creatinine levels are increasing (Lemmer, Rickenbacker, & Vlahakes, 2003). Given that cardiac surgery patients may exhibit a relative diuresis of 200–400 mL/hr owing to the effects of hemodilution and osmotic agents sometimes administered during cardiopulmonary bypass (Lee & Sladen, 2002; Stafford-Smith & Newman, 2006), urinary output may not be indicative of perfusion for the first several hours after the surgery. Following the initial few hours postoperatively, urinary output should be at least 0.5 mL/kg/hr.

BLOOD PRESSURE MONITORING

In the immediate postoperative period, maintaining hemodynamic stability is the priority. Intra-arterial pressure monitoring provides for the direct measurement of arterial blood pressure, and in many clinical situations is more accurate than the auscultatory measurement. Variables such as cuff size can influence indirect (noninvasive) pressure readings. Indirect pressure readings can underestimate actual systolic pressures by several mm Hg in hypotensive patients (Borrow & Newburger, 1982; Cloud, Rajkumar, Kooner, Cooke, & Bulpitt, 2003). This difference occurs because of the Korotkoff sounds produced by blood flow. As blood flow diminishes, the sound becomes less audible, to the point that the faint early sounds may be missed. Indirect measurement of blood pressure, whether obtained manually or with a noninvasive automated pump, provides the best estimate of SBP but underestimates DBP when the patient is at rest (Griffin, Robergs, & Heyward, 1997).

Intra-arterial monitoring is indicated in situations when the patient's condition necessitates close hemodynamic observation. Patients who undergo mechanical manipulation of the heart as in cardiac surgery, those who receive drug therapy, and those in whom an intra-aortic balloon pump (IABP; discussed in Chapter 10) is used will all require frequent assessment of arterial pressure postoperatively. An intra-arterial line will also assist in assessing perfusion associated with dysrhythmias. When an intra-arterial catheter

Box 9–5 Mean Arterial Pressure Calculation

$$\text{MAP} = \frac{\text{Systolic blood pressure} + (\text{Diastolic blood pressure} \times 2)}{3}$$

For example, if the patient's blood pressure is 120/80, the MAP can be calculated as follows:

$$\frac{120 + (80 \times 2)}{3} =$$

$$\frac{120 + 160}{3} \text{ or } \frac{280}{3} = 93 \text{ mm Hg}$$

is in place in a peripheral artery, the SBP readings may be falsely elevated because of the amplitude of the waveform. However, mean arterial pressure (MAP) and DBP data are accurate (Griffin et al., 1997).

MAP is the driving force for peripheral blood flow and the preferred pressure to be evaluated in unstable patients. On the monitor screen, it appears as a digital readout adjacent to the displayed blood pressure, usually in parentheses. MAP can also be calculated by the nurse using the formula given in Box 9–5. MAP readings do not change as the pressure waveform moves distally along the arterial tree. This pressure is measured electronically by first integrating the area under the arterial pressure waveform and then dividing by the duration of the cardiac cycle. Many clinical conditions may be reflected by changes in the arterial waveform.

Pulsus alternans (see Figure 9–2) is believed to be a sign of decreased myocardial contractility. A paradoxical pulse is an exaggeration of the normal variation in the pulse during the inspiratory phase of respiration, in which the pulse becomes weaker as the person inhales and stronger as the person exhales. Pulsus alternans is an indicator of the presence of severe ventricular systolic failure (Weber, 2003) and can be a sign of several conditions, including cardiac tamponade, which is a concern following cardiac surgery.

Complications associated with an intra-arterial catheter include ischemia or thrombosis of the affected extremity, infection, and bleeding. Prolonged hyperextension of the wrist can cause nerve conduction deficits. Close assessment for proper positioning and for signs of any complications related to indwelling intra-arterial catheters (e.g., presence of paresthesias, redness, extremity temperature and color) is an essential nursing responsibility and should be included in routine assessments (Srejic & Wenker, 2003).

CENTRAL VENOUS PRESSURE MONITORING

Because of the lack of supportive data on current use of pulmonary artery catheters (PACs), central venous pressure (CVP) catheters are being used more often in the cardiac surgical population. In one study,

Figure 9–2 Pulsus alternans.

Source: Illustrated by James R. Perron

researchers compared low-risk patients undergoing coronary artery bypass grafting with CVP with patients undergoing the same procedure with a PAC. Patients who had surgery with a PAC in place had higher weight gain and longer intubation time. Further, it is also speculated that the PAC may be associated with increased morbidity and resource utilization (Stewart, Psyhojos, Lahey, Levitsky, & Campos, 1998). Circumstances in which a PAC may be used include patients with pulmonary hypertension, low cardiac output, or predicted postoperative hemodynamic instability following cardiac surgery (Handa, Kyo, & Miyao, 2003).

It can be anticipated that patients will manifest a decrease in blood and plasma volume within the first 24 hours following cardiac surgery. Etiologic factors for this phenomenon include the patient's underlying cardiac disease, medications (preoperative, anesthesia, and vasoactive agents), procedure-induced hypothermia, rewarming, and bleeding. There is no reported agreement on which data should be used to guide fluid therapy in these patients. Filling pressures (i.e., CVP and pulmonary artery occlusive pressure) are often misleading as signs of optimal left ventricular filling, especially in patients with alterations in ventricular compliance (Boldt, 2005). In a landmark study, significant variations were reported in hemodynamic data following cardiac surgery. Because hemodynamic reference data had not been previously reported and great variability existed among the participants in this study, it remains difficult to use hemodynamic data as the sole basis for treatment decisions; indeed, using acceptable values to guide treatment may result in over-treatment of some patients (Sloth et al., 2008). Rather, correlating hemodynamic data with the patient's clinical presentation may be the most advantageous course of action.

Causes of elevated CVP readings may include hypervolemia, increased venous tone, RV or LV dysfunction, valve disease (mitral, tricuspid), pulmonary hypertension, atrial fibrillation, high pericardial pressures (such as seen in tamponade), high intrathoracic pressure (such as seen in pneumothorax or with positive pressure ventilation), and high intra-abdominal pressure. A low CVP value is most often indicative of hypovolemia or a decrease in cardiac output (Kazerooni & Gross, 2003). Volume repletion with a crystalloid, colloid, blood, or blood product, along with identifying and treating the source of fluid loss, will resolve the problem. The most common sources of hypovolemia are overzealous diuresis, third spacing, and hemorrhage, but causes may also include diaphoresis and vasodilation. CVP readings are influenced by the relationships among intravascular volume status, ventricular compliance, and intrathoracic pressure. As a consequence, trending data and correlating them with the patient's clinical status is more likely to optimize the patient's hemodynamic status than evaluating and treating just one isolated numeric value.

To further help assure the accuracy of CVP readings, pressure waveforms are read at end-expiration. Reading the tracing at this point minimizes the influence of intrathoracic pressure on the values.

In addition to aligning the transducer to the phlebostatic axis and interpreting the waveforms at end-expiration, analysis of waveform morphology is essential when the nurse is collecting hemodynamic data. A typical CVP tracing consists of three waves and two descents. An "a" wave represents contraction of the right atrium and corresponds with the P wave on an ECG tracing. An "a" wave will not be seen in patients with tricuspid stenosis, right ventricular hypertrophy, pulmonary hypertension, pulmonary stenosis, or atrial fibrillation. Giant "a" waves may be visible if the right atrium is attempting to eject blood into the right ventricle through a closed tricuspid valve, as

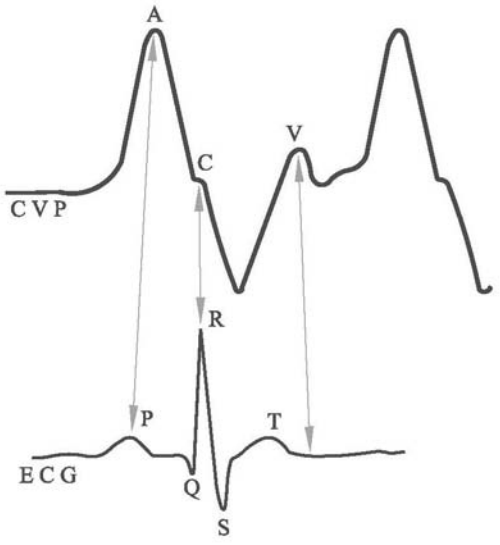

Figure 9–3 CVP waveform.
Source: Illustrated by James R. Perron

occurs in tricuspid stenosis (Mathew & Newman, 2001). Pericardial constriction may be reflected by a prominent "a" wave.

A "c" wave is produced with bulging of the tricuspid valve into the right atrium at the start of ventricular systole. It corresponds with the start of the QRS complex on an ECG tracing (Mathew & Newman, 2001). A large "c" wave may be present in patients who have tricuspid regurgitation.

The "X descent" represents atrial relaxation and corresponding displacement of the tricuspid valve during ventricular systole (Mathew & Newman, 2001). Absence of the "X descent" may be present in patients who have tricuspid regurgitation.

A "v" wave represents filling of the right atrium with a closed tricuspid valve. It corresponds to the area immediately following the T wave on an ECG tracing. A giant "v" wave may be seen where an acute increase in pressure in the right ventricle occurs (Muehlschlegel, Dobija, & Lobato, 2007), as is seen in patients who have tricuspid regurgitation. Pericardial constriction may be reflected by a prominent "v" wave.

Finally, a "Y descent" represents opening of the tricuspid valve. At this time, blood is flowing from the right atrium (causing an associated decrease in right atrial pressure) into the RV (Mathew & Newman, 2001) (see Figure 9–3). An attenuated "Y descent" may be seen in tricuspid stenosis, reflecting obstruction to right atrial emptying.

The nurse must keep in mind that alterations in waveforms may result in inaccurate numeric displays and that analysis of the waveforms is essential to obtain accurate hemodynamic data.

Table 9–1 lists complications associated with use of a CVP catheter. Some of these complications are site dependent—for example, pneumothorax is associated with internal jugular or subclavian insertion sites but not external jugular or femoral site use.

The risk of vascular injuries may be reduced with use of real-time ultrasound imaging during catheter insertion. Infectious complications may be minimized when steps to prevent infection are taken—and such steps should be part of every hospital's protocol. Preventive strategies include hand hygiene,

Table 9–1 Complications Associated with CVP Catheters

Pneumothorax (usually occurs during catheter placement)
Thrombus
Infection
Air embolism
Adjacent vessel perforation
Catheter shearing and embolization
Thrombophlebitis
Extravasation of fluid or medication into the mediastinum, pericardium, retroperitoneum, or pleural cavity
Hemothorax
Vascular injuries (e.g., local hematoma, arterial laceration, perforation of the superior vena cava, pericardial perforation)
Arterial puncture
Subpleural hematoma
Uncontrolled venous bleeding

Sources: Gerhardt & Skeehan, 2007; Savolainen et al., 2004.

maximal barrier precautions, chlorahexidine skin antisepsis, optimal catheter site selection, use of antibiotic-impregnated catheters, and daily review of catheter necessity (Barbieto & Mark, 2006).

MONITORING USING A PULMONARY ARTERY CATHETER

A PAC may be used to assess cardiac function, cardiac output/index, and intracardiac pressures (Vender & Szokol, 2002). Achieving a cardiac index in the range of 2.5–4.2 L/min/m^2 is a goal for most postoperative cardiac surgery patients. Obtaining these hemodynamic data directly from the LV would be ideal. Unfortunately, because of the potential for both damage to the left ventricular wall and dysrhythmias, it is not possible to directly monitor these pressures on a continuous basis.

Left atrial pressure is an alternative parameter to evaluate, as this pressure is the earliest indicator of left ventricular preload if no obstruction to flow is present (e.g., mitral stenosis). The line may be used for direct vasoactive medication infusion when the drug administered may be deleterious if routed through the pulmonary system before reaching the left heart. However, because the possibility of tamponade with catheter removal and entry of air or catheter dislodgement exist while the catheter is in place, a left atrial pressure line is not a routine choice for most clinicians.

A PAC may be the next choice because it sits in the pulmonary artery and would provide an earlier indication of changes in the LV than a CVP line. With no obstruction to flow, pulmonary artery pressure (PAP) will indirectly reflect left atrial pressure and approximate value of left ventricular end-diastolic pressure (left-sided preload). Values obtained with a PAC include pulmonary artery systolic (PAS), pulmonary artery diastolic (PAD), pulmonary artery mean (PAM), and PAOP.

- The PAS reflects pressure measured from the tricuspid to the mitral valve and is a good overall indicator of pulmonary artery pressures. Conditions such as COPD, acute respiratory distress syndrome, and pulmonary hypertension are likely to increase PAS pressure.
- The PAD reflects pressure in the area between the pulmonic and aortic valves. If there is no obstruction to blood flow, PAD is a good indicator of LV function.
- The pressure in the pulmonary artery is dynamic; it increases when blood is ejected from the right ventricle and then decreases until the next ejection of blood. The mean pulmonary artery pressure is the continuous average of the pressure in the pulmonary artery during one complete cardiac cycle (from the start of ejection of blood to the next) (Costanzo, 2008).
- The PAOP, obtained by inflating the PAC balloon, reflects the pressure between the tip of the PAC and the aortic valve. Because it assesses less surface area, the PAOP is more reflective of left ventricular function than is the PAD. In most circumstances, the PAOP is thought to closely equate to left atrial pressure and left ventricular end-diastolic pressure (LVEDP) or LV preload.

Fluid therapy and titration of vasoactive agents are based on these data. In some conditions, the PAOP is reported as greater than LVEDP—for example, in mitral valve disease, increased pulmonary vascular resistance, use of positive-pressure ventilation with associated increase in intrathoracic pressure, tachycardia, and COPD. In other conditions, the PAOP is reported as less than LVEDP—for example in the presence of aortic regurgitation, a noncompliant left ventricle, or pulmonary embolism (Tuman, Carroll, & Ivankovitch, 1989).

Normally, the PAD is slightly higher than the PAOP, and the normal correlation is less than 5 mm Hg (Marini & Leatherman, 2005). To obtain the PAOP, the balloon must be inflated, which increases the potential risk of

pulmonary artery rupture, pulmonary infarction, pulmonary thrombosis or embolism, and pulmonary artery hemorrhage. Obtaining PAOP readings may not be performed routinely but may be done if an acute change in the patient's clinical status or PAD occurs, or if no correlation between the PAD and PAOP exists. Unit-specific protocols for obtaining hemodynamic data should be followed.

When obtaining PAOP readings, balloon inflation time should be minimized. The balloon should be inflated slowly to avoid migration of the catheter into a smaller pulmonary artery or vessel rupture. The balloon should be left deflated at all other times (Marino, 2006).

CARDIAC OUTPUT MEASUREMENT

The PAC also allows measurement of cardiac output (CO) via thermodilution or the assumed Fick method. A bolus of either normal saline or D_5W is injected into the RA (proximal) port. The fluid mixes with the blood as it travels past the tricuspid valve, through the RV, and into the PA. The overall temperature of the mixed blood and injectate is measured by the thermistor (a temperature-sensing device) at the tip of the catheter. The amount of time it takes the cooler blood to pass the thermistor is used to calculate CO. The longer it takes for the cooler blood to pass, the lower the CO. An electronic display of the time–temperature curve and calculated numerical CO value are displayed on the monitor.

Several variables must be assessed to assure the accuracy of the CO displayed. Accuracy of CO results is essential because many of the hemodynamic calculations listed earlier and subsequent therapeutic modalities are based on accurate CO determination (Gawlinski, 2004). Intracardiac shunts produce shunting of cold injectate into the left heart, which decreases PA cooling and lowers the peak of the time–temperature curve, as seen with a right-to-left shunt; this condition also results in an underestimation of CO. Tricuspid regurgitation causes underestimation of the CO because the injectate will reflux back into the right atrium and prevent adequate mixing.

In patients with a left-to-right shunt, increased right heart volume dilutes the injectate, resulting in an overestimation of CO. Temperature of the injectate, injectate technique, minimal manipulation of the injectate-filled syringe, time between measurements, and lack of obstruction to a smooth injection must be confirmed and the patient's body position assessed to ensure accuracy of measurements. The monitor must be preset with the gauge of the PAC in place and the amount of injectate to be infused (5 mL or 10 mL). In addition, forward flow—so that adequate mixing occurs—is important (Gawlinski, 2004). Dysrhythmias, such as atrial fibrillation, will prevent thorough mixing. Thus the trend in CO values obtained is extremely important to monitor.

Continuous Cardiac Output

Potential causes of errors in obtaining intermittent measurements of CO have been discussed. Continuous cardiac output (CCO) catheters use a tracer that is not cool but warm; a 10-cm thermal filament is placed on the outside of the catheter at the level of the RV. The filament warms the catheter every 30–60 seconds, with low levels of heat energy being transferred to the blood that is adjacent to the filament. The same process is used to determine CO as with the intermittent injectate method. The only difference is that with this technology CO is calculated based on the amount of time it takes the warmed blood to pass the thermistor instead of cooled blood. The CO value is averaged over 3–6 minutes, and a numeric display of the calculated value appears on the monitor screen (Headley, 1997).

Benefits of contour cardiac output include avoidance of individual variations in the volume and speed of infusion of the tracer bolus, and the fact that CO is based on a time-weighted average versus a single instantaneous measurement. Drawbacks include the expense and lack of data supporting improved patient outcomes with its use (Headley, 1997).

Alternative Methods to Determine Cardiac Output

Even as the incorporation of goal-directed therapy using CO or similar parameters to guide intravenous fluid and inotropic therapy continues to increase, other, less invasive options for monitoring CO are being adopted in many practices. Technologies that are based on arterial pressure can provide CO determinations and measure other clinically important variables, such as stroke volume variation (SVV), pulse pressure variation (PPV), and systolic pressure variation (SPV). Clinical use of these parameters is emerging as a means for determining the patient's ability to respond to changes in fluid levels. SVV occurs due to changes in intrathoracic pressure during spontaneous breathing; blood pressure decreases during inhalation and increases during exhalation. The opposite changes are observed when a patient is receiving positive pressure ventilation.

Arterial Pulse Contour CCO

Arterial pulse contour CCO monitoring estimates CO based on pulse contour analysis; it is an indirect method based on analysis of the arterial pressure pulsation waveform. This technology relies on the concept that the contour of the arterial pressure waveform is proportional to stroke volume. The arterial pressure waveform is used to calculate CO, stroke volume variance, intrathoracic volumes, and extravascular lung water. These data are then used to predict response to fluid therapy (Uchino et al., 2006). The arterial waveform is typically recorded from an intra-arterial catheter, although noninvasive recordings have also been used. The efficacy of arterial pulse contour-based CO technology has been demonstrated in patients who underwent coronary artery bypass grafting procedures (de Waal, Kalkman, Rex, & Buhre, 2007).

Three of the currently available pulse contour cardiac output systems use intra-arterial waveform analysis. The PiCCO™ system uses thermodilution for calibration and requires femoral or axillary arterial catheterization. It incorporates use of a catheter with a thermistor on the tip. The catheter records aortic pressure waveforms, and CO is then calculated using a formula based on the area under the systolic portion of the waveform (Button et al., 2007). Data from several studies have led some researchers to question the correlation between CO measurements obtained using this technology and the intermittent injectate method in hypothermic patients, including those undergoing cardiopulmonary bypass and patients with an upper-body warming device in use (Böttiger et al., 1995; Ong, Gillies, & Bellomo, 2004; Spackman & Abenstein, 1993). When the PiCCO system is used, it is suggested that the arterial waveform be calibrated preoperatively and that recalibration be avoided until the patient is admitted to the ICU (Rauch et al., 2002).

The second pulse contour cardiac output system available is LiDCO™, which uses lithium dilution for calibration and arterial pulse wave analysis from PulseCO™. The radial or brachial artery is used as the access site. With this technique, a small dose of intravenous lithium chloride is administered. Cardiac output is then determined by a dilution curve made by a lithium-sensitive electrode that is attached to the intra-arterial catheter (Jonas & Tanser, 2002).

FloTrac™/Vigileo™, the third method of pulse contour analysis, does not employ a calibration process to improve monitor precision but instead uses a formula or algorithm to continually update a constant that is used

to determine CO (Compton, Zukunft, Hoffmann, Zidek, & Schaefer, 2008). The FloTrac sensor and Vigileo monitor together constitute the FloTrac system. As with the other pulse contour cardiac output systems, SVV may be calculated. Data used to calculate SVV include the patient's blood pressure, age, gender, and body surface area. The patient's CO is determined from the stroke volume and heart rate. An accurate arterial pressure waveform is essential for accurate contour cardiac output determination. Any factor that may alter the tracing (e.g., dysrhythmias, hypotension, equipment issues) may affect the results.

Recently, a study was conducted to compare the efficacy of these three pulse contour cardiac output systems in determining CO in patients who have undergone cardiac surgery. The data suggest that each of the methods is comparable to using a PAC with the intermittent injectate method (Button et al., 2007).

Stroke Volume Variation

SVV produces data on changes in preload that occur with mechanical ventilation. It is "the difference between the maximum and minimum stroke volume during one mechanical breath relative to the mean stroke volume" (Berkenstadt et al., 2005, p. 721). SVV monitoring can provide data that suggest whether a patient's stroke volume will improve with volume repletion (Reuter, Felbinger, Kilger, et al., 2002).

Currently, there are conflicting data regarding the ability of SVV to predict response to fluid therapy. Reuter and colleagues (Reuter, Felbinger, Schmidt, et al., 2002) determined that SVV predicted preload responsiveness in cardiac surgery patients. Conversely, Wiesenack and colleagues (2003) reported that SVV did *not* predict an increase in CO or stroke volume in cardiac surgery patients. Possible explanations for the discrepancies in results include differences in tidal volumes used and differences in the cardiac stability of the two groups of patients (Pinsky, 2003).

Pulse Pressure Variation

PPV is "the difference between the maximum and minimum values of the arterial pulse pressure during one mechanical breath divided by the mean of the two values" (Berkenstadt et al., 2005, p. 721). Reports suggest that variations in PPV can accurately predict response to fluid therapy in patients with shock and in surgical procedures. Upon evaluating the Frank-Starling curve, an increase in preload is associated with a decrease in PPV; conversely, a decrease in preload is associated with an increase in PPV and contractility. It has been suggested that PPV is more accurate in predicting fluid response than CVP and PAOP, SPV, and SVV (Michard, Lopes, & Auler, 2007).

Systolic Pressure Variation

SPV is "the difference between the maximum and minimum systolic blood pressure during one mechanical breath" (Berkenstadt et al., 2005, p. 721). It can reportedly indicate decreases in CO from blood loss and predict a patient's response to volume repletion. This parameter is used to estimate circulating volume (Gouvêa & Gouvêa, 2005).

In a study of patients in the ICU who underwent coronary artery bypass grafting, researchers determined that PPV and SPV were both able to predict whether a patient would respond to volume repletion with an increase in CO. While PPV was demonstrated to be superior to SPV at predicting response to fluid therapy, the researchers concluded that both PPV and SPV were far superior to CVP and PAOP data (Kramer, Zygun, Hawes, Easton, & Ferland, 2004).

Doppler Methods

Doppler-based methods use ultrasound and the Doppler effect to determine CO. When ultrasound waves strike moving objects, the waves are reflected back to their source at a

different frequency, which is directly related to the velocity of the moving objects and the angle at which the ultrasound beam strikes these objects. Proper probe placement is essential when using these methods to monitor CO. Several different Doppler-based methods may be used to measure CO, each of which uses a slightly different site in the body for measuring blood flow (Berton & Cholley, 2002). Data suggest that ultrasound determination of CO correlates with data from a PAC and central venous saturation percentage (Knobloch et al., 2005).

Electrical Bioimpedence

Electrical bioimpedence is a noninvasive method to determine CO. Using this technology, CO is measured based on changes in impedance that occur as blood is ejected from the left ventricle into the aorta and is calculated from changes in thoracic impedance. With this method, changes in thoracic blood volume during the cardiac cycle can be used to calculate CO. This technique is a successful method of monitoring CO because the algorithm eliminates the impedance due to body tissue and lung volume changes, instead using only the change in thoracic blood volume for CO determination. An alternative approach uses a specially designed endotracheal tube to measure electrical impedance changes in the ascending aorta (Ramsay, 2006).

ASSESSMENT OF OXYGENATION PARAMETERS

Venous Oxygen Saturation

In addition to direct pressure measurements and CO assessment, other hemodynamic data may assess a patient's condition following cardiac surgery. Another type of PAC provides for continuous monitoring of venous oxygenation saturation (SvO_2). SvO_2 reveals the association between oxygen delivery (the amount of oxygen that is carried to the tissues each minute) and oxygen consumption (the amount of oxygen used by the tissues) (Jesurum, 2004).

Mixed venous blood represents the amount of oxygen in the systemic circulation after the blood's passage through the tissues. Venous oxygen saturation data reflect tissue oxygenation and cardiopulmonary function and can be used to discover whether a patient is clinically deteriorating. It has been suggested that changes in SvO_2 may occur prior to changes in other aspects of the hemodynamic profile (Jesurum, 2004).

Normal SvO_2 is in the range of 60% to 80%. Trends and changes in oxygen delivery, oxygen consumption, or tissue oxygenation may be identified by reviewing data related to venous oxygen saturation. These data can also be used to determine the efficacy of interventions implemented to optimize these variables as well as procedures performed by the ICU nurse while caring for a postoperative cardiac surgery patient (Jesurum, 2004). With continuous digital readout of SvO_2 measurements, early recognition and prompt intervention to eradicate effects of poor tissue oxygenation can be implemented by the ICU nurse. Causes of changes in SvO_2 are many and include most variables affecting preload, afterload, and contractility. Although not specific to any one factor, any change in SvO_2 alerts the ICU nurse to quickly investigate.

Central Venous Oxygen Saturation

Newer catheters that allow for assessment of central venous oxygen saturation ($ScvO_2$) are being used in some cardiac surgical programs as the transition away from PACs continues. With this monitoring approach, a blood sample is obtained from a central venous catheter and is analyzed. A normal $ScvO_2$ is 70% or greater. If the value is less than 70%, it indicates that the tissues are extracting more oxygen than is normal and that the tissues do not perceive that their oxygen needs are being met (Goodrich, 2006; Rivers, Ander, & Powell, 2001).

There is some concern that $ScvO_2$ may not accurately reflect global hypoxia. $ScvO_2$ and SvO_2 values are not equivalent, but trends in both values are similar enough to allow them to be substituted for each other. Both levels have been shown to be correlated with patient outcomes, and both respond rapidly to changes in blood flow and oxygenation (Reinhart, Rudolph, Bredle, Hannemann, & Cain, 1989). In any event, changes in trends are more important to track than any change in one parameter.

POSTOPERATIVE HEMODYNAMIC ASSESSMENT

The ICU nurse caring for a postoperative cardiac surgery patient must be aware of both normal and baseline parameter values so that any clinical deterioration or improvement in the patient may be promptly noted. Some patients, because of their comorbidities or their disease process (such as valve disease), may require higher filling pressures postoperatively to maintain an adequate cardiac output/index.

An adequate cardiac index in the range of 2.5–4.2 L/min/m^2 will be sustained by normalizing heart rate and stroke volume as soon as possible. Many variables may affect heart rate and rhythm in the postoperative period. The most common causes in the postoperative cardiac surgery patient include hypovolemia and pain, both of which should be addressed promptly. Despite sedation, the nurse should assess for other signs and symptoms that indicate the presence of pain. Pain assessment and management are discussed in detail in Chapter 14.

Dysrhythmias that may be seen in the postoperative period include atrial fibrillation, premature ventricular contractions, and ventricular tachycardia; the latter two dysrhythmias may occur due to electrolyte imbalance. All of the dysrhythmias may arise as a result of cardiac irritability from operative manipulation. Ventricular fibrillation, although rare, may also occur. The presence of any dysrhythmia may affect a patient's hemodynamic status and requires rapid intervention. The etiology and management of postoperative dysrhythmias are discussed in detail in Chapter 15.

After assessing heart rate, an adequate stroke volume should be ensured. Variables that influence stroke volume—preload, afterload, and contractility—often are affected in the intraoperative and postoperative periods. For example, preload may be decreased as the patient undergoes the rewarming process, which may lead to vasodilation. Bleeding from chest tubes or third spacing that results from the inflammatory process may also decrease preload, resulting in a decrease in CO. Postoperative bleeding is always a concern for the cardiac surgical patient. Blood loss will decrease the oxygen-carrying capacity to vital organs and tissues (Gespard, 2006). Logically, decreased circulating volume will decrease preload, stroke volume, and CO.

The causes of postoperative bleeding are many. For instance, the cardiopulmonary bypass circuit may cause platelet destruction as the blood circulates through it, in addition to decreasing levels of clotting factors. Inadequate hemostasis from incomplete heparin reversal or excessive protamine administration is another potential cause of altered hemostasis, as is a surgical bleed from a suture site.

If chest tube drainage exceeds 100 mL/hr for more than 3 hours, 200 mL/hr for 3 hours, or 300 mL in the first hour following surgery, the surgeon should be notified. Transfusion of blood or blood products may be ordered if the coagulation studies are outside of the normal range or if the patient's hematocrit level is low. If the patient is hypertensive, the blood pressure must be decreased to prevent stress on the suture sites, which may cause further bleeding. The patient may need to undergo surgical reexploration. Decreases in blood pressure, cardiac filling pressures, and urinary output are signs of hypovolemia that must be evaluated. Adjustments to volume administra-

tion are frequently necessary as well. Volume repletion is accomplished by administration of isotonic crystalloids (e.g., lactated Ringer's or normal saline) or colloids (e.g., albumin, blood, or blood products) as determined by the patient's lab results. Conversely, if preload indices are too high, diuretics, vasodilators (e.g., nitroglycerin), or both may be used.

An increased afterload may result from severe left ventricular dysfunction, hypovolemia, vasoconstriction, hypothermia, or increased catecholamine stimulation from the surgical procedure. Along with volume-related interventions and use of a warming blanket, arterial vasodilator administration may be beneficial in such cases. A decreased afterload may be the result of significant vasodilation from warming; this condition may be treated with administration of an agent that causes vasoconstriction.

Decreased contractility in the postoperative period may be the result of an increase or decrease in preload, an increase in afterload, or factors that affect myocardial contractility directly (e.g., ischemia, right or left ventricular failure, and aneurysms). Electrolyte imbalance and tamponade may also alter contractility. In such a scenario, preload and afterload are optimized while other interventions to treat the underlying cause are completed. If indicated, administration of positive inotropic agents is initiated. If afterload reduction is needed, an IABP is added. The IABP can increase CO by as much as 1 liter and may be necessary to support the patient through an acute event. IABP therapy is discussed in detail in Chapter 10. The use of biventricular pacing has also been reported to improve contractility following bypass procedures (Bakhtiary et al., 2007).

If blood builds up inside the mediastinum, cardiac tamponade may occur, resulting in physical compression of the heart, limitation of diastolic filling time, and a decrease in CO. Cardiac tamponade and several other postoperative complications are discussed in detail in Chapter 13.

SUMMARY

The number of cardiac surgical patients with pulmonary artery catheters has decreased worldwide. Interestingly, data have not supported the assertion that any specific hemodynamic monitoring technique affects patient outcomes (Nesbitt, 2006). Although little published evidence exists to associate use of patient monitoring with improved clinical outcomes, this lack of evidence does not necessarily equate to a lack of benefit. Thus catheters will still be used, albeit with caution.

ICU nurses play a pivotal role in monitoring the postoperative hemodynamic status of patients following cardiac surgery. They must obtain accurate data, integrate those monitoring data with information gained by assessing the patient's clinical status, and use clinical judgment to select the best interventions to optimize the patient's status given the patient's current condition and past medical history. Having expertise helps to ensure that obtained parameters are not reflecting nonphysiologic events such as patient turning, artifact, and inaccurate leveling, and that values are assessed at end-expiration. The ICU nurse with high levels of critical judgment and clinical inquiry competencies will use accurate information and evidence-based guidelines to determine when activities can be clustered or when oxygen consumption is too high to do so.

By definition, the cardiac surgical patient always has underlying cardiac pathology that will have a major impact on postoperative recovery. Monitoring that incorporates a clinical evaluation, review of physiology, and expected responses relative to the type of cardiac surgery performed is essential. Invasive catheters may be used to augment—but not replace—monitoring for subtle changes. The expert ICU nurse validates signs and intervenes quickly. Each of these competencies is essential to achieve an optimal patient outcome following cardiac surgery.

CASE STUDY

A 76-year-old male patient, with a history of MI and angioplasty to the left anterior descending artery 3 years ago, is admitted to the ICU after on-pump cardiac surgery. Triple bypass was completed on the LAD, circumflex, and right coronary artery. At the time of admission, the patient data were as follows: BP 101/70; MAP 80 mm Hg; HR 110; PAS 22 mm Hg; PAD 9 mm Hg; PAOP 7 mm Hg; CVP 5 mm Hg; temperature 35.4 °C; CI 2.2 L/min/m^2; Hct 26%.

Critical Thinking Questions

1. What else should be part of this patient's initial admission assessment?
2. Which of the parameters given in the case study may indicate hypovolemia?
3. What are the best options for fluid replacement for this patient?
4. What are two reasons why tachycardia might occur in the immediate postoperative period?

Answers to Critical Thinking Questions

1. Following cardiac surgery, the ICU nurse will connect the patient to the bedside monitor upon receipt from the operating room. The ECG leads are connected to the bedside monitor from the transport monitor, and heart rate and rhythm are assessed. The pulse oximetry probe is connected to either the finger, earlobe, or forehead. Pulse oximetry is a simple, noninvasive method of monitoring the percentage of hemoglobin that is saturated with oxygen. The target oxygen saturation (SpO_2) is 95% or greater.
2. CVP 5 mm Hg
3. Fluid bolus with normal saline
4. Tachycardia may arise as a result of cardiac irritability from intraoperative manipulation, electrolyte imbalance, pain, or anxiety.

SELF-ASSESSMENT QUESTIONS

1. Pulsus alternans is indicative of
   a. left ventricular systolic dysfunction.
   b. left ventricular diastolic dysfunction.
   c. right ventricular systolic dysfunction.
   d. right ventricular diastolic dysfunction.
2. Causes of an increased CVP may include
   a. hypervolemia, mitral regurgitation, and third spacing.
   b. ascites, pulmonary hypertension, and diuresis.
   c. cardiac tamponade, pneumothorax, and third spacing.
   d. hypervolemia, pneumothorax, and tricuspid stenosis.
3. An acceptable correlation between the PAD and PAOP, with the PAD being higher, is
   a. 3–8 mm Hg.
   b. 1–2 mm Hg.
   c. 1–4 mm Hg.
   d. 4–8 mm Hg.
4. You suspect that the tip of the PA catheter has slipped from the PA to the RV. Two changes on the waveform that might indicate this dislodgement are
   a. higher diastolic pressure and presence of a dicrotic notch.
   b. lower diastolic pressure and absence of a dicrotic notch.
   c. higher diastolic pressure and absence of a dicrotic notch.
   d. lower diastolic pressure and presence of a dicrotic notch.
5. Positive inotropic therapy to improve cardiac output includes
   a. IABP, milrinone, and labetalol.
   b. dobutamine, milrinone, and IABP.
   c. digoxin, atenolol, and IABP.
   d. IABP, metoprolol, and dobutamine.

6. A large "v" wave seen on the PAOP wave tracing may indicate
   a. mitral regurgitation.
   b. mitral stenosis.
   c. tricuspid regurgitation.
   d. tricuspid stenosis.
7. The presence of a paradoxical pulse is often indicative of
   a. ARDS.
   b. hypertrophic cardiomyopathy.
   c. mitral stenosis.
   d. cardiac tamponade.
8. SvO_2 represents the
   a. percentage of oxygen remaining in the blood, assessed upon return to the right side of the heart after passage through the systemic circulation and tissues.
   b. percentage of oxygen removed from the blood, assessed upon return to the right side of the heart after passage through the systemic circulation and tissues.
   c. percentage of lactic acid removed from the blood, assessed upon return to the right side of the heart after passage through the systemic circulation and tissues.
   d. percentage of lactic acid remaining in the blood, assessed upon return to the right side of the heart after passage through the systemic circulation and tissues.
9. Interventions to increase preload include
   a. crystalloids, blood, and epinephrine.
   b. nitroglycerin, crystalloids, and furosemide.
   c. epinephrine, norepinephrine, and furosemide.
   d. crystalloids, blood, and nitroglycerin.
10. The PAD would not reflect left ventricular pressure in the presence of
    a. mitral stenosis.
    b. tricuspid stenosis.
    c. pulmonic stenosis.
    d. tricuspid regurgitation.

Answers to Self-Assessment Questions

1. a
2. d
3. c
4. b
5. b
6. a
7. d
8. a
9. a
10. a

Clinical Inquiry Box

Question: Does the level of experience of a critical care nurse affect the hemodynamic decision making in postoperative cardiac surgery patients?

Reference: Currey, J., & Botti, M. (2006). The influence of patient complexity and nurses' experience on haemodynamic decision-making following cardiac surgery. *Intensive and Critical Care Nursing, 22*(4), 194–205.

Objective: To uncover the decision-making utilized by critical care nurses during the first 2 hours after cardiac surgery. Specifically, a nurse's level of experience and complexity of decision making were investigated.

Methods: This descriptive study used continuous nonparticipant observation of clinical practice for a 2-hour period after cardiac surgery. Observations were recorded in field notes, and semistructured interviews occurred among 28 nurses and were audio recorded. Content analysis of the observation and interview data were performed.

Results: Experience of the nurses and support of colleagues were associated with higher-quality decision making. These two factors—nurses' utilization of evidence for practice and the experience levels of both nurses and their colleagues—are critical for the delivery of quality patient care.

Conclusion: These findings suggest staffing units with experienced nurses is essential, especially to ensure that new nurses have individuals to use as resources in the decision-making process.

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Chapter 10

Intra-aortic Balloon Pump

Barbara Hutton-Borghardt

INTRODUCTION

The intra-aortic balloon pump (IABP) is a mechanical device that is temporarily used to improve cardiac function. In many situations, the IABP is life-saving in its ability to stabilize patients as they await procedures such as heart transplant, coronary artery bypass grafting (CABG), or percutaneous coronary interventions (PCI) such as PTCA/stent placement (Tommaso, 2002).

An IABP may be further indicated in the management of cardiogenic shock (Reid & Cottrell, 2005). Medications such as vasodilators and inotropes are used initially to improve cardiac function. If they are not effective, the IABP may be used alone or with pharmacotherapy to assist left ventricular (LV) function and improve cardiac output (CO) (Stenz, 2006).

Since its introduction in the late 1960s, IABP has become a widely used device in preoperative, intraoperative, and postoperative cardiac surgery patients (Ahmad, 2006). Worldwide, it is estimated that 100,000 IABP catheters are inserted annually (Stenz, 2006). A description of how an IABP improves cardiac function appears in Box 10–1.

COMPONENTS OF AN IABP

The IABP consists of two main parts: (1) a double-lumen catheter with an inflatable balloon attached to the distal end and (2) a console that regulates the inflation and deflation of the balloon. One lumen of the balloon catheter is attached to a pressure-transducer device that monitors the patient's arterial aortic pressure; the other lumen (with the balloon) is attached to a gas reservoir. The console allows for appropriate timing of balloon inflation and houses the helium (or CO_2) tanks. The tanks contain the gas that will be used to inflate the balloon during therapy.

Box 10–1 Goals of IABP Therapy

The IABP achieves its goals of stabilizing cardiac function by several mechanisms:

- It improves cardiac function (cardiac output) by decreasing left ventricular end-diastolic volume (preload).
- It improves myocardial oxygen supply by increasing blood flow to the coronary arteries.
- It decreases myocardial oxygen demand by decreasing left ventricular wall tension.
- It stabilizes cardiac function in patients with dysrhythmias and myocardial ischemia.

Source: Laurent & Shinn, 2005.

Additionally, the console has a monitor that displays the arterial waveforms, electrocardiogram (ECG), and balloon-pressure waveforms. Waveforms assist practitioners in determining whether the timing of balloon inflation/deflation is appropriate and allow for any necessary adjustments to be made (Metules, 2003).

Physiology of Balloon Function

The IABP is timed to inflate and deflate in opposition to the cardiac cycle. The goal of inflation of the IABP balloon is to enhance perfusion. The balloon inflates at the beginning of diastole and deflates before ventricular systole, a process known as counterpulsation. To correlate the inflation and deflation to the ECG, the balloon begins to inflate in the middle of the T wave and to deflate before the end of the QRS complex (Ahmad, 2006).

Inflation of the balloon at the beginning of diastole displaces blood upward toward the aortic root and augments the diastolic pressure between the balloon and the aortic origin. The increase in diastolic pressure, which is known as diastolic augmentation, forces blood back into the coronary arteries, which are normally perfused during diastole. Consequently, blood flow to the coronary arteries is increased, with a resultant improvement in myocardial oxygen supply. IABP inflation further causes a decrease in heart rate and afterload and enhances LV function. Ischemia of the myocardial muscle is diminished or relieved with the ensuing improved CO (Boehmer & Popjes, 2006). During inflation, blood is also pushed forward to the periphery. In this way, blood flow is increased below the inflated balloon, which may enhance perfusion of the renal arteries and systemic blood vessels (Paul & Rasmusson, 2007).

Deflation of the balloon immediately before systole occurs pulls blood forward away from the left ventricle, allowing for more complete emptying. This enhanced LV emptying decreases preload (or end-diastolic volume) and myocardial oxygen demand. The tension caused by the pressure of blood on the left ventricle as it ejects blood (afterload) is diminished as well, further decreasing myocardial oxygen demand and increasing CO and ejection fraction (EF). Systolic blood pressure is noted to be lower with the reduction in afterload (Metules, 2003).

Secondary effects of the IABP placement result from the improvement in cardiac function as well. Heart rate, pulmonary artery diastolic (PAD), and pulmonary artery occlusive pressures (PAOP) are decreased; mean arterial pressure (MAP), CO, and perfusion to vital organs are increased (Laham & Aroesty, 2008).

INDICATIONS FOR IABP THERAPY

The IABP is used in a variety of clinical situations, such as in cardiogenic shock. Cardiogenic shock is a complication in approximately 7.5% of patients with an acute myocardial infarction (AMI) and carries a mortality rate in the range of 70% to 80%. Early revascularization with angioplasty, fibrinolysis, or bypass surgery is initiated to improve mortality in such circumstances (Hochman et al., 2006). In the case of the patient with cardiogenic shock, the IABP may be used to reduce myocardial ischemia and improve cardiac function, especially as the patient is prepared for a revascularization procedure (Metules, 2003). Data suggest that the use of the IABP in reducing ischemia in AMI can preserve and prevent the loss of viable myocardial muscle and improve survival rates in cardiogenic shock (Lindholm et al., 2003). In post-MI patients, persistent ischemia and reinfarction may also be prevented through use of an IABP (Duvernoy & Bates, 2005).

For patients with unstable angina who are receiving maximum medical therapy but who still experience chest pain/discomfort, the IABP has been successful in reducing or

entirely eliminating symptoms. The patient's condition can be stabilized in preparation for surgery or revascularization procedure (Ahmad, 2006; Laham & Aroesty, 2008).

In post-MI patients, structural damage, such as a ventriculoseptal defect (VSD) or mitral regurgitation may occur. The IABP can help hemodynamically stabilize these patients until surgical repair can be performed. With a VSD, there is an abnormal opening between the right and left ventricles. Because pressure is higher in the left ventricle than in the right ventricle, blood is shunted into the right ventricle, resulting in a lower CO and right ventricular failure. The decrease in afterload produced by the IABP decreases the right-to-left shunt (Thiele et al., 2003).

Mitral regurgitation in post-MI patients is often due to papillary muscle dysfunction or rupture. The papillary muscles, which are located in the mid- to lower ventricles, are connected to the valve leaflets by the string-like cordinae tendinae. When left ventricular systole occurs, the papillary muscles contract and pull on the cordinae. This action prevents the mitral valve leaflets from inverting. In the setting of papillary muscle dysfunction, the mitral valve becomes incompetent and regurgitant blood flow occurs. Blood is then forced back up into the left atrium during ventricular systole, increasing the pressure in that chamber. The increased left atrial pressure is transmitted into the pulmonary vasculature, causing pulmonary congestion and edema. Use of the IABP to decrease afterload can diminish this regurgitant blood flow, thereby relieving pulmonary congestion as the patient awaits surgical repair (Antman et al., 2004).

The IABP may be essential to assist cardiac function in patients with end-stage cardiac disease or damage while they are awaiting transplant (i.e., as a bridge to transplant). The IABP may be used on a longer basis—as long as 6 months—in these instances. Its use may enable these patients to ambulate as they await transplant (Boehmer & Popjes, 2006).

Refractory ventricular dysrhythmias may also be responsive to IABP therapy. Poor LV function, coupled with an increase in afterload and increased myocardial oxygen demand, will produce ventricular stretching. Ventricular stretching increases irritability, resulting in difficult to treat dysrhythmias. The use of the IABP improves coronary blood flow, thereby helping to reduce irritability. Additionally, because the IABP decreases preload and afterload, the ventricle will be less distended, which further decreases irritability and arrhythmogenicity (Fotopoulos et al., 1999).

It is often difficult to wean postoperative on-pump cardiac surgery patients from cardiopulmonary bypass (CPB) due to preexisting poor cardiac function and the effects of CPB itself. Placing patients on CPB involves stopping the heart, usually with the use of a cold electrolyte solution (cardioplegia), and inducing a controlled state of ischemia. In the postoperative period, the myocardial muscle is stunned and may need assistance to function effectively (Dixon, Santamaria, & Campbell, 2005; Henke & Eigsti, 2003). The IABP stabilizes the hemodynamic profile of these cardiac surgery patients and allows them to be weaned more slowly with less risk of organ damage from a failing heart. Myocardial stunning is discussed in detail in Chapter 13.

Preoperative use of the IABP in patients who are considered at high risk for cardiac surgery has been shown to lower the postoperative mortality rate and shorten intensive care recovery (Christenson, Cohen, Miller, Ohman, & Urban, 2002). High-risk patients include those with two of the following characteristics: poor LV function (EF less than 30%), unstable angina, left main coronary artery stenosis of greater than 70%, and undergoing a redo bypass procedure (Christenson, Simonet, & Schmuziger, 2000).

Other indications for use of IABP therapy include progressive heart failure, prevention

of restenosis of the coronary artery in a post-PTCA procedure, need for cardiac support during noncardiac surgery (Stone et al., 2003; Tremper, 2006), right ventricular failure, and septic shock (Kern, King, Douglas, & Franch, 2004).

In a worldwide study known as the Benchmark Registry, more than 16,000 patients who had undergone IABP support were evaluated. The most common indications for initiating IABP therapy were to provide hemodynamic support during or after a cardiac catheterization procedure, cardiogenic shock, postoperative cardiac surgery in which CPB was used, preoperative cardiac support in high-risk patients, and unstable angina refractory to medical therapy (Ferguson et al., 2001).

CONTRAINDICATIONS TO IABP THERAPY

Contraindications to the use of the IABP are few and can be divided into absolute and relative contraindications. Absolute contraindications are those in which the patient should not receive IABP therapy; they include abdominal aortic aneurysm, aortic dissection, aortic insufficiency, and irreversible brain damage (Little, 2004). Relative contraindications are those in which the potential risk of using the IABP must be weighed against the potential benefit; they include patients with peripheral vascular disease, coagulopathies or thrombocytopenia, terminal diseases, and end-stage cardiomyopathies that are not suitable for transplant (Little, 2004; Tremper, 2006).

INSERTION OF AN IABP

Insertion of an IABP catheter may be performed at the bedside in the ICU, in the catheterization lab, or in the operating room. Generally, institutional policy requires an informed consent to be signed and reviewed for completeness prior to insertion.

After preparation of the area and administration of a local anesthetic, the balloon catheter is inserted into either the right or left femoral artery. It is threaded up into the descending aorta so that the tip of the catheter is located 1 to 2 centimeters below the subclavian branch of the aortic arch and above the branches of the renal arteries (Kern et al., 2004; Metules, 2003) (see Figure 10–1). In bridge-to-transplant patients, the catheter is usually inserted in the subclavian fossa, with the distal end being located above the renal arteries (Boehmer & Popjes, 2006).

Traditionally, the balloon catheter is inserted through an introducer sheath, although many newer catheters are designed to be sheathless. The latter design results in a smaller-diameter catheter in the femoral artery, decreasing the chance of ischemic complications to the lower extremity (Erdogan et al., 2006).

The catheter may be inserted under fluoroscopy, which facilitates direct visualization—a key consideration in ensuring proper placement of the catheter. If fluoroscopy is not used, a radiograph film will be checked immediately following the procedure (Little, 2004). When viewed, the tip of the catheter should be located at the second or third intercostal space. Proper positioning is essential, as a catheter placed too high will obstruct blood flow to the subclavian artery, which supplies the head and upper extremities. A catheter placed too low can obstruct blood flow to the renal arteries (Zellinger, 2007).

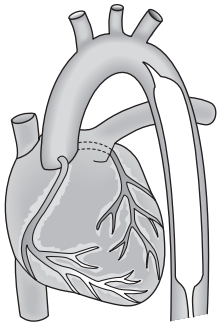

Figure 10–1 Inflated balloon catheter in descending aorta.

Source: Illustrated by James R. Perron

After the catheter is secured in place, a sterile dressing is applied to the insertion site. The patient's ECG tracing is displayed on the console's monitor; review of the ECG is important to maintain proper triggering of the pump. The central lumen of the balloon catheter is attached to a pressure-monitoring device with continuous flush to monitor the arterial pressure waveform. The balloon lumen is attached to the gas reservoir of the IABP console.

Upon its initial setup and every hour thereafter, the balloon will be inflated with a syringe, or by the autofill function on some consoles, with the appropriate volume of helium or CO_2. Helium is beneficial, especially with faster heart rates, because it is lighter in density than CO_2 and can travel faster in and out of the balloon circuit. However, in the event helium is released into the descending aorta (e.g., from a leak or balloon rupture), the gas will rapidly move to the cerebral and coronary vasculature—an immediately fatal complication (Little, 2004). Carbon dioxide, while slower in moving through the balloon circuit, has the advantage of being a more soluble gas and may cause less harm and decreases the potential for gas embolism development should the gas leak into the bloodstream via a ruptured or torn balloon (Metules, 2003). Balloon volume size varies from 30 to 34 mL for a smaller adult to 50 mL for a larger adult. Most balloons used are 40 mL in size (Quaal, 2005).

TIMING

Correct timing of balloon inflation and deflation is imperative to achieve the optimal benefit. Usually, the ECG is used to trigger the pump: The pump identifies the "R" wave to signify ventricular systole. Other triggers, such as an arterial pressure waveform or pacer spikes, may also be used (Metules, 2003). If the designated trigger is not noted, the pump will not initiate inflation and deflation of the balloon. In such a case, the trigger must be restored or a different trigger selected for the pump to work.

The arterial waveform displayed on the console's monitor is used to identify whether the timing of inflation and deflation is accurate. Balloon inflation should start at the beginning of diastole; deflation occurs just before systole. Initially, the inflation frequency is set at 1:2 (every other beat assisted) so that the unassisted and assisted waveforms can be compared. Later, the frequency may be switched to 1:1 (every beat assisted) if the patient's status requires this timing. As the patient's condition improves, the frequency may be weaned to 1:2, 1:3, 1:4, or 1:8 before IABP therapy is discontinued.

To confirm that the timing of inflation and deflation is correct, specific characteristics are observed on the arterial waveform. First, it is necessary to become familiar with the normal arterial waveform, noting the dicrotic notch (see Figure 10–2). Next, the arterial waveform of a patient receiving IABP therapy is observed. The unassisted systole, the dicrotic notch signifying closure of the aortic valve, and the unassisted aortic end-diastolic pressure should be identified (see Figure 10–3). Following the dicrotic notch of an assisted beat will be diastolic augmentation. The dicrotic notch should form a distinct "V" shape between the unassisted systole and the augmented diastolic, indicating that pressure increased in the aortic root during balloon inflation. Following the augmented diastolic is the assisted end-diastolic pressure, which is lower than the unassisted diastolic pressure because deflation of the balloon results in lower aortic pressure.

Balloon inflation is optimal when (1) a sharp "V" is noted at the dicrotic notch and (2) following the dicrotic notch, the augmented diastolic is as high as or higher than the previous systolic blood pressure. Balloon deflation is optimal when (1) the assisted end-diastolic pressure is lower, usually by 5 to 10 mm Hg,

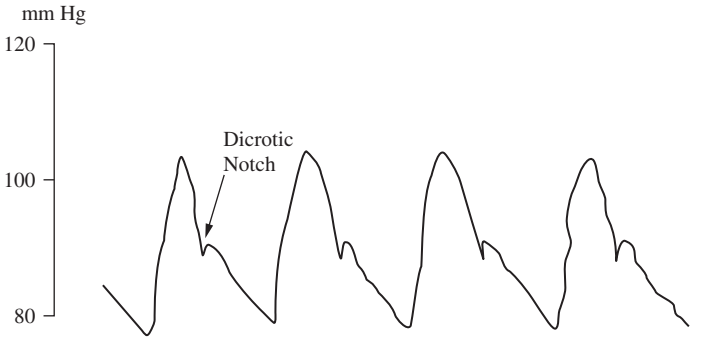

Figure 10–2 Normal arterial waveform.
Source: Illustrated by James R. Perron

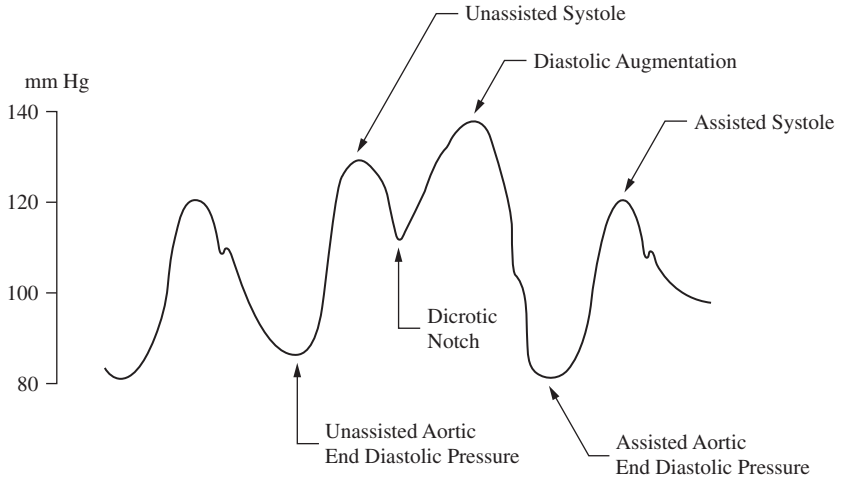

Figure 10–3 Arterial waveform of IABP patient, 1:2 counterpulsation.
Source: Illustrated by James R. Perron

than the unassisted aortic end-diastolic pressure and (2) the assisted systolic blood pressure is 5 to 10 mm Hg lower than the unassisted systolic pressure (Tremper, 2006).

Timing Errors

Although most IABPs have automatic timing, it is essential that continuous monitoring be maintained. Often, manual adjustments to optimize timing are needed. With timing errors, not only are patients not receiving optimal benefit, but they may also suffer deleterious consequences, especially when inflation is not timed correctly (Pantalos et al., 2003).

Timing errors occur when there is early or late *inflation* or early or late *deflation* of the balloon. With early balloon inflation, the balloon inflates before closure of the aortic valve. This action forces the valve to close early, resulting in aortic regurgitation and subsequent reduction in stroke volume, as well as increases in end-diastolic volume and myocardial oxygen demand. In such a case, the arterial waveform will lose its characteristic "'V" shape before diastolic augmentation (see Figure 10–4).

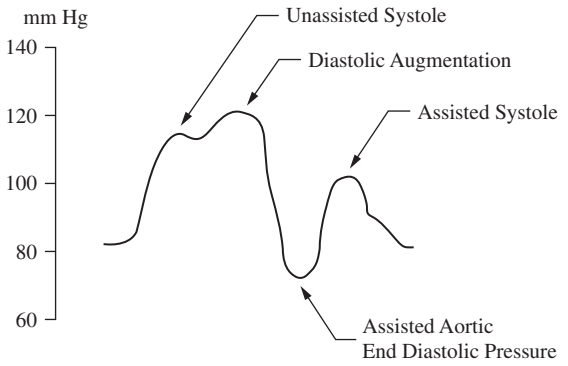

Figure 10–4 Arterial waveform with early balloon inflation.

Source: Illustrated by James R. Perron

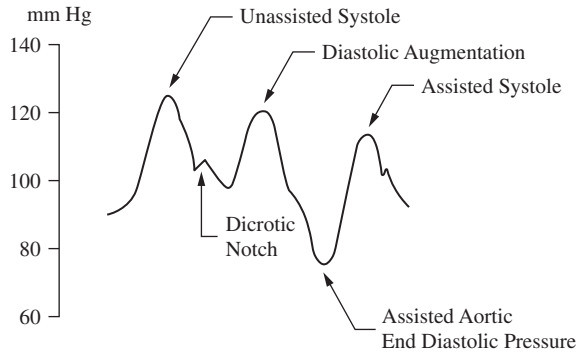

Figure 10–5 Arterial waveform with late balloon inflation.

Source: Illustrated by James R. Perron

With late inflation, the balloon inflates later than the appropriate time after closure of the aortic valve, with resultant lower augmented diastolic and coronary perfusion pressures. As a result, the IABP's key benefit—improving blood and oxygen supply to the coronary arteries—is lost or reduced. On the waveform, the peak of the augmented diastolic will be farther away from the dicrotic notch and will be lower, instead of higher, than the unassisted systolic (see Figure 10–5).

Normally, balloon deflation occurs just before the beginning of systole. If it occurs too far before the onset of systole, however, the patient's diastolic pressure will rise, leading to increases in afterload and myocardial oxygen demand. The arterial waveform reveals a sharp drop-off in the augmented diastolic curve, followed by a "U"-shaped curve before the next systolic upstroke (see Figure 10–6). When the balloon deflates later than the optimal time, its volume decreases as the aortic valve opens instead of before it opens. This results in the loss of the afterload reduction benefit; it may also increase afterload (and myocardial oxygen demand) as the inflated balloon impedes the ejection of blood from the left ventricle. The waveform will reveal a widened augmented diastolic wave and a slow rise of the next assisted systole (see Figure 10–7).

Most IABP consoles have a display for the balloon pressure waveform. This waveform represents the pressure as gas is propelled in and out of the balloon catheter. Monitoring this waveform is beneficial as it will assist the ICU nurse in determining whether the balloon is functioning effectively (Quaal, 2005) (see Figure 10–8). Caregivers may find it necessary to follow the specific manufacturer's directions for many settings on such devices, as

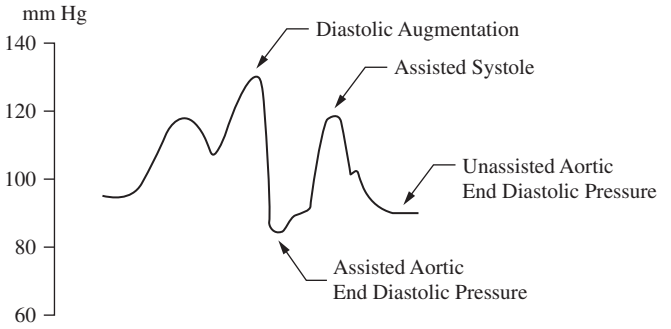

Figure 10–6 Arterial waveform with early balloon deflation.

Source: Illustrated by James R. Perron

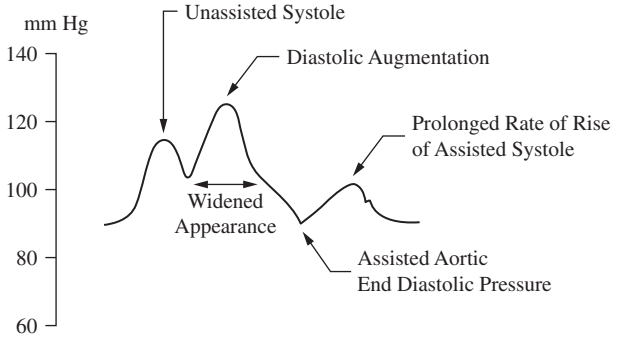

Figure 10–7 Arterial waveform with late balloon deflation.

Source: Illustrated by James R. Perron

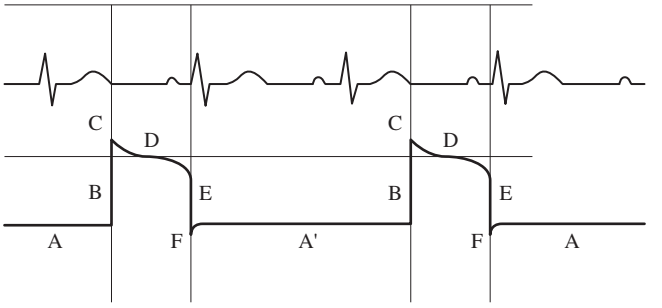

Figure 10–8 IABP waveform.

A = Balloon Pressure baseline; B = Rapid inflation; C = Peak inflation artifact; D = Balloon Pressure Plateau (balloon is completely inflated); E = Rapid deflation; F = Balloon deflation artifact; A' = Return to baseline (balloon completely deflated)

Source: Illustrated by James R. Perron

Box 10–2 IABP Therapy Waveform Definitions

Dicrotic notch: An area on the downstroke of the arterial waveform that results from the slight pressure increase created by closure of the aortic valve.

Diastolic augmentation: The increase in pressure in the aorta above the balloon catheter that results with balloon inflation during diastole. It increases perfusion in the coronary arteries and myocardial oxygen supply.

Unassisted aortic end-diastolic pressure: The pressure in the aorta at the end of diastole when counterpulsation via the balloon pump has not assisted that cardiac cycle.

Assisted aortic end-diastolic pressure: The pressure in the aorta at the end of diastole when counterpulsation has assisted the cardiac cycle. It is usually lower than the unassisted end-diastolic pressure.

Unassisted systole: The systolic aortic pressure when counterpulsation has not assisted the cycle.

Assisted systole: The systolic aortic pressure when counterpulsation has assisted the cardiac cycle. It is usually lower than the unassisted systole due to the action of balloon deflation.

Source: Laurent & Shinn, 2005.

consoles offered by different companies may have unique properties.

Refer to Box 10–2 for definitions related to IABP therapy.

COMPLICATIONS OF IABP THERAPY

Although the mortality rate associated with the use of the IABP is low, the rate of complications is reported to range from 2% to 40% (Boehmer & Popjes, 2006; Stenz, 2006; Tremper; 2006). In the largest study of IABP use, the complication rate was 7%, with major complications occurring in only 2.8% of cases (Ferguson et al., 2001).

The most prevalent complications are vascular in nature, with the most common being lower-limb ischemia below the insertion site. Fortunately, catheter sizes are becoming smaller and many do not require a sheath for placement (Erdogan et al., 2006). Over the years, smaller catheter size and sheathless introducers have helped to reduce complication rates (Boehmer & Popjes, 2006). Some patients, however, are more prone to limb ischemia. Especially vulnerable populations include the elderly, diabetics, females, obese patients, and individuals with peripheral vascular disease (Hurwitz & Goodman, 2005). Limb ischemia can occur while the catheter is in place or within hours of its removal and is related to presence of a clot at the catheter site. Thrombectomy is usually required to treat this complication (Marino & Sutin, 2006).

Other vascular complications include bleeding or hemorrhage from the insertion site, perforation of the femoral artery, superficial or deep vein thrombosis, stroke, aortic dissection or perforation, and compartment syndrome (Kern et al., 2004; Tremper, 2006). Vascular complications can result in severe consequences, to the point that the patient may require an amputation, thrombectomy, blood transfusions, or vascular surgery (Boehmer & Popjes, 2006; Stone et al., 2003). Mortality rates associated directly with the use of the IABP are reported to be less than 1% (Ferguson et al., 2001; Stone et al., 2003).

Balloon-related complications can occur as well. Balloon rupture will result in the release of a helium or CO_2 bolus into the bloodstream, as the gas will no longer be contained within the balloon circuit. Blood noted within the gas tubing, inability to maintain augmentation, and low pressure/gas alarms are indicators of possible balloon rupture (Boehmer & Popjes, 2006). Balloon rupture is more likely to

occur in patients with atherosclerosis, in whom the balloon will be inflating against rough calcium deposits in the aorta (Laurent & Shinn, 2005).

Balloon migration within the aorta is also possible. If the balloon migrates upward, blood supply to the upper extremities and head may be compromised. If the balloon migrates downward, blood supply to the renal arteries will be impaired (Tremper, 2006).

The ICU nurse should monitor for a variety of other complications when an IABP is used. Infection at the insertion site or in the systemic circulation may occur due to the presence of an indwelling catheter; red blood cell hemolysis and thrombocytopenia are possible due to the action of the balloon on blood components as they pass through the aorta (Tremper, 2006). Other complications cited in the literature include spinal cord ischemia, visceral ischemia, renal failure, and peripheral neuropathy (Marino & Sutin, 2006).

WEANING FROM IABP THERAPY

The IABP is a temporary device that is usually discontinued postoperatively. Occasionally, some patients who are awaiting cardiac transplant may have it in place longer. Others may have IABP therapy discontinued intraoperatively if it was used to stabilize the patient's condition in the preoperative period. Unless complications occur, the IABP is removed after a period of weaning. The ICU nurse, while monitoring the patient on an ongoing basis, is often the first to assess readiness for weaning (Krau, 1999). Although the orders for weaning will be instituted by a physician or mid-level provider, the ICU nurse is responsible for determining the patient's tolerance to the weaning process. Some of the parameters that may suggest readiness for weaning are listed in Box 10–3.

Weaning from the IABP involves decreasing the frequency of assisted beats, decreasing the volume in the balloon over time, or both. Frequency weaning involves switching from 1:1 (every beat assisted by IABP) to 1:2 (every other beat assisted). Switching from a 1:1 to 1:2 ratio provides the most marked decrease in blood flow to the coronary arteries—more than switching from 1:2 to 1:3 or 1:4, or from 1:4 to 1:8 (Krau, 1999). As a consequence, the patient who is weaned in this manner will require frequent monitoring, especially during the first stage of weaning.

Volume weaning involves gradually reducing the amount of gas in the balloon. Usually,

Box 10–3 Parameters for IABP Weaning

- Stable hemodynamic parameters: Stable on low doses of vasoactive medications; MAP > 65–70 mm Hg; PAOP < 18 mm Hg; cardiac index > 2.0 L/min/m^2; SVR < 2000 dynes/sec/cm^{-5}; urine output > 0.5 mL/kg/hr; SvO_2 60–80%.
- No unstable dysrhythmias: Heart rate should be normal or near normal without dysrhythmias that compromise hemodynamic parameters.
- Low or normal serum lactate levels.
- Normal electrolyte levels.
- Acceptable hemoglobin/hematocrit levels.
- No chest pain/discomfort or dyspnea.
- No mental status changes indicative of poor cerebral perfusion.

MAP = mean arterial pressure; PAOP = pulmonary artery occlusive pressure; SvO_2 = mixed venous oxygen saturation; SVR = systemic vascular resistance (afterload).

Sources: Krau, 1999; Quaal, 2005.

the volume is lowered by 20% with each reduction. Because there is an increased risk of thrombus formation in the balloon folds, volume weaning is not recommended as readily as frequency weaning. Volume weaning, however, may be better tolerated by patients who do not tolerate frequency weaning (Krau, 1999). Weaning is successful when the patient is able to remain hemodynamically stable with IABP therapy off.

Anticoagulation used during IABP therapy should be tapered and ultimately discontinued prior to catheter removal. Frequency of balloon inflation can be set to 1:8 while the heparin effect is allowed to wear off (Trost & Hillis, 2006).

Once the catheter is removed, pressure should be applied to the site for 30 to 45 minutes, followed by application of a sterile pressure bandage for 2 to 4 hours (Quaal, 2005). After placement of the pressure bandage, the patient should be checked for bleeding every 30 minutes for 2 to 4 hours, then every 2 hours for 24 hours. Monitoring should confirm that a hematoma is not developing under the bandage, as hematomas can be a significant source of blood loss. The patient should be instructed to keep the head of the bed at 30 degrees or less, with no flexion of the leg for at least 8 hours following IABP catheter removal (Quaal, 2005).

TROUBLESHOOTING THE IABP

With the IABP, as with any mechanical device, problems may occur that need to be addressed promptly. Often it is best to refer to the manufacturer's troubleshooting guide, as it contains a complete reference of problems likely to be encountered. A few of the most common problems are discussed here.

Low Diastolic Augmentation

Poor augmentation occurs in approximately 39% of unsuccessful IABP attempts (Ferguson et al., 2001). Potential causes of low diastolic augmentation include incorrect balloon timing, dysrhythmias that result in low stroke volume, hypotension or low vascular resistance, balloon leak or rupture, incorrect balloon catheter placement or balloon migration, inappropriate balloon size, and balloon not fully opened.

If the balloon or balloon catheter is found to be faulty, it should be removed as soon as possible (within 30 minutes) to avoid thrombus development on an idle catheter (Trost & Hillis, 2006). Most consoles have an alarm system that warns providers when there is a gas leak or a rapid loss of gas. Slow leaks may be the result of a hole in the balloon or a loose connection in the gas tubing. A rapid loss of gas, in contrast, is usually the result of balloon rupture or a disconnected gas circuit. In this scenario, the gas line should be clamped off immediately. If blood is noted in the balloon catheter or gas circuit, it is recommended that the pump be stopped immediately because the balloon or catheter has a leak (Reid & Cottrell, 2005). Continuing to pump will introduce gas into the bloodstream, producing an air embolus.

Faulty Trigger

On occasion, the ECG trigger may not function properly. Common causes of a faulty trigger include poor electrode placement, low ECG voltage, faulty electrode pads or cables, dysrhythmias, and other equipment's interference with the ECG signal (Tremper, 2006). If the problem cannot be easily rectified, switching to the arterial pressure trigger will be necessary until the problem can be solved.

Autofill Failure

The autofill feature on the IABP maintains the volume of gas within the balloon. Should this feature not function, an autofill alarm will sound. The cause of this problem could be an insufficient amount of gas in the tank or occlusion of the gas outlet. The amount of gas in the tank should be checked and the tank replaced as needed. Also, the provider

should assess for and correct any kinks or leaks in the tubing and ensure that the valve on the tank is in the open position (Tremper, 2006).

In the event of pump failure or if pumping needs to be stopped, the IABP balloon should be manually inflated with a syringe every 5 minutes. The syringe should be filled with a volume of gas that is 10 mL less than balloon capacity so as to prevent thrombus formation (Weil, 2007).

ICU nurses caring for a patient receiving IABP therapy must be knowledgeable about the potential complications that can occur during catheter/balloon insertion and removal and during therapy. They must equally be aware of management strategies and preventive measures to implement to avoid or minimize associated morbidity. Patients receiving IABP therapy need to have continuous monitoring, and the nurse-to-patient ratio is encouraged to be 1:1. Often, these patients are critically ill and have complex problems related to their condition as well as the difficulties experienced by dependence, even for a few days, on highly specialized equipment. Box 10–4 lists nursing interventions required by the patient who is receiving IABP therapy.

MONITORING FOR COMPLICATIONS OF IABP THERAPY

- **Limb Ischemia**. Perform peripheral vascular checks, including checking pulses, capillary refill time, skin color, and temperature of lower extremities. Distal pulses should be checked at least every 4 hours until the patient is discharged. Patients should be instructed to notify the nurse if they note any changes in circulation (Reid & Cottrell, 2005).
- **Bleeding at the Insertion Site**. Check the dressing and under the patient's thigh for bleeding every 2 hours. Check for hematoma development under dressing.
- **Anemia and Thrombocytopenia**. Obtain a daily CBC. Transfuse platelets and red blood cells as indicated.
- **Infection**. Monitor for signs and symptoms of infection: temperature $>$ 101 °F, WBC count greater than 10,000 cells/mm^3, chills, mental status changes. If infection is suspected, send specimens for peripheral blood, urine, and sputum cultures. Culture the IABP port as well. Change the insertion site dressing according to facility policy and examine the skin around site for redness, increased temperature, or purulent drainage. Institute antibiotics promptly as prescribed (Quaal, 2005; Reid & Cottrell, 2005).
- **Catheter Migration**. Monitor the patient's pulses, skin color, and temperature; assess for altered sensation in the left upper extremity every 1–2 hours. Report urine output of less than 0.5 mL/kg/hr, increasing BUN/creatinine, or flank pain. Assess the patient for increased abdominal girth or discomfort with absent bowel sounds. Monitor the level of consciousness and evaluate the patient for unilateral neurological impairment. Obtain a chest radiograph and anticipate repositioning or reinsertion of the catheter if migration is suspected (Quaal, 2005).
- **Aortic Dissection**. This complication occurs in 1% to 4% of IABP insertions. Assess the patient for abdominal, back, intrascapular, or shoulder pain, usually of sudden onset. The pain may be described as "tearing." Other symptoms include increased abdominal girth and absent or unequal peripheral pulses with

Box 10–4 Nursing Interventions for the Patient Receiving IABP Therapy

Pre-insertion Interventions

- Provide as calm an environment as possible because the patient will likely be overwhelmed.
- Provide reassurance that the IABP therapy is temporary.
- Explain the procedure and the steps to help ensure safety (as time permits).
- Allow families to participate in discussions and to express concerns.
- Ascertain that consent is signed and complete if required.
- Obtain a 12-lead ECG; insert a urinary catheter.
- Assist with the insertion of invasive lines such as an arterial line and a pulmonary artery catheter.
- Obtain baseline hemodynamic readings: HR, RR, BP, MAP, PAP, PAOP, CVP, CO or CI, SVR, and urine output.
- Obtain baseline blood work: ABG, mixed venous blood gas, chemistries with BUN/creatinine, CBC with platelets and differential, coagulation profile, and type and crossmatch.
- Perform a peripheral vascular assessment, including checking ankle-brachial index,* skin temperature, presence and strength of pulses, and capillary refill in lower extremities.
- Monitor for the presence of a left radial pulse. Inform the physician if the pulse is lost so that the catheter can be repositioned.

Post-insertion Interventions

- Monitor and record hemodynamic measurements every 15 to 30 minutes until the patient is stable, then hourly and PRN.
- Obtain an ECG and chest radiograph daily and PRN.
- Titrate vasopressors/inotropic agents as required to desired hemodynamic parameters. Hemodynamic stability is essential to maintain optimal perfusion to the limb.
- Maintain IV fluid therapy as ordered to maintain an acceptable preload.
- Assess for pain/discomfort, anxiety, and mental status changes hourly.
- Document IABP settings hourly; include the assisted and unassisted pressures.
- Print and document the arterial waveform tracing every 12 hours and PRN with changes.
- Assess for presence and strength of distal pulses, indices of adequate limb perfusion, and sensorimotor function of both lower extremities every 15 minutes for 1 hour, then 30 minutes for 1 hour, and then hourly or according to unit protocol.
- Assess the ankle-brachial index every 4 hours.
- Monitor for the presence of a left radial pulse. Loss of pulse indicates that the catheter has migrated upward, is occluding the left subclavian artery, and requires repositioning.
- Maintain and titrate the heparin infusion to desired anticoagulation as ordered. Obtain coagulation studies 6 hours after dosage changes or follow the facility protocol.
- Obtain daily blood work: chemistries, CBC with platelets, coagulation profile, ABG, lactate level, and mixed venous blood gas.
- Monitor respiratory status: Assess breath sounds every 4 hours. Maintain oxygen and/or ventilator therapy. Encourage coughing and deep breathing/incentive spirometry every 2 hours. Keep the head of bed at a 30- to 45-degree angle to prevent aspiration. Perform chest physiotherapy when the patient is logrolled.

continues

Box 10–4 Nursing Interventions for the Patient Receiving IABP Therapy (continued)

- Maintain NPO or clear liquids as tolerated. If tolerated, maintain tube feedings via feeding tube. Check residual every 4 hours and notify the physician if it is greater than 200 mL.
- Prevent skin breakdown related to immobility. Maintain the patient on bed rest, with sedation if needed. Encourage the patient not to flex the hip on affected side. Use a leg immobilizer if necessary. If tolerated, logroll the patient every 4 hours; perform meticulous skin care. Provide passive range-of-motion exercises for the lower extremity without a catheter and for upper extremities every 4 hours.

***To check ankle-brachial index:**

- With patient supine and at rest, apply blood pressure cuff around both ankles and arms.
- Inflate blood pressure cuffs above patient's normal systolic blood pressure (SBP).
- Deflate blood pressure cuffs. Obtain blood pressure readings using a Doppler and record SBP measurements from the arms and ankles.
- Divide ankle systolic pressure by the highest arm pressure; this will yield an ABI value for each leg.

An index value of 0.9 to 1.3 is considered normal. Values greater than the normal range indicate the presence of some degree of peripheral vascular disease. Presence of mild, moderate, or severe peripheral vascular disease warrants reevaluation of vein selection for cardiac surgery.

ABG = arterial blood gas; ABI = ankle-brachial index; BP = blood pressure; BUN = blood urea nitrogen; CBC = complete blood count; CI = cardiac index; CO = cardiac output; CVP = central venous pressure; ECG = electrocardiogram; HR = heart rate; MAP = mean arterial pressure; PAOP = pulmonary artery occlusive pressure; PAP = pulmonary artery pressure; RR = respiratory rate; SVR = systemic vascular resistance (afterload).

Sources: Creager & Libby, 2004; Laurent & Shinn, 2005; Little, 2004; Marino & Sutin, 2006; Quaal, 2005; Reid & Cottrell, 2005. Vascular Disease Foundation, 2009.

concomitant decreased blood pressure and urine output. Obtain a CT scan or MRI if dissection is suspected. Treatment consists of prompt surgical repair (Hurwitz & Goodman, 2005; Reid & Cottrell, 2005).

- **Compartment Syndrome**. This complication may occur during therapy or after removal of the catheter. The patient should be assessed for increased girth, tenderness, pain, loss of sensation, pressure, paresthesia, or paralysis of the affected extremity. The leg may also develop pallor when elevated if compartment syndrome is present. Treatment may involve a fasciotomy (Reid & Cottrell, 2005).

SUMMARY

Care of the patient receiving IABP therapy is complex and challenging. Patients require prompt intervention for problems and empathy for their critical illness. For the ICU nurse, additional instruction both in theory and in hands-on experience is required to be able to maintain and troubleshoot the complex IABP apparatus. Management of this device is accomplished while balancing evidence-based care that involves critical thinking and decision making, preventing and detecting complications, and providing emotional support to patients and families who are experiencing one of the most vulnerable times in their lives.

CASE STUDY

M.H. is a 63-year-old male with a history of diabetes, hypertension, and coronary artery disease who presented to the emergency department with crushing substernal chest pain radiating down his left arm, without relief from sublingual nitroglycerin. Based on his ECG findings and serum troponin levels, the diagnosis of acute anterior wall MI was made and the patient was admitted to the ICU.

Although he was initially stable, within hours after his admission, M.H.'s blood pressure began to drop; he also developed bibasilar crackles and low urine output. A pulmonary artery catheter was inserted. Despite appropriate interventions and vasoactive agents, however, the patient's condition continued to deteriorate. It was noted that M.H. was becoming confused at this time. Owing to his instability, he was taken to the cardiac catheterization lab, where an IABP catheter was inserted via the right femoral artery. The patient underwent coronary angiography and rotational atherectomy with intercoronary stent placement for a high-grade lesion in the LAD.

After M.H.'s readmission to the ICU, the initial frequency of balloon inflation and deflation was at a 1:1 ratio. In the next few hours, his condition stabilized, with the patient showing improvements in vital signs, urine output, and mental status.

Over the next 2 days, M.H. was weaned from vasoactive agents; weaning from the IABP also commenced. By day 4, the IABP had been successfully removed. On day 7 of hospitalization, M.H. was transferred out of the ICU to a progressive care unit and then to the general unit; 1 week later, he was discharged from the hospital with frequent follow-up appointments scheduled.

Critical Thinking Questions

1. Identify and discuss M.H.'s risk factors, and explain how these factors led to his current diagnosis of acute MI.
2. During M.H.'s IABP therapy, the diastolic augmentation is noted to be low. Discuss why diastolic augmentation is a critical factor for this patient. Describe the possible reasons for this outcome, and identify the interventions that will be required.
3. M.H. will require support and teaching for his recovery phase. Identify and discuss the major areas for teaching that will be needed, including medications and lifestyle alterations.

Answers to Critical Thinking Questions

1. M.H.'s history of diabetes is a significant risk factor for CAD because hyperglycemia causes vascular damage. Approximately two-thirds of all persons with diabetes will die from complications of heart or vascular disease (Morton, 2005).

   Hypertension confers a three to four times greater risk of developing CAD. Current criteria for the diagnosis of hypertension are a systolic blood pressure greater than 120 mm Hg or a diastolic blood pressure greater than 80 mm Hg. In the setting of hypertension, changes occur to the vessel wall possibly by sheer stress and endothelial dysfunction (Henri & Rugg, 2006).

   M.H.'s history of CAD raises significant suspicion that his symptoms are related to a serious cardiac event. Often such a patient will have stable angina with predictable

pain on exertion (when the heart's oxygen demand rises), which later changes with thrombus formation in the coronary artery to unstable angina or AMI.

2. Diastolic augmentation is an important function of the IABP. For M.H., diastolic augmentation increases blood flow into the coronary arteries during diastole. This increases blood flow to the heart muscle, resulting in relief from ischemia and improved function of the left ventricle. In addition, because preload and afterload are diminished, forward flow (cardiac output) is improved, resulting in improved hemodynamic parameters.

   Reasons for low diastolic augmentation include the following issues:
   - Incorrect timing of balloon inflation and deflation. To correct this problem, readjust the timing according to the arterial waveform.
   - Leak in the balloon catheter. Occasionally balloons can rupture or tear. This problem warrants prompt changing of the balloon catheter.
   - Leak in the gas circuit resulting in poor inflation. Check all connections from the catheter to the gas cylinder and tighten them as necessary.
   - Balloon not unwrapped completely. This issue results in poor or incomplete opening of the balloon. Check the position of the catheter with an x-ray. Attempt to inflate the balloon with an appropriate-sized syringe. If this measure is unsuccessful, the catheter will need to be replaced.
   - Poor cardiac function or low vascular resistance. If appropriate, add positive inotropic agents and vasoactive agents.
   - Dysrhythmias such as atrial fibrillation or ventricular tachycardia that result in low stroke volume. To correct these conditions, administer antiarrhythmics as ordered, and improve oxygen and electrolyte imbalances.
3. Some of the major areas for teaching may include the following topics:
   - Control hyperglycemia and blood pressure through diet and exercise. A diet that is low in cholesterol and saturated fats with an increase in fiber is recommended. M.H. should also continue to follow the dietetic recommendations and carbohydrate limitations of his diabetic diet.
   - Attend smoking cessation programs (if appropriate).
   - Use stress reduction techniques.
   - Engage in weight reduction (if appropriate) through diet and exercise. An exercise program that is physician guided and increases over time as the patient tolerates more activity is the most appropriate strategy.

   Medications for M.H. that will require teaching as to mechanisms of action, side effects, and contraindications may include these agents:
   - Lipid-lowering medications to reduce cholesterol levels.
   - Nitrates to relieve symptoms of chest pain and lower blood pressure.
   - Antiplatelet medications to reduce the inflammatory response involved in coronary artery thrombus formation.
   - Beta-adrenergic blocking agents. These agents block sympathetic stimulation by epinephrine and norepinephrine on beta receptors. They lower heart rate, contractility, and blood pressure, which in turn decrease myocardial oxygen demand. In addition, they exert an antiarrhythmic effect.
   - Angiotensin-converting enzyme (ACE) inhibitors to lower blood pressure and decrease the work of the heart.

- Calcium channel blockers to cause coronary artery and systemic vasodilation, decrease contractility of the heart, blood pressure, heart rate, and conduction through the AV node. All of these effects can decrease myocardial oxygen demand. These agents also dilate coronary arteries, which helps to increase myocardial oxygen supply and have a negative inotropic effect on the heart muscle.

M.H.'s patient education should include recognition of warning signs and symptoms for progressing angina and symptoms of LV dysfunction that may occur after an MI. Early treatment of these symptoms may result in improved survival and quality of life.

SELF-ASSESSMENT QUESTIONS

1. Positive effects of IABP therapy include which of the following?
   i. Decreased afterload
   ii. Decreased preload
   iii. Decreased stroke volume
   iv. Increased coronary artery perfusion
   a. iv only
   b. i and ii
   c. i, ii, and iv
   d. iii and iv
2. If the ECG is not sufficient as a trigger, which of the following should be used as a trigger?
   a. T-P interval
   b. Pacer spikes
   c. T wave
   d. Arterial waveform
3. The most common complication that results from the use of IABP therapy is
   a. lower limb ischemia.
   b. aortic dissection.
   c. bleeding.
   d. infection.
4. The urine output of a patient receiving IABP therapy has suddenly dropped to less than 0.5 mL/kg/hr over the past 2 hours. A complication of IABP therapy that should be suspected is
   a. infection.
   b. anemia.
   c. catheter migration.
   d. balloon rupture.
5. If the balloon's inflation/deflation cycle must be stopped temporarily, which action must be taken by the nurse?
   a. Administer a heparin bolus.
   b. Flush the catheter with saline every 2 hours.
   c. Inflate the balloon manually every 5 minutes with a syringe.
   d. Remove the balloon catheter.
6. Indications for IABP use include all of the following *except*
   a. postoperative cardiopulmonary bypass.
   b. cardiogenic shock after acute myocardial infarction.
   c. mitral regurgitation with papillary muscle dysfunction.
   d. uncomplicated myocardial infarction.
7. Which of the following is considered an absolute contraindication to IABP therapy?
   a. Heart failure
   b. Aortic insufficiency
   c. Peripheral vascular disease
   d. Myocardial infarction
8. The effectiveness of the timing of balloon inflation and deflation can be assessed by
   a. arterial pressure waveform.
   b. daily lactate level.
   c. urine output.
   d. daily ECG.

9. After removal of the catheter, the nurse should expect all of the following *except*
   a. explaining to the patient mobility limitations (not to flex affected leg) for the next 8 hours.
   b. holding pressure on the site of catheter removal for 30 to 45 minutes.
   c. checking the site every 2 hours only if blood is noted on the dressing.
   d. continuing to check the site every 30 minutes for 1 hour, then every 2 hours for the next 24 hours.
10. If the diastolic augmentation is low during IABP therapy, which of the following observations will be made?
   a. The augmented diastolic wave will be lower than the previous systolic and coronary artery perfusion will be increased.
   b. The augmented diastolic wave will be higher than the previous systolic and coronary artery perfusion will be decreased.
   c. The augmented diastolic wave will be lower than the previous systolic and coronary artery perfusion is decreased.
   d. The augmented diastolic wave will be higher than the previous systolic and coronary artery perfusion will be increased.

Answers to Self-Assessment Questions

1. c
2. d
3. a
4. c
5. c
6. d
7. b
8. a
9. c
10. c

Clinical Inquiry Box

Question: Does the use of IABP prior to cardiac surgery ensure better outcomes?

Reference: Dyub, A. M., Whitlock, R. P., Abouzahr, L. L., & Cinà, C. S. (2008). Preoperative intra-aortic balloon pump in patients undergoing coronary bypass surgery: A systematic review and meta-analysis. *Journal of Cardiac Surgery, 23*(1), 79–86.

Objective: To assess the effectiveness of preoperative IABP placement in high-risk patients undergoing coronary bypass surgery as evidenced by hospital mortality and IABP-related complications (bleeding, leg ischemia, aortic dissection).

Methods: A meta-analysis was conducted using a random effects model. Studies were chosen that met the following criteria: randomized controlled trials (RCT) and cohort studies with controls; adults (older than 18 years of age) undergoing elective or urgent coronary bypass surgery; and documentation of at least hospital mortality. The treatment was defined as insertion of IABP before surgery. The control groups consisted of patients who did not receive IABP therapy preoperatively and those who received IABP therapy either intraoperatively or postoperatively. The outcome variables for the analysis were hospital mortality and IABP-related complications (e.g., bleeding, leg ischemia, aortic dissection).

Results: Ten publications fulfilled the eligibility criteria (four were RCTs and six were cohort studies with controls). Both statistical and clinical heterogeneity were noted among the included studies. A total of 1034 patients received preoperative IABP; 1329 did not receive preoperative IABP. The overall risk for hospital mortality in patients treated with preoperative IABP was 41%. Broken down by type of study, the risk of hospital mortality was 18% in RCT and 54% in cohort studies (studies following patients over a period of time). Overall, 3.7% (13 of 349) of patients who received preoperative IABP developed either limb ischemia or hematoma at the IABP insertion site, and most of these complications improved after discontinuation of IABP therapy.

Conclusion: Evidence from this meta-analysis supports the use of preoperative IABP in high-risk patients to reduce hospital mortality. Implications for nurses are that preoperative IABP therapy poses an increased risk of mortality and hence these individuals will more than likely present with higher acuity.

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WEB RESOURCES

Intra-aortic balloon pump transport: This short video demonstrates the proper procedure to safely load and transport a patient who has IABP therapy in place. http://video.google.com/videoplay?docid=7160026936252015006&q=IABP&ei=widCSOiZMoO8rwKAsYSOCQ&hl=en

Avoid Hazards of IABP (FDA): http://www.fda.gov/cdrh/medicaldevicesafety/tipsarticles/balloonpump.html

Hazard video: http://video.google.com/videosearch?hl=en&q=IABP&um=1&ie=UTF8&sa=N&tab=wvhl=en&sitesearch=&q=intraaortic%20balloon%20pump

IABP information: http://www.cprworks.com/IABP.html

Chapter 11

Mechanical Ventilation After Cardiac Surgery

Mary Jane Bowles

INTRODUCTION

Mechanical ventilation may be essential in the postoperative management of patients undergoing cardiac surgery and cardiopulmonary bypass. Prior to the 1990s, patients were mechanically ventilated until the morning after surgery before weaning was attempted. In more recent decades, the need for cost containment has resulted in "fast-tracking" patients by implementing early weaning protocols and reversible sedation. This strategy has been reported to lead to shorter mechanical ventilation times and ICU stays for cardiac surgery patients. Other reported benefits of earlier extubation include improved preload, decreased hemodynamic compromise, and decreased neurologic compromise (in elderly patients) (Kern et al., 2001).

The majority of patients are extubated within 24 hours following coronary artery bypass grafting (CABG) procedures. However, prolonged mechanical ventilation may occur in as many as 50% of cardiac surgery patients (Yende & Wunderink, 2002).

Nurses in the ICU who are caring for patients who underwent cardiac surgery in the immediate postoperative phase must have an understanding of the pathophysiology of the lungs, use of mechanical ventilation, weaning protocols, and ability to interpret the clinical significance of diagnostic tests such as arterial blood gases and radiographic findings. They must be able to identify complications of patients on mechanical ventilation and implement measures to prevent morbidities associated with therapy so optimal patient outcomes can be attained.

PREDICTORS OF PROLONGED MECHANICAL VENTILATION

Several preoperative and postoperative factors have been identified as predictors of the need for prolonged mechanical ventilation following cardiac surgery.

Preoperative Predictors

Preoperative predictors of prolonged mechanical ventilation include the presence of valvular disease, recent myocardial infarction, arterial hypertension, diabetes, previous cardiac surgery, chronic peripheral vascular disease, involvement of three or more vessels, surgical priority, elevated serum creatinine, age (greater than 75 years), gender (females have a higher risk than males), impaired left ventricular function (ejection fraction less than 40%), and COPD (Doering, Imperial-Perez, Monsein, & Esmailian, 1998; Hammermeister, Burchfield, Johnson, & Grover, 1990; Kern et al., 2001; Natarajan, Patil, Lesley, & Ninan, 2006;

Pappalardo et al., 2004; Thompson et al., 1997; Tu, Jaglal, & Naylor, 1995; Wong et al., 1999).

Postoperative Predictors

Postoperative predictors include hypoxia, decreased mentation, excessive postoperative bleeding, renal or cardiovascular failure, need for intra-aortic balloon pump (IABP), parenteral nutrition, inotropic therapy, acute respiratory distress syndrome (ARDS), pulmonary edema, and prolonged surgical or bypass time (Guller et al., 2004; Kern et al., 2001; Thompson et al., 1997; Wong et al., 1999).

Other researchers have reported CABG patients (aged 65 years or older) who required 6 to 12 hours of postoperative mechanical ventilation have higher mortality in comparison to those patients requiring less than 6 hours of mechanical ventilation (Peterson et al., 1999). Other predictors for prolonged mechanical ventilation include duration of surgery, perioperative heart failure, serum glucose level, postoperative transfusion, and PaO_2/FiO_2 ratio (Suematsu et al., 2000).

Delays Related to Bypass Complications

Several clinical conditions, when present, are likely to result in failure of ventilator weaning. When these conditions are compounded with complications associated with cardiopulmonary bypass, ventilation time may be prolonged due to a decrease in surfactant production, potential for pulmonary microemboli, and interstitial fluid accumulation in the lungs. Further, red blood cell damage in the pump circuit may potentially occur, decreasing the number of oxygen-carrying capacity cells (Alsoufi, 2006; Khalpey, Ganim, & Rawn, 2008).

COMPLICATIONS OF PROLONGED VENTILATION

Prolonged use of mechanical ventilation is not without risk. In one study, patients who required prolonged ventilation (more than 72 hours) experienced sepsis, endocarditis, gastrointestinal bleeding, stroke, renal failure, or deep sternal wound infection (Durham & Gold, 2008). A higher mortality rate is also associated with patients who require prolonged ventilation.

PATHOPHYSIOLOGY OF THE LUNGS

The main function of the respiratory system is to transport air into the lungs so that oxygen can enter the body and carbon dioxide can be eliminated. Air enters the nose or mouth and moves through the trachea into the bronchi and then into each lung. Once air is in the lungs, gas exchange occurs in the approximately 300 million alveoli. Oxygen and carbon dioxide can cross between the lung capillaries and the alveolar spaces, allowing gas exchange to occur. The nasal passages and bronchi warm and moisten the air before it enters the alveoli as a method of preventing damage to delicate alveolar structures.

The mechanism of breathing involves the diaphragm and the intercostal muscles. During normal breathing, inspiration is an active process and expiration is a passive process. Inspiration involves contraction of the diaphragm and the intercostal muscles to allow for the movement of air into the respiratory tract. The diaphragm and intercostal muscles then relax during expiration.

The respiratory center, which is located in the medulla oblongata (the lower part of the brain stem), receives neural, chemical, and hormonal signals that can control the rate and depth of movements of the diaphragm and other respiratory muscles. An increase in carbon dioxide or a decrease in oxygenation, for example, will increase the rate or depth of breathing. Injury, medications, and disease processes can affect the respiratory center's ability to respond to changes in carbon dioxide or oxygen, resulting in respiratory com-

promise. The use of mechanical ventilation may be needed in these circumstances.

The autonomic nervous system is also involved in breathing. The parasympathetic nervous system may stimulate bronchoconstriction, whereas stimulation of the sympathetic nervous system may cause bronchodilation.

ASSESSMENT OF READINESS FOR WEANING

While many postoperative cardiac surgery patients are extubated prior to their admission to the ICU and many others remain on mechanical ventilation for only a few hours after surgery, all intubated patients should be evaluated for their readiness for weaning. Despite the relatively short amount of time during which patients are intubated postoperatively, it has been reported that as many as 20% of patients experience difficulty with weaning. A number of criteria have been suggested for the ICU nurse to use to determine patient readiness, including the patient's general physiologic and hemodynamic stability, pulmonary mechanics, adequacy of gas exchange, ability to breathe spontaneously, and mental status (Hanneman, 2004).

General Physiologic and Hemodynamic Stability

The patient's overall condition should be assessed, as a number of conditions may potentially influence the success of weaning from mechanical ventilation. Presence of excessive bleeding or an electrolyte imbalance may affect the patient's ability to oxygenate or eliminate carbon dioxide (Hanneman, 2004). Similarly, if the patient is not hemodynamically stable—a common finding in the postoperative cardiac surgery patient—success with weaning may be impaired. The ICU nurse should assess vital signs and hemodynamic parameters, and evaluate the patient for presence of dysrhythmias, tachycardia, bradycardia, weak peripheral pulses, and signs of heart failure (e.g., increase in pulmonary artery occlusive pressure or decrease in cardiac output or mixed venous saturation) (Hanneman, 2004).

A number of clinical conditions have been found to influence the ability to wean by affecting either the capacity of or the demand on the respiratory system. In addition to hemodynamic instability and electrolyte imbalances, the presence of an acid–base imbalance, volume overload, alterations in mental status, myocardial ischemia, new-onset dysrhythmia, or need for vasopressors may delay weaning from mechanical ventilation (Eskandar & Apostolakos, 2007).

Electrolyte imbalances can decrease muscle contractility and, therefore, may influence success with weaning. Specifically, phosphorus, calcium, magnesium, and potassium deficits should be corrected prior to attempting to wean the patient from mechanical ventilation (Eskandar & Apostolakos, 2007).

A patient's mental status should be adequate enough to allow for maintenance of a patent airway and ability to cooperate with coughing and deep breathing to prevent postextubation respiratory compromise and complications (Eskandar & Apostolakos, 2007).

Pulmonary Mechanics

Evaluation of certain parameters is suggested to evaluate patient readiness to wean—namely, vital capacity, minute ventilation (or volume), respiratory rate, tidal volume, and negative inspiratory pressure (or force) (Hanneman, 2004; Soo Hoo & Park, 2002).

- Vital capacity is the amount of air that can be exhaled forcibly following a full inspiration (Steltner et al., 2004).
- Minute ventilation is the volume of gas exchange (inhaled and exhaled) in 1 minute. It is measured by multiplying respiratory rate and tidal volume (Seymour, Halpern, Christie, Gallop, & Fuchs, 2008).

- Respiratory rate is the number of breaths taken by a patient in a minute.
- Tidal volume refers to the amount of air inhaled by the patient during a normal breath (versus a forced inhalation.) If tidal volume is too low, it is surmised that the patient will develop atelectasis post-extubation.
- Negative inspiratory pressure refers to the amount of negative pressure that the patient generates during a forced inspiration when working against an obstruction to flow (Soo Hoo & Park, 2002). It is a reflection of the patient's ability to take a deep breath and generate a cough that is strong enough to clear secretions.

The ICU nurse should be mindful that these physiologic weaning parameters are not perfect predictors of a patient's success with successful extubation. Rather, when assessed in combination with the other criteria discussed in this section, these data will provide some insight into the patient's condition and possible tolerance to breathing without mechanical support (Soo Hoo & Park, 2002).

Respiratory Physiologic Issues

Infrequently, a postoperative patient may require prolonged ventilator support for more than several days. Failure to wean has two primary causes: failure of gas exchange at the alveolar level and failure to ventilate adequately.

Atelectasis

One of the most common reasons for deficiency in gas exchange in the postoperative cardiac surgery patient is atelectasis. Atelectasis affects as many as 70% of cardiac surgery patients. It usually results from single-lung ventilation and associated intraoperative intentional lung collapse (Sladden & Berkowitz, 1993). Pain from median sternotomy or thoracotomy incisions inhibits deep breathing efforts, which can result in atelectasis and pneumonia (Silvestry, 2008). Aggressive pulmonary toileting and pain management are needed post-extubation to prevent further respiratory compromise. Atelectasis is discussed in more detail in Chapter 13.

Left Ventricular Failure

Persistent left ventricular failure after cardiac surgery causes an increase in hydrostatic pressure, with resultant fluid extravasation into alveoli. Interstitial fluid in the alveoli inhibits oxygen transfer, increases shunting, decreases compliance, increases secretions, and facilitates atelectasis that can progress to pneumonia (Salenger, Gammie, & Vander Salm, 2003).

Pleural Effusion

Postoperative cardiac surgery patients may also develop a pleural effusion, usually on the left side. Although the specific etiology of this condition is not known, contributing factors are thought to include volume overload, hypoalbuminemia, inflammation of the pericardium and pleura (postpericardiotomy syndrome), atelectasis, pneumonia, and pulmonary embolism (Khalpey et al., 2008). Development of a pleural effusion may lead to hypoxia, thereby affecting the success of weaning from mechanical ventilation.

A small pleural effusion is common in the early postoperative course following CABG procedures. It occurs with less frequency in patients who have undergone mitral or aortic valve replacement surgery, and typically occurs more commonly on the left side than on the right side. Effusions may necessitate thoracentesis or occasionally placement of a chest tube. Pleural effusions can present with different symptoms depending on the size of the effusion. Typically, the ICU nurse can expect to percuss dullness or decreased resonance and to auscultate diminished or inaudible breath sounds or a pleural friction rub. Pleural effusions rarely result in an

increased mortality rate or increased lengths of stay (Heffner, 2008). This complication is discussed in more detail in Chapter 13.

Phrenic Nerve Injury

Another potential cause of weaning failure is phrenic nerve injury. During cardiac surgery, cold preservation techniques for myocardial protection are often utilized—one study reported the incidence of phrenic nerve injury ranges from 10% to 85% of cardiac surgery patients (Dimopoulou et al., 1998). The use of ice slush has been associated with phrenic nerve injury (Dimopoulou et al., 1998; McGarvey, Cheung, & Stecker, 2006) and, therefore, has been suggested as a measure that should be avoided (Dimopoulou et al., 1998).

Other factors have also been implicated in the development of phrenic nerve injury. They include use of the left internal mammary artery (Abd, Braun, Baskin, O'Sullivan, & Alkaitis, 1989; DeVita, Robinson, Rehder, Hattler, & Cohen, 1993), preexisting diabetes (Efthimiou, Butler, Benson, & Westaby, 1991), low preoperative cardiac performance (Abd et al., 1989), and surgical technique (Benjamin, Cascade, Rubenfire, Wajszczuk, & Kerin, 1982). Data are not consistent, however, regarding these factors' implications for phrenic nerve injury (Dimopoulou et al., 1998).

Phrenic nerve injury may be associated with either unilateral or bilateral paralysis. Most cases have unilateral involvement, with the graft side more likely to be affected (Abd et al., 1989). Because the phrenic nerve is responsible for diaphragmatic contraction, when partial injury of one or both phrenic nerves occurs, lower lobe atelectasis—particularly on the left side—may result in delay of weaning or decreased ability to clear secretions after extubation. In one study, the researchers did not find difference in duration of mechanical ventilation time or hospital lengths of stay between patients with unilateral and bilateral phrenic nerve injury (Dimopoulou et al., 1998).

Arterial Blood Gas

Another method of evaluating the effectiveness of breathing and determining readiness for weaning from mechanical ventilation following cardiac surgery is by obtaining an arterial blood gas (ABG). An ABG provides data with which to evaluate the patient's condition and the need for potential intervention; specifically, it includes pH, PaO_2, SaO_2, $PaCO_2$, and HCO_3 levels. Accurate interpretation will assist in determining the patient's acid–base balance and any required interventions. Table 11–1 lists normal values for an ABG.

Acid–Base Disorders

The pH is a measurement of acidity and alkalinity of the blood. If a patient has an acidic pH, a decrease in myocardial contractility, vascular response to catecholamines, and response to effects and actions of certain

Table 11–1 Components and Normal Values of Arterial Blood Gas

ABG Component	Normal Value
pH	7.35–7.45
PaO_2	80–100 mm Hg
$PaCO_2$	35–45 mm Hg
HCO_3	22–26 mEq/L
SaO_2	94–100%
Base excess	–2 to +2 (A negative base excess indicates a base deficit in the blood.)

medications may result. An alkalotic pH may result in interference with tissue oxygenation, normal neurological functioning, and normal muscular functioning.

RESPIRATORY ACIDOSIS

The definition of respiratory acidosis is a pH of less than 7.35 with a $PaCO_2$ greater than 45 mm Hg. It is important to treat a respiratory acidosis, because its presence increases the minute ventilation required to normalize pH (Eskandar & Apostolakos, 2007). Table 11–2 lists common causes of respiratory acidosis in the postoperative cardiac surgery patient.

Signs and symptoms of respiratory acidosis are respiratory, neurological, and cardiovascular in nature. Respiratory symptoms may include dyspnea, respiratory distress, and shallow respirations. Headache, restlessness, combativeness, hallucinations, and confusion are neurological symptoms. If CO_2 levels continue to increase, symptoms can progress to stuporousness, constricted pupils, drowsiness, seizures, and coma. Cyanosis may be present if the acidosis is accompanied by hypoxemia (Adrogué & Madias, 1998a; Hayes, 2005).

Cardiac response to acidosis includes flushed warm skin, bounding pulses, diaphoresis, tachycardia, dysrhythmias, cen-

Table 11–2 Causes of Respiratory Acidosis

Impaired Respiratory Muscle Function Related to:

- Neuromuscular blocking agents

Pulmonary Disorders

- Atelectasis
- Pneumonia
- Pneumothorax
- Pulmonary edema
- Pulmonary embolism

Increased CO_2 Production

- Shivering
- Sepsis

Hypoventilation Secondary to:

- Pain
- Sternal incision
- Residual anesthesia
- Awakening with inadequate analgesia and impaired respiratory mechanics
- Opioid side effects

User Error

- Inappropriate ventilator settings
- Hypoventilation during transfer from the operating room

Sources: Chikwe, Beddow, & Glenville, 2006; Gerhardt, 2007; Gothard, Kelleher, & Haxby, 2003.

tral and peripheral cyanosis, and pulmonary hypertension. In severe cases, decreased cardiac output, hypotension, peripheral edema, dysrhythmias, and prerenal azotemia may develop (Adrogué & Madias, 1998a). Treatment for respiratory acidosis entails treating the underlying cause of hypoventilation and increasing ventilation.

RESPIRATORY ALKALOSIS

Respiratory alkalosis is defined as a pH greater than 7.45 with a $PaCO_2$ less than 35 mm Hg. Conditions that cause hyperventilation can result in respiratory alkalosis. Table 11–3 lists common causes of respiratory alkalosis in the postoperative cardiac surgery patient.

Respiratory alkalosis is associated with both nervous and cardiac system sequelae. Lightheadedness, dizziness, agitation, numbness or tingling of the extremities, laryngospasm, confusion, and blurred vision are common neurologic symptoms. Cardiac symptoms may include chest pain, ischemic changes on ECG, peripheral vasoconstriction, dysrhythmias, and palpitations. The patient often experiences dry mouth, diaphoresis, muscle twitching, weakness, and tetanic spasms of the arms and legs; some patients may also develop seizures (Adrogué & Madias, 1998b; Edgren, 2008).

Treatment of respiratory alkalosis focuses on eradicating the underlying cause. The patient must be monitored for respiratory muscle fatigue and acute respiratory failure. If these situations occur, temporary reinstitution of mechanical ventilation may be indicated.

METABOLIC ACIDOSIS

Metabolic acidosis is defined as a bicarbonate level less than 22 mEq/L and a pH less

Table 11–3 Causes of Respiratory Alkalosis

Hypoventilation Secondary to:

- Anxiety or fear
- Pain or generalized discomfort

Increased Oxygen Demand

- Fever
- Sepsis

Pulmonary Disorders

- Pneumonia
- Pulmonary edema

Medications

- Respiratory stimulants

User Error

- Inappropriate ventilator settings
- Hyperventilation during transfer from OR

Sources: Chikwe, Beddow, & Glenville, 2006; Gerhardt, 2007; Gothard, Kelleher, & Haxby, 2003; Rimailho, Goldstein, & Vincent, 1985.

Table 11–4 Causes of Metabolic Acidosis

Hemodynamics

- Decreased cardiac output
- Inadequate systemic perfusion
- Decreased cardiac function
- Decreased peripheral perfusion
- Hypotension
- Hypovolemia
- Vasoconstriction from hypothermia

Physiologic Conditions (increasing acids)

- Sepsis
- Renal failure
- Regional ischemia
- Diabetic ketoacidosis
- Anaerobic metabolism

Sources: Chikwe, Beddow, & Glenville, 2006; Gerhardt, 2007; Gothard, Kelleher, & Haxby, 2003.

than 7.35. Table 11–4 lists possible causes of metabolic acidosis in the postoperative cardiac surgery patient.

Metabolic acidosis symptoms arise in relation to the neurologic, cardiovascular, gastrointestinal, and respiratory systems. Headache, confusion, restlessness progressing to lethargy, stupor, and coma are possible. Kussmaul respirations occur when the body attempts to maintain a normal pH by blowing off CO_2. Cardiac dysrhythmias; decreased cardiac contractility and cardiac output; hypotension; warm, flushed skin; nausea and vomiting; insulin resistance; and hyperkalemia may also be observed (Adrogué & Madias, 1998a).

The ICU nurse should attempt to identify the underlying cause of the metabolic acidosis. Hypoxia of any tissues will produce metabolic acids from anaerobic metabolism even if the PaO_2 is normal. The only way to treat acidosis is to restore tissue perfusion, thereby preventing further hypoxemia and hypoxia from developing. If renal failure is the etiology of the metabolic acidosis, the ICU nurse should attempt to attain and maintain normovolemia, administer diuretics based on the patient's hemodynamic profile, and possibly support the patient during dialysis or hemofiltration (Gothard et al., 2003). Treatment may also entail administration of sodium bicarbonate (depending on the severity of the acidosis or pH level), treatment of the patient's hypothermia, and initiation of measures to optimize the patient's hemodynamic profile (Gerhardt, 2007).

Table 11–5 Causes of Metabolic Alkalosis

Loss of Acids

- Nasogastric suctioning
- Excessive administration of diuretics
- Hypochloremia

Hypokalemia

Massive Transfusion (from citrate)

Sources: Chikwe, Beddow, & Glenville, 2006; Gothard, Kelleher, & Haxby, 2003.

METABOLIC ALKALOSIS

Metabolic alkalosis is defined as a bicarbonate level greater than 26 mEq/L with a pH greater than 7.45. Table 11–5 lists conditions that may cause a metabolic alkalosis in the postoperative cardiac surgery patient.

Metabolic alkalosis symptoms are primarily associated with the neurologic and musculoskeletal systems. Dizziness, headache, lethargy, stupor, disorientation, delirium, seizures, and coma may be expected. Musculoskeletal symptoms may include weakness, muscle cramps, muscle twitching, and tetany. The patient can progress to respiratory depression—evidenced as hypoventilation and hypoxemia—and may develop supraventricu-

lar or ventricular dysrhythmias. Electrolyte imbalances associated with metabolic alkalosis include hypokalemia, hypocalcemia, hypomagnesemia, and hypophosphatemia. Of note, patients with a mild to moderate metabolic alkalosis have few or no symptoms. However, if the bicarbonate level is severe (greater than 40 mEq/L), symptoms will likely develop (Adrogué & Madias, 1998b).

Treatment of metabolic alkalosis can be difficult. Acetazolamide (Diamox®) is commonly given after cardiac surgery when excess diuretics have been administered. It may take hours to days to resolve the alkalosis. Acetazolamide blocks the action of carbonic anhydrase, thereby promoting renal excretion of sodium, potassium, phosphorus, bicarbonate, and water. Renal excretion of potassium and phosphorus may be excessive with acetazolamide therapy, however. In severe cases, IV administration of hydrochloric acid may be necessary. The ICU nurse should be aware of the fluid load associated with this therapy (Adrogué & Madias, 1998b). If an electrolyte disturbance occurs in conjunction with therapy for metabolic alkalosis, repletion according to facility protocol is indicated (Gothard et al., 2003).

Intrapulmonary Shunt

Intrapulmonary shunt (IPS) is the percentage of cardiac output that does not participate in gas exchange. This blood passes through the lungs but is not exposed to ventilated alveoli, so gas exchange does not take place; as a consequence, the blood leaves the lungs in a desaturated state. IPS can occur as a result of a number of conditions (e.g., collapsed or fluid-filled alveoli) and is a major cause of hypoxemia in the ICU. A frequent cause of IPS following cardiac surgery is atelectasis (Magnusson & Spahn, 2003). A normal shunt is in the range of approximately 2–5%. Some patients, however, may have a shunt as high as 40% or 50% (e.g., patients with acute respiratory distress syndrome). Because the desaturated blood has not been exposed to ventilated alveoli, increasing oxygen delivery will not correct the resultant hypoxia. Instead, correction of the underlying pathology is necessary to resolve this condition.

A-a Gradient

In addition to assessing acid–base balance, another assessment criterion that may be used to determine patient readiness to wean from mechanical ventilation is calculation of the Alveolar-arterial oxygen gradient (A-a gradient), a method of measuring IPS. This calculation determines the difference between the percentage of alveolar oxygen entering the alveoli and the percentage of oxygen diffusing into the arterial blood. The result of this calculation will aid the clinician in assessing for the presence of dysfunction in oxygenation as well as the degree of IPS (Marini & Wheeler, 2005). The higher the A-a gradient, the more severe the problem with oxygen reaching the blood. If the shunt is too extensive, the patient is not ready for weaning from mechanical ventilation.

Hypoventilation during cardiac surgery results in atelectasis, increasing A-a gradient. As the alveoli reexpand postoperatively, the A-a gradient normalizes ($<$ 300 mm Hg), revealing the patient's readiness for weaning (Markou, Myrianthefs, & Baltopoulos, 2004).

The formula for calculating an A-a gradient is complex. Fortunately, Internet sources offer calculator programs installed to facilitate the process. The ICU nurse would need to insert the local barometric pressure P_B (which is preset at 760 mm Hg), PaO_2 and $PaCO_2$ data from the ABG, and the patient's FiO_2 level. Once the nurse clicks the "Calculate A-a gradient" button, the result appears (GlobalRPh.com, 2008). A-a gradient calculation has been criticized for its complexity, its age-dependent nature, and its relationship with FiO_2 in patients with constant ventilation/perfusion mismatch (Markou et al., 2004).

PaO_2/FiO_2 Ratio

A suggested alternative to the A-a gradient that is easy to calculate and considered a reliable indicator of gas exchange is the PaO_2/FiO_2 (P/F) ratio (Markou et al., 2004). This ratio is an index of oxygenation that is commonly used by clinicians because of its ease in calculation. A PaO_2/FiO_2 ratio of less than 200 is associated with a significant shunt. Criticisms of the PaO_2/FiO_2 ratio include the fact that it is affected by changes in $PaCO_2$ and SvO_2, it is reportedly not equally sensitive across the entire range of FiO_2, and it cannot provide information about the functional status of the lungs based on interventions to augment oxygenation (e.g., positive end-expiratory pressure [PEEP], lateral or prone positioning) (Marini & Wheeler, 2005).

$PaO_2/(FiO_2 \times$ Mean $P_{aw})$

Another oxygenation index, $PaO_2/(FiO_2 \times$ mean P_{aw}, where P_{aw} is mean airway pressure), takes into account the effects of PEEP. It has been widely used in neonatal and pediatric venues, but has not been well adapted for use in the adult population as yet (Hess & Kacmarek, 2002). In a study of cardiac surgery patients, data suggested that $PaO_2/(FiO_2 \times$ mean $P_{aw})$ measurements may be more reliable than other oxygenation measurements in reflecting intrapulmonary shunt (El-Khatib & Jamaleddine, 2004).

POSTOPERATIVE MECHANICAL VENTILATION

Prolonged mechanical ventilation following cardiac surgery is associated with increased ICU and hospital lengths of stay, resource use, costs, and poorer physiologic outcomes (Natarajan et al., 2006). Results of "fast-track" programs have shown that postoperative intubation can be safely limited to 4 to 8 hours (Doering, Esmailian, & Laks, 2000).

If not extubated in the OR, the patient is placed on mechanical ventilation upon arrival to the ICU. The mode of ventilation and settings used will depend on the patient's clinical status. Cardiac surgical patients have multiple risk factors for postoperative respiratory dysfunction. Most patients are resilient and will be weaned from mechanical ventilation within 24 hours; 5% will require prolonged support (Chikwe, Beddow, & Glenville, 2006).

Initial Postoperative Ventilator Settings

For patients who remain on mechanical ventilation in the postoperative period, the settings used should be based on a plan intended to optimize gas exchange, decrease work of breathing, and minimize complications associated with positive-pressure ventilation (Chikwe et al., 2006). Table 11–6 shows the initial ventilator settings.

Patient Monitoring

When the patient is admitted to the ICU, the nurse should auscultate breath sounds to confirm good bilateral air entry and absence of bronchospasm. The postoperative cardiac surgery patient will be monitored with pulse oximetry and potentially with capnometry (end tidal carbon dioxide [$ETCO_2$]) (Khalpey et al., 2008). These noninvasive monitoring devices provide the ICU nurse with continuous estimates of the patient's oxygenation and ventilation status, respectively and will likely expedite the weaning process. For capnography, an infrared gas analyzer is placed in the exhalation port of the ventilator or closest to the endotracheal tube. The normal $ETCO_2$ is 2–6 mm Hg less than the $PaCO_2$. Researchers have found continuous, noninvasive monitoring of SpO_2 and $ETCO_2$ to be a reliable means of weaning patients from mechanical ventilation after cardiac surgery if adjustments are made for the

Table 11–6 Initial Postoperative Ventilator Settings

Mode	SIMV, Assist Control, Pressure Support Ventilation, or Pressure Control
FiO_2	Range = 0.4–1.0. Depends on the patient's ABG results and SpO_2 measurements. It is modified to the lowest level while maintaining SpO_2 levels at least 92% or what is reasonable according to the patient's baseline and past medical history.
Tidal volume	8–12 mL/kg ideal body weight. Tidal volume may be increased to decrease carbon dioxide levels, and vice versa. Tidal volume may also be adjusted to maintain pH within appropriate limits for the patient. It may also be adjusted to maintain peak inspiratory pressure less than 35 cm H_2O.
Rate	8–18 breaths/minute. Respiratory rate may be increased to decrease carbon dioxide levels.
Minute volume	100–120 mL/kg/min. Minute volume may be increased by increasing the rate, the tidal volume, or both to decrease carbon dioxide levels.
PEEP	5–10 cm H_2O. PEEP levels may be increased to improve oxygenation.
Pressure support	5–10 cm H_2O.
Inspiratory:expiratory (I:E) ratio	1:2. If a patient has difficulty with oxygenation, the ratio may be changed to either 1:1 or 2:1 (inverse I:E ratio).
Inspiratory flow rate	30–60 L/min.

ABG = arterial blood gas; PEEP = positive end-expiratory pressure; SIMV = synchronized intermittent mandatory ventilation.

Sources: Chikwe, Beddow, & Glenville, 2006; Herlihy, Koch, Jackson, & Nora, 2008; Khalpey, Ganim, & Rawn, 2008; Lytle & Brown, 2008.

$PaCO_2$–$ETCO_2$ gradient that can occur at high ventilatory rates (Thrush, Mentis, & Downs, 1991).

WEANING CRITERIA

Anesthesia traditionally utilizes short-acting anesthetic agents so that the patient will wake up quickly. The ICU nurse assesses the patient for readiness to wean on an ongoing basis.

Initiating the weaning process commences when the patient is hemodynamically stable, normothermic, and adequately resuscitated; does not have any clinically significant dysrhythmias; is draining less than 100 mL/hour from the chest tube; is not shivering; and is on minimal vasoactive support. The patient must be awake, oriented, able to cooperate with instructions, and triggering the ventilator by taking spontaneous breaths (Heijmans, Maessen, & Roekaerts, 2004).

The patient should also demonstrate adequate muscle strength as demonstrated by either a strong hand grasp or a sustained head lift for 5 seconds (Lytle & Brown, 2008). A chest radiograph should be reviewed prior to extubation to ascertain the presence of any indicators that may indicate the patient might not tolerate extubation. Lab values (e.g., electrolytes, lactate level) should be within normal ranges. The ABG results should be at or close to the patient's baseline or normalized.

The patient should be normothermic before weaning is attempted, as shivering

causes an increase in carbon dioxide production. Shivering following hypothermic cardiopulmonary bypass (CPB) causes a twofold to threefold increase in oxygen consumption and predisposes the patient to develop respiratory and metabolic acidosis (Bhattacharya, Bhattacharya, Jain, & Agarwal, 2003).

WEANING FROM MECHANICAL VENTILATION

Weaning may be accomplished in several different ways, depending on the mode of ventilation and the patient's condition. The first goal is to wean the patient as tolerated while maintaining a $SpO_2 \geq 92$–94% on FiO_2 .40 and PEEP 5 cm H_2O. If the patient is receiving pressure support, as the patient's respiratory effort increases, pressure support levels can be gradually titrated down (Heijmans et al., 2004; Khalpey et al., 2008).

Once physiologic parameters have been met, the amount of support the patient receives from the ventilator is gradually decreased or else the patient undergoes a spontaneous breathing trial with either pressure support or a t-piece. If using pressure support, the patient is placed on 5 cm H_2O with no preset breaths to be delivered. The patient's minute volume and respiratory rate should remain within clinically acceptable limits. The patient will remain on these settings (if tolerated) for 30 minutes, at which point an ABG is obtained.

An alternative to a spontaneous breathing trial for weaning is to gradually and incrementally decrease the amount of support from pressure support ventilation or the synchronized intermittent mandatory ventilation (SIMV) rate. Data suggest that patients who are weaned with spontaneous breathing trials are successfully extubated two to three times earlier than patients who are weaned with either of the alternative methods (Esteban et al., 1995). Further, SIMV has been found to be the least effective method of weaning and no added benefit existed between pressure support and spontaneous breathing trials (Hemant, Chacko, & Singh, 2006).

Regardless of the method used, once the patient has satisfactory ABG results and has demonstrated the ability to breathe independently without signs of distress, extubation can be considered. In addition to assessment of pulmonary mechanics, if the patient is able to maintain a patent airway and manage secretions, the patient is considered ready to be extubated if the criteria in Box 11–1 are met.

The ICU nurse plays a pivotal role in assessing tolerance to weaning. Signs and symptoms that would indicate poor tolerance to weaning include a respiratory rate of 35 or greater; SpO_2 less than 90%; heart rate greater than 140; systolic or diastolic blood pressure higher than 180 or 90 mm Hg, respectively; and presence of agitation, diaphoresis, or anx-

Box 11–1 Readiness for Extubation Criteria

NIP $\geq$ –25 cm H_2O
RR $\leq$ 25 bpm
HR $<$ 140
Minute volume (V_E) $\leq$ 10 L/min
Vital capacity (VC) $\geq$ 10–15 mL/kg

Cardiac status:
- No signs of ischemia
- Not receiving vasopressor therapy or low dose inotropic agents

Neurologic status:
- Alert
- Able to respond to commands
- Cough and gag reflex
- Able to protect airway and clear secretions
- Able to sustain a head lift for at least 5 seconds

NIP = negative inspiratory pressure; RR = respiratory rate.

Sources: Hanneman, 2004; Hemant, Chacko, & Singh, 2006; Khalpey, Ganim, & Rawn, 2008.

iety (Khalpey et al., 2008). Further, if the patient has an inadequate minute volume, tidal volume, episodes of apnea lasting more than 25 seconds, mental status changes, a decrease in SpO_2 to less than 92%, or $ETCO_2$ greater than 55 mm Hg, the trial is stopped, the patient is restored to the prior ventilator settings, and an ABG is obtained. It has been recommended that spontaneous breathing trials be attempted hourly until weaning is successful (Lytle & Brown, 2008).

When ABG results are obtained and are within the appropriate range, collaboration with the physician regarding extubation is indicated. If the ABG results are not acceptable, the patient should be placed back on the previous support settings and reassessed in an hour (Lytle & Brown, 2008).

Researchers have compared intubation times using *SmartCare*™, a knowledge-based system for automated weaning with conventional physician-controlled weaning after off-pump coronary artery bypass. No complications or increase in reintubations occurred with this computer-driven weaning system, and SmartCare reduced the duration of mechanical ventilation (Kataoka et al., 2007). Many factors associated with cardiopulmonary bypass would need to be studied before the use of SmartCare could be routinely recommended in this population, however.

Another study evaluated the Siemens Servo 300A ventilator, which has an automode function allowing for automated weaning from mechanical ventilation. Data suggest that the automode decreased ventilation time by 2 hours, decreased peak airway pressure during spontaneous ventilation, and improved patients' cardiac index (Hendrix, Kaiser, Yusen, & Merk, 2006).

Weaning from Prolonged Ventilation

In long-term weaning, the physiologic index is a reliable predictive indicator of failure to wean and extubation. The physiologic index is determined by assessing the minute frequency of spontaneous ventilation (*f*) and dividing this value by the tidal volume (Vt) in liters. When this index is high, it reflects a clinical picture of a patient with rapid, shallow breathing. When *f*/Vt is less than 105, 78% of patients can be weaned and extubated successfully. When *f*/Vt is greater than 105, 95% of patients cannot be weaned and extubated successfully. A Vt of 325 mL is a good threshold value for predicting weaning success or failure (Yang & Tobin, 1991).

POST-EXTUBATION CARE

Upon extubation, the patient is assessed for a patent airway and absence of laryngeal edema. The ICU nurse should ask the patient to speak a few words. Afterward, the patient should be placed on a humidified face mask set to deliver a FiO_2 10% greater than what was received when the patient was on mechanical ventilation. The FiO_2 level may be titrated down according to SpO_2 values, which should initially be maintained above the range of 97% to 98%. After the initial post-extubation period, FiO_2 can be titrated to maintain SpO_2 at least 95% for the first 2 to 3 days. After that point, a nasal cannula can be used to maintain SpO_2 at least 90% (Salenger et al., 2003).

Extubation failure in postoperative cardiac surgery patients has a reported overall incidence of 5%. The incidence is 14% among patients with COPD; it is 10% for those with a history of stroke. Other identified risk factors of extubation failure include renal failure, IABP requirement, longer surgical time, and longer time on bypass (Khalpey et al., 2008).

In a qualitative study of cardiothoracic patients, four nursing dimensions of care emerged as critical issues during the immediate post-extubation period: clinical physiologic data, communication, early physical activities,

and available resources. Specifically, ICU nurses identified these four aspects of care as their responsibility (De Beer, Nel, & Arries, 2002).

Post-extubation, the ICU nurse should initially observe for laryngospasm for as long as 1 hour and stridor for as long as 24 hours—both conditions may result in the need for reintubation. Prophylactic administration of dexamethasone has been shown to be effective in decreasing the incidence of post-extubation stridor in patients who are at risk for developing laryngeal edema (Lee, Peng, & Wu, 2007).

After cardiac surgery, many patients will have decreased breath sounds secondary to lower lobe atelectasis (Khalpey et al., 2008). For this reason, the critical care nurse must frequently evaluate the patient in terms of work of breathing, respiratory rate, use of accessory muscles, and expiratory phase of breathing. Nursing care must include encouraging mobility, use of incentive spirometry, bronchial hygiene, and frequent auscultation of breath sounds. Chest physiotherapy will promote lung expansion, mobilize secretions, encourage coughing, and prevent the side effect of retained secretions, which might otherwise cause atelectasis and potentially pneumonia (Salenger et al., 2003).

One of the sequelae of bypass procedures is activation of the inflammatory response, which can cause marked pulmonary dysfunction (Khalpey et al., 2008). A variety of interventions are being studied for their potential to mitigate the deleterious effects of bypass procedures that can cause delays in weaning from mechanical ventilation. These interventions include use of a leukocyte filtration to reduce the effects of cardiopulmonary bypass; intraoperative use of heparin-bonded circuits designed to prevent complement activation and subsequent increase in neutrophil activation; and use of antioxidants and anti-inflammatory drugs with the serine protease inhibitor activity of Aprotinin in combination with leukocyte-reduction filters. The last combination has been shown to improve post-bypass lung performance by reducing inflammatory response and its sequelae (Olivencia-Yurvati, Ferrara, Tierney, Wallace, & Mallet, 2003).

SUMMARY

Caring for patients following cardiac surgery is often challenging. While many patients are admitted to the ICU having already been extubated, others require management with mechanical ventilation for either a short or prolonged period of time. Mechanical ventilation is suggested to be associated with—and may even cause—lung damage and many other complications (Pappalardo et al., 2004).

Prolonged use of mechanical ventilation is correlated with increased mortality rate. The mortality rate can be 50% or more in patients who develop acute lung injury or acute respiratory distress syndrome (El-Chemaly, Abreu, & Krieger, 2003). The cardiac surgery ICU nurse must continuously assess the post-cardiac surgery patient for tolerance to therapy, prevent complications associated with mechanical ventilation, minimize the effects of the patient's comorbidities and the procedure-associated complications, and assess the patient's readiness for and tolerance of weaning from mechanical ventilation. Although the majority of patients are quickly weaned from mechanical ventilation and extubated, extubation failure must be minimized or recognized promptly. Using high levels of clinical judgment and caring practices will affect the ICU nurse's ability to optimize outcomes of the postoperative cardiac surgery patient.

CASE STUDY

A 65-year-old, moderately obese female has a 3-year history of coronary artery disease with a stent placed in her right coronary artery 18 months prior to her current admission. Her history includes elevated cholesterol, hypertension, and diet-controlled diabetes. The patient is being treated with 325 mg aspirin daily, atorvastatin (Lipitor®), and an angiotensin-converting enzyme (ACE) inhibitor.

The patient presented with nausea and vomiting as well as "heaviness" in her chest. She had a recent bout with bronchitis and had not been feeling well for 2 days, but felt her condition was related to the bronchitis. Upon her admission, a 12-lead ECG revealed ST-segment elevation in leads II, III, and aVF. The patient reported her chest pain to be 7 out of 10; she was treated with sublingual nitroglycerin, which decreased the pain to 5 out of 10. Initial electrolytes and complete blood count were within normal limits although troponin I was elevated. Cardiac catheterization revealed the following:

- Severe triple vessel coronary artery disease.
- The right coronary artery (RCA) had 90% dominant obstruction.
- The left anterior descending (LAD) coronary artery had 80% obstruction.
- The aortic and mitral valves were normal without significant stenosis or regurgitation.

The patient was scheduled for coronary artery bypass grafting. The left internal mammary artery was used to bypass the LAD, and radial arteries were used to bypass the remaining blockages. The intraoperative course was uneventful, and the patient was admitted to the ICU postoperatively.

Critical Thinking Questions

1. For which post-extubation complications should the ICU nurse assess on this patient?
2. Which pulmonary interventions will be necessary to facilitate respiratory functioning in this patient?
3. What are possible pulmonary complications from cardiac surgery?
4. How would the nurse assess for a positive Hoover sign?
5. How should you assess for air leak syndrome?

Answers to Critical Thinking Questions

1. In the post-extubation patient, the nurse should initially observe for laryngospasm for as long as 1 hour and stridor for as long as 24 hours; either of these complications may result in the need for reintubation. After cardiac surgery, the patient would be expected to have decreased breath sounds secondary to lower lobe atelectasis. The patient will need frequent assessment in terms of work of breathing, respiratory rate, use of accessory muscles, and the expiratory phase of breathing, which can indicate compromised pulmonary function.
2. Nursing care must include mobility and bronchial hygiene, and frequent auscultation of breath sounds. Bronchial hygiene will promote lung expansion, mobilize secretions, and prevent retention of secretions that cause atelectasis and potentially pneumonia. This care should include pulmonary toileting of effective coughing and

incentive spirometry. Incentive spirometry has only limited effectiveness, however, because many patients are unable to cooperate adequately to use it correctly.

3. Potential pulmonary complications include phrenic nerve injury, pulmonary atelectasis, pleural effusions, and air leak syndrome.
4. A positive Hoover sign indicates a pleural effusion. To assess for this condition, the nurse should observe the patient for an asymmetric expansion of the thoracic cage. With a positive Hoover sign, the nurse will note a lagging expansion on the affected side where the pleural effusion is located.
5. Assess the patient for symptoms of a sensation of fullness in the chest, pleuritic chest pain that may radiate to the shoulders, dyspnea, coughing, hoarseness, and dysphagia. Crepitus in the neck due to associated subcutaneous emphysema may be present. Upon auscultation, a crackling sound may be heard over the heart during systole (Hamman sign).

SELF-ASSESSMENT QUESTIONS

1. An initial set of blood gas results are as follows: pH = 7.31; PCO_2 = 50 mm Hg; and HCO_3 = 22 mEq/L. What is the correct interpretation of these results?
   a. Uncompensated respiratory acidosis
   b. Compensated respiratory acidosis
   c. Uncompensated metabolic acidosis
   d. Compensated metabolic acidosis
2. Based on the post-cardiac surgery ABG results given in Question 1, which of the following is the potential cause?
   a. Bleeding
   b. Hypoventilation
   c. Hyperglycemia
   d. Preexisting cardiac history
3. Which of the following criteria would indicate that the patient is not ready for extubation?
   a. Minute volume of 3 L/minute
   b. Vital capacity ≥ 10–15 mL/kg
   c. Negative inspiratory pressure of –20 cm H_2O
   d. Respiratory rate of 24 bpm
4. After 4 hours of mechanical ventilation, your patient is stable on a FiO_2 of 0.30, rate of 4, and pressure support of 5 cm H_2O. Which of the following indicators would deter extubation?
   a. Chest tube draining of 225 mL/hr
   b. Temperature of 37.0 °C
   c. CI of 2.6 L/min/m^2
   d. MAP of 75 mm Hg
5. After 8 hours in the ICU, your patient has received 4 units of PRBCs, 2 units of FFP, and 6 units of platelets. Chest tube output is minimal and the patient has remained hemodynamically stable. The patient is again being evaluated for extubation. She is alert, respiratory rate = 7, and $PaCO_2$ = 75 mm Hg. You should anticipate
   a. a decrease in tidal volume.
   b. an increase in rate.
   c. proceeding with extubation.
   d. administering opioids.
6. After reassessment, the patient has the following characteristics: alert and cooperative, NIP –20 cm H_2O, minute volume 10 L/min, vital capacity 14 L/kg, hemodynamically stable, chest tube draining minimal. Your assessment of the patient is that she is
   a. ready for extubation.
   b. not ready for extubation.

Answers to Self-Assessment Questions

1. a	4. a
2. b	5. b
3. a	6. b

Clinical Inquiry Box

Question: Is there a difference in pulmonary complications following on-pump versus off-pump cardiac surgery?

Reference: Groeneveld, A. B., Jansen, E. K., & Verheij, J. (2007). Mechanism of pulmonary dysfunction after on-pump and off-pump cardiac surgery: A prospective cohort study. *Journal of Cardiothoracic Surgery, 2*(1). http://www.cardiothoracicsurgery.org/content/2/1/11

Objective: To investigate whether there are differences in pulmonary complications between on-pump and off-pump cardiac surgical approaches.

Methods: The study enrolled 31 patients who underwent on-pump surgery and 8 patients who underwent off-pump surgery. Data collected included postoperative pulmonary leak index (PLI), extravascular lung water (EVLW), transfusion history, radiographs, and ventilatory and gas exchange variables.

Results: There was no significant difference between PLI, EVLW, transfusion of red blood cell (RBC) concentrates, occurrence of atelectasis, ventilatory variables, and duration of mechanical ventilation. The PLI was significantly correlated to the number of RBC concentrates infused.

Conclusion: Cardiopulmonary bypass does not cause lung vascular injury. Atelectasis is the major factor contributing to pulmonary dysfunction. Therefore, nurses must be vigilant in their assessment of postoperative patients' pulmonary status and implement measures to prevent or treat this pulmonary complication.

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WEB RESOURCES

Respiratory assessment: http://www.youtube.com/watch?v=IepL5u5lAtE

Mechanical ventilation tutorial: http://www.ccmtutorials.com/rs/mv/

Patient education guides on mechanical ventilation: http://www.chestnet.org/patients/guides/mech_vent/

Ventilator case studies: http://www.ventworld.com/education/casestudies.asp

Chapter 12

Pharmacologic Support Following Cardiac Surgery

Timothy E. McMurry, Roberta Kaplow, and Sonya R. Hardin

INTRODUCTION

As described in Chapters 8 and 13, hemodynamic compromise following cardiac surgery is common and challenging to manage. The etiology of the instability may be the patient's underlying cardiac disease, postoperative filling pressures, dysrhythmias, decreased ventricular compliance, loss of vasomotor tone, increased capillary permeability, excessive bleeding, increased urinary output, inflammatory response to cardiopulmonary bypass (CPB), poor myocardial protection during aortic cross-clamping, pulmonary edema, cardiac tamponade, or ventricular dysfunction. Even though the surgery has been completed, there may not be an immediate improvement in contractility in some patients (Salenger, Gammie, & Vander Salm, 2003).

In the care of the postoperative cardiac surgery patient, the ICU nurse must be aware of the intricate balance between physiological data and the medications utilized to treat and prevent complications. This chapter discusses several medications used in the immediate postoperative setting, including their mechanism of action, therapeutic uses, and side effects. In addition, nurse precautions that are utilized in the delivery of care are described. Many of the medications profiled in this chapter have a number of mechanisms of action and indications. Because of the potential for interaction of some of these medications and their sometimes burdensome side effect profiles, the ICU nurse needs a high level of clinical judgment to help optimize the patient's outcome.

AGENTS USED TO MANAGE POSTOPERATIVE HYPERTENSION

Hypertension may occur in as many as 60% of postoperative cardiac surgery patients (Talmor & Lisbon, 2005). This condition is frequently linked to vasoconstriction (Silvestry, 2008). Development of hypertension, vasoconstriction, or both may be due to decreased oxygen levels or inflammatory responses to CPB (Salenger et al., 2003; Silvestry, 2008). Hypertension leads to increased afterload with resultant metabolic acidosis, increased systemic vascular resistance (SVR), decreased cardiac output (CO), and increased myocardial oxygen consumption (Katz, 2007). As noted in Chapters 8 and 13, potential causes of increased afterload include hypothermia, hypovolemia, hypercarbia, inadequate rewarming, volume overload, cardiogenic shock, pain, and anxiety. The latter two causes arise as a result of increased sympathetic nervous system stimulation (Khalpey, Ganim, & Rawn, 2008; Talmor & Lisbon, 2005). If vasoconstriction is extreme, patients are at risk of

developing life-threatening hypertension and decreased CO (Khalpey et al., 2008). Controlling hypertension is also important after cardiac surgery to reduce bleeding from surgical sites and enhance CO. Refer to Table 12–1 for a summary of medications used to treat hypertension following cardiac surgery.

Vasodilators

Vasodilators are the agents of choice to decrease hypertension in the immediate postoperative cardiac surgery patient (Katz, 2007). Vasodilators are utilized to control hypertension, reduce afterload, and prevent angina pectoris, myocardial infarction (MI), and heart failure, all of which could occur in the postoperative cardiac surgery patient. These agents may also be used in postoperative cardiac surgery patients who have normal blood pressure despite poor pump function (Khalpey et al., 2008). Agents may dilate either the arterial or venous system, or both. The most commonly used vasodilators in this patient population are nitroglycerin (NTG, Tridil®), nitroprusside (Nipride®), nicardipine (Cardene®), and fenoldopam mesylate (Corlopam®).

Care must be taken to correct hypovolemia in hypertensive patients prior to administering a vasodilator. Abrupt, life-threatening hypotension may develop when vasodilators are used and there is an inadequate volume to fill the vasculature. The ICU nurse should always be prepared to administer a rapid fluid bolus when starting any vasodilator, should hypotension occur. As with all vasoactive agents, use of the smallest dose necessary to accomplish the desired effect is recommended. The risk of side effects escalates with higher infusion rates.

Nitroglycerin

HEMODYNAMIC EFFECTS AND INDICATIONS

NTG has many uses in postoperative cardiac surgery patients. It decreases preload and, in higher doses, afterload. Patients with high preload benefit because NTG lowers pulmonary artery pressure (PAP) and central venous pressure (CVP) via its vasodilatory action. NTG also decreases SVR and pulmonary vascular resistance (PVR) (Salenger et al., 2008). Whenever ischemia is suspected postoperatively, NTG may be ordered because of its ability to dilate the coronary arteries and increase coronary blood flow. This agent also decreases pulmonary congestion and myocardial oxygen consumption (Katz, 2007).

In addition to treating hypertension, decreasing preload or afterload, and treating myocardial ischemia, NTG is also used on a short-term basis (24 to 48 hours) to prevent spasm of internal mammary arteries in the postoperative period.

DOSAGE

Infusion rates for NTG may be set as low as 5–10 mcg/min. The rate is titrated in 10-mcg increments until a mean arterial pressure (MAP) goal has been attained. Titration to effect can occur as often as every 5 to 10 minutes owing to the short half-life of NTG. This agent has an immediate onset of action and the drug effects last 30 minutes (Katz, 2007).

SIDE EFFECTS

One potential side effect of NTG is hypoxia—a condition caused by the drug's inhibition of pulmonary arterial vasoconstriction, which in turn increases blood flow through poorly oxygenated lung areas (Katz, 2007; Massé & Antonacci, 2005). Other side effects that are often reported with NTG administration include lightheadedness, headache, hypotension, tachycardia, dizziness, and flushing of the face and neck (Doucet et al., 2000; Silver, 2002). Although rare, methemoglobinemia has been reported as being associated with intravenous administration of NTG (Anderson, Woodside, Spencer, & Hunter, 2004).

Table 12–1 Antihypertensive Agents Used in Postoperative Cardiac Surgery Patients and Hemodynamic Effects

Agent	Dose	Mechanism of Action	Hemodynamic Effects
Vasodilators			
Nitroglycerin (Tridil®)	5–10 mcg/min; titrated in 10-mcg increments every 5 to 10 minutes.	Venous and arterial vasodilation (dose dependent). Increases coronary blood flow, dilates coronary arteries.	Decreases preload and afterload (dose dependent). Decreases PAP, CVP, SVR, PVR, myocardial oxygen consumption.
Nitroprusside (Nipride®)	0.3 mcg/kg/min; titrate every 10 minutes up to 10 mcg/kg/min.	Smooth muscle relaxant; arterial vasodilation. Generates nitric oxide.	Decreases SVR and PVR; increases venous capacitance, decreases coronary vascular resistance.
Nicardipine (Cardene®)	Infusion at 5 mg/hr. Dose may be slowly increased by 2.5 mg/hr to a maximum of 15 mg/hr. Once blood pressure endpoint is reached, a maintenance infusion may be run at 3 mg/hr.	Blocks flow of calcium. Acts directly on arterioles. Also been shown to dilate the coronary vasculature.	Peripheral vascular and coronary vasodilation and lower blood pressure.
Fenoldopam mesylate (Corlopam®)	Initial dose of 0.03–0.1 mcg/kg/min. Titration in increments of 0.05–0.1 mcg/kg/min every 5–15 min, to maximum of 1.6 mcg/kg/min, to achieve desired blood pressure. Doses must be administered as a continuous infusion. Should not be used for more than 48 hrs.	Selective dopamine-1-receptor agonist and moderately binds to alpha$_2$ receptors.	Vasodilator; increases renal blood flow; decreases SVR and PVR and enhances cardiac output.

continues

Table 12–1 Antihypertensive Agents Used in Postoperative Cardiac Surgery Patients and Hemodynamic Effects (continued)

Agent	Dose	Mechanism of Action	Hemodynamic Effects
Beta Blockers			
Esmolol (Brevibloc®)	Loading dose: 500 mcg/kg IV bolus over 1 minute. Maintenance dose: 50 mcg/kg/min over 4 min. If additional dosing is required after 5 min, use same loading dose followed by 100 mcg/kg/min over 4 min. May continue to titrate by increasing the maintenance dose in 50 mcg/kg/min increments until desired endpoint or maintenance dose of 200 mcg/kg/min is reached. When endpoint is reached, loading dose should be eliminated or titration may take place every 10 min.	Cardioselective beta-adrenergic receptor blocker. Inhibits effects of $beta_1$ receptors. Inhibits $beta_2$ receptors at higher doses.	Decreases heart rate, blood pressure, contractility, and cardiac output.
Labetalol (Normodyne®, Trandate®)	10 mg IV over 2 minutes. Additional 10mg to 20 mg doses every 10 min up to a maximum of 300 mg in 24 hours may be given.	Non-cardioselective adrenergic blocking agent. Exerts inhibitory effects on $beta_1$, $beta_2$, and $alpha_1$ receptors.	
ACE Inhibitors			
Enalaprilat (Vasotec®)	0.625 to 1.25 mg, infused over 5 min. Additional doses, up to a maximum of 5 mg every 6 hours, may be administered.	Prevents conversion of angiotensin I to angiotensin II (a potent vasoconstrictor) by inhibiting ACE in the pulmonary and systemic vascular endothelium.	Vasodilation; decreases SVR.

Table 12–1 Antihypertensive Agents Used in Postoperative Cardiac Surgery Patients and Hemodynamic Effects (continued)

Agent	Dose	Mechanism of Action	Hemodynamic Effects
ARBs			
No specific ARB recommendations noted in literature.	Dosage is drug dependent.	Blocks production of angiotensin II from sources of angiotensin II other than the liver (i.e., blood vessels, in the adrenals, and within all other tissues.)	The adrenal-related blockage results in a decrease in aldosterone levels, thereby leading to increased excretion of sodium and water from kidneys.
Calcium Channel Blockers			
Nicardipine	See page 207 under vasodilators.		
Clevidipine (Cleviplex®)	1–2 mg/hr via continuous infusion. Dose may be doubled in 90-second intervals. Once blood pressure begins to approach goal, incremental dosing should be every 5–10 min and be less than double the dose. A maximum initial dose is 16 mg/hr. Total 24-hr dosing should not exceed 21 mg/hr.	Smooth muscle relaxant and arterial vasodilator.	Decreases MAP and SVR.
Selective Dopamine-1-Receptor Agonist			
Fenoldopam Mesylate	See page 207 under vasodilators.		

Sources: Cheung et al., 1999; Katz, 2007; Khalpey et al., 2008; Lemmer, Richenbacher, & Vlahakes, 2003; Levy, Tanaka, & Bailey, 2008; Micromedex Online, 2008; Salenger et al., 2008; Singla et al., 2008.

NURSING IMPLICATIONS

Abrupt discontinuation of NTG can cause coronary vasospasm. For this reason, close monitoring of rhythm, blood pressure, and hemodynamic parameters is warranted when the infusion is stopped. The drug dosage used depends on the desired effect, the patient's blood pressure, and hemodynamics, bearing in mind that increasing coronary blood flow may improve cardiac function. The advantages of NTG are its ease of titration and short half-life.

Nitroprusside

HEMODYNAMIC EFFECTS AND INDICATIONS

Nitroprusside is a smooth muscle relaxant that is used to control hypertension and reduce afterload (SVR and PVR). A powerful arterial vasodilator, it lowers blood pressure by generating nitric oxide. Nitroprusside also increases venous capacitance and decreases coronary vascular resistance (Katz, 2007; Salenger et al., 2008).

DOSAGE

For afterload reduction, initial doses as low as 0.3 mcg/kg/min should be used and slowly titrated (every 10 minutes) up to 10 mcg/kg/min to maintain the blood pressure within specified limits. Nitroprusside has an immediate onset of action (the peak effect occurs in 2 minutes), and its effects dissipate rapidly (within 3 minutes) when the infusion is discontinued (Katz, 2007; Massé & Antonacci, 2005). It rapidly reduces blood pressure and is converted in the body to cyanide and then thiocyanate when administered in doses greater than 10 mcg/kg/min. Its adverse effects can be attributed mainly to excessive hypotension and excessive cyanide accumulation; thiocyanate toxicity may also occur, especially in patients with renal impairment. Typically, this effect occurs more often in patients who receive an infusion over a period of 72 to 96 hours (Massé & Antonacci, 2005).

SIDE EFFECTS

Administration of nitroprusside may produce reflex tachycardia, hypotension, and renal dysfunction. Rarely, patients may develop a decreased platelet count or hypothyroidism (thiocyanate impairs iodine transport). Owing to its dilation of the pulmonary arterioles, nitroprusside can decrease arterial oxygen content and cause—or worsen—any existing ventilation/perfusion mismatch. Methemoglobinemia may also occur, which will decrease the blood's oxygen-carrying capacity (Benowitz, 2004). Cerebral vasodilation with resultant increased intracranial pressure may occur. Nitroprusside may also inhibit platelet function (Massé & Antonacci, 2005).

An excessive amount of cyanide in the plasma (more than 80 ng/mL) following nitroprusside administration—as a consequence of overdosage or depletion of endogenous thiosulfate (which converts cyanide to thiocyanate)—may result in nausea, disorientation, confusion, psychosis, weakness, muscle spasm, or convulsions. These symptoms are related to the effects of thiocyanate toxicity (Habal, 2008). Metabolic acidosis may be the first sign of cyanide toxicity. Thiocyanate levels should be monitored daily (Massé & Antonacci, 2005); excess amounts can be removed with dialysis.

NURSING IMPLICATIONS

Nitroprusside can cause sudden, life-threatening hypotension if its use is not closely monitored. Care should be taken not to flush or initiate new medications in lines that contain nitroprusside, as doing so can result in abrupt hypotension. When nitroprusside is discontinued, the line should be aspirated and then flushed to avoid this possibility.

Like NTG, nitroprusside can cause pulmonary vasodilation with shunting of blood to atelectatic areas of the lung, resulting in lowered oxygen saturation and a need for higher oxygen delivery. This effect is usually

seen immediately and can be dose dependent. If it occurs, another therapy may be chosen. Increasing positive end-expiratory pressure (PEEP) is helpful in resolving atelectasis.

Beta Blockers

Depending on the etiology of hypertension, a beta blocker may be considered part of the pharmacological arsenal to combat this complication. The net effects of beta blockers are a decrease in heart rate, blood pressure, contractility, and CO. Beta blockers are discussed in detail later in this chapter.

Esmolol (Brevibloc®)

MECHANISM OF ACTION

Esmolol is an ultra-short-acting, cardioselective, beta-adrenergic receptor blocker. It inhibits the effects of $beta_1$ receptors. At higher doses, this agent inhibits $beta_2$ receptors located in bronchial musculature and blood vessels (Micromedex Online, 2008).

INDICATIONS

Esmolol is indicated for postoperative hypertension. It is also indicated as part of the management of acute MI, intraoperative hypertension, and intraoperative and postoperative tachyarrhythmias, including supraventricular dysrhythmias (Micromedex Online, 2008).

DOSAGE

For postoperative hypertension, the dose of esmolol is 500 mcg/kg, given as an IV bolus administered over 1 minute. This bolus should be followed by a maintenance dose of 50 mcg/kg/min given over 4 minutes. If additional dosing is required after 5 minutes, the same loading dose followed by 100 mcg/kg/min may be infused over 4 minutes. This titration may continue by increasing the maintenance dose in 50 mcg/kg/min increments until the desired therapeutic endpoint or a maintenance dosage of 200 mcg/kg/min is reached. When the endpoint is reached, the loading dose should be eliminated or titration may take place every 10 minutes rather than every 5 minutes (Micromedex Online, 2008). Because esmolol has a short half-life, it is a practical choice for treating patients with a labile blood pressure (Khalpey et al., 2008).

SIDE EFFECTS

Side effects commonly associated with esmolol include bradycardias, chest pain, hypotension, confusion, headache, dizziness, agitation, dyspnea, wheezing, fatigue, constipation, and nausea and vomiting. Serious, but less common side effects include seizures, bronchospasm, and pulmonary edema (Micromedex Online, 2008).

NURSING IMPLICATIONS

Logically, any patient who requires an agent that causes beta-receptor stimulation should not receive beta-blocker therapy (Khalpey et al., 2008). Esmolol is contraindicated in patients with cardiogenic shock, hemodynamic compromise, second- or third-degree heart block, first-degree heart block (if the PR interval is greater than 0.24 sec) (Khalpey et al., 2008), or severe sinus bradycardia. Caution should be exercised when this agent is administered to patients with heart failure, bronchospastic disease, atrial fibrillation (AF) with associated hypotension, diabetes, renal impairment, or hyperthyroidism. Because esmolol may require large volumes of fluid for its administration, thought should be given as to whether it is the appropriate drug for patients who may not be able to tolerate this excessive fluid intake.

The ICU nurse should monitor heart rate, blood pressure, and for signs of heart failure in patients receiving esmolol. Similarly, patients with diabetes should have their blood glucose monitored on a regular basis. Sudden withdrawal of therapy should be avoided (Micromedex Online, 2008).

Labetalol (Normodyne®, Trandate®)

MECHANISM OF ACTION

Labetalol is a nonselective, adrenergic blocking agent that exerts inhibitory effects on $beta_1$, $beta_2$, and $alpha_1$ receptors (Khalpey et al., 2008; Micromedex Online, 2008).

INDICATIONS

Labetalol is used on an off-label basis for postoperative hypertension. Data suggest it is effective when used on postoperative vascular surgery patients.

DOSAGE

For postoperative hypertension, patients receive 10 mg intravenously over 2 minutes. If additional doses are needed, 10 to 20 mg may be given every 10 minutes, up to a maximum dose of 300 mg in a 24-hour period.

SIDE EFFECTS

When labetalol is administered, no adverse effects or hemodynamic consequences have been reported (Orlowski, Vidt, Walker, & Haluska, 1989). When it is given for on-label conditions, serious side effects have included bronchospasm, hyperkalemia, and ventricular dysrhythmias. Commonly experienced side effects include bradycardias, edema, orthostatic hypotension, diaphoresis, increased liver enzymes, dizziness, paresthesias, elevated renal function tests, dyspnea, wheezing, and fatigue (Micromedex Online, 2008).

NURSING IMPLICATIONS

Like esmolol, labetalol is contraindicated in patients with cardiogenic shock, second- or third-degree heart block, or severe sinus bradycardia. It is also contraindicated in patients with bronchial asthma or COPD. Caution should be exercised when labetalol is administered to patients with heart failure, bronchospastic disease, diabetes, heart failure, ischemic heart disease, liver disease, peripheral vascular disease (PVD), or hyperthyroidism. Monitoring by the ICU nurse should include heart rate, blood pressure, and signs of heart failure. Similarly, patients with diabetes should have their serum glucose monitored on a regular basis. Sudden withdrawal of therapy should be avoided (Micromedex Online, 2008). As with esmolol, any patient who requires an agent that causes beta-receptor stimulation should not receive beta-blocker therapy (Khalpey et al., 2008).

Angiotensin-Converting Enzyme Inhibitors

Enalaprilat (Vasotec®)

HEMODYNAMIC EFFECTS

Angiotensin-converting enzyme (ACE) inhibitors act on the renin–angiotensin–aldosterone system (RAAS). Specifically, they prevent the conversion of angiotensin I to angiotensin II (a potent vasoconstrictor) by inhibiting ACE in the pulmonary and systemic vascular endothelium, resulting in vasodilation (Levy, Tanaka, & Bailey, 2008). These agents cause a decrease in SVR and typically have little effect on heart rate. With the dosage described below, patients should experience improvements in both blood pressure and CO (Lemmer, Richenbacher, & Vlahakes, 2003).

INDICATIONS

ACE inhibitors may be administered early after cardiac surgery to patients with mild left ventricular (LV) dysfunction, even in the face of moderate renal impairment (Manché, Galea, & Busuttil, 1999).

DOSAGE

The initial dose of enalaprilat is 0.625 to 1.25 mg, infused over 5 minutes. Additional doses, up to a maximum of 5 mg every 6 hours, may be administered (Lemmer et al., 2003).

SIDE EFFECTS

The most common side effects with enalaprilat are cough, hyperkalemia, and renal failure. The cough is thought to occur due to the accumulation of bradykinin in the lung and vasculature (Levy et al., 2008). Hyperkalemia occurs when aldosterone is inhibited secondary to the inhibition of angiotensin II. In one study, hypotension and a transient decline in renal function occurred; these conditions were corrected with dopamine (Manché et al., 1999). Rare side effects such as dysgeunia (altered sense of taste) and neutropenia can be serious complications, however. The ICU nurse should observe for signs of onset of these complications and anticipate possible discontinuation of the medication if they occur.

NURSING IMPLICATIONS

For patients who have a history of renal insufficiency and who are receiving enalaprilat, nurses should monitor serum creatinine levels (Lemmer et al., 2003). Meticulous monitoring of the patient's hemodynamic profile and hourly measurement of urinary output may help avoid the development of renal failure sometimes associated with ACE inhibitors. In any event, caution should be used when administering ACE inhibitors to patients with significant LV dysfunction (Manché et al., 1999).

Angiotensin-Receptor Blockers

Angiotensin-receptor blockers (ARBs) influence the RAAS by blocking production of angiotensin II from sources of angiotensin II other than the liver. The blocking of angiotensin receptors occurs on blood vessels, in the adrenals, and within all other tissues. The adrenal-related blockage results in a decrease in aldosterone levels, thereby leading to increased excretion of sodium and water from the kidneys.

ARBs do not increase potassium levels or cause many of the side effects associated with ACE inhibitors. They are utilized predominately for hypertension management and require blood pressure monitoring after their initiation. Both ACE inhibitors and ARBs are contraindicated in patients with bilateral renal artery stenosis.

Calcium Channel Blockers

Nicardipine

HEMODYNAMIC EFFECTS

As the classification connotes, nicardipine blocks the flow of calcium. It acts directly on arterioles to cause peripheral vascular and coronary vasodilation and lower blood pressure. It has little effect on contractility or atrioventricular (AV) node conduction. Nicardipine has also been shown to dilate the coronary vasculature (Khalpey et al., 2008; Lemmer et al., 2003; Levy et al., 2008). Administration did not affect ventricular preload or afterload or CO despite significant decreases in blood pressure (Cheung et al., 1999).

INDICATIONS

Nicardipine is indicated for postoperative hypertension.

DOSAGE

Therapy is initiated at an infusion rate of 5 mg/hr. The dose may be slowly increased by 2.5 mg/hr to a maximum of 15 mg/hr. Once the blood pressure endpoint is reached, a maintenance infusion may be run at 3 mg/hr (Micromedex Online, 2008).

SIDE EFFECTS

The most common side effects of nicardipine are headache, hypotension, nausea, vomiting, peripheral edema, headache, dizziness, and tachycardia. Serious adverse events that have

been reported include angina, MI, and a rare dysrhythmia (Micromedex Online, 2008).

NURSING IMPLICATIONS

Because of nicardipine's potential to cause negative inotropic effects, especially in patients with heart failure, portal hypertension, or significant LV dysfunction, caution should be exercised when administering this agent with a beta blocker. Close blood pressure and heart rate monitoring are required during therapy. Nicardipine is contraindicated in patients with advanced aortic stenosis. Diastolic pressure and afterload reduction may worsen rather than improve myocardial oxygen balance (Micromedex Online, 2008).

Clevidipine (Cleviplex®)

HEMODYNAMIC EFFECTS

Clevidipine is a newly FDA-approved, ultrashort-acting intravenous calcium channel blocker. It functions as both a smooth muscle relaxant and an arterial vasodilator (Singla et al., 2008). This agent causes a decrease in MAP and SVR, but it does not reduce filling pressures (Micromedex Online, 2008).

INDICATIONS

Clevidipine is used to treat postoperative hypertension without impairing cardiac function. In one study of postoperative cardiac surgery patients, treatment with this calcium channel blocker was effective in 91.8% of patients (Singla et al., 2008).

DOSAGE

The initial dose of clevidipine is 1–2 mg/hr via continuous infusion. The dose may be doubled in 90-second intervals. Once the patient's blood pressure begins to approach the goal, incremental dosing should be less frequent (every 5–10 minutes) and be less than double the dose. A maximum initial dose of 16 mg/hr is recommended, and a therapeutic effect is achieved for most patients at infusion rates of 4–6 mg/hr. The total 24-hour dosing should not exceed an average of 21 mg/hr because of lipid load restrictions (Micromedex Online, 2008).

SIDE EFFECTS

Reported side effects of clevidipine include headache, sinus tachycardia, hypotension, nausea, vomiting, and dizziness (Singla et al., 2008). Other side effects that have been reported include AF and acute renal failure. Although rare, cardiac arrest, MI, hypotension, and reflex tachycardia have occurred with use of this agent (Micromedex Online, 2008).

NURSING IMPLICATIONS

Administration of clevidipine is contraindicated in patients with an allergy to soy or egg products or with alterations in lipid metabolism (e.g., hyperlipidemia). It is also contraindicated in patients with severe aortic stenosis because clevidipine may reduce myocardial oxygen delivery secondary to afterload reduction. Caution should be exercised when administering clevidipine concomitantly with a beta blocker. Heart failure symptoms may be exacerbated due to this agent's negative inotropic effects. Patients may develop hypotension and reflex tachycardia when rapid titration takes place in an effort to increase the dosage. Rebound hypertension may develop following extended infusions of clevidipine (Micromedex Online, 2008).

Clevidipine is prepared in a phospholipid emulsion. Any unused medication must be discarded after 4 hours of spiking the stopper.

When a patient is receiving clevidipine, the ICU nurse should continuously monitor heart rate and blood pressure during the infusion and until vital signs become stable. Blood pressure monitoring should continue for a minimum of 8 hours following discontinuation of clevidipine if the patient is not converted to another antihypertensive agent. Patients should also be monitored for

exacerbation of heart failure symptoms (Micromedex Online, 2008).

Selective Dopamine-1-Receptor Agonists

Fenoldopam Mesylate

HEMODYNAMIC EFFECTS

Fenoldopam mesylate is a dopamine-1-receptor agonist. The dopamine-1 (D_1) receptors are located in the coronary, mesenteric, and renal vasculature; when stimulated, they cause vasodilation (Levy et al., 2008). Fenoldopam also moderately binds to $alpha_2$ receptors, which results in lowered SVR and PVR and enhanced CO. This agent has rapid action as a vasodilator and increases renal blood flow (Lemmer et al., 2003).

INDICATIONS

Fenoldopam is indicated for the treatment of severe postoperative hypertension (Micromedex Online, 2008). It is believed to be especially useful in patients with renal insufficiency when it is administered in the prescribed dose range. Fenoldopam causes an increase in glomerular filtration rate, renal blood flow, and sodium excretion (Lemmer et al., 2003; Levy et al., 2008).

DOSAGE

The initial dose of fenoldopam is 0.03–0.1 mcg/kg/min. Titration can occur in increments of 0.05–0.1 mcg/kg/min every 5–15 minutes, to a maximum of 1.6 mcg/kg/min, to achieve the desired blood pressure. The doses must be administered as a continuous infusion; no bolus administration should be performed. Fenoldopam should not be used for more than 48 hours (Lemmer et al., 2003; Micromedex Online, 2008).

SIDE EFFECTS

Possible adverse effects of fenoldopam include hypotension, tachyarrhythmias, flushing, nausea, vomiting, dizziness, headache, angina, cardiac dysrhythmias, heart failure, MI, and serum creatinine elevation (Micromedex Online, 2008).

NURSING IMPLICATIONS

Caution should be used when fenoldopam is administered to patients who are concomitantly receiving beta blockers or in patients with hypokalemia, hypotension, liver disease, tachycardia, or glaucoma. During administration of this agent, the ICU nurse should monitor blood pressure, heart rate, and serum electrolytes, particularly potassium (Micromedex Online, 2008).

AGENTS USED TO MANAGE POSTOPERATIVE LOW CARDIAC OUTPUT AND HYPOTENSION

Some degree of myocardial depression, low CO, and hypotension is common in the immediate postoperative period following cardiac surgery. These conditions can be related to preexisting cardiac disease, postischemic dysfunction, or reperfusion injury (Levy et al., 2008).

Low CO following cardiopulmonary bypass (CPB) procedures is primarily due to LV dysfunction. This LV dysfunction may occur secondary to cardioplegic arrest, decreased preload, loss of vasomotor tone, intraoperative blood loss, increased capillary permeability, increased urinary output from hypothermia, dysrhythmias, or intraoperative MI (Aranki, Cutlip, & Aroesty, 2008). Low cardiac output syndrome (LCOS), which may occur in postoperative cardiac surgery patients, is a decrease in CO secondary to a brief episode of myocardial dysfunction (Massé & Antonacci, 2005).

Contributing factors to postoperative hypotension include hypovolemia, vasodilation (relative hypovolemia), anemia, pneumothorax, hemothorax, cardiac tamponade, electrolyte imbalance, hemorrhage, metabolic alterations, and dysrhythmias.

Effective treatment of low CO and hypotension depends on quickly identifying the causes and initiating the appropriate treatment. Detrimental complications can occur even with brief periods of hypotension, so aggressive and prompt intervention is warranted.

When low CO or hypotension is accompanied by low CVP and pulmonary artery occlusive pressure (PAOP), volume resuscitation is needed to correct hypovolemia. A combination of crystalloids, colloids, and blood products may be used for this purpose. An in-depth discussion of volume resuscitation appears in Chapter 17.

If hypotension persists after volume resuscitation, significant vasodilation may occur. In this scenario, adrenergic agonists or vasopressors may be required to normalize blood pressure if the patient has normal pump function and remains unresponsive to volume repletion alone (Khalpey et al., 2008). Pharmacologic intervention is also suggested to begin once the patient has adequate filling pressures and acid–base and electrolyte balance has been achieved (Massé & Antonacci, 2005).

As many as 40% of postoperative cardiac surgery patients require vasopressor support, and as many as 20% require inotropic support (St. Andre & DelRossi, 2005). Patients who have poor LV function or CO may require inotropic support to augment contractility. In these patients, volume repletion, administration of vasodilators, pacing, or any combination of these may not be adequate (Khalpey et al., 2008; Silvestry, 2008). Typically, patients who improve with inotropic support are those with a cardiac index (CI) less than 2 L/min/m^2 with an optimal heart rate, cardiac rhythm, filling pressures, afterload, and absence of tamponade (Silvestry, 2008).

Adrenergic Agonists

Adrenergic agonists are used to normalize blood pressure when all known causative factors are corrected but hypotension persists. Any patient receiving an adrenergic agonist should be continuously assessed for hypovolemia, which may occur even after adequate volume repletion. Adrenergic agonists are often referred to as sympathomimetics, reflecting their ability to activate adrenergic receptors by direct receptor binding, promotion of norepinephrine (NE) release, blockade of NE reuptake, and inhibition of NE inactivation.

Adrenergic agonists are classified as either catecholamines or non-catecholamines. Catecholamines include epinephrine (Adrenaline®), norepinephrine (Levophed®), dopamine (Intropin®), and dobutamine (Dobutrex®). An example of a non-catecholamine is phenylephrine (Neosynephrine®).

Adrenergic agonists are notable for their specificity, with the various agents acting on $alpha_1$, $alpha_2$, $beta_1$, $beta_2$, or a combination of these receptors (see Table 12–2). The precise ability of a drug to selectively activate certain receptors to the exclusion of others is depends on the dosage, however. Clinical activation of $alpha_1$ receptors results in vasoconstriction. Activation of $alpha_2$ receptors inhibits NE release. When $beta_1$ receptors are activated, patients experience a positive inotropic effect (increased force of contraction), increased blood pressure, heart rate, CO, and impulse conduction through the AV node. Activation of $beta_2$ receptors can also have positive inotropic (increase in contractility) and chronotropic (increase in heart rate) effects on the heart and cause peripheral vasodilation (especially in skeletal and muscle vasculature). When $beta_2$ receptors in the lung are stimulated, bronchodilation occurs.

Stimulation of dopamine-1 (D_1, post-synaptic) receptors, by contrast, causes direct vasodilation. Stimulation of dopamine- 2 (D_2, pre-synaptic) receptors causes vasodilation by inhibiting the release of NE (Salenger et al., 2003).

Use of adrenergic agonists is typically initiated in the operating room during cardiac surgery, and patients can be weaned from

Table 12–2 Adrenergic Receptors and Effects when Stimulated

Adrenergic Receptor Type	Effects when Stimulated
β_1	Increased heart rate, blood pressure, contractility (increased inotropic effect), cardiac output, conduction velocity, and automaticity
β_2	Bronchodilation
α_1	Vasoconstriction
α_2	Vasodilation by inhibition of norepinephrine release
D_1, post-synaptic	Direct vasodilation
D_2, pre-synaptic	Vasodilation by inhibition of norepinephrine release
V_1 (on vascular smooth muscle)	Increased peripheral vascular resistance and vasoconstriction of capillaries and arterioles

Sources: Katz, 2007; Salenger et al., 2003.

their agents rapidly after recovery from anesthesia. These drugs are titrated so as to maintain blood pressure within the ordered parameters—typically a MAP of more than 65 mm Hg or a systolic blood pressure (SBP) of at least 90 mm Hg. Higher pressures may be required to perfuse organs when patients have a history of extreme hypertension, carotid artery disease, PVD, or renal dysfunction (Khalpey et al., 2008).

Six adrenergic agonists are typically used after cardiac surgery: phenylephrine, norepinephrine, epinephrine, vasopressin (antidiuretic hormone), dopamine, and dobutamine. These medications are used to elevate blood pressure for patients in hypotensive states.

Phenylephrine

HEMODYNAMIC EFFECTS AND INDICATIONS

Phenylephrine is a vasoconstrictor that is often used after cardiac surgery to manage mild to moderate hypotension. It causes vasoconstriction by activating alpha$_1$ receptors; no other adrenergic receptors are stimulated. The vasoconstrictor effects lead to an increase in SVR. Phenylephrine is also valuable in patients with a high CI who are profoundly vasodilated. A decrease in CO is seen with use of this agent, and either an increase or a decrease in heart rate may be seen (Micromedex Online, 2008; Salenger et al., 2003).

DOSAGE

Phenylephrine should be started at a dose relative to the clinical situation. Effects are often seen immediately. The dose range is 2–200 mcg/min (St. Andre & DelRossi, 2005).

SIDE EFFECTS

Because of its vasoconstrictor activity, phenylephrine causes hypoperfusion to tissues and end organs, which can lead to visceral and renal ischemia. It also causes an increase in myocardial oxygen consumption and may exacerbate metabolic acidosis (Salenger et al., 2003). Other reported side effects include hypertension, MI, tachyarrhythmias, ventricular dysrhythmias, and pulmonary edema (Micromedex Online, 2008).

NURSING IMPLICATIONS

The patient should receive adequate volume resuscitation prior to receiving phenylephrine or receiving a significantly increased dose of this agent. Phenylephrine is contraindicated for use in patients with severe hypertension and tachycardia. Caution should be exercised when administering this drug to patients with bronchial asthma, diabetes, or hypertension. Monitoring of blood pressure and heart rate is advisable (Micromedex Online, 2008).

Norepinephrine

HEMODYNAMIC EFFECTS AND INDICATIONS

Norepinephrine is a powerful vasopressor and adrenergic agonist that stimulates $alpha_1$ and $beta_1$ receptors, causing vasoconstriction, increased inotropic effects, and cardiac stimulation. A small amount of $beta_2$-receptor stimulation occurs as well. Norepinephrine is classified as a vasopressor and an inotrope (Levy et al., 2008; Salenger et al., 2003). It is typically used in profound hypotension when volume repletion is inadequate; it can also be administered concomitantly with fluid resuscitation if the patient's blood pressure and CO are significantly impaired (Katz, 2007). In addition, norepinephrine is the most common treatment for vasodilatory hypotension/shock associated with CPB (Aranki, Cutlip, & Aroesty, 2008).

DOSAGE

Norepinephrine is initially started at 2–20 mcg/min and titrated so as to reach the desired response, usually a MAP of at least 70 mm Hg (Katz, 2007; Salenger et al., 2003).

SIDE EFFECTS

The most clinically significant side effects experienced by the postoperative cardiac surgery patient are an increase in myocardial workload and oxygen consumption. End-organ damage (e.g., damage to the kidneys and mesentery) may also occur secondary to $alpha_1$-receptor stimulation (Katz, 2007). Norepinephrine exacerbates hyperglycemia and metabolic acidosis; the latter effect is related to an increase in lactate production (Salenger et al., 2003).

NURSING IMPLICATIONS

High doses and long-term use of norepinephrine cause decreased perfusion to the skin and can lead to tissue necrosis and limb loss. Patients should be assessed regularly for cyanosis, decreased capillary refill time, and diminished peripheral pulses, all of which are signs of decreased perfusion (Margereson, 2003). They should receive adequate volume resuscitation prior to receiving this therapy or receiving a significantly increased dose of norepinephrine.

Epinephrine

HEMODYNAMIC EFFECTS AND INDICATIONS

Epinephrine stimulates $alpha_1$, $beta_1$, and $beta_2$ receptors (Levy et al., 2008). While not typically administered in other situations in postoperative patients, this powerful catecholamine may be used after cardiac surgery as an inotrope to improve cardiac function and enhance stroke volume (SV), as an adrenergic agonist and vasopressor for refractory hypotension, or as a positive chronotropic agent to increase heart rate in bradycardia (Massé & Antonacci, 2005). Epinephrine is also useful in the cardiac arrest situation owing to its ability to enhance automaticity (Katz, 2007).

DOSAGE

Epinephrine's effects on different adrenergic receptors vary with the dosage used. At low doses (less than 0.02 mcg/kg/min), epinephrine causes stimulation of $beta_2$ receptors with resultant vasodilation and relaxation of the bronchial smooth muscle. At higher doses (0.008–0.06 mcg/kg/min), $beta_1$ stimulation results in an increased blood pressure, CO, and contractility. At doses of 0.5–4.0 mcg/min,

positive chronotropic effects are noted. At the highest dosage (more than 2 mcg/min), $alpha_1$-receptor stimulation causes vasoconstriction. The blood pressure effects of epinephrine vary in postoperative cardiac surgery patients. In particular, patients who are postoperative CPB demonstrate inconsistent hemodynamic responses to epinephrine administration. Variable responses in CO, heart rate, and MAP have been reported (Salenger et al., 2003; Silvestry, 2008).

SIDE EFFECTS

When higher doses of epinephrine are administered, patients may develop atrial or ventricular ectopy and tachyarrhythmias owing to $beta_1$-receptor stimulation. The higher the dose, the more likely that atrial or ventricular ectopy and tachyarrhythmias will be seen (Katz, 2007; Salenger et al., 2003).

Epinephrine can raise the serum glucose levels so profoundly that insulin drips should be anticipated. Higher than normal doses of insulin may be required to maintain adequate glycemic control. The hyperglycemia is attributable to increased gluconeogenesis and the stress response to epinephrine administration (Katz, 2007). Hyperglycemia typically occurs in patients who receive epinephrine within the first 6–8 postoperative hours and usually disappears within a few hours after epinephrine is discontinued (St. Andre & DelRossi, 2005).

Patients receiving epinephrine may also develop metabolic acidosis; serum bicarbonate levels are typically between 17 and 21 mEq/L, although a serum bicarbonate level of 15 mEq/L has been reported. This metabolic acidosis may occur secondary to the inadequate metabolism and lactate buildup that occurs in response to $beta_1$ stimulation. It is not related to hypoperfusion, as patients' cardiac performance is acceptable when the acidosis develops. CO and mixed venous saturation levels also remain within acceptable parameters (Katz, 2007). As with hyperglycemia, metabolic acidosis typically occurs in patients who receive epinephrine within the first 6–8 postoperative hours and usually disappears within a few hours of epinephrine's discontinuation (St. Andre & DelRossi, 2005).

NURSING IMPLICATIONS

While on epinephrine, the patient must be monitored closely for tachycardia and signs of myocardial ischemia—administration of this agent will increase PVR, SVR, lactate, and myocardial oxygen consumption. Adequate oxygenation should be maintained and the patient monitored for signs of ischemia, given that epinephrine increases myocardial oxygen demand.

The ICU nurse should be prepared to quickly wean the patient from insulin if the epinephrine drip is reduced or discontinued. Hyperglycemia usually resolves within a few hours (6 or fewer) after the epinephrine infusion is discontinued (St. Andre & DelRossi, 2005).

While increasing blood pressure and CO/CI are goals of therapy, vasodilators may be necessary to control elevated blood pressure when epinephrine must be used at high doses to maintain CO. Similarly, when epinephrine is infused at higher doses, $alpha_1$ stimulation causes an increase in myocardial workload, SVR, and PAOP (Katz, 2007).

Adrenergic agonists—including epinephrine—cause vasoconstriction, such that significant tissue damage can occur if extravasation of these agents into the subcutaneous tissue occurs. Decreased blood flow to tissue as a result of vasoconstriction may lead to tissue death. Immediate treatment with an appropriate agent should be utilized promptly after extravasation of adrenergic agonists is suspected or identified. Epinephrine should be given ideally via a central line to limit the risk of extravasation.

Vasopressin

CPB frequently causes the release of vasopressin, antidiuretic hormone, that may

contribute to post-bypass vasoconstriction. Data indicate that vasopressin levels may diminish as hypotension continues. This finding suggests that the body may have a limited supply of vasopressin that is exhausted with the initial bout of hypotension (Levy et al., 2008).

Approximately 10% of cardiac surgery patients develop vasodilatory shock (Argenziano et al., 1998). Features of vasodilatory shock include decreased MAP, organ hypoperfusion, lactic acidosis, decreased SVR, and maldistribution of blood volume. End-organ failure is the ultimate outcome if the condition is not reversed (Albright, Zimmerman, & Selzman, 2002).

HEMODYNAMIC EFFECTS AND INDICATIONS

Vasopressin is used to treat vasodilatory shock following CPB procedures in patients with profound hypotension (MAP less than 70 mm Hg) despite fluid resuscitation, afterload reduction, inotropic therapy, and norepinephrine administration. Postoperative CPB patients who have protracted hypotension demonstrate poor vascular smooth-muscle response to catecholamines. Vasopressin, when administered in high doses, promotes contraction of vascular smooth muscle, which in turn causes vasoconstriction of the capillaries and small arterioles and can increase MAP (Albright et al., 2002). It is also believed that some patients have low vasopressin concentrations, such that exogenous administration may improve these patients' clinical status (Aranki et al., 2008; Raja & Dreyfus, 2004). The postoperative cardiac surgery patients with vasodilatory shock who benefit most from vasopressin are those with a deficiency and those with a low ejection fraction (EF) who take ACE inhibitors (Argenziano et al., 1998).

Vasopressin also has indications in cardiac arrest situations as an early substitute for epinephrine in patients with ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) (Katz, 2007).

Vasopressin may be effective in milrinone-related hypotension (Gold et al., 2000); milrinone is discussed later in this chapter. Vasopressin stimulates V_1 receptors on vascular smooth muscle, which causes an increase in peripheral vascular resistance and vasoconstriction of capillaries and arterioles (Katz, 2007).

Vasopressin increases secretion of corticotropin, a hormone produced by the anterior pituitary gland that stimulates the adrenal cortex. The adrenal cortex produces cortisol, a major hormone responsible for blood pressure regulation.

DOSAGE

The dosage of vasopressin needed to achieve vasoconstrictor effects is 0.01–0.1 unit/min by continuous IV infusion (Albright et al., 2002; Katz, 2007).

SIDE EFFECTS

Side effects of vasopressin are rare but include end-organ damage from vasoconstriction, leading to hypoperfusion, hyponatremia, and increased SVR. All of these effects occur secondary to the drug's vasoconstriction effects (Katz, 2007).

NURSING IMPLICATIONS

Extreme caution should be used in patients with vascular disease who are receiving vasopressin because of the potential for extreme vasoconstriction associated with this agent. The ICU nurse should monitor for a number of adverse effects in the postoperative cardiac surgery patient, including decreased CO, chest pain, myocardial ischemia, ventricular dysrhythmias, bronchoconstriction, metabolic acidosis, tremors, gastrointestinal infarction, abdominal cramping, and water intoxication (Albright et al., 2002).

Dopamine and Dobutamine

The use of dopamine and dobutamine is required in many postoperative cardiac surgery patients even when careful attention is paid to intraoperative myocardial protection. Prolonged surgery, myocardial edema, advanced age, reperfusion injuries, and poor preoperative cardiac function are all factors that put the patient at higher risk for low CO postoperatively. Both dopamine and dobutamine cause an increase in CO and heart rate (Silvestry, 2008).

Before these agents are administered, CO/CI should be high enough to sustain end-organ perfusion and deliver adequate amounts of oxygen to tissues. This criterion should be judged subjectively for each patient based on adequate urine output, normal capillary refill time, appropriate mentation, adequate blood pressure, warm skin temperature, and lack of acidosis. Objectively, CI should be more than 2 L/min/m^2 before use of dopamine and dobutamine is considered; normal CI in the nondiseased heart is in the range of 2.5–4.5 L/min/m^2. When preload has been optimized and SV remains low, poor contractility is the likely etiology and inotropes are indicated. Adding inotropes will increase the amount of contractile force and result in an improved SV and CO/CI.

Dopamine

HEMODYNAMIC EFFECTS AND INDICATIONS

Like epinephrine, dopamine's effects on different adrenergic receptors vary with dosage. Dopamine stimulates $alpha_1$, $beta_1$, and $beta_2$ receptors, resulting in either vasoconstriction or positive inotropic and chronotropic effects (Levy et al., 2008). Stimulation of $beta_2$ receptors is less than that seen with the other adrenergic agents. Dopamine is used to increase blood pressure, CO, and perfusion through the renal vasculature. At higher doses, this drug has vasopressor properties, as it stimulates the release of endogenous NE. Dopamine also stimulates D_1 and D_2 receptors when administered in doses less than 8 mcg/kg/min (Albright et al., 2002; Salenger et al., 2003).

DOSAGE

Renal vasodilation occurs due to stimulation of dopaminergic receptors at doses of 0.5–3.0 mcg/kg/min. At an infusion rate of 4–10 mcg/kg/min, $beta_1$ stimulation is seen. Positive inotropic and chronotropic effects lead to an increase in heart rate, blood pressure, contractility, and CO. At doses exceeding 10 mcg/kg/min, $alpha_1$ stimulation occurs, along with associated vasoconstriction and increased SVR. While receptor stimulation overlap occurs at these higher infusion rates, the dopaminergic effect is lost (Salenger et al., 2003). Dopamine's effect on renal perfusion in terms of long-term outcomes remains controversial (Massé & Antonacci, 2005). This agent should be started at a low dose and doses titrated upward slowly to achieve the desired effect.

SIDE EFFECTS

Common side effects of dopamine include chest pain, hypertension, palpitations, tachyarrhythmias, headache, anxiety, dyspnea, oliguria, nausea, and vomiting. Serious side effects include ectopic beats (including ventricular dysrhythmias), widening QRS complex, and gangrenous disorder (Micromedex Online, 2008).

NURSING IMPLICATIONS

Systolic pressures are often elevated with dopamine use, making it a poor choice in patients with pulmonary hypertension. Dopamine is also contraindicated in patients with tachyarrhythmias. Caution should be exercised when administering this agent to patients with angina, hypovolemia, or ventricular dysrhythmias (Micromedex Online, 2008).

Like epinephrine, dopamine causes vasoconstriction, such that significant tissue damage can result if extravasation into the subcutaneous tissue occurs. Decreased blood flow to tissue from vasoconstriction may lead to tissue sloughing and death. Immediate infiltration with an appropriate agent (e.g., phentolamine [Regitine®] 10–15 mL) to the ischemic area should be implemented promptly after extravasation of adrenergic agonists is suspected or identified.

Dobutamine

HEMODYNAMIC EFFECTS AND INDICATIONS

Dobutamine is a synthetic catecholamine and positive inotrope that acts primarily as a $beta_1$ agonist. It causes an increase in CO/CI, while lowering SVR and increasing heart rate (Massé & Antonacci, 2005). It achieves these effects by increasing contractility and causing peripheral vasodilation (Khalpey et al., 2008). Dobutamine causes minimal amounts of $alpha_1$-receptor stimulation and a small amount of $beta_2$-receptor stimulation (Salenger et al., 2003). This agent is useful when patients have low CO with high SVR or PVR and cannot tolerate vasodilators to decrease afterload. Dobutamine administration also results in enhanced coronary blood flow and decreased LV preload and afterload—more so than is noted with dopamine (Salenger et al., 2003; Silvestry, 2008).

Patients with high pulmonary pressures (e.g., those who have undergone mitral valve replacement), with or without a history of pulmonary hypertension, and with low heart rates may benefit more from dobutamine than from dopamine. This preference arises because dobutamine administration is associated with a decrease in pulmonary artery pressure, left ventricular stroke work index, CI, PAOP, and SVR (Micromedex Online, 2008).

DOSAGE

The onset of action of dobutamine is rapid, and it is rapidly cleared (2–3 minutes) when discontinued, allowing for rapid titration of the drug. The dose is 2–20 mcg/kg/min (Katz, 2007).

SIDE EFFECTS

While administering dobutamine, the ICU nurse should observe for hypotension, tachycardia, ventricular dysrhythmias, and myocardial ischemia (Katz, 2007; Khalpey, Ganim, & Rawn, 2008; Leyh et al., 2003). Other reported side effects include angina, dyspnea, tachyarrhythmias, hypertension, and headache (Micromedex Online, 2008). Of note, dobutamine is less likely to cause dysrhythmias than other positive inotropic agents.

NURSING IMPLICATIONS

Like other agents in this category, dobutamine should not be given to hypovolemic patients. Monitoring of blood pressure, heart rate, PAP, PAOP, CVP, CO, SVR, and urinary output should be performed on an ongoing basis to determine the drug's efficacy and the patient's tolerance of therapy. Evaluation of the patient's ECG and electrolyte status should also be performed on a regular basis (Micromedex Online, 2008).

Phosphodiesterase Inhibitors

Another category of medications that may be used to treat low CO after cardiac surgery comprises the phosphodiesterase (PDE) inhibitors. Two direct phosphodiesterase inhibitors—inamrinone (formerly amrinone, Inocor®) and milrinone (Primacor®)—are especially well-known agents.

Inamrinone

HEMODYNAMIC EFFECTS AND INDICATIONS

Inamrinone increases CO with its inotropic effects as well as its vasodilating systemic and pulmonary vasculature (Salenger et al., 2003). It enhances CO by directly inhibiting phosphodiesterase from metabolizing cyclic adenosine monophosphate (cyclic AMP) in

myocardial cells. An increase in cyclic AMP causes an increase in the amount of calcium that moves into cells through ion channels, thereby resulting in a more forceful contraction (inotropic effect).

Inamrinone also produces venous and arterial vasodilation, and decreases SVR, PVR, and LV preload (PAOP), while minimally affecting myocardial oxygen demand (Levy et al., 2008; Silvestry, 2008). All of these effects contribute to an improvement in CO/CI (Khalpey et al., 2008). The drug has little effect on heart rate, however (Levy et al., 2008). Inamrinone also produces vasodilation in vascular smooth muscle by decreasing intracellular calcium concentration. This effect causes relaxation of the vasculature and ventricles, thereby increasing stroke volume and CO/CI and lowering afterload (Levy et al., 2008; Massé & Antonacci, 2005). In addition, inamrinone promotes myocardial relaxation and improves coronary skeletal muscle and mesenteric blood flow (Salenger et al., 2003).

Inamrinone is indicated for the management of ventricular failure in the postoperative cardiac surgery patient (Katz, 2007). Because of its vasodilator properties, this agent is useful in the management of patients with pulmonary vasoconstriction and right ventricular (RV) dysfunction (Levy et al., 2008).

DOSAGE

A loading dose of 0.75 mg/kg of inamrinone should be administered over a period of 2–3 minutes. It is followed by a continuous infusion of 10–30 mg/kg/min. Additional loading doses at 0.75 mg/kg may be administered as clinically indicated, although the total daily dose should not exceed 10 mg/kg/day.

The peak effect of inamrinone occurs within 10 minutes of administration. The duration of the drug's effect is dose dependent, lasting approximately 30 minutes to 2 hours. Inamrinone has a long half-life, making it important to slowly wean patients from the drug and to monitor cardiac function hours after the drug has been discontinued.

SIDE EFFECTS

Inamrinone may cause thrombocytopenia and has largely been replaced by milrinone because of this effect. Occasional side effects that have been reported include nephrogenic diabetes insipidus, elevated liver function tests, fever, flu-like symptoms, and exacerbation of an underlying dysrhythmia (DiDomenico, 2001).

NURSING IMPLICATIONS

Patients receiving inamrinone may require concomitant administration of an adrenergic agonist owing to the profound vasodilation that occurs with use of this drug (Katz, 2007).

Milrinone

HEMODYNAMIC EFFECTS AND INDICATIONS

Milrinone is a positive inotrope with vasodilator properties. Its administration will cause a decrease in SVR and PVR, making it an ideal agent for patients with RV failure. Milrinone also decreases coronary vascular resistance and, therefore, has a highly favorable effect on myocardial oxygen consumption (Salenger et al., 2003).

Like inamrinone, milrinone enhances CO by directly inhibiting phosphodiesterase from metabolizing cyclic AMP in myocardial cells. An increase in cyclic AMP causes an increase in the amount of calcium that moves into cells through the ion channels, thereby resulting in a more forceful contraction (inotropic effect).

Milrinone also produces venous and arterial vasodilation, and decreases SVR, PVR, and LV preload (PAOP), while minimally affecting myocardial oxygen demand (Levy et al., 2008; Silvestry, 2008). All of these actions contribute to an improvement in CO/CI (Khalpey et al., 2008). The drug has little effect on heart rate, however (Levy et al., 2008). It also produces vasodilation in vascular smooth muscle by decreasing intracellular calcium concentration. This effect causes relaxation of the

vasculature and ventricles, thereby increasing SV and CO/CI and lowering afterload (Levy et al., 2008; Massé & Antonacci, 2005). In addition, milrinone promotes myocardial relaxation and improves coronary skeletal muscle and mesenteric blood flow (Salenger et al., 2003).

Milrinone is indicated for the management of ventricular failure in the postoperative cardiac surgery patient (Katz, 2007). Because of its vasodilator properties, it is a valuable option in the management of patients with pulmonary vasoconstriction and RV dysfunction (Levy et al., 2008).

DOSAGE

A loading dose of 50–75 mcg/kg of milrinone should be followed by a continuous infusion at a rate of 0.25–0.75 mcg/kg/min (Salenger et al., 2003). Milrinone has a rapid onset of action, and its effects last 2–4 hours following titration or discontinuation (Katz, 2007). Milrinone has a shorter half-life (36 minutes) than inamrinone (Levy, Bailey, & Deeb, 2002), and may be weaned more quickly than inamrinone. This half-life is still longer than that for dobutamine, making PDE inhibitors more challenging to titrate (Levy et al., 2008).

SIDE EFFECTS

VT or supraventricular tachycardia (SVT) may occur when milrinone is given, owing to the drug's proarrhythmic properties. Hypotension should be anticipated related to the vasodilatory properties of milrinone (Salenger et al., 2003).

NURSING IMPLICATIONS

Patients receiving milrinone may require concomitant administration of an adrenergic agonist to counteract the profound vasodilation that occurs (Katz, 2007). Aggressive replacement of potassium and magnesium are recommended as well, because the dysrhythmias are more likely to occur when an electrolyte imbalance is present. Patients receiving milrinone should also be observed for hypotension during therapy. Patient improvement may be reflected by increased CO, decreased PAOP, and favorable changes in clinical indices. See Table 12–3 and Box 12–1 for a summary of medications used to treat low cardiac output syndrome and hypotension following cardiac surgery.

Other Agents Used to Control Postoperative Hypotension

Methylene Blue (Urolene Blue®)

HEMODYNAMIC EFFECTS AND INDICATIONS

Methylene blue is an inhibitor of nitric oxide, which is released in large quantities in patients following CPB. Nitric oxide produces profound vasodilation and vasoplegia (hypotension with normal or high CO, low CVP, low PAOP, and low peripheral vascular resistance) (Katz, 2007). Methylene blue is indicated in vasodilatory shock in the immediate postoperative CPB period. Despite its frequent use for this indication, vasodilatory shock is not currently an FDA-approved on-label indication for methylene blue (Micromedex Online, 2008).

DOSAGE

The dosage of methylene blue is 1–2 mg/kg, administered as a slow IV push (Katz, 2007; Micromedex Online, 2008). In one study, the dose was administered over 20 minutes (Leyh et al., 2003)

SIDE EFFECTS

The two main side effects of methylene blue administration are hypertension and a brief period of factitious low oxygen saturation on pulse oximetry (Katz, 2007). Other reported side effects include hypertension, hypotension, abdominal pain, dizziness, headache, confusion, nausea, vomiting, and diarrhea. Serious adverse events reported include

Table 12–3 Select Agents Used to Manage Postoperative Low Cardiac Output and Hypotension and Hemodynamic Effects

Agent	Dose	Mechanism of Action	Hemodynamic Effects
Adrenergic Agonists			
Epinephrine (Adrenaline®)	0.008–0.06 mcg/kg/min by continuous IV infusion	Stimulation of β_1 receptors	Increased contractility and stroke volume, and cardiac stimulation.
	0.5–4.0 mcg/min by continuous IV infusion		Increased heart rate.
	Less than 0.02 mcg/kg/min by continuous IV infusion	Stimulation of β_2 receptors	Vasodilation and relaxation of the bronchial smooth muscle.
	Greater than 2 mcg/min by continuous IV infusion	Stimulation of α_1 receptors	Vasoconstriction; increased SVR.
Norepinephrine (Levophed®)	2–20 mcg/min by continuous IV infusion	Stimulation of α_1 receptors	Vasoconstriction; increased SVR; decreased cardiac output; increase or decrease in heart rate.
		Stimulation of β_1 receptors	Increased inotropic effects and cardiac stimulation.
Dopamine (Intropin®)	0.5–3.0 mcg/kg/min	Stimulation of dopaminergic receptors	Renal vasodilation.
	4–10 mcg/kg/min by continuous IV infusion	Stimulation of β_1 receptors	Increased heart rate, blood pressure, contractility, and cardiac output.
	Greater than 10 mcg/kg/min	Stimulation of α_1 receptors	Vasoconstriction; increased SVR.
	Less than 8 mcg/kg/min by continuous IV infusion	Stimulation of D_1 and D_2 receptors	Vasodilation.
Dobutamine (Dobutrex®)	2–20 mcg/kg/min by continuous IV infusion	Stimulation of β_1 receptors (increased contractility) and peripheral vasodilation	Increased cardiac output/cardiac index, and heart rate; decreased SVR and PAOP (more so than dopamine), pulmonary artery pressure, left ventricular stroke work index, enhanced coronary blood flow.

continues

Table 12–3 Select Agents Used to Manage Postoperative Low Cardiac Output and Hypotension and Hemodynamic Effects (continued)

Agent	Dose	Mechanism of Action	Hemodynamic Effects
Adrenergic Agonists (cont.)			
Dobutamine (Dobutrex®) (cont.)		Minimal amounts of α_1 stimulation	Vasoconstriction.
		Small amount of β_2 receptor stimulation	Relaxation of the bronchial smooth muscle.
Phenylephrine (Neosynephrine®)	2–200 mcg/min by continuous IV infusion	Activation of α_1 receptors	Vasoconstriction; increased SVR; decreased cardiac output; increase or decrease in heart rate.
Vasopressin (antidiuretic hormone)	0.01–0.1 unit/min by continuous IV infusion	Stimulates V_1 receptors	Contraction of vascular smooth muscle, which causes vasoconstriction of the capillaries and small arterioles and can increase mean arterial pressure.
Phosphodiesterase (PDE) Inhibitors			
Inamrinone (Inocor®)	Loading dose: 0.75 mg/kg administered over 2–3 min. Maintenance: 10–30 mg/kg/min by continuous infusion of. Additional loading doses at 0.75 mg/kg may be administered. Total daily dose should not exceed 10 mg/kg/day.	PDE inhibitor; venous and arterial vasodilation; vasodilation in vascular smooth muscle by decreasing intracellular calcium concentration.	Increased cardiac output/cardiac index and stroke volume; decreased SVR, PVR, and PAOP; promotes myocardial relaxation and improves coronary skeletal muscle, and mesenteric blood flow.
Milrinone (Primacor®)	Loading dose: 50–75 mcg/kg. Maintenance dose: 0.25–0.75 mcg/kg/min by continuous infusion.	PDE inhibitor; positive inotrope and vasodilator.	Increased cardiac output/cardiac index and stroke volume; decreased SVR, PVR, and PAOP; promotes myocardial relaxation and improves coronary skeletal muscle, and mesenteric blood flow.
Other agent			
Methylene Blue (Urolene Blue®)	1–2 mg/kg, administered as a slow IV push	Inhibitor of nitric oxide	Vasodilation.

PAOP = pulmonary artery occlusive pressure; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance.

Sources: Albright et al., 2002; Katz, 2007; Khalpey et al., 2008; Levy et al., 2008; Massé & Antonacci, 2005; Micromedex Online, 2008; Salenger et al., 2003; Silvestry, 2008; St. Andre & DelRossi, 2005.

Box 12–1 Vasoactive Agents Used to Manage Postoperative Low Cardiac Output and Hypotension and Hemodynamic Effects

Agent	MAP	PAP	PAOP	CO/CI	SVR	PVR
Epinephrine	+	+/–	+/–	+	+/–	+/–
Norepinephrine	++	++	++	+	++	++
Dopamine	+/–	+/–	+/–	+	+/–	+/–
Dobutamine	+/–	–	–	+	–	–
Phenylephrine	++	~	+	~	++	++
Vasopressin	++	–	~	~	++	–/~
Inamrinone	–	–	–	+	–	–
Milrinone	–	–	–	+	–	–
Methylene Blue	+	+	~	~	+	+

CI = cardiac index; CO = cardiac output; MAP = mean arterial pressure; PAOP = pulmonary artery occlusive pressure; PAP = pulmonary artery pressure; PVR = pulmonary vascular resistance; SVR = systemic vascular resistance.

+ = increase; – = decrease; ~ = no change

Sources: Albright et al., 2002; Katz, 2007; Khalpey et al., 2008; Levy et al., 2008; Massé & Antonacci, 2005; Micromedex Online, 2008; Salenger et al., 2003; Silvestry, 2008; St. Andre & DelRossi, 2005.

cardiac dysrhythmias, malignant hyperthermia, and methemoglobinemia (Micromedex Online, 2008).

NURSING IMPLICATIONS

Following administration of methylene blue, the ICU nurse should observe for hypertension, urine discoloration, and transiently low oxygen saturation on pulse oximetry. The last effect typically lasts less than 10 minutes (Katz, 2007) and results from interference with light absorption. If the patient's oxygen saturation is in question during this time, evaluation with an arterial blood gas should be performed (Touma, 2007). The ICU nurse should anticipate immediate increases in SVR and MAP and the need to significantly lower the infusion rate of norepinephrine (Leyh et al., 2003). The ICU nurse should also monitor methemoglobin levels, complete blood count results, and blood pressure during administration of methylene blue. Caution should be exercised when this agent is administered to patients with renal impairment or G6PD deficiency (Micromedex Online, 2008). In one study, serum lactate levels decreased significantly within 12 hours after administration of methylene blue to patients with vasoplegia following CPB (Leyh et al., 2003).

Dexamethasone (Decadron®)

The inflammatory process and the sequelae that occur as a result of CPB surgery have been the focus of several studies (Whitlock, Rubens, Young, & Teoh, 2005). CPB stimulates a systemic inflammatory response, with an associated release of pro-inflammatory mediators. The results include development of hemodynamic instability (Bruins et al., 1997).

Despite supportive data, and possibly because the data are not consistent (Chaney, Nikolov, Blakeman, Bakhos, & Slogoff, 1998), the use of steroids remains controversial in cardiac surgery patients. Their mechanism of action and the pathophysiologic changes that occur during cardiac surgery have been cited as justifications for their administration in this scenario. In one study, patients who received preoperative and postoperative doses of steroids experienced less systemic inflammation as compared with patients who did not receive prophylactic steroids (Kilger et al., 2003).

Any postoperative cardiac surgery patient exhibiting protracted vasodilatory shock should be suspected of having adrenal insufficiency. In a stressed patient (e.g., a cardiac surgery patient), a low or normal cortisol level can be assumed to be associated with adrenal insufficiency (Khalpey et al., 2008). Adrenal insufficiency responds to steroids, which raise cortisol levels. Cortisol plays a vital role in regulating blood pressure by increasing the sensitivity of the vasculature to endogenous epinephrine and norepinephrine. In the absence of normal cortisol levels, widespread vasodilation occurs secondary to the effects of pro-inflammatory mediators. A cosyntropin stimulation test can be performed for diagnosis of adrenal insufficiency. In the meantime, intravenous dexamethasone may be administered (Khalpey et al., 2008).

MECHANISM OF ACTION

Steroids decrease inflammation by suppressing neutrophil migration, decreasing production of pro-inflammatory mediators, and reversing the increase in capillary permeability (Uptodate.com Lexi Corp, 2008).

INDICATIONS

Steroids are used in cases involving postoperative hemodynamic instability associated with a CPB-induced inflammatory response.

DOSAGE

The dosage of dexamethasone for treatment of shock (e.g., related to adrenal insufficiency that is responsive to steroid therapy) is 4–10 mg IV. This dose may be repeated as needed. For shock that is unresponsive to steroids, the dose is 1–6 mg/kg IV or up to a maximum of 40 mg. Doses may be repeated every 2–6 hours while shock persists. Each dose should be administered over a span of 5–10 minutes. (Uptodate.com Lexi Corp, 2008).

SIDE EFFECTS

A number of clinically significant adverse reactions have been reported in association with dexamethasone administration. These reactions include, but are not limited to, dysrhythmia, bradycardia, cardiac arrest, cardiomyopathy, heart failure, circulatory collapse, edema, myocardial rupture (if administered to a patient following MI), thromboembolism, depression, emotional instability, euphoria, headache, increased intracranial pressure, seizure, adrenal suppression, diabetes mellitus, hyperglycemia, metabolic alkalosis, sodium retention, abdominal distention, gastrointestinal (GI) hemorrhage or perforation, nausea, hepatomegaly, glucosuria, and pulmonary edema (Uptodate.com Lexi Corp, 2008).

NURSING IMPLICATIONS

The ICU nurse should monitor hemoglobin, potassium, and glucose levels, and evaluate for occult blood loss when caring for a patient who is receiving steroids. If the patient is receiving concomitant calcium channel blockers, these agents may decrease the metabolism of steroids. Steroid therapy should, therefore, be monitored to determine its efficacy.

Caution should be exercised if steroids are administered to patients with diabetes mellitus, as hyperglycemia related to alteration in glucose production/regulation may occur. Caution should also be exercised when administering steroids to patients with GI disease because of the risk of perforation. In patients with renal disease, fluid retention may develop when steroids are given. Patients with a history of a seizure disorder should be monitored closely, as adrenal crisis in conjunction with steroid therapy may precipitate seizures. Withdrawal and discontinuation of a corticosteroid should be done slowly, with gradual tapering of the dose (Uptodate.com Lexi Corp, 2008).

Hydrocortisone (Solu-Cortef ®)

MECHANISM OF ACTION

As described earlier, steroids decrease inflammation by suppressing neutrophil migration, decreasing production of pro-inflammatory mediators, and reversing the increase in capillary permeability (Uptodate.com Lexi Corp, 2008).

INDICATIONS

Hydrocortisone is used in cases involving postoperative hemodynamic instability associted with a CPB-induced inflammatory response.

DOSAGE

The dosage of hydrocortisone is based on the indication. For acute adrenal insufficiency, the recommended dose is 100 mg IV bolus, then 300 mg/day in divided doses every 8 hours. Alternatively, the drug may be administered as a continuous infusion for 48 hours. If hydrocortisone is being administered to counteract the stress of surgery in patients who have adrenal insufficiency, the dose is 100–150 mg/day (50 mg every 8–12 hours) for 2–3 days. The dose should be diluted to 50 mg/mL and administered over a period of 30 seconds to several minutes, depending on the dose (Uptodate.com Lexi Corp, 2008).

In one study, patients received 100 mg of hydrocortisone preoperatively. Postoperatively, the patients received 10 mg/hr for 24 hours, 5 mg/hr for 24 hours, 3 × 20 mg/day, and 3 × 10 mg/day (Kilger et al., 2003).

SIDE EFFECTS

Side effects that are reported as being related to administration of hydrocortisone include insomnia, diabetes mellitus, epistaxis, and arthralgia. Rare but potentially life-threatening adverse events include, but are not limited to, hypertension, edema, headache, delirium, hallucinations, seizure, mood swings, bruising, hypokalemia, hyperglycemia, Cushing's syndrome, sodium and water retention, abdominal distention, ulcerative esophagitis, and immunosuppression (Uptodate.com Lexi Corp, 2008).

NURSING IMPLICATIONS

As with dexamethasone, withdrawal and discontinuation of hydrocortisone should be done slowly, with gradual tapering of the dose (Uptodate.com Lexi Corp, 2008). The nursing implications described earlier for dexamethasone apply to patients receiving hydrocortisone as well.

AGENTS USED TO PREVENT OR CONTROL POSTOPERATIVE DYSRHYTHMIAS

As discussed in Chapter 15, postoperative dysrhythmias are common in cardiac surgery patients. Several potential etiologic factors have been identified, including preexisting cardiac conditions (e.g., pericarditis), atrial infarction, ischemia, or enlargement; respiratory complications; electrolyte imbalance (e.g., hypokalemia, hyperkalemia, hypomagnesemia); surgical trauma (intraoperative injury to the atrium, inadequate cardioprotection during CPB); hypothermia; hyperadrenergic state; acid–base imbalance; anxiety; and pain (Bharucha & Marinchak, 2007). Prior to intervening with pharmacotherapy, any underlying causes should be treated (Massé & Antonacci, 2005). Atrial and ventricular dysrhythmias, as well as bradyarrhythmias and tachyarrhythmias, may be experienced by cardiac surgery patients.

The most common antiarrhythmic medications used in the immediate postoperative phase are categorized as Class I, II, III, or IV agents. Class I agents are sodium channel

Box 12–2 Categories of Antiarrhythmic Therapy

Category	Mechanism of Action
Class I	Sodium channel blockers
Class II	Beta blockers
Class III	Delays repolarization; prolongs action potential
Class IV	Calcium channel blockers

blockers and include quinidine (Quinaglute®), procainamide (Pronestyl®), disopyramide (Norpace®), lidocaine (Xylocaine®), propafenone (Rythmol®), flecanide (Tambocor®), and encainide (Enkaid®). Class II agents are beta blockers and include metoprolol (Lopressor®) and sotalol (Betapace®). Class III agents delay repolarization and include amiodarone (Cordarone®), ibutilide (Corvert®), and sotalol. Class IV agents include the calcium channel blockers diltiazem (Cardizem®) and verapamil (Calan®) (see Box 12–2). The agents used most often for postoperative cardiac surgery patients are discussed here and are summarized in Table 12–4.

Agents Used to Manage Atrial Dysrhythmias

As noted in Chapter 15, AF is a common dysrhythmia that may occur in postoperative cardiac surgery patients. Its reported incidence ranges from 10% to 65% in such patients. The incidence varies with type of procedure performed, with 20–40% of patients who have undergone coronary artery bypass grafting (CABG) procedures, as many as 50% of patients who have undergone valve surgery, and as many as 60% of patients who have undergone combination surgical procedures being affected by this complication. The onset of occurrence is 1–3 days following surgery (Aranki et al., 2008; Khalpey et al., 2008). Given that most patients remain in the ICU for 24 hours or less, AF may not appear until after the patient leaves the ICU.

A wide array of medications is used to treat atrial dysrhythmias after cardiac surgery. The particular medication selected will depend on the drug's mechanism of action, the suspected cause of the dysrhythmia, and the drug's side effect profile.

Prior to initiating treatment of AF, three criteria are considered. First, determination is made as to whether the patient is hemodynamically stable or unstable with the presence of AF. The ICU nurse can identify the presence of hemodynamic compromise by assessing for hypotension, altered mental status, presence of chest pain, shortness of breath, poor peripheral perfusion, decreased urinary output, signs of impaired CO, or increased preload (Khalpey et al., 2008).

Next, precipitating factors should be identified. These conditions may include ischemia, increased sympathetic tone, electrolyte or acid–base imbalance, or pulmonary disorders (Khalpey et al., 2008).

Lastly, the goal of therapy (rate or rhythm control) needs to be decided. The ultimate goal is hemodynamic stability (Khalpey et al., 2008). Agents that may be used to control rate include beta blockers, calcium channel blockers, and possibly digoxin.

Class II Agents

Metoprolol

Metoprolol is currently recommended as first-line therapy for AF. Treatment with a beta blocker or amiodarone (which is discussed later in this chapter) has an efficacy

Table 12–4 Agents Used to Manage Postoperative Dysrhythmias

Dysrhythmia	Agent	Dose	Mechanism of Action
Atrial Fibrillation	Metoprolol	**PO**: Initial dose of 50 mg, followed by additional doses of 25 mg until heart rate is less than 100 beats/min. **IV**: 5–15 mg (usually 5 mg) over 2.5 minutes. Additional doses may be given at 7.5-minute intervals.	Class II cardioselective beta blocker.
	Carvedilol	**PO**: 6.25 mg BID. **Patients with heart failure**: 3.125 mg PO daily.	Class II cardioselective beta blocker.
	Amiodarone	**IV**: 150 mg given over 10 minutes, followed by a 24-hour infusion given at a rate of 1 mg/min for the first 6 hours and at a rate of 0.5 mg/min for the next 18 hours, if required.	Class III but possesses properties in all four categories of agents. Blocks potassium channels, which prolongs the duration of the action potential and decreases membrane excitability. Slows heart rate by depressing SA node. Increases refractoriness of AV node. Decreases impulse conduction by indirectly blocking sodium channels, and blocking beta-adrenergic receptors. Increases atrial and ventricular refractoriness. Inhibits alpha-adrenergic receptors.
	Ibutilide	**IV: For patients who weigh ≥ 60 kg**: 1 mg over 10 min. **For patients who weigh less < 60 kg**: 0.01 mg/kg over 10 min. A second dose of equal strength may be administered over 10 min if conversion does not take place with initial dose.	Class III antiarrhythmic.

continues

Table 12–4 Agents Used to Manage Postoperative Dysrhythmias (continued)

Dysrhythmia	Agent	Dose	Mechanism of Action
Atrial Fibrillation (cont.)	Sotalol	**PO: For AF prevention**: 80 mg BID. Dose may be increased in 3 days to 120 mg and then to 160 mg BID if no QT prolongation.	Classes II and III. Non-cardioselective beta blocker.
	Diltiazem	**IV**: Initial bolus: 0.25 mg/kg over 5-10 min. Subsequent bolus: 0.35 mg/kg over 5–10 min. after 15 minutes if needed. Maintenance infusion: 5–15 mg/hr.	Class IV. Blocks calcium ion influx during depolarization of cardiac and vascular smooth muscle. Decreases vascular resistance and causes relaxation of the vascular smooth muscle, resulting in a decrease in blood pressure. Negative inotropic effect.
	No specific ACE inhibitor or ARB recommendations noted in literature.	Dose is drug dependent.	Agents that act on the Renin–Angiotensin–Aldosterone System. It is not clear how angiotensin inhibition helps prevent development of AF.
	Digoxin	**IV or PO**: Loading dose 0.25 mg every 2 hrs up to a maximum of 1.5 mg. **PO maintenance dose**: is 0.125–0.375 mg daily. **IV maintenance dose**: 0.125–0.25 mg daily.	May control ventricular rate. Slows conduction at the AV node and increases refractory period. Positive inotrope. Decreases sympathetic response and renin–angiotensin system effect.
	Adenosine	**IV Initial dose**: 6 mg rapid IV push followed by 20 mL normal saline. **Subsequent IV doses**: 12 mg IV push followed by 20 mL normal saline. Two subsequent doses may be given.	Transient depression of LV function. Slows SA node impulse formation. Slows conduction through the AV node. Can interrupt reentry pathways through the AV node. Coronary vasodilation.

Table 12–4 Agents Used to Manage Postoperative Dysrhythmias (continued)

Dysrhythmia	Agent	Dose	Mechanism of Action
Atrial Fibrillation (cont.)	Dexamethasone	**Anti-inflammatory IV dose**: 0.75–9 mg over 5–10 min. daily in divided doses every 6 hrs.	Decreases effect of activation of the complement system following CPB. Decreases release of pro-inflammatory mediators.
	Hydrocortisone	**Anti-inflammatory IV dose**: 15–240 mg IV every 12 hrs.	Decreases effect of activation of the complement system following CPB. Decreases release of pro-inflammatory mediators.
Ventricular Dysrhythmias	Amiodarone	**VF, pulseless VT Initial bolus**: 300 mg IV push. A maximum of 2.2 g may be given in 24 hrs. **Continuous infusion**: 1 mg/min for the first 6 hrs and at a rate of 0.5 mg/min for the next 18 hours, if required.	See description in AF.
	Lidocaine	**VF, pulseless VT Initial bolus**: 1–1.5 mg/kg. Subsequent bolus doses: 0.5–0.75 mg/kg every 5–10 min. **Continuous infusion**: 1–4 mg/min. Maximum dose is 3 mg/kg in 24 hrs.	Class I antiarrhythmic agent.
	Sotalol	**PO**: 80 mg BID. If necessary, may increase dose to 240–320 mg/day.	Classes II and III. Non-cardioselective beta blocker.

AF = atrial fibrillation; CPB = cardiopulmonary bypass; VF = ventricular fibrillation; VT = ventricular tachycardia.

Sources: Bharucha & Marinchak, 2007; Engelman et al., 1995; Katz, 2007; Khalpey et al., 2008; Levy et al., 2008; Micromedex Online, 2008; Uptodate.com Lexi Corp, 2008.

rate of 52–65% in terms of reducing the frequency of AF (Aranki et al., 2008).

HEMODYNAMIC EFFECTS

As the name of the category connotes, beta-adrenergic receptor blockers decrease tissue response to catecholamines (especially epinephrine and NE). Beta blockers are classified as either cardioselective or non-cardioselective based on whether they exert their efforts on alpha receptors, $beta_1$ receptors, $beta_2$ receptors, or a combination of these (Levy et al., 2008). Metoprolol is a Class II antiarrhythmic and a cardioselective beta blocker. It has a negative inotropic effect and decreases heart rate, contractility, CO, and blood pressure.

INDICATIONS

Metoprolol is used to decrease the workload of the heart by reducing heart rate and prophylactically to prevent AF or atrial flutter after cardiac surgery. In addition to promoting rate control for AF (Fuster et al., 2006), this drug may be used for rhythm conversion (Khalpey et al., 2008). Beta blockers, because of their mechanism of action, are not particularly effective in the treatment of dysrhythmias related to catecholamine stimulation (Levy et al., 2008). Typically, these agents are used in the period after the initial 24 postoperative hours, as myocardial function has generally improved by that time (St. Andre & DelRossi, 2005).

DOSAGE

Metoprolol may be administered either intravenously or orally. The initial oral dose is 50 mg, followed by additional doses of 25 mg until the patient's heart rate is less than 100 beats/min (Khalpey et al., 2008). The intravenous dose is 5–15 mg (usually 5 mg) administered over 2.5 minutes. Additional doses may be given at 7.5-minute intervals.

SIDE EFFECTS

Side effects of metoprolol include bradycardia, AV block, heart failure symptoms, and widening QRS complexes. Other potential side effects include hypotension and bronchospasm (Lúcio et al., 2004).

NURSING IMPLICATIONS

Valve replacement surgeries make patients prone to heart block; metoprolol is contraindicated in these cases. Metoprolol should be used with caution in patients with COPD and asthma because it may lead to airway constriction. Likewise, it should be used with caution in patients with ongoing pump dysfunction, as immediate correction does not occur postoperatively.

On rare occasions, beta blockers may mask signs of hypoglycemia in patients with diabetes (Levy et al., 2008). Caution should be exercised when administering these agents to patients with hypotension or heart failure (Fuster et al., 2006).

If patients do not convert to normal sinus rhythm or become clinically unstable, synchronized cardioversion (SCV) may be performed once rate control has been achieved. SCV typically will not be effective until rate control has been attained in this patient population, as intense adrenergic activity is occurring at this time. Patients should be considered for anticoagulation therapy despite their postoperative status if AF persists for more than 48 hours (St. Andre & DelRossi, 2005).

Carvedilol (Coreg®)

HEMODYNAMIC EFFECTS

Carvedilol is a non-cardioselective beta blocker. By blocking beta receptors, it decreases heart rate, blood pressure, contractility, and CO, thereby decreasing myocardial workload. This drug also blocks alpha receptors, causing arterial vasodilation.

INDICATIONS

While carvedilol is primarily prescribed in the management of hypertension and heart failure, data suggest that it is more effective in preventing postoperative AF in cardiac surgery patients than either metoprolol or atenolol (Celik, Iyisoy, Celik, Gunay, & Isik, 2008; Merritt, 2003). Its use for atrial dysrhythmias is a non-FDA-labeled indication (Micromedex Online, 2008).

DOSAGE

The dosage of carvedilol for patients with hypertension is 6.25 mg orally twice daily. The dosage for patients with heart failure is 3.125 mg orally daily.

SIDE EFFECTS

Side effects of carvedilol include hypotension, bradycardia, hyperglycemia, dizziness, diarrhea, and fatigue. Serious but rare adverse events that have been reported include heart block, worsening heart failure, asthma with status asthmaticus, Stevens-Johnson syndrome, and aplastic anemia (Micromedex Online, 2008).

NURSING IMPLICATIONS

Carvedilol is contraindicated in patients with second- or third-degree heart block, bronchial asthma, cardiogenic shock, severe sinus bradycardia (if the patient does not have a pacemaker), decompensated heart failure being treated with an inotrope, sick sinus syndrome, or hepatic impairment (Micromedex Online, 2008). It should be used with caution in patients with diabetes, as carvedilol may potentiate insulin-induced hypoglycemia and hinder glucose level upturn. It may also exacerbate hyperglycemia in patients with heart failure. As noted earlier, the drug may mask symptoms of hypoglycemia. There is an increased risk of renal impairment secondary to hypotension in patients with a systolic blood pressure less than 100 mm Hg. Chest pain may develop in patients with Prinzmetal's variant angina. Carvedilol may also cause or exacerbate adrenal insufficiency in those patients with PVD.

The ICU nurse should monitor heart rate and blood pressure while the patient remains on carvedilol therapy. Renal function test data should be evaluated in patients with ischemic heart disease. The ICU nurse should anticipate a decrease with any signs of heart failure or angina (Micromedex Online, 2008).

Class III Agents

Amiodarone

MECHANISM OF ACTION

Amiodarone, while placed in the Class III category of drugs, is unique in that it possesses properties in all four categories of agents described earlier. It creates its antiarrhythmic effect by blocking potassium channels, which prolongs the duration of the action potential and decreases membrane excitability. Amiodarone slows heart rate by depressing sinoatrial (SA) node automaticity (the heart's inherent ability to initiate a beat) and increases refractoriness of the AV node (Levy et al., 2008). This agent also decreases impulse conduction by indirectly blocking sodium channels, blocking beta-adrenergic receptors (causing beta blockade), increasing atrial and ventricular refractoriness, and inhibiting alpha-adrenergic receptors and calcium channels, producing antianginal effects. The vasodilatory effects, including coronary vasodilation, further contribute to its antianginal effects (Brantman & Howie, 2006; Levy et al., 2008). Collectively, these actions make amiodarone one of the most effective antiarrhythmic agents in postoperative cardiac surgery patients (Katz, 2007).

INDICATIONS

Amiodarone is used in postoperative cardiac surgery patients to convert AF, and in the management of VF and VT. It has replaced diltiazem as the treatment of choice for SVT (Levy et al., 2008). Amiodarone is generally recommended if a beta blocker or calcium channel blocker is ineffective in the management of a patient with AF (Fuster et al., 2006).

DOSAGE

The dosage of amiodarone for AF is 150 mg given intravenously over 10 minutes, followed by a 24-hour infusion given at a rate of 1 mg/min for the first 6 hours and at a rate of 0.5 mg/min for the next 18 hours, if required.

SIDE EFFECTS

Amiodarone is associated with a wide array of side effects, including pulmonary and liver toxicity with its long-term use. Myocardial depression and heart block may develop as well. If the drug is administered too quickly, significant hypotension may result (Khalpey et al., 2008).

NURSING IMPLICATIONS

With intravenous administration of amiodarone, a patient's heart rate and blood pressure may decrease. Minimal change is noted in the QRS and QT complexes (QTc), however (Levy et al., 2008). The hypotension seen in postoperative cardiac surgery patients is typically temporary. If the patient experiences sustained or clinically significant bradycardia during that time, the epicardial pacing wires can be used to reestablish a normal heart rate (Katz, 2007).

The ICU nurse should closely monitor patients on amiodarone for AV block, bradycardia, electrolyte imbalance, hypotension, LV dysfunction, and new or worsened arrhythmias. Amiodarone should be used with caution in conjunction with QTc-prolonging drugs (e.g., quinidine, sotalol, erythromycin, and haloperidol) and in the presence of electrolyte imbalance, pulmonary disease, and hepatic disease. Electrolyte imbalance may result in diminished drug efficacy or a dysrhythmia. Patients with hepatic disease will demonstrate decreased drug clearance. Pulmonary toxicity (e.g., acute respiratory distress syndrome, pulmonary fibrosis or infiltrates, and pneumonitis) has been reported as well (Micromedex Online, 2008).

Ibutilide

MECHANISM OF ACTION

Ibutilide is a Class III antiarrhythmic agent (Bharucha & Marinchak, 2007).

INDICATIONS

Ibutilide may be used to attempt to convert AF to normal sinus rhythm if SCV has not proved successful (Bharucha & Marinchak, 2007).

DOSAGE

For patients who weigh at least 60 kg, the dosage of ibutilide is 1 mg. If patients weigh less than this amount, the dosage is 0.01 mg/kg. Either dose is administered over 10 minutes. A second dose of equal strength may be administered over 10 minutes if conversion does not take place upon completion of the initial dose (Bharucha & Marinchak, 2007; Khalpey et al., 2008). The half-life of ibutilide is 2 to 12 hours, with an average of 6 hours (Bharucha & Marinchak, 2007).

SIDE EFFECTS

The primary side effect noted with ibutilide is development of torsade de pointes. The risk of developing torsade de pointes is increased if ibutilide is given in combination with amiodarone (Bharucha & Marinchak, 2007; Khalpey et al., 2008). Other significant side effects include, but are not limited to, monomorphic VT, SVT, hypotension, QT prolongation, AV block, and bradycardia (Bharucha & Marinchak, 2007).

NURSING IMPLICATIONS

Administration of ibutilide is contraindicated in patients with a QT complex greater than 440 milliseconds. The ICU nurse should monitor patients who are receiving this Class III drug for development of QT prolongation, torsade de pointes, and heart block. Prior to and during therapy, patients should have any hypokalemic and hypomagnesemic conditions corrected. Patients should not receive concomitant therapy with another agent that can cause QT prolongation (Bharucha & Marinchak, 2007).

Sotalol

MECHANISM OF ACTION

Sotalol has both Class II and Class III mechanisms of action. As a beta blocker, it is noncardioselective; it affects both $beta_1$ and $beta_2$ receptors. Net effects include a decrease in heart rate and AV node conduction and an increase in AV node refractoriness. As a Class III agent, sotalol prolongs the atrial and ventricular action potentials (Bharucha & Marinchak, 2007).

INDICATIONS

Sotalol was initially indicated to help prevent development of AF after cardiac surgery in patients who were not candidates for the usual beta-blocker therapy. The 2004 ACC/AHA guidelines for postoperative CABG patients, however, downgraded this recommendation, as the efficacy of sotalol in preventing AF is not well established based on the most recent data (Bharucha & Marinchak, 2007).

Sotalol may also be indicated in the treatment of life-threatening ventricular dysrhythmias (Bharucha & Marinchak, 2007; Levy et al., 2008).

DOSAGE

If used for AF prevention, the dosage of sotalol is 80 mg PO BID. This dose may be increased in 3 days to 120 mg and then to 160 mg BID if the response is inadequate and no associated undue QT prolongation (not greater than 520 milliseconds) occurs. The onset of action for sotalol is 1–2 hours, with a peak effect being noted in 2.5 to 4 hours. The duration of effect is 8–16 hours; the half-life is 12 hours (Bharucha & Marinchak, 2007).

SIDE EFFECTS

A number of side effects have been reported in conjunction with administration of sotalol. These include, but are not limited to, bradycardia, chest pain, palpitations, fatigue, dizziness, dyspnea, hypotension, edema, confusion, headache, sleep disturbances, bleeding, upper respiratory problems, nausea, and vomiting (Bharucha & Marinchak, 2007).

NURSING IMPLICATIONS

Monitoring of QT complexes, serum magnesium and potassium levels, and ECG reports is recommended when this drug is used. Sotalol is contraindicated for patients with bronchial asthma, bradycardias (sinus, Mobitz II, or complete heart block, unless the patient has a pacemaker), prolonged QT complex, cardiogenic shock, heart failure, or a creatinine clearance less than 40 mL/min. Patients with diabetes who are receiving sotalol may develop hypoglycemia, although the therapy may mask the symptoms of a decreased serum glucose level. Patients who are receiving concomitant calcium channel blocker therapy should be observed for bradycardia or heart block development. Those receiving concomitant therapy with other agents that can prolong QT complexes are at increased risk for this complication given the additive effect exerted by sotalol (Bharucha & Marinchak, 2007).

Class IV Agents

Two calcium channel blockers sometimes used in postoperative cardiac surgery patients are diltiazem and verapamil. Both slow the

ventricular response in patients with AF, atrial flutter, or SVT (Davison et al., 1985). However, verapamil is seldom used because of its negative inotropic effects (Levy et al., 2008). There are no convincing data that calcium channel blockers prevent development of AF after cardiac surgery, and they are not typically prescribed for that reason. Diltiazem may be administered to help decrease ischemic events. Calcium channel blockers may be effective in controlling the ventricular rate associated with AF, but are reportedly not as effective as beta-blocker therapy (Bharucha & Marinchak, 2007).

Diltiazem

MECHANISM OF ACTION

Diltiazem blocks calcium ion influx during depolarization of cardiac and vascular smooth muscle (Levy et al., 2008). It decreases vascular resistance and causes relaxation of the vascular smooth muscle, resulting in a decrease in blood pressure (Micromedex Online, 2008). A negative inotropic effect occurs as well, as contractile strength is regulated by calcium ions flowing in and out of the cell.

INDICATIONS

Diltiazem is used to lower the ventricular response in AF, atrial flutter, and SVT when the ventricular rate is greater than 100 beats per minute. It may also be used for rhythm conversion (Khalpey et al., 2008). In addition, diltiazem may be used to prevent vasospasm in patients with internal mammary grafts by relaxing vascular smooth muscle and stabilizing the vessel.

DOSAGE

An initial bolus of 0.25 mg/kg, followed by 0.35 mg/kg after 15 minutes if needed, is typically ordered. Doses should be administered over a course of 5–10 minutes. The medication can be slowed or stopped if a reduction in blood pressure is noted or if the target heart rate is reached prior to completing the initial bolus. The bolus should be followed by a continuous infusion at 5–15 mg/hr (Levy et al., 2008); this rate may be titrated to maintain the heart rate within the prescribed parameters. Conversion to normal sinus rhythm is commonly seen after administration of diltiazem; however, the primary goal when this agent is used is rate control.

SIDE EFFECTS

AV block may occur with diltiazem use. This side effect occurs more often in patients who have undergone valve operations, and particularly those involving the mitral valve. In this case, diltiazem therapy should be placed on hold (Mooss et al., 2000). Other side effects commonly observed with this calcium channel blocker include bradycardias, heart failure, peripheral edema, dizziness, and headache. Cardiac dysrhythmias are rare side effects (Micromedex Online, 2008).

NURSING IMPLICATIONS

Diltiazem can be safely administered to patients with marginal blood pressure if it is given slowly. Reduction of heart rate will improve diastolic filling, which will in turn improve CO and reduce myocardial workload after rate control is achieved. If the patient is on vasodilators to treat hypertension, consideration should be given to lowering the dose or stopping the vasodilators altogether prior to administering diltiazem, as co-administration may potentiate a hypotensive effect. When administering diltiazem with adrenergic agonists, the ICU nurse should continuously monitor the patient's heart rate and blood pressure.

Diltiazem may increase the effects of anesthetics. Patients who were taking diltiazem preoperatively may not awaken as quickly or may be more difficult to arouse after receiving this medication during the first 24 hours following cardiac surgery (Micromedex Online, 2008).

Agents Acting on the Renin–Angiotensin–Aldosterone System

The renin–angiotensin–aldosterone system (RAAS) is implicated in the causal pathway of AF. Inhibition of this pathway with ACE inhibitors and ARBs may, therefore, prevent occurrence of AF (Manché et al., 1999). Data from trials and observations suggest that ACE inhibitors and ARBs may decrease the incidence of postoperative AF in patients who have undergone CABG (Arnsdorf & Podrid, 2006). In one meta-analysis, preoperative and postoperative administration of an ACE inhibitor significantly decreased the incidence of postoperative AF following CABG (Mathew et al., 2004). These data are not consistent, however. Other investigators have reported no significant reduction in postoperative AF following cardiothoracic surgery (Coleman, Makanji, Kluger, & White, 2007).

It is not clear how angiotensin inhibition helps prevent development of AF. One hypothesis is that it helps control risk factors for AF, heart failure, and hypertension (Arnsdorf & Podrid, 2006). Another suggested mechanism focuses on the decrease in atrial stretch in the setting of increased left atrial pressure. Administration of ACE inhibitors and ARBs cause improved ventricular function and a decrease in left atrial pressure, thereby decreasing triggers for the development of AF (Webster, Fitzpatrick, Nicholls, Ikram, & Wells, 1985). ACE inhibitors such as enalaprilat and ARBs (e.g., losartan [Cozaar®]) do not currently have an FDA-approved indication for AF, but rather are used on an off-label basis for this condition (Micromedex Online, 2008).

Other Agents to Treat Atrial Dysrhythmias

Digoxin (Lanoxin®)

Digoxin may be effective in controlling the ventricular rate associated with AF, but is reportedly not as effective as beta-blocker therapy for this indication (Bharucha & Marinchak, 2007).

HEMODYNAMIC EFFECTS

Digoxin slows conduction at the AV node and increases the refractory period (Levy et al., 2008). It also has a positive inotropic effect and causes a decreased sympathetic response and renin–angiotensin system effect (Micromedex Online, 2008).

INDICATIONS

Digoxin may be considered in the treatment of postoperative AF in patients with poor ejection fraction, heart failure, or other contraindications to beta-blocker therapy (Fuster et al., 2006; Khalpey et al., 2008). However, neither preoperative nor postoperative administration of digoxin has been shown to decrease the development of AF. Digoxin may add to the effectiveness of beta blockers or calcium channel blockers if monotherapy with either type of agent proves effective for rate control (Bharucha & Marinchak, 2007; Fuster et al., 2006). Oral digoxin is not indicated as monotherapy for treatment of paroxysmal AF (Fuster et al., 2006).

DOSAGE

The loading dose of digoxin for AF is 0.25 mg IV or orally every 2 hours up to a maximum of 1.5 mg. The maintenance dose is 0.125–0.375 mg orally daily or 0.125–0.25 mg IV daily. The half-life of digoxin is typically 38–48 hours but is longer in patients with impaired renal function (Khalpey et al., 2008). Digoxin should be administered over a period of at least 5 minutes (Micromedex Online, 2008).

SIDE EFFECTS

Side effects associated with digoxin include nausea, vomiting, anorexia, diarrhea, headache, and visual disturbances. Cardiac dysrhythmias may also occur (Micromedex Online, 2008).

NURSING IMPLICATIONS

Several drugs (e.g., amiodarone) increase serum digoxin levels (Khalpey et al., 2008). Caution should be exercised when administering digoxin to any patient with acute MI, AV block, electrolyte imbalance (e.g., hypokalemia, hypocalcemia, hypercalcemia, hypomagnesemia), hypoxia, severe bradycardia, heart failure, pulmonary disease, VT, Wolff-Parkinson-White syndrome, renal disease, sick sinus syndrome, or premature ventricular contractions (Micromedex Online, 2008). The ICU nurse should observe the patient's blood pressure, heart rate, and cardiac rhythm during and following administration of digoxin. Evaluation of renal function and electrolyte status should ideally be made prior to administration.

The therapeutic range for digoxin levels is 0.8–2 ng/mL; levels should be obtained immediately prior to administering the next dose of the drug (Micromedex Online, 2008). The determination of digoxin toxicity is based more on the presence of a "dig toxic" rhythm rather than exclusively on blood levels.

Indications for digoxin immune Fab (Digibind®) include presence of ventricular dysrhythmias, bradyarrhythmias, second- or third-degree heart block, or hyperkalemia (greater than 5.0 mEq/L). If the patient has bradycardia and Digibind is not available, atropine may be indicated. Extreme caution should be exercised in patients who have received digoxin and who require cardioversion, as digoxin potentiates the effects of electricity and the patient may develop a "dig toxic" rhythm.

Adenosine (Adenocard®)

MECHANISM OF ACTION

Adenosine produces no negative inotropic effects, although transient depression of LV function does occur. The latter effect slows SA node impulse formation, slows conduction through the AV node, and can interrupt reentry pathways through the AV node. Adenosine produces coronary vasodilation, possibly related to its action on smooth muscle receptors (Micromedex Online, 2008).

INDICATIONS

Adenosine is indicated for the treatment of paroxysmal SVT (Micromedex Online, 2008). The drug is also used on an off-label basis to counteract an etiology of narrow or wide QRS complex supraventricular tachyarrhythmias (Micromedex Online, 2008).

DOSAGE

The initial dose of adenosine is 6 mg delivered via rapid IV administration. This dose should be followed by infusion of 20 mL of normal saline. Administration should take place as close to the hub as possible. Two subsequent doses of 12 mg each (maximum dose) may be administered, if needed. Adenosine has an ultra-short half-life (less than 10 seconds) (Micromedex Online, 2008).

SIDE EFFECTS

Side effects that have been reported following adenosine's termination of the tachycardic rhythm include chest pain, lightheadedness, flushing, nausea, headache, and dyspnea. Serious adverse events that have been reported include bradycardias, dysrhythmias, heart block, and bronchospasm (in patients with asthma) (Micromedex Online, 2008).

NURSING IMPLICATIONS

Prior to administration of adenosine, the ICU nurse should alert the patient to anticipate "a strange sensation." Monitoring of the patient during treatment should include continuous cardiac monitoring and vital signs. A rapid decrease in heart rate with a brief episode (6 seconds) of ventricular asystole should be anticipated. For this reason, emergency resuscitative equipment should be immediately available

during administration of adenosine. A baseline and repeat ECG are also recommended.

Administration of adenosine is contraindicated in patients with second- or third-degree heart block, symptomatic bradycardia, or sick sinus syndrome. Caution should be exercised when administering this agent to patients with hypertension, hypotension, MI, unstable angina, or bronchoconstrictive disorders (Micromedex Online, 2008). It is also recommended that adenosine administration be avoided in heart transplant recipients, revascularized patients, and individuals with AF or atrial flutter (Khalpey et al., 2008).

Dexamethasone (Decadron®)

The use of steroids as a prophylactic measure for development of postoperative AF following cardiac surgery is based on the mechanism of action of steroids and the pathophysiologic changes that occur during the intraoperative and postoperative periods. Activation of the complement system occurs during CPB and during the first few postoperative days. In addition, increasing levels of C-reactive protein supplement activation of the complement system. Activation of the complement system is associated with development of postoperative dysrhythmias (Bruins et al., 1997). Steroid administration has been shown to decrease this effect as well as the release of pro-inflammatory mediators (Engelman et al., 1995).

Data are not consistently supportive of steroid use despite studies suggesting these agents' efficacy. In one study, patients received a preoperative dose of steroids, followed by additional doses for the first 24 postoperative hours. A significant difference in the incidence of postoperative AF was reported between patients who did and did not receive steroids—21% and 51%, respectively (Prasongsukarn et al., 2005).

In another study, patients who underwent CABG, aortic valve replacement, or both had a 33% lower incidence of postoperative AF when they received preoperative and postoperative steroids (Halonen et al., 2007).

MECHANISM OF ACTION

As previously described, steroids decrease inflammation by suppressing neutrophil migration, decreasing production of pro-inflammatory mediators, and reversing the increase in capillary permeability (Uptodate .com Lexi Corp, 2008). Levels of complement, C-reactive protein complex, the number of white blood cells, and the amount of pro-inflammatory mediators—all of which serve as indicators of an inflammatory response—are higher in patients who develop AF as compared with patients who remain in normal sinus rhythm after cardiac surgery (Halonen et al., 2007). It has been suggested that intraoperative inflammation may contribute to the development of AF; dexamethasone, as an anti-inflammatory agent, may help prevent AF from developing (Uptodate.com Lexi Corp, 2008).

INDICATIONS

Prophylactic administration of dexamethasone may prevent development of AF associated with cardiac surgery.

DOSAGE

While not recommended specifically for prevention of postoperative AF, the anti-inflammatory dose of dexamethasone is 0.75–9 mg/day, given in divided doses every 6 hours. The drug should be administered as an IV bolus over 5–10 minutes (Uptodate.com Lexi Corp, 2008).

In one study, patients received 1 g of methylprednisolone preoperatively. This initial dose was followed by dexamethasone 4 mg every 6 hours for 24 hours following surgery (Prasongsukarn et al., 2005).

SIDE EFFECTS

A number of clinically significant adverse reactions have been reported to be associated with dexamethasone administration. These side effects were listed in the section addressing management of postoperative hypotension.

NURSING IMPLICATIONS

Because steroids have a large number of physiologic effects, additional data are necessary before recommending their routine use in prevention of postoperative cardiac surgery dysrhythmias. Withdrawal and discontinuation of a corticosteroid should be done slowly, with gradual tapering of the dose (Uptodate.com Lexi Corp, 2008). The other nursing implications described in the section on hypotension apply here as well.

Hydrocortisone (Solu-Cortef ®)

MECHANISM OF ACTION

As described earlier, steroids decrease inflammation by suppressing neutrophil migration, decreasing production of pro-inflammatory mediators, and reversing the increase in capillary permeability (Uptodate.com Lexi Corp, 2008). Levels of complement, C-reactive protein complex, the number of white blood cells, and the amount of pro-inflammatory mediators—all of which are indicators of an inflammatory response—are higher in patients who develop AF as compared with patients who remain in normal sinus rhythm after cardiac surgery (Halonen et al., 2007).

INDICATIONS

Prophylactic administration of hydrocortisone is intended to prevent development of AF associated with cardiac surgery.

DOSAGE

When administered as an anti-inflammatory agent, the recommended dose is 15-240 mg IV every 12 hours (Uptodate.com Lexi Corp, 2008). In one study, patients received 100 mg of hydrocortisone preoperatively. This was followed by 1 mg every 8 hours for 3 days (Halonen, et al., 2007).

SIDE EFFECTS

Side effects that are reported as being related to administration of hydrocortisone are delineated in the section addressing postoperative hypotension.

NURSING IMPLICATIONS

As with dexamethasone, withdrawal and discontinuation of hydrocortisone should be done slowly, with gradual tapering of the dose (Uptodate.com Lexi Corp, 2008). The other nursing implications described earlier apply here as well.

Agents Used to Treat Postoperative Ventricular Dysrhythmias

VF, VT, and torsade de pointes may occur after heart surgery. The development of paroxysmal VT is common following cardiac surgery—its incidence ranges from 17% to 97%. This dysrhythmia is usually an indication of intraoperative ischemia–reperfusion injury, electrolyte abnormalities (e.g., hypokalemia and hypomagnesemia), or an increase in sympathetic stimulation (Aranki et al., 2008; Khalpey et al., 2008).

The incidence of sustained VT, VF, and torsade de pointes ranges from 1% to 3% (Aranki et al., 2008). The presence of sustained VT (i.e., VT lasting for more than 30 seconds or associated with significant hemodynamic compromise) requires a more aggressive management approach. In addition to treating the underlying cause, administration of inotropes should be minimized. Pharmacologic intervention with amiodarone or lidocaine may be implemented. SCV should be performed if sustained VT causes significant compromise (Khalpey et al., 2008).

VF should be treated with immediate defibrillation. Following the initial shock, car-

diopulmonary resuscitation should be performed. This procedure should be followed by administration of epinephrine and an antiarrhythmic such as amiodarone or lidocaine (American Heart Association, 2006).

Torsade de pointes is usually associated with an intraoperative MI in patients with risk factors such as intraoperative hemodynamic instability, increased sympathetic activity, or metabolic derangements (Aranki et al., 2008). It is often terminated by correcting low magnesium levels or by removing the causative agent (if the dysrhythmia was initiated in response to a medication that causes prolongation of the QT complex).

Amiodarone

Amiodarone was discussed earlier in the section on atrial dysrhythmias. The dosage for VF or pulseless VT is an initial bolus of 300 mg IV push. A maximum dose of 2.2 g may be administered in 24 hours. If indicated, a continuous infusion may be initiated.

Lidocaine

MECHANISM OF ACTION

Lidocaine is a Class I antiarrhythmic agent. It controls cardiac rate and rhythm by blocking the sodium channels, thereby decreasing the duration of the action potential (Katz, 2007; Levy et al., 2008). It also blocks the initiation and conduction of nerve impulses by decreasing membrane permeability to sodium ions. This latter effect inhibits depolarization and blocking of conduction (Uptodate.com Lexi Corp, 2008), which includes slowing conduction in the ischemic myocardium (Levy et al., 2008). Lidocaine also inhibits automaticity of conduction tissue by intensifying the electrical stimulation threshold of the ventricle and the His–Purkinje system.

INDICATIONS

Lidocaine is indicated for management of VF or pulseless VT following defibrillation, cardiopulmonary resuscitation, and epinephrine (Uptodate.com Lexi Corp, 2008).

DOSAGE

For VF or pulseless VT, the initial dose is 1–1.5 mg/kg. Additional bolus doses may be administered every 5–10 minutes at a dose of 0.5–0.75 mg/kg. The bolus dose is followed by an infusion of 1–4 mg/min if indicated. The maximum dose in a 24-hour period is 3 mg/kg.

The onset of action for a bolus dose of lidocaine is 45–90 seconds. The duration of action is 10–20 minutes (Uptodate.com Lexi Corp, 2008).

SIDE EFFECTS

A number of side effects have been reported related to lidocaine administration. These effects include dysrhythmias, bradycardia, cardiovascular collapse, heart block, hypotension, seizures, somnolence, slurred speech, drowsiness, confusion, dizziness, metallic taste, bronchospasm, dyspnea, and respiratory depression or arrest (Uptodate.com Lexi Corp, 2008).

NURSING IMPLICATIONS

Patients need to receive continuous cardiac monitoring while receiving lidocaine therapy. Lidocaine should be used with caution in patients with severe liver dysfunction, as this condition increases the risk of lidocaine toxicity. Caution should also be taken when lidocaine is administered to patients with Wolff-Parkinson-White syndrome, heart failure, hypovolemia, shock, or severe respiratory depression. Lidocaine is contraindicated for patients with severe heart block unless the patient has a pacemaker (Uptodate.com Lexi Corp, 2008). Patients should be monitored for development of neurologic toxicity during therapy.

Sotalol

Sotalol was discussed previously in the section addressing management of atrial

dysrhythmias. The initial dosage for management of ventricular dysrhythmias is 80 mg given orally on a twice-daily basis. If necessary, this dose may be increased to 240–320 mg/day. It is suggested to allow 3 days between dose adjustments to allow for a steady-state plasma concentration to be attained and to monitor QT intervals. Most patients require 160–320 mg/day in divided doses to help ensure effectiveness. However, for patients with refractory ventricular dysrhythmias, doses as high as 480–640 mg/day may be required. When these higher doses are considered, clinicians should determine that the potential benefits of sotalol administration outweigh the risks (Uptodate.com Lexi Corp, 2008).

Agents to Treat Postoperative Bradycardia

As described in Chapter 15, bradycardia and heart blocks may develop postoperatively after cardiac surgery (Brister & Lenkei-Kerwin, 2005). Cold cardioplegia, valve repair, hypothermia, MI, medications (e.g., beta blockers, calcium channel blockers, digoxin, or amiodarone), and surgical trauma near the SA and AV nodes may all be causative factors. The most common bradyarrhythmias include complete heart block, sinus node dysfunction, and junctional rhythms (Aranki et al., 2008).

A normal rate is typically restored within 24–48 hours after cardiac surgery, with epicardial, transvenous, or transcutaneous pacing being used in the interim (Khalpey et al., 2008; Zevola, Raffa, & Brown, 2002). Some patients require permanent pacemaker placement after surgery to maintain a normal rate and rhythm (Morris & St. Claire, 1999). It is important to aggressively treat bradycardia or heart blocks that depress cardiac function; otherwise, they may quickly lead to cardiogenic shock.

Agents to Treat Electrolyte Imbalances

Correcting electrolyte imbalances is paramount in preventing and correcting all dysrhythmias. The goal of treatment is to lower the heart rate, thereby reducing the workload on the heart and promoting conversion back to sinus rhythm as soon as possible. As described in Chapter 17, numerous factors related to cardiac surgery put the patient at risk for developing postoperative acid–base and electrolyte disturbances. These factors include anesthesia, induced hypothermia, physiologic effects of CPB techniques, shock resulting in renal insult, cardioplegia, rapid fluid and electrolyte shifts across fluid compartments following CPB, stress associated with surgery, intraoperative volume repletion, hemodilution, and the rewarming process that follows hypothermia (Margereson, 2003; Pezzella, Ferraris, & Lancey, 2004). An in-depth discussion of the management of the common electrolyte imbalances experienced by postoperative cardiac surgery patients appears in Chapter 17.

OTHER AGENTS THAT MAY BE REQUIRED IN POSTOPERATIVE CARDIAC SURGERY PATIENTS

Naloxone (Narcan®)

MECHANISM OF ACTION

Naloxone is an opioid antagonist. It has the greatest affinity for the mu receptor but competes for the mu, kappa, and sigma opiate receptor sites in the central nervous system (Micromedex Online, 2008).

INDICATIONS

Naloxone may be indicated if hypoventilation is present following narcotic administration. When this problem occurs, small doses of naloxone should be enough to stimulate respiration.

DOSAGE

For reversal of opioid-induced respiratory depression, the dose is 0.4–2 mg IV. It may be repeated every 2 to 3 minutes as needed until the desired effect is achieved (Micromedex Online, 2008).

SIDE EFECTS

Side effects associated with use of naloxone include cardiac dysrhythmias, hypertension, hypotension, VF, pulmonary edema, and hepatotoxicity (Micromedex Online, 2008).

NURSING IMPLICATIONS

Care should be taken to closely monitor for continued hypoventilation owing to the relatively short half-life of naloxone as compared to some narcotics. The dose should be titrated to patient effect. The ICU nurse should monitor blood pressure, heart rate, and respiratory rate following administration of naloxone. In addition, a decline in opioid medication effects should be anticipated (Micromedex Online, 2008).

PROPHYLACTIC ANTIBIOTICS

Data suggest a 50% incidence of postoperative infection in patients who have undergone CPB and who did not receive prophylactic antibiotic therapy. Administration of prophylactic antibiotics preoperatively and continuing for 24 hours postoperatively significantly decreases postoperative infection rates. No added benefit is reported when antibiotics are continued for additional time. Antibiotic selection is area and facility specific (Salenger et al., 2003).

AGENTS USED TO CONTROL POSTOPERATIVE BLEEDING

As discussed further in Chapter 13, the incidence of excessive postoperative bleeding (defined as loss of more than 500 mL of blood in the first postoperative hour) following cardiac surgery is reported to range from 3% to 14% (Bowman et al., 2008). Postoperative bleeding may be surgical in origin, related to platelet dysfunction from exposure to CPB circuitry, or attributable to inadequate heparin reversal at the end of CPB (Mullen-Fortino & O'Brien, 2008; Talmor & Lisbon, 2005). Depending on the etiology of the bleeding, pharmacologic intervention may be warranted.

Protamine Sulfate

MECHANISM OF ACTION

Protamine sulfate combines with heparin to form an inactive salt. This salt has no anticoagulation activity (Caravati, 2004).

INDICATIONS

Protamine sulfate is indicated for patients with a postoperative coagulopathy that is due to inadequate heparin reversal (Katz, 2007).

DOSAGE

The dosage is 1 mg of protamine sulfate for every 100 units of heparin that needs to be reversed, up to a maximum dose of 50 mg. This dose is administered over 10 minutes. Heparin is neutralized within 5 minutes of administration and the effect lasts for 2 hours (Katz, 2007).

SIDE EFFECTS

Side effects of protamine sulfate administration include hypotension, elevated PAP (from pulmonary vasoconstriction secondary to a non-immunologic reaction), bradycardia, and non-cardiogenic pulmonary edema (Katz, 2007; St. Andre & DelRossi, 2005).

A protamine reaction may manifest in any of several ways. If this medication is administered too quickly, a Type I reaction occurs:

Hypotension develops as a result of histamine release, with resultant decreases in SVR and PVR. These effects can be reversed with administration of an alpha-receptor agonist.

A Type II reaction is either an anaphylactic or anaphylactoid reaction with associated hypotension, tachycardia, bronchospasm, flushing, and pulmonary edema. This reaction is often related to immunoglobulin E or G (IgE or IgG) causing release of histamine, leukotrienes, and kinins. The release of these substances results in capillary leak, hypotension, and pulmonary edema. Type II reactions may occur within the first 10–20 minutes (or more) following administration of protamine.

A Type III reaction causes catastrophic pulmonary vasoconstriction, with associated increases in pulmonary artery pressure, hypotension (secondary to peripheral vasodilation), decreased left atrial depression, right ventricular dilation, and myocardial depression. This kind of reaction is hypothesized to result from activation of various mediators of the inflammatory response. Complement activation leads to leukocyte aggregation, which causes pulmonary edema; the arachidonic acid pathway stimulates production of thromboxane, which causes constriction of pulmonary vasculature. The latter effect subsides in approximately 10 minutes (Bojar, 2004).

NURSING IMPLICATIONS

Patients with allergies to fish have a high risk of the anaphylactoid type of protamine reaction, as protamine sulfate is made of a protein found in fish sperm. In addition, caution should be used when administering protamine to men who are infertile or who have undergone a vasectomy, as anti-protamine antibodies may be present in these individuals (Katz, 2007). There is also a 30- to 50-fold increased risk of protamine reaction in patients who take NPH insulin (Bojar, 2004).

While a protamine reaction is more likely to occur in the OR, administration of protamine also may take place in the ICU if the patient experiences inadequate heparin reversal. If a protamine reaction occurs, in addition to administration of an alpha-receptor agonist to increase SVR, management strategies that may be implemented include administration of the following therapies: 500 mg intravenous calcium chloride to increase SVR and promote contractility; an inotropic agent (e.g., low-dose epinephrine, dobutamine, inamrinone, milrinone) to decrease PVR; a vasodilator (e.g., nitroglycerin, nitric oxide) to decrease preload and PVR; aminophylline to manage wheezing; and heparin to reverse a protamine reaction (Bojar, 2004).

Recombinant Activated Factor VII (NovoSeven®)

MECHANISM OF ACTION

Recombinant activated factor VII activates the extrinsic pathway of the coagulation system. This action stimulates the generation of thrombin and leads to a subsequently rapid correction of the patient's prothrombin time (Khalpey et al., 2008). Factor VII also expedites platelet activation and ultimate fibrin clot formation (Bowman et al., 2008).

INDICATIONS

As described further in Chapter 13, recombinant activated factor VII may prove helpful in achieving hemostasis in cardiac surgery patients, thereby reducing their transfusion requirements (Enomoto & Thorborg, 2005). It is administered to patients with clotting deficiencies or antibodies to replacement of factors. Factor VII is used frequently in an off-label manner following cardiac surgery—in this case, postoperatively in coagulopathic patients who received blood and blood products. More data are needed before recommending its widespread use in the cardiac

surgery population, however (Katz, 2007). If this clotting factor is administered to cardiac surgery patients who do not have hemophilia, factor VII deficiency, or factor VIII deficiency, this practice would be considered off-label use at this time (Micromedex Online, 2008).

DOSAGE

No dosage data are available that are specifically applicable to the cardiac surgery patient without a history of hemophilia, factor VII deficiency, or factor VIII deficiency. The smallest possible dose or a single dose of 2.4–4.8 mg or 45 mcg/kg is suggested for consideration (Johnson, Ross, & Moores, 2007). In one study, when 1.2 mg was administered to postoperative cardiac surgery patients, significant improvements in intractable bleeding were reported (Romagnoli et al., 2006). Administration takes place via a slow IV push.

SIDE EFFECTS

Serious adverse effects that have been reported with recombinant activated factor VII use include ischemic heart disease, MI, SVT, arterial thromboembolism, bleeding, coagulopathies, venous thromboembolism, cerebral artery occlusion, cerebral ischemia, acute renal failure, and pulmonary embolism (Micromedex Online, 2008).

NURSING IMPLICATIONS

The ICU nurse must carefully observe for and anticipate thromboembolic complications when the patient receives recombinant activated factor VII (Katz, 2007). Monitoring of coagulation profile results (i.e., prothrombin time, activated partial thromboplastin time, platelets, and international normalized ratio), obtaining factor VII levels, and assessing for decreased postoperative bleeding are all steps that should be taken to determine the efficacy of this treatment. Caution should be exercised when administering recombinant activated factor VII to patients with advanced atherosclerotic disease, coagulopathies, or septicemia because of the increased risk of thrombotic events associated with use of this medication (Micromedex Online, 2008).

Aminocaproic Acid (Amicar®)

MECHANISM OF ACTION

Aminocaproic acid is an anti-fibrinolytic agent. It works by preventing plasminogen from binding to fibrin, thereby stopping the activation of plasmin and preventing clot breakdown (Katz, 2007).

INDICATIONS

Aminocaproic acid is indicated for patients with postoperative bleeding that occurs secondary to fibrinolysis.

DOSAGE

The dose of aminocaproic acid is 4–5 g administered over 1 hour, followed by a continuous infusion at a rate of 1 g/hr for 8 hours or until bleeding is controlled (Katz, 2007).

SIDE EFFECTS

Side effects of aminocaproic acid include thrombocytopenia, dysrhythmias, and thrombosis formation; all of these side effects are rare (Katz, 2007). Reported serious adverse events include bradyarrhythmias, hypotension, renal failure, and rhabdomyolysis (Micromedex Online, 2008).

NURSING IMPLICATIONS

Administration of aminocaproic acid is contraindicated in patients with coagulopathies. This medication should be used with caution in patients with cardiac, hepatic, or renal insufficiency. A definitive diagnosis of primary fibrinolysis must be made before administering aminocaproic acid.

Aminocaproic acid should not be administered rapidly. The ICU nurse should monitor the patient's complete blood count and coagulation profile prior to and after therapy. Evaluation for bradycardia, hypotension, thrombosis, dyspnea, pulmonary embolism, and renal function tests should be conducted as well (Micromedex Online, 2008).

SUMMARY

Patients who undergo cardiac surgery may develop several alterations in their hemodynamic profile and their cardiac rate and rhythm in the immediate postoperative period. Alterations in preload, afterload, and CO may be treated with a variety of agents, each of which has its own side effect profile. An understanding of the pharmacologic agents used in the immediate postoperative period is essential. Part of the role of the ICU nurse is to stay current with data regarding pharmacologic agents used in the management of postoperative cardiac surgery patients. Implementation of recommendations published in updates and Black Box Warnings issued by the Food and Drug Administration (FDA) is essential to help assure patient safety and optimal patient outcomes (see Table 12–5). The ICU nurse must be vigilant in managing the complexities associated with administration of these agents and use clinical judgment to help ensure optimal patient outcomes.

Table 12–5 Black Box Warnings Issued by the FDA

Agent	Black Box Warning
amiodarone HCL (Cordarone®)	Grapefruit juice decreases absorption of the medication. Skin can turn a gray-blue color. Regular blood work for thyroid problems should be planned.
beta blockers (oral dosage forms): metoprolol, (Lopressor®)	Abrupt withdrawal may result in angina pectoris, the occurrence of MI, and ventricular arrhythmias. Gradual reduction over several weeks is recommended.
ibutilide (Corvert®)	Continuous ECG monitoring and personnel trained in identification and treatment of acute ventricular arrhythmias are required due to the potential for fatal arrhythmias. Patients with atrial fibrillation of more than 2–3 days must be adequately anticoagulated for ≥ 2 weeks.
nitroprusside (Nipride®)	Medication must be diluted. Frequent blood pressure monitoring required due to hypotension. Cyanide toxicity can occur; therefore monitoring of acid-base balance and venous oxygen concentration is needed.
norepinephrine (Levophed®), dopamine (Intropin®)	Infiltration requires the use of phentolamine mesylate (Regitine®) as soon as possible to treat extravasation.
phenylephrine (Neosynephrine®)	The complete contents of the package insert should be reviewed prior to prescribing.
sotalol (Betapace®)	ECG monitoring required when starting medication. Adjust dosage in renal impairment. Creatinine clearance should be calculated prior to dosing.

Source: www.fda.gov.

SELF-ASSESSMENT QUESTIONS

1. Which of the following agents would be most useful to treat postoperative hypertension in a cardiac surgery patient with a labile blood pressure?
   a. Nitroprusside
   b. Methylene blue
   c. Nitroglycerin
   d. Esmolol
2. Which of the following should the ICU nurse anticipate when caring for a patient receiving dobutamine?
   a. Coronary ischemia
   b. Increased myocardial oxygen consumption
   c. Increased afterload
   d. Methemoglobinemia
3. Your patient is receiving methylene blue. You note a decrease in SpO_2 on the pulse oximeter. Which of the following actions is indicated?
   a. Increase the FIO_2
   b. Immediately stop the infusion
   c. Obtain an arterial blood gas
   d. Collaborate with the physician for nitric oxide administration
4. Your postoperative cardiac surgery patient develops atrial fibrillation. Metoprolol is prescribed. Which of the following conditions should prompt the ICU nurse to question the order?
   a. The patient underwent a combined CABG and valve repair procedure.
   b. The patient has a first-degree AV block with a PR interval of 0.28 second.
   c. The patient has a cardiac index of 2.7 L/min/m^2 on a milrinone infusion.
   d. The patient was taking digoxin preoperatively.
5. Which of the following agents should be considered for rate control in a patient with atrial fibrillation with a rapid ventricular response and a history of a 30% ejection fraction?
   a. Digoxin
   b. Metoprolol
   c. Diltiazem
   d. Amiodarone
6. You are caring for a patient with a failing right ventricle and elevated pulmonary vascular resistance. Which of the following agents is indicated?
   a. Nitroglycerin
   b. Milrinone
   c. Nitroprusside
   d. Nicardipine
7. Which of the following measurements should indicate to the ICU nurse the need to temporarily hold off on administering nitroprusside to a patient with hypertension?
   a. SVR 1600 dyne/sec/cm^{-5}
   b. PAOP 10 mm Hg
   c. MAP 95 mm Hg
   d. CVP 1 mm Hg
8. Which of the following arterial blood gas results should indicate to the ICU nurse the presence of a complication related to nitroprusside administration?
   a. 7.25/pCO_2 42/pO_2 73/SaO_2 93%/HCO_3 17
   b. 7.49/pCO_2 31/pO_2 81/SaO_2 95%/HCO_3 25
   c. 7.50/pCO_2 39/pO_2 68/SaO_2 91%/HCO_3 31
   d. 7.25/pCO_2 52/pO_2 70/SaO_2 93%/HCO_3 23
9. You are caring for a patient with advanced aortic stenosis and postoperative hypertension. The ICU nurse should question use of which of the following medications?
   a. Nitroprusside
   b. Nicardipine
   c. Clevidipine
   d. Enalaprilat

10. Your postoperative cardiac surgery patient is receiving an epinephrine infusion. For which of the following conditions should the ICU nurse observe?
    a. Respiratory acidosis
    b. Hyperglycemia
    c. Adrenal insufficiency
    d. Afterload reduction

Answers to Self-Assessment Questions

1. d
2. a
3. c
4. b
5. a
6. b
7. d
8. a
9. b
10. b

Clinical Inquiry Box

Question: What is the postoperative impact of intraoperative administration of catecholamines to adult elective cardiac surgery patients based exclusively on the clinical judgment of the anesthesiologist?

Reference: Fellahi, J.-L., Parienti, J.-J., Hanouz, J.-L., Plaud, B., Riou, B., & Quattara, A. (2008). Perioperative use of dobutamine in cardiac surgery and adverse cardiac outcome. *Anesthesiology, 108*(6), 979-987.

Objective: To determine the clinical outcome of catecholamine administration based on clinical judgment. The endpoints for this study were major cardiac morbidity and mortality.

Methods: Consecutive patients (nonrandomized; $n = 657$) were divided into two groups. One group received catecholamines; the other (control group) did not. Ninety percent of the patients who received catecholamines had also received dobutamine. The endpoint of major cardiac morbidity was defined as any one of the following conditions: (1) sustained ventricular arrhythmia that required treatment; (2) need for postoperative intra-aortic balloon pump therapy; or (3) postoperative myocardial infarction. The endpoint of overall mortality was defined as death during the patient's hospitalization.

Results: Of the 657 patients, 84 (13%) received catecholamines either intraoperatively or during the first few postoperative hours. The incidence of major cardiac morbidity was 30% among the patients who received catecholamines compared with 9% among the patients who were in the control group. No patients required intra-aortic balloon pump therapy. A clinically significant relationship between catecholamine administration and ventricular arrhythmias was identified. The relationship between catecholamine administration and myocardial infarction was not statistically significant. The authors noted, however, that only low-risk patients were evaluated in this study. The overall mortality in the patients who received catecholamines was 8%, as compared with 1% in the control group. The relationship between catecholamine administration and mortality did not achieve statistical significance.

Conclusion: Intraoperative administration of dobutamine to low-risk cardiac surgery patients who undergo cardiopulmonary bypass, when use of that therapy is based solely on the clinical judgment of the cardiac anesthesiologist, is associated with major cardiac morbidity. ICU nurses caring for postoperative cardiac surgery patients who received intraoperative catecholamines should anticipate the possibility of a major cardiac event in the immediate postoperative period.

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WEB RESOURCES

Federal Drug Administration: http://www.fda.gov/

Safety-related drug labeling changes: http://www.fda.gov/medwatch/safety.htm

FDA Safety News (recalls and safety alerts and preventing medical errors): http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/psn/index.cfm

Adverse event reporting system (case reports): http://www.fda.gov/cder/aers/extract.htm

Herbal–drug interaction poster: http://www.coloradopatientsafety.org/Herbal-Drug-Poster.pdf

Scientific Review of Alternative Medicine: http://www.sram.org/

National Center for Complementary and Alternative Medicine: http://nccam.nih.gov/

Clinical drug trials (lists studies by drug intervention): www.clinicaltrials.gov

Micromedex Online: www.micromedex.com

Chapter 13

Postoperative Complications of Cardiac Surgery and Nursing Interventions

Beverly Jones

INTRODUCTION

More than 600,000 coronary artery bypass grafting (CABG) and valve procedures are performed in the United States annually (Silvestry, 2008). Patients who become candidates for cardiac surgery present with a number of comorbidities. Some of these comorbidities are directly related to the need for surgery, whereas others are attributable to age or other noncardiac issues. An extensive presurgical evaluation should always be performed, as discussed in Chapter 4. From these data and the patient's condition, management of the patient's complex problems may take place before, during, and following cardiac surgery.

Despite the trend toward cardiac surgery being performed on older persons and those with more complex health issues, the 30-day mortality associated with these types of procedures continues to decline (Brown et al., 2008). The overall mortality rate is now reported to be less than 5%. For patients undergoing their first cardiac surgery procedure, the mortality rate is 1–2%, even among those patients who experience postoperative complications (St. Andre & DelRossi, 2005).

An important focus for the patient undergoing cardiac surgery is an assessment of cardiac risk. Calculation of risk potential affords patients and their families insight into the risk of complications and possible mortality of the surgical procedure. It also heightens the healthcare team's awareness of the high-risk patient for whom more aggressive therapy may be warranted and alerts caregivers to the potential for postoperative complications (Adams, Filsoufi, & Antman, 2005). This chapter describes the most common postoperative complications associated with cardiac surgery and the ICU nursing management of these complications.

RISK FACTORS FOR POSTOPERATIVE COMPLICATIONS

The incidence of postoperative complications after cardiac surgery is reported according to the specific complication, presence of comorbidities, and patient-related factors (e.g., age). Multiple cardiac surgery databases have been developed that compile information and allow for calculating risk factors for postoperative complications. Table 13–1 lists the most common risk factors that have been shown to have predictive value for postoperative complications and higher rates of mortality.

In one study, risk of predetermined complications was evaluated among participants of the Medicare program. Patients who sustained postoperative cardiac surgery complications tended to be older than 75 years, female, and non-Caucasian, and had a history

Table 13–1 Risk Factors of Postoperative Complications

Older age (greater than 65 years)
Emergent need for procedure (versus urgent need)
Reoperations
Preexisting preoperative comorbidities: renal dysfunction (dialysis dependent), COPD, diabetes, cerebrovascular disease, peripheral vascular disease, heart failure, acute myocardial infarction, pulmonary hypertension
CABG-valve surgery
Low ejection fraction
Pulmonary dysfunction
Preoperative use of intra-aortic balloon pump

Sources: Grover, Shroyer, & Hammermeister, 1996; Kurki &, Kataja, 1996; Tu, Jaglal, & Naylor, 1995; Tuman, McCarthy, March, Najafi, & Ivankovich, 1992.

of heart failure, COPD, acute myocardial infarction (MI), cardiogenic shock, atrial fibrillation (AF) or flutter, ventricular fibrillation (VF), nonsustained tachycardia, valve disease, or type I diabetes. The overall incidence of complications was 13.64%; 10.99% of patients developed one complication, and 2.64% developed two or more complications (Brown et al., 2008).

Another study evaluated older patients (mean age of 79 years) with an elevated body mass index (BMI). The incidence of postoperative complications was 23.1% in this patient group (Maurer, Luchsinger, Wellner, Kukuy, & Edwards, 2002).

Complications of cardiac surgery have negative and variable effects on patient outcomes. They can occur as a result of change in cardiac performance from preexisting comorbidities, preoperative preparation for surgery, the surgical procedure itself, intraoperative care, or any combination of these factors (St. Andre & DelRossi, 2005).

Postoperative complications may occur secondary to patient comorbidities, the surgical procedure, or cardiopulmonary bypass (CPB). Examples of the latter two causes are effects of anesthesia, transient left ventricular (LV) dysfunction, increased capillary permeability, hypothermia, bleeding, and infection (Silvestry, 2008).

Advances in surgical management, critical care, technology, understanding of CPB hypothermia, and pharmacologic management have positively influenced postoperative management and outcomes, allowing for more complex surgeries, operations on older persons, and consideration of patients with multiple comorbidities as surgical candidates. Data further suggest that postoperative strict glycemic control is correlated with lower morbidity and mortality rates (Talmor & Lisbon, 2005).

Despite all the improvements in cardiac surgery, a few complications continue to be associated with a mortality rate of 50% or greater. Even as ever more technological advances emerge, it is important to identify the deleterious side effects of CPB, circulatory arrest, hypothermia, and aortic cross-clamping, as they can cause physiologic abnormalities in major organs that may persist in the postoperative period. Knowledge and early identification of potential postoperative complications are essential to successful patient outcomes from cardiac surgery.

CARDIAC COMPLICATIONS

Adequate cardiac function is the most pivotal factor associated with recovery from cardiac surgery. Patients with low cardiac output (CO) have a higher mortality risk. Hence, the ICU nurse plays a vital role in preventing or identifying and then treating cardiac complications.

Hemodynamic compromise in the cardiac surgery patient is challenging to manage, as the status of such patients tends to be labile in the immediate postoperative period. The etiology of hemodynamic compromise is multifactorial. It may be caused by the patient's

underlying cardiac disease, factors that affect CO (e.g., filling pressures, dysrhythmias), decreased ventricular compliance, loss of vasomotor tone, increased capillary permeability, excessive bleeding, increased urinary output, inflammatory responses to CPB, poor myocardial protection during aortic cross-clamping, pulmonary edema, cardiac tamponade, or ventricular dysfunction. The factor that influences decreased cardiac performance most in the immediate postoperative period, however, is the underlying preoperative cardiac pathology. Even though surgery has been performed, the patient will not experience an immediate improvement in contractility (Salenger, Gammie, & Vander Salm, 2003).

In addition to the inflammatory response of CPB, data suggest that a release of inflammatory mediators occurs in patients who undergo cardiac surgery without bypass, contributing to these individuals' postoperative hemodynamic instability. Secretion of prostaglandins and other pro-inflammatory mediators (cytokines) stimulates release of nitric oxide, leading to profound vasodilation (Scollan-Boring, 2005). Nitric oxide causes resistance to vasopressors by preventing vessels of some patients from vasoconstricting. A decrease in vasopressin levels further contributes to a relative hypovolemia despite normal intravascular volume (Bridges & Dukes, 2005).

When decreased ventricular function is present, compensatory mechanisms such as sympathetic nervous system (SNS) stimulation and endogenous catecholamine production cause an increase in heart rate, contractility, and vasoconstriction. In turn, both preload and afterload increase (Talmor & Lisbon, 2005). Initially, these compensatory factors will improve CO and blood pressure, albeit usually at the cost of increasing myocardial oxygen consumption, which can exacerbate myocardial ischemia. The compensatory mechanisms are temporary, however; when they are exhausted, poor tissue perfusion will ensue. Initial signs of poor tissue perfusion include tachycardia, diminished peripheral pulses, delayed capillary refill time (CRT), decreased urinary output, hypotension, and (possibly) metabolic acidosis (Margereson & Riley, 2003).

One of the primary foci of caring for a postoperative cardiac patient is obtaining a balance between oxygen supply and demand so that oxygen delivery to the tissues can be enhanced without stressing the heart as it recovers from surgery (Khalpey, Ganim, & Rawn, 2008). Optimal hemodynamic parameters in a postoperative cardiac surgery patient include a CI of greater than 2 L/min/m^2, pulmonary artery occlusive pressure (PAOP) of approximately 15 mm Hg, central venous pressure (CVP) less than 15 mm Hg, mean arterial pressure (MAP) greater than 65 mm Hg, systolic blood pressure (SBP) in the range of 90–140 mm Hg, left atrial pressure (LAP) in the range of 5–15 mm Hg, systemic vascular resistance index in the range of 1400–2800 dyne/sec/cm^{-5}/m^2, and heart rate less than 100 bpm. The patient should also have warm, well-perfused extremities and urine output greater than 0.5 mL/kg/hr. These goals, however, should be individualized based on the patient's comorbidities and clinical status (Khalpey et al., 2008; Salenger et al., 2003).

Low Cardiac Output

To help ensure oxygen delivery, CO (the amount of blood ejected by the heart each minute) must be adequate (Salenger et al., 2003). A related parameter to CO is cardiac index (CI), the amount of blood ejected by the heart each minute in relation to a patient's body surface area. Adequate tissue perfusion is dependent on satisfactory CO. Cardiac output and index are functions of stroke volume (SV, the amount of blood ejected by the heart with each beat) and heart rate. SV depends on myocardial contractility, preload (the amount of volume returning to the right or left side of the heart), and afterload (the amount of work

the heart has to do to eject blood) (Massé & Antonacci, 2005; Silvestry, 2008).

Low cardiac output syndrome (LCOS) is often seen after cardiac surgery and is associated with increased morbidity and mortality. The reported incidence of this condition is 6%, 12%, and 23% for patients with a LV ejection fraction (LVEF) greater than 40%, between 20% and 40%, and less than 20%, respectively. LCOS has been defined as the need for administration of inotropic therapy for more than 30 minutes or for intra-aortic balloon pump (IABP) therapy; it is a common complication following aortic valve surgery (Maganti, Rao, Borger, Ivanov, & David, 2005).

Postoperative cardiac surgery patients may develop LCOS due to a transient decrease in perfusion due to LV dysfunction. Cardiac arrest from cardioplegia with resultant myocardial stunning and diastolic dysfunction, decreased preload, increased afterload, dysrhythmias, and MI have also been implicated etiologies for LCOS. The ultimate results are an imbalance between oxygen supply and demand and a metabolic acidosis (Aranki, Cutlip, & Aroesty, 2008; Massé & Antonacci, 2005; Talmor & Lisbon, 2005).

The ICU nurse must monitor for signs and symptoms of impaired CO. These include altered mental status, hypotension, decreased MAP, narrow pulse pressure, tachycardia, decreased peripheral pulses, increased CRT, cool extremities, poor perfusion, oliguria, or anuria (Massé & Antonacci, 2005; Mullen-Fortino & O'Brien, 2008).

If monitoring equipment is available, SvO_2 levels will be lower than normal in the setting of LCOS. SvO_2 (i.e., mixed venous oxygen saturation) is the percentage of hemoglobin saturated with oxygen in the pulmonary artery after blood has circulated systemically and oxygen has been extracted based on cellular need. Normal SvO_2 values are in the range of 70–75%. If SvO_2 is less than 70%, it indicates that cells are sensing hypoperfusion or an increased metabolic rate. SvO_2 monitoring provides data on the balance between oxygen supply and demand. Lab data that will help support a diagnosis of low CO include a metabolic acidosis or increasing base deficit on arterial blood gas and an elevated serum lactate level (Massé & Antonacci, 2005).

Management of the patient with low CO depends on the underlying cause, hemodynamic profile, and patient assessment findings. Low CO is usually brief in duration. Use of fluids, vasopressors, and inotropic agents will vary based on whether the patient has low preload or cardiac index and whether the SVR is elevated or low (Aranki, Cutlip, & Aroesty, 2008; Liu & Gropper, 2002). If inotropic support is required, the chosen drug's efficacy must be carefully monitored, as inotropic agents increase myocardial workload and metabolic rate (Massé & Antonacci, 2005). Epinephrine, norepinephrine (Levophed®), dopamine (Intropin®), or dobutamine (Dobutrex®) may be administered if contractility (EF) is below the expected values for the patient. These agents may have decreased efficacy in patients with chronic systolic dysfunction due to downregulation of beta receptors. Use of a phosphodiesterase inhibitor (e.g., milrinone [Primacor®]) may be a more effective means to augment contractility in this group of patients (Talmor & Lisbon, 2005). If vasodilation is the cause of the LCOS, administration of a vasoconstrictor (e.g., phenylephrine [Neosynephrine®], norepinephrine, or vasopressin) is warranted (Talmor & Lisbon, 2005).

If afterload is elevated, administration of nitroprusside (Nipride®) or IABP therapy may be indicated (Aranki et al., 2008; Massé & Antonacci, 2005). Regardless of the cause of low CO, the primary goal of management will focus on decreasing metabolic demand. Interventions such as preventing hyperthermia, administering sedation and analgesia, decreasing work of breathing with mechanical ventilation, and preventing or treating tachycardia, dysrhythmias, and electrolyte

and acid–base imbalances may need to be considered (Massé & Antonacci, 2005).

Preload Issues

Preload refers to the amount of volume returning to the right or left heart at the end of filling (diastole). It may be assessed by CVP and PAOP, which are the filling pressures of the right and left heart, respectively. PAOP is a reflection of left ventricular end-diastolic pressure (LVEDP), from which estimates of left ventricular end-diastolic volume can be made (Silvestry, 2008). The majority of patients are admitted to the ICU from the OR with alterations in preload despite having a positive fluid balance. The volume, however, is not in the intravascular space; instead, much of the fluid is located in the interstitium or other third space (e.g., pleural cavity).

Adequate preload is essential to maintain a satisfactory CO and tissue perfusion. Decreased preload in the immediate postoperative cardiac surgery patient can result from several factors, including excessive fluid output from diuresis or hypothermia, vasodilation during rewarming, inadequate intraoperative fluid resuscitation, intraoperative or postoperative bleeding, loss of vasomotor tone, infusion of vasodilator agents, decreased LV compliance, or capillary leak leading to third spacing of fluid (Khalpey et al., 2008; Massé & Antonacci, 2005; Silvestry, 2008).

The type and amount of fluid resuscitation required will be based on the patient's history, the amount and type of fluid lost, and the hematocrit level. If the patient experiences excessive postoperative bleeding, transfusion of blood and blood products should be initiated while the source of the blood loss is being determined. The hemoglobin requirement should be determined by the patient's cardiac status, age, and other clinical issues pertinent to the situation (Ferraris et al., 2007). Coagulation factors such as fresh frozen plasma and cryoprecipitate may also need to be given to correct coagulopathies usually caused by CPB.

If bleeding is not present, bolus administration of isotonic crystalloids (e.g., 0.9% normal saline, lactated Ringer's solution) or colloid (e.g., 5% albumin, hetastarch [up to 2 L]) may be used to optimize preload, usually to a PAOP of 18–20 mm Hg. Administration of inotropic agents is not recommended for patients with decreased preload (Khalpey et al., 2008; Marino, 2006; St. Andre & DelRossi, 2005). Patients with a history of ventricular hypertrophy or diastolic dysfunction usually require a higher preload (Khalpey et al., 2008). Volume requirements may decrease after the patient has been removed from positive pressure ventilation, as this change is often associated with an increase in venous return owing to the decrease in intrathoracic pressure (Khalpey et al., 2008). Vasodilation is reported as the major contributor to a decrease in preload in the initial postoperative period. Volume repletion typically occurs most within the first 5 hours following surgery (St. Andre & DelRossi, 2005).

A therapeutic endpoint for volume resuscitation may be the MAP. The goal of a MAP in the range of 70–80 mm Hg is suggested. Tachycardia is not believed to be an appropriate indicator of adequacy of preload, given the many preoperative and intraoperative factors that can affect the correlation between heart rate and hypovolemia (St. Andre & DelRossi, 2005). Ongoing monitoring of the patient's hemodynamic profile must take place concomitantly with volume repletion. Care must be taken not to overstretch the ventricle with excessive volume, as an impaired CO may ensue (Massé & Antonacci, 2005).

Cardiac Dysrhythmias

Dysrhythmias often occur after cardiac surgery, as discussed in detail in Chapter 15. AF is the most frequently encountered dysrhythmia, occurring in as many as 50% of patients

(Auer et al., 2005). It usually occurs on the second and third postoperative days and, therefore, may not manifest while the patient is in the ICU. Ventricular dysrhythmias are less common and may be indicative of myocardial dysfunction or injury (Tineli et al, 2005). Dysrhythmias following cardiac surgery may result from a number of etiologies (Table 13–2).

Dysrhythmias can compromise CO when they interfere with diastolic filling. If a disturbance in heart rhythm is present, prompt identification and close assessment of the patient are essential. Assessment of the patient with cardiac dysrhythmias following cardiac surgery requires evaluation of the rhythm and its effects on systemic perfusion, as well as evaluation of precipitating factors. Treatment is based on whether the goal of therapy is to control the rate or to convert the rhythm. Pharmacologic management of dysrhythmias is discussed in detail in Chapters 12 and 15.

Table 13–2 Common Causes of Dysrhythmias Following Cardiac Surgery

Cardiac Problems
Preexisting heart disease
Poor intraoperative myocardial protection
Myocardial ischemia
Respiratory Issues
Hypoxia
Hypocarbia/hypercarbia
Endotracheal tube misplacement
Pneumothorax
Electrolyte Disturbances
Hypokalemia
Hyperkalemia
Hypomagnesemia
Surgical Trauma
Atriotomy
Ventriculotomy
Surgical correction near the conduction system
Medications
Digoxin
Vasoactive medications
Other
Hypothermia
Disorders in acid–base balance
Anxiety
Pain
Anemia
Rebound adrenergic tone

Source: Brister & Lenkei-Kerwin, 2005.

Diastolic Dysfunction

Diastolic dysfunction may result from impaired systolic relaxation, stiffness of the left ventricle, or decreased diastolic stretching or compliance. Dysfunction commonly occurs as a result of aortic stenosis with LV hypertrophy or poorly preserved intraoperative myocardial protection. Patients with postoperative decreased ventricular compliance will have diastolic dysfunction (Salenger et al., 2003; Silvestry, 2008). This complication often persists for at least 3 hours following CABG (Ekery et al., 2003). If the left ventricle becomes stiff during filling (diastole), it may not be able to fill completely. As a result, fluid may back up to the lungs, and heart failure may ensue.

A number of intraoperative and postoperative etiologic factors of ventricular dysfunction have been identified. They include inadequate myocardial protection during aortic cross-clamping, intraoperative pulmonary edema or inadequate coronary blood flow, graft occlusion or thrombosis from MI or ischemia, MI from an air embolism or graft vasospasm, and cardiac tamponade (Silvestry, 2008).

The consequence of diastolic dysfunction is low CO with a small left ventricle. The hemo-

dynamic picture is one of elevated PAOP and low CO. Treatment of diastolic dysfunction includes volume administration to maximize preload and administration of vasodilators. Patients with diastolic dysfunction from decreased LV compliance will require a higher PAOP to maintain adequate preload than do patients with a reduced preload from the other etiologies listed earlier. Because the ventricle has decreased compliance in this case, the PAOP may be elevated despite the need for additional preload (Silvestry, 2008; St. Andre & DelRossi, 2005). Infusion of an inotropic agent with either a catecholamine (e.g., dopamine) or other agent to augment CO may also be required. Milrinone, a phosphodiesterase inhibitor, or nicardipine (Cardene®), a calcium channel blocker, may cause relaxation of the ventricle and may be of benefit in patients with diastolic dysfunction (Salenger et al., 2003). If the patient does not respond to traditional therapies, an echocardiogram should be obtained to determine the presence of valvular incompetence or cardiac tamponade (Silvestry, 2008).

Low Cardiac Output due to Right Ventricular Failure

Although most low output failure following cardiac surgery is attributable to LV failure, occasionally the right ventricle fails. Etiology of this complication may include ischemia, infarction, or increased PVR. Preexisting conditions such as pulmonary hypertension, aortic stenosis, mitral valve disease, tricuspid regurgitation, or right ventricular hypertrophy can also lead to postoperative right ventricular (RV) failure. Inadequate output of the right ventricle leads to decreased filling of the left ventricle, LV output, and poor systemic perfusion. The right ventricle then becomes distended, with RV failure being the ultimate outcome. The diagnosis of RV failure is based on the presence of elevated CVP and low PAOP and CO (Khalpey et al., 2008).

Management of RV failure includes providing sufficient preload, reducing right ventricular afterload (pulmonary vascular resistance, PVR), and maintaining systemic blood pressure. Volume repletion is essential to optimize left heart function. Care must be taken, however, to avoid overdistention of the right ventricle (Khalpey et al., 2008). Inotropic support may be required; the key is to use medications that provide ventricular support without increasing PVR. Milrinone is commonly used in the treatment of RV failure, as this agent increases contractility and causes vasodilation without increasing PVR (Khalpey et al., 2008).

In the case of severe RV failure, inhaled nitric oxide or a prostaglandin E infusion may be warranted. These medications cause dilation of the pulmonary vasculature, thereby reducing PVR (Khalpey et al., 2008; Vlahakes, 2005).

Decreased Myocardial Contractility

Contractility is the shortening of myocardial fibers during systole (ventricular emptying) and the force produced by the myocardium to eject blood. It is evaluated with EF by echocardiography (Massé & Antonacci, 2005).

Cardiac contractility may be impaired postoperatively due to such factors as hypoxia, acidosis, electrolyte imbalance, narcotics, anesthesia, transient ischemic/reperfusion injury, impaired preoperative function (EF less than 35%), inadequate intraoperative myocardial protection, duration and extent of postoperative hypothermia, CPB time (especially if longer than 120 minutes), tamponade, valve function, or myocardial ischemia or infarction (Khalpey et al., 2003; Massé & Antonacci, 2005; Salenger et al., 2003; St. Andre & DelRossi, 2005).

Myocardial function usually declines approximately 5 hours after surgery, possibly as a result of reperfusion from cardioplegia arrest. This effect usually lasts about

24 hours, though the duration and extent of diminished function may be influenced by any recent ischemic events, intraoperative course, and degree of dysfunction preoperatively (St. Andre & DelRossi, 2005). Decreased contractility may require inotropic support with vasoactive medications to support cardiac function. Both inotropic and vasodilator support with medications such as dobutamine, dopamine, milrinone, and epinephrine, used alone or in combination, may prove effective in improving cardiac contractility (Khalpey et al., 2003; Salenger et al., 2003). Epinephrine, however, is associated with the development of temporary but significant hyperglycemia, metabolic acidosis, and increased serum lactate when used in the initial 6–8 postoperative hours. These effects usually resolve in 12 hours (St. Andre & DelRossi, 2005).

In addition to titrating medications according to the patient's hemodynamic profile, the ICU nurse must monitor for signs and symptoms of inadequate perfusion related to the impaired contractility. Evaluation of cardiac index, hypotension, mottling, end-organ dysfunction (e.g., inadequate urinary output), and presence of a metabolic acidosis is vital. Urinary output may be increased in the initial postoperative period, however, so it is considered a less reliable indicator of poor perfusion (St. Andre & DelRossi, 2005).

Myocardial Stunning and Hibernation

Cardiovascular research has led to the identification of two important phenomena: myocardial stunning and myocardial hibernation.

Myocardial stunning is a period of impaired contractility following temporary ischemia, in which the dysfunction persists despite return of blood flow (Shavelle, 2006; Wang et al., 2003). Myocardial stunning may occur after CPB, and postoperative cardiac dysfunction (i.e., decreased ventricular function) is often attributed to its effects (Wang et al., 2003). The impaired ventricular function related to myocardial stunning is reported to terminate within 1–2 days and is not affected by preload or afterload manipulation (Kloner, Przyklenk, & Kay, 1994).

Hibernating myocardium is a condition of impaired LV function when the patient is at rest; it reflects a chronic reduction in blood flow. Heart function can be partially or totally normalized by improving blood flow or decreasing oxygen demand (Shavelle, 2006). Myocardial hibernation is considered a compensatory or protective mechanism to safeguard the capacity and integrity of the myocardium during times of decreased blood flow (Schipke & Birkenkamp-Demtröder, 2001).

Increased Systemic Vascular Resistance (Afterload)

As with preload, right- and left-sided afterload can be evaluated to help determine cardiac performance. Right-sided afterload is reflected by PVR; left-sided afterload is reflected by SVR. Most of the discussion in this section refers to left-sided afterload.

Afterload is determined by intraventricular systolic pressure and the thickness of the ventricular wall. The latter factor is minimally affected with cardiac surgery. SBP will have the greatest effect on afterload and, therefore, SV and myocardial oxygen demand. By decreasing afterload, CO will improve (Salenger et al., 2003).

Increased SVR is also often a compensatory result of the SNS response to low CO. Increased SVR may be poorly tolerated in a patient with already poor myocardial function.

Hypertension occurs in as many as 60% of postoperative patients (Talmor & Lisbon, 2005) and is often associated with vasoconstriction (Silvestry, 2008). Development of hypertension, vasoconstriction, or both may be related to decreased oxygen levels in the muscle with concomitant metabolic acidosis or inflammatory responses to CPB (Salenger

et al., 2003; Silvestry, 2008). Other potential causes of increased afterload include hypothermia, hypovolemia, hypercarbia, inadequate rewarming, volume overload, cardiogenic shock, pain, and anxiety. The latter two etiologies result from increased SNS stimulation (Khalpey et al., 2008; Talmor & Lisbon, 2005). If vasoconstriction is extreme, the patient is at risk of developing life-threatening hypertension and decreased CO (Khalpey et al., 2008).

Treatment of increased afterload may entail administration of vasodilator therapy with medications such as sodium nitroprusside, nitroglycerin (Tridil®), or milrinone. Sodium nitroprusside is the treatment of choice (Silvestry, 2008; St. Andre & DelRossi, 2005). Given that vasodilators cause a decrease in preload, concomitant administration of fluids may be required to maintain adequate intravascular volume during their use. As the potential for abrupt hypotension exists when nitroprusside is administered, frequent blood pressure monitoring is essential, especially during rewarming (Silvestry, 2008). In severe cases of LV failure, IABP counterpulsation may be used to reduce afterload. IABP therapy is discussed in detail in Chapter 10.

Decreased Systemic Vascular Resistance

While an increase in afterload is common following cardiac surgery, some patients develop a decreased SVR postoperatively. This condition, which is also referred to as vasodilatory shock, is associated with a CO that is either normal or increased (Aranki et al., 2008). The incidence of vasodilatory shock is reported to range from 5% to 8%. Patients who are at higher risk for the development of a decreased SVR are those who have a low EF (less than 35%) and those with end-stage heart failure requiring assist device insertion. Vasodilatory shock may be caused by an inflammatory response to CPB (Silvestry, 2008). Its treatment entails administration of a vasoconstrictor agent such as phenylephrine or norepinephrine (Khalpey et al., 2008; Salenger et al., 2003; Silvestry, 2008). If patients do not respond to this therapy, vasopressin administration may be attempted. Finally, methylene blue administration may be considered, as this agent inhibits nitric oxide production (Aranki et al., 2008; Silvestry, 2008).

Mechanical Issues

A number of mechanical issues can contribute to the development of hemodynamic compromise in the postoperative cardiac surgery patient. These complications include cardiac tamponade, coronary artery graft spasm, prosthetic valve regurgitation, pneumothorax, and hemothorax (Silvestry, 2008).

Cardiac Tamponade

During cardiac surgery, the pericardial sac is entered and is usually not sutured back together before chest closure. This leaves a communication between the heart and mediastinum, which can lead to the potential accumulation of blood or fluid (Lemmer, Richenbacher, & Vlahakes, 2003; St. Andre & DelRossi, 2005). The accumulation compresses the atria, restricts venous return to the heart and ventricular filling, and results in a decrease or cessation of preload, causing a potential precipitous fall in CO (Massé & Antonacci, 2005). Early tamponade is usually a result of persistent mediastinal bleeding not being evacuated by chest tubes.

Cardiac tamponade is one of several potential complications that may result in ventricular dysfunction (Silvestry, 2008), and typically occurs within the first 12 postoperative hours (St. Andre & DelRossi, 2005). Diagnosis may be difficult because hypotension, tachycardia, and elevated filling pressures are common scenarios in most immediate postoperative cardiac surgery patients. In addition, some of the other characteristic symptoms of cardiac

tamponade (e.g., muffled heart sounds, pulsus paradoxus, and neck vein distention) are not helpful in the cardiac surgery patient. While the patient may experience equalization of intracardiac pressures (CVP equal with PAOP or PAD), other signs and symptoms will likely suggest the presence of cardiac tamponade prior to this manifestation (St. Andre & DelRossi, 2005). Heightened awareness for tamponade should be present when the patient develops the signs and symptoms listed in Table 13–3.

Continuous hypotension that does not respond to fluid administration and the presence of signs and symptoms listed in Table 13–3 requires prompt intervention, including a bedside echocardiogram (Talmor & Lisbon, 2005). The patient may need to return to the OR for clot evacuation or bleeding site repair. When an echocardiogram is not feasible or there is impending cardiac arrest, emergency mediastinal exploration is warranted for accurate diagnosis (Mullen-Fortino & O'Brien, 2008; Talmor & Lisbon, 2005). Box 13–1 lists the steps undertaken in an emergency resternotomy.

Table 13–3 Signs and Symptoms of Cardiac Tamponade

Sudden decrease or cessation of mediastinal bleeding
Dyspnea
Low cardiac output with hypotension
Narrowing pulse pressure
Inappropriately fluctuating MAP
Increased central venous pressure
Low cardiac output/index
Sudden oliguria
Altered mental status
Diaphoresis
Dysrhythmias, including tachycardia
Cyanosis or pallor
Anxiety
Restlessness
Low-voltage QRS on ECG
Electrical alternans on ECG
"Water bottle heart" and cardiac enlargement on chest radiograph
Hepatomegaly

ECG = electrocardiogram; MAP = mean arterial pressure.

Sources: Kaplow & Reid, 2006; St. Andre & DelRossi, 2005; Talmor & Lisbon, 2005.

Coronary Vasospasm

A frequently unrecognized cause of sudden cardiovascular collapse in the early postoperative period is coronary vasospasm. This complication usually presents itself as acute hypotension, ST-segment elevation in multiple leads, and low CO. All types of coronary grafts are implicated in the development of coronary vasospasm—saphenous vein grafts, arterial conduits, and normal cardiac vessels alike (St. Andre & DelRossi, 2005). The etiology of coronary graft spasm is not completely understood. Several hypotheses have proposed release of platelet thromboxane A_2, increased alpha-adrenergic tone, hypothermia, or electrolyte imbalances of magnesium or calcium as possible causes (Lemmer et al., 2003).

Vasospasm usually resolves on its own. If it does not, treatment is aimed at supporting hemodynamic instability and administration of vasodilators (Saxena, Konstantinov, Koniuszko, Singh, & Newman, 2006).

Myocardial Ischemia and Infarction

Myocardial ischemia, whether transient or leading to MI, may occur after cardiac surgery. The risk of postoperative myocardial ischemia or MI is greater given the increased age, comorbidities, extent of coronary artery disease, and degree of LV dysfunction that cardiac surgery patients tend to have prior to surgery (Moosbauer, Hofer, & Gombotz, 2007). Specific risk factors for MI include cardiomegaly, long CPB time, redo surgery, and

Box 13–1 Emergency Resternotomy Procedures

1. Alert the surgeon and operating team.
2. Obtain an emergency chest opening tray.
3. Obtain an electrocautery device, and apply the ground pads to the patient's skin to prevent a Bovie burn.
4. Set up sterile suction.
5. Obtain personal protective equipment, sterile gowns, antiseptic solution, and drapes.
6. Remove the dressing.
7. Pour antiseptic on the patient's skin.
8. Place sterile towels on the patient's skin.
9. Assist the surgeon by supplying wire cutters or a scalpel.
10. Open the wound down to the sternum with the scalpel.
11. Cut the sternal wires with the wire cutters.
12. Place the sternal retractor to expose the heart.
13. Assist with controlling bleeding and suctioning, if needed.
14. Assist in irrigation of mediastinum with warm saline or antibiotics.
15. Assist in closing the sternum.
16. Apply a dressing to the incision, securing the epicardial pacer wires and chest tube sites.
17. Assess the patient's cardiovascular and hemodynamic status every 15 minutes until stable.
18. Monitor coagulation and hematology laboratory studies as needed.
19. Monitor chest tube drainage.

CABG combined with another cardiac surgical procedure (Aranki et al., 2008).

In one study, predictive factors for postoperative MI included female gender, combined valvular surgery and preoperative renal failure, LVEF less than 40%, pulse pressure greater than 70 mm Hg, and faster heart rate. The last factor is felt to be related to the correlation between heart rate and sympathetic tone. As the SNS is highly activated during cardiac surgery, this condition increases the patient's risk for developing ischemia secondary to an imbalance between oxygen supply and demand. Increased SNS activity is also associated with increased platelet aggregation and decreased fibrinolytic activity, both of which contribute to coronary thrombosis development (Aboyans, Frank, Nubret, Lacroix, & Laskar, 2008).

The incidence of postoperative MI has been reported to range from 7% to 26% (Aboyans et al., 2008; Fransen, Diris, Maessen, Hermens, & van Dieijen-Visser, 2002; Gilchrist, 2001). The incidence specifically for patients who have undergone CABG is reported to be in the range of 2% to 4% (Silvestry, 2008). Patients may be started on aspirin, clopidogrel, or both within the initial few postoperative hours to reduce prevalence of MI (St. Andre & DelRossi, 2005).

Mechanisms for myocardial ischemia include reperfusion injury from poor myocardial protection with cardioplegia, incomplete revascularization, coronary vasospasm, or coronary artery or intracoronary embolism (Khalpey et al., 2008). MI may also occur in the early postoperative period related to closure of a bypass graft (Lemmer et al., 2003).

The diagnosis of MI is initially determined by ECG changes (e.g., the presence of Q waves or ST elevation) and the presence of elevated cardiac markers (troponin I or creatine kinase [CK-MB]). ST segment changes are common in the immediate postoperative period after cardiac surgery and are usually not clinically significant. Other suspect ECG findings include new bundle branch block, ventricular dysrhythmias, or complete heart block (Khalpey et al., 2008). Development of new Q waves is indicative of MI even in the presence of normal cardiac markers. When evaluating cardiac markers, it is important to account for the elevation in the patient's baseline levels due to the surgical injury to the heart (Wu, 1998).

Troponin I is a myocardial protein that is a very sensitive and specific marker for myocardial damage. Elevation of troponin I to more

than 15–20 mcg/L within 12 hours and a peak of more than 35 mcg/L at 24 hours are indicative of MI (Gensini et al., 1998). Following an MI, troponin I levels peak in 36 hours and remain elevated longer than CK-MB levels (Lemmer et al., 2003). Of note, troponin levels are usually elevated after cardiac surgery, making diagnosis of MI difficult in these patients. For this reason, it has been recommended that troponin I levels not be used for definitive MI diagnosis after cardiac surgery (Abramov et al., 2006).

CK-MB levels can also be used in the diagnosis of MI. Some release of CK-MB isoenzymes occurs in most patients who undergo cardiac surgery. However, if a cardiac surgery patient has an intraoperative MI, the CK-MB peak level will be higher and the elevation will last longer. Data suggest that in a patient who has undergone CABG surgery, peak postoperative CK-MB levels of less than 20 U/L with no ECG changes indicate no significant myocardial injury. If CK-MB levels exceed 50 U/L or are five times the upper limit of normal or greater, MI is suggested (Aranki et al., 2008; Lemmer et al., 2003). Most recent data suggest that troponin levels are more useful for detecting necrosis and should be used to either supplement or replace CK-MB measurements (Aranki et al., 2008).

Data also suggest that troponin T may be used for up to 2 days postoperatively to diagnose an MI. In one study, CK-MB levels were almost normal by day 2 after cardiac surgery (Abdal Aziz, Ali, Roberts, & Al Khaja, 2000).

Patients with suspected MI or persistent ischemia follow the same course as uncomplicated postoperative patients, with beta blockade and intravenous nitroglycerin being administered to them if the blood pressure permits (Khalpey et al., 2008; Lemmer et al., 2003). Serial troponin and CK-MB levels should be obtained and monitored as well as 12-lead ECG for the presence of new Q waves. IABP therapy is suggested to diminish inotrope use, infarct size, and myocardial oxygen demand (Khalpey et al., 2008).

Cardiac Arrest

Cardiac arrest is the most serious complication in postoperative cardiac surgery patients. Low CO and sepsis are the leading causes of cardiac arrest in this population. Mechanical factors such as tamponade and coronary artery graft occlusion are also causes of sudden cardiac arrest in hemodynamically stable patients during the immediate postoperative period (Anthi et al., 1998).

Basic and advanced cardiac life support protocols set forth by the American Heart Association should be initiated in any patient who experiences cardiac arrest (St. Andre & DelRossi, 2005). As resuscitation efforts are started, evaluation for potential causes—including those unique to cardiac surgery patients—should begin. Checking the position of the endotracheal tube, signs of hypovolemia, patient temperature, chest tube drainage, proper ventilator functioning, results from chest radiograph (for widened mediastinum, tension pneumothorax, or tamponade), arterial blood gas, and electrolytes may help identify the underlying cause of cardiac arrest. Noting the infusion rates of vasoactive agents may provide additional clues. Treating and reversing the cause is the priority here, with concomitant high-quality cardiopulmonary resuscitation (CPR) being a pivotal part of this care.

Many patients will experience pulseless electrical activity in the postoperative cardiac surgery scenario (St. Andre & DelRossi, 2005). If, however, the underlying rhythm requires defibrillation, this procedure (which is outlined in Box 13–2) can be accomplished with internal defibrillation.

Adequate cardiac function has been identified as the most critical factor affecting recovery from cardiac surgery (Silvestry, 2008). Continuous monitoring of the patient's hemodynamic status by the ICU nurse is essential. Evaluation of the patient's cardiac rate and rhythm, blood pressure, hemodynamic profile data, chest tube drainage, uri-

Box 13–2 Internal Defibrillation Procedure

1. Follow the procedure for open sternotomy.
2. Follow the procedures for advanced cardiac life support (ACLS).
3. Prepare the defibrillator for internal defibrillation by gathering sterile internal defibrillation paddles.
4. Assist with positioning the internal paddles on the heart.
   a. One paddle is placed over the right atrium or right ventricle.
   b. The other paddle is placed over the apex of the heart.
5. Charge the defibrillator paddles (5–20 joules).
6. Verify providers are clear of the patient and all equipment before defibrillation.
7. Assess the patient's cardiac rhythm for conversion and presence of pulse.
8. If needed, repeat the defibrillation, following advanced cardiac life support guidelines.
9. Assist with transport to the operating room or closure at the bedside.
10. Monitor the patient's neurologic, cardiac, and pulmonary status until stable.

nary output, and lab data, combined with ongoing patient assessment, will provide the valuable data required to titrate vasoactive infusions to help attain and maintain optimal hemodynamic function.

PULMONARY COMPLICATIONS

Postoperative pulmonary complications are noted to be primary contributors to increased morbidity for the cardiac surgery patient (Silvestry, 2008). The incidence of pulmonary dysfunction following CABG ranges between 30% and 60% (Mullen-Fortino & O'Brien, 2008). Most patients are able to tolerate pulmonary dysfunction without major disruption in oxygenation and ventilation.

Preoperative identification of patients with pulmonary risk factors should facilitate provision of proper perioperative intervention. Presence of preoperative risk factors such as tobacco use, age older than 65 years, obesity, diabetes, preexisting pulmonary disease (e.g., COPD), heart failure, and LV dysfunction place the patient at higher risk for postoperative pulmonary complications (Mullen-Fortino & O'Brien, 2008). Identification and early intervention can prevent the incidence of most postoperative pulmonary dysfunction (Margereson & Riley, 2003).

Cardiac surgery patients are especially prone to pulmonary complications as a result of several procedure-related factors: use of general anesthesia, need for a median sternotomy incision, cooling for myocardial protection, use of CPB, and harvest of the internal mammary artery (IMA), which requires pleural dissection (Wynne & Botti, 2004). Developing an understanding of the postoperative changes in pulmonary function, routine pulmonary management, and contributory factors of pulmonary dysfunction allows for the early identification and management of such problems.

Postoperative Effects on Pulmonary Function

The development of pulmonary dysfunction after cardiac surgery is associated with inconsistencies in gas exchange, ventilation/perfusion mismatch, and pulmonary shunting. Patients will often manifest signs including shortness of breath and decreased oxygen saturation (Mullen-Fortino & O'Brien, 2008).

Cardiac surgery patients are at risk for developing postoperative pulmonary complications due to increased age, comorbidities, and the surgical procedure itself. In one study, patients who were hypoalbuminemic had a higher incidence of pulmonary dysfunction, nosocomial infections including pneumonia, and increased duration of mechanical ventilation than patients with normal albumin

levels (Rady, Ryan, & Starr, 1997). In a study of the "oldest old" patients, the effect of an elevated BMI was evaluated on development of complications. Patients in the lowest BMI group (20.8 ± 1.6) and the highest BMI group (29.5 ± 3.8) had the highest incidence of pulmonary complications following cardiac surgery (Maurer et al., 2002). Table 13–4 lists other factors contributing to higher risk of pulmonary dysfunction.

Table 13–4 Factors Contributing to Development of Pulmonary Dysfunction after Cardiac Surgery

Contributing Factors	**Effects on Pulmonary System**
General anesthesia Paralytics Narcotics Supine positioning	Decreased central respiratory drive leading to decreased use of respiratory muscles. Upward shift of diaphragm. Chest wall relaxation. Changes in compliance of chest wall.
Cardiopulmonary bypass	Pulmonary edema from fluid overload and hemodilution. Interstitial pulmonary edema from a systemic inflammatory response, which produces capillary leak. Complement activation, release of cytokines, and neutrophil activation, which cause increased endothelial permeability. Insufficient alveolar distention to activate production of surfactant, which may lead to alveolar collapse, retention of secretions, and atelectasis.
Cooling for myocardial protection	Phrenic nerve injury.
Median sternotomy incision and chest tubes	Chest wall splinting, which reduces the patient's ability to take deep breaths.
Use of IMA for coronary artery bypass conduit	Use of IMA requires pleural dissection, which causes a potential decrease in chest wall compliance.
Sternal or thoracotomy incisional pain Obesity Age Diaphragmatic injury Smoking history History of COPD, heart failure	Decreased respiratory muscle use.

IMA = internal mammary artery.

Sources: Taggart, 2000; Wynne & Botti, 2004.

Atelectasis

Atelectasis occurs in the dependent parts of the lungs, most commonly in the left lower lobe, in most patients who have had anesthesia. Incidence is reported to be as high as 70% of cardiac surgery patients, and the complication typically occurs secondary to single-lung ventilation and deliberate intraoperative lung collapse as part of the procedure (Silvestry, 2008). The development of atelectasis is associated with decreased lung compliance, impaired oxygenation, and development of some degree of lung injury (Weissman, 2004). These adverse effects continue into the postoperative period and can have a substantial impact on patient recovery.

Techniques or devices that encourage patients to inspire deeply are beneficial. The aim of therapy is to produce a large and sustained increase in the transpulmonary pressure, thereby distending the lung and reexpanding the collapsed alveoli. Several methods, such as deep breathing exercises, incentive spirometry, and chest physiotherapy, have been shown to be helpful in reexpansion of the collapsed lung units (Wynne & Botti, 2004). Vigorous pulmonary toileting along with early ambulation are generally effective therapies for the postoperative cardiac surgery patient who is recovering from atelectasis.

Pleural Effusion

Postoperative pleural effusions, which comprise a collection of fluid in the pleural space, are common in postoperative cardiac surgery patients, occurring in 41–87% of these individuals (Mullen-Fortino & O'Brien, 2008). Typically, the effusion develops in the immediate postoperative period (the first 24 hours) (Talmor & Lisbon, 2005) and can be visualized on chest radiograph. The effusion is usually present in the left lower lobe and is small in size, but can also be bilateral (Heidecker & Sahn, 2006).

Patients with small pleural effusions (less than 500 mL) are usually asymptomatic. Small pleural effusions will likely resolve on their own (Mullen-Fortino & O'Brien, 2008; Talmor & Lisbon, 2005). Preexisting lung disease and moderate to large effusions (occupying more than 50% of the lung) may cause dyspnea. In this situation, a thoracentesis or chest tube insertion is indicated (Mullen-Fortino & O'Brien, 2008; Talmor & Lisbon, 2005).

Phrenic Nerve Injury

Phrenic nerve injury with diaphragmatic dysfunction has been documented in 10–85% of postoperative cardiac surgery patients. The primary etiology of this complication is cold injury to the phrenic nerve from use of iced slush in the pericardial region (Dimopoulou et al., 1998). The phrenic nerve may also be injured in the takedown of the IMA (Tripp & Bolton, 1998). Unilateral nerve injuries produce few respiratory symptoms, and patients can usually be extubated without difficulty. In contrast, bilateral phrenic nerve injury may cause paradoxical breathing, tachypnea, and carbon dioxide retention when attempts are made at extubation. Chest radiograph may reveal an elevated hemidiaphragm at end-expiration with spontaneous ventilation; the elevation will not be apparent while the patient is mechanically ventilated owing to the effects of the positive pressure ventilation. Treatment may involve plication of the diaphragm, which attempts to stabilize the diaphragmatic muscle and prevents paradoxical motion with breathing (Mertens, 2006). Phrenic nerve injury is discussed in more detail in Chapter 11.

Pneumothorax

A pneumothorax may occur after cardiac surgery because of direct injury to the lung during surgery, central venous cannulation, or barotrauma during positive pressure ventilation. It is usually noted in the immediate

postoperative period and is considered a residual effect of surgery (Talmor & Lisbon, 2005). The overall incidence of pneumothorax after cardiac surgery is approximately 1–2%, although the incidence following CABG with IMA harvesting is as high as 5.3% (Weissman, 2004). The incidence may also increase in patients with bullous lung disease or in those requiring high levels of positive end-expiratory pressure (PEEP). A pneumothorax typically presents on the left side of the chest and occurs when the left parietal pleura is opened and the left IMA is dissected. This complication is managed by connecting the chest tube to suction (Talmor & Lisbon, 2005).

A tension pneumothorax can develop quickly in patients who are placed on mechanical ventilation. This complication usually arises after the patient develops a right pneumothorax if the right parietal pleura is accidentally cut (Talmor & Lisbon, 2005). In this situation, the patient acutely decompensates. Although breath sounds may be diminished, it may be difficult to assess given ventilator sounds and various alarms in the unit. Other signs and symptoms of tension pneumothorax can include distended neck veins, hypotension, and tracheal deviation away from the collapsed lung. If the patient is hemodynamically unstable and a tension pneumothorax is suspected, decompression with a 16-gauge needle at the second intercostal space, midclavicular line, is indicated. A rush of air and an improvement in hemodynamics confirm the diagnosis (Goodrich, 2005). A chest radiograph should be obtained to assess the involved structures and the severity of the tension pneumothorax. Treatment also includes placement of a chest tube, usually at the fifth intercostal space, anterior axillary line, for the residual pneumothorax (Weissman, 2004).

Prolonged Mechanical Ventilation

A small number of patients will require prolonged mechanical ventilation after cardiac surgery. Prolonged mechanical ventilation is often necessitated by cardiac dysfunction, continual postoperative bleeding, neurologic complications, acute renal failure, surgical reexploration, or need for blood transfusions (Weissman, 2004). In these patients, continual attempts at extubation are made; failure to wean after 24 hours is usually due to difficulty with oxygenation, ventilation, or both. When weaning failure occurs, cardiac and volume status are optimized, metabolic abnormalities corrected, narcotics and sedation reduced, and nutrition is initiated.

One important goal in the mechanically ventilated patient is reduction of the FiO_2 to less than 0.50. Prolonged exposure to FiO_2 greater than 0.50 has been shown to cause harmful effects on the lungs (e.g., through production of oxygen free radicals); for this reason, using the lowest FiO_2 to achieve an acceptable oxygen saturation is important (Peruzzi & Shapiro, 2002). Addition of PEEP may allow for a decrease in FiO_2 and improve oxygenation. Patients who develop fluid overload may need aggressive diuresis over a course of days to decrease interstitial lung water or pleural effusion. Patients who are ventilated for 10–14 days with multiple unsuccessful attempts at weaning may require a tracheostomy. In one study of patients requiring prolonged mechanical ventilation, 99% were ultimately successfully weaned and 85% were discharged (Engoren, Buderer, & Zacharias, 2000). Care of the patient requiring prolonged ventilation is addressed in detail in Chapter 11.

Acute Respiratory Distress Syndrome and Acute Lung Injury

Acute respiratory distress syndrome (ARDS) is an extreme form of acute lung injury (ALI) that is characterized by inflammation of the lung parenchyma and increased microvascular permeability, which causes leakage of fluid into the alveolar space, hypoxemia, increased work of breathing, and pulmonary infiltrates

on chest radiograph. Both ARDS and ALI usually occur after the first 24 hours following cardiac surgery (Talmor & Lisbon, 2005).

ARDS is an uncommon complication after cardiac surgery but can be associated with a high mortality rate—as high as 15% to 70% (Talmor & Lisbon, 2005). The reported incidence of ARDS following cardiac surgery is less than 2%, however (Silvestry, 2008). Previous cardiac surgery, shock, and number of blood products received are important predictive factors for this complication (Milot et al., 2001). CPB has been implicated as a causative factor in ARDS because the extracorporeal circulation stimulates a systemic inflammatory response and release of cytokines and endothelial-derived factors (e.g., nitric oxide).

Treatment of ARDS is mainly supportive, but maintaining adequate oxygenation is the primary goal. The use of smaller tidal volumes (6 mL/kg) and PEEP have been associated with lower mortality (Sakr et al., 2005). Data support the use of smaller tidal volumes in patients with established ALI or ARDS. ARDS specifically in cardiac surgery patients has not been studied, however. Data are also not available on the efficacy of smaller tidal volumes to prevent these complications from developing (Talmor & Lisbon, 2005).

Pneumonia

Postoperative pneumonia may develop after the first 24 hours following cardiac surgery. Patients at high risk are those who require mechanical ventilation for more than 48 hours or who develop diaphragmatic dysfunction (Talmor & Lisbon, 2005). As discussed in Chapter 11, patients with persistent LV failure are also at higher risk for pneumonia. Persistent LV failure after cardiac surgery ultimately results in extravasation of fluid into the alveoli. Interstitial fluid in the alveoli decreases compliance, increases secretions, and facilitates atelectasis, which may then progress to pneumonia (Salenger et al., 2003). Patients with incisional pain from sternotomy or thoracotomy sites are at risk for developing pneumonia if the pain interferes with effective coughing and deep breathing (Silvestry, 2008).

One key way to prevent postoperative pneumonia is to follow evidence-based guidelines for managing ventilator-associated pneumonia. These guidelines can be accessed from the American Association of Critical-Care Nurses' Web site, in the "Practice Alerts" section.

HEMATOLOGIC COMPLICATIONS

Bleeding and Coagulopathies

Bleeding is a common postoperative scenario in the cardiac surgery patient population. The incidence of excessive bleeding is reported to range from 3% to 14%, with reexploration being required in as many as 5% of patients (Bowman et al., 2008). The etiology of postoperative bleeding may be surgical, related to platelet dysfunction from exposure to CPB circuitry, or associated with inadequate heparin reversal at the end of CPB (Mullen-Fortino & O'Brien, 2008; Talmor & Lisbon, 2005). Excessive bleeding is defined as loss of more than 500 mL of blood in the first postoperative hour.

If excessive bleeding is present, the source must be quickly identified. Possible sources include, but are not limited to, anastomoses sites—that is, a branch of the saphenous veins, IMAs, cannulation sites, or sternal wire sites. If the bleeding is quite excessive or an anatomic source has been identified or suspected, treatment entails emergently returning the patient to the OR (Salenger et al., 2003).

An estimated 30% of patients who undergo CABG require blood transfusion (Silvestry, 2008). Cardiac surgery patients are transfused with 10–25% of all blood products used in the United States every year (Whitlock, Crowther, & Ng, 2005). Preoperative risk factors for bleeding include use of pharmacologic agents that

cause platelet dysfunction (e.g., ASA, clopidigrel [Plavix®], dipyridamole [Aggrenox®], abciximab [ReoPro®], and tirofiban [Aggrastat®]) and agents that prevent clotting (e.g., warfarin [Coumadin®], heparin, enoxaparin [Lovenox®]). Other patients at higher risk for postoperative bleeding include those with inherited disorders of coagulation (e.g., Von Willebrand's disease, hemophilia) or acquired coagulopathies (e.g., from end-stage renal disease, hepatic impairment) (Adams, Manson, Turner, Sindram, & Lawson, 2007). In these cases a hematologist should be involved with the case prior to surgery.

Excessive postoperative bleeding has also been associated with the following risk factors: previous cardiac surgery, preoperative cardiogenic shock, emergency cardiac surgery, female gender, small BMI, older age, peripheral vascular disease, lower preoperative hemoglobin, and renal insufficiency (Maurer et al., 2002; Silvestry, 2008, Whitlock et al., 2005). Patients who have been identified as being at higher risk for requiring blood transfusions typically have the following characteristics: (1) advanced age; (2) preoperative anemia or small body size; (3) preoperative use of antiplatelet or antithrombotic drugs; (4) redo operations or complex procedures; (5) emergency operations; and (6) noncardiac comorbidities (Ferraris et al., 2007). Table 13–5 lists surgery-specific factors that can cause excessive bleeding in the postoperative cardiac surgery patient.

Postoperative bleeding in the ICU setting is categorized as either surgical (bleeding vessel, anastomosis, or other suture line) or nonsurgical (coagulopathy). General guidelines for excessive mediastinal bleeding are characterized by continuous chest tube output of greater than 200 mL/hr for 2 hours. When bleeding exceeds these guidelines, efforts must be made to determine whether the bleeding is correctable by replacement of coagulation factors or whether the patient should return to the OR for exploration. Typical indications for surgical reexploration are blood loss greater than 200 mL/hr for 4 hours, 300 mL/hr for 3 hours, 400 mL/hr for 2 hours, or 500 mL/hr for 1 hour (St. Andre & DelRossi, 2005).

Table 13–5 Potential Causes of Bleeding in the Postoperative Cardiac Surgery Patient

Surgical bleeding
Platelet dysfunction/depletion
Hypotension
Deficiency/depletion of plasma clotting factors
Residual effects of heparin due to incomplete reversal with protamine
Hemodilution
Hypothermia
Increased fibrinolytic activity
Consumption coagulopathy

Sources: Mullen-Fortino & O'Brien, 2008; Silvestry, 2008; St. Andre & DelRossi, 2005; Talmor & Lisbon, 2005.

Packed red blood cells, platelets, cryoprecipitate, and fresh frozen plasma should be administered to correct abnormalities in hemoglobin, platelets, fibrinogen, and coagulation factors, respectively, if the cause is thought to stem from an acquired coagulopathy. Medications that improve platelet function (e.g., desmopressin [DDAVP®]) or prevent fibrinolysis (e.g., aminocaproic acid [Amicar®]) may also help in improving postoperative coagulopathy (Levi & Mannucci, 2007; Silvestry, 2008). Protamine sulfate is administered to neutralize the heparin given during CPB; its potential side effects include bradycardia, hypotension, and pulmonary vasoconstriction secondary to a non-immunologic reaction (St. Andre & DelRossi, 2005). Protamine sulfate reactions are described in detail in Chapter 12.

A newer medication that may help in the patient with a perioperative coagulopathy is factor VIIa. In the past, recombinant activated factor VII was used only for treatment of bleeding episodes in patients with deficiencies of factor VII or IX or hemophilia who developed bleeding following an invasive procedure. This therapy works by expediting platelet activation and ultimate fibrin clot formation (Bowman et al., 2008). Current evidence suggests that factor VIIa may be helpful in achieving hemostasis in cardiac surgery patients, thereby reducing transfusion requirements (Enomoto & Thorborg, 2005). After deficiencies in coagulation factors are corrected, if bleeding persists (greater than 300 mL/hr for 2 hours), urgent reexploration may be warranted. In one study, administration of recombinant activated factor VII resulted in a significant decrease in chest tube output, blood and blood product administration, and need for reexploration. Reported incidence of thrombosis in this patient group was 11.1% (Bowman et al., 2008).

Heparin-Induced Thrombocytopenia

Thrombocytopenia affects some 23–41% of all critically ill patients (Napolitano, Warkentin, Almahameed, & Nasraway, 2006). This condition has numerous etiologies in cardiac surgery patients. Factors such as effects of the CPB circuitry (e.g., mechanical destruction of platelets), hemodilution, platelet dysfunction, depletion of platelets, intravascular devices, use of IABP therapy, and effects of medications (e.g., antibiotics, antiarrhythmics) are common causes of thrombocytopenia in this population. A multitude of medications used in the cardiac surgery setting can also cause thrombocytopenia. One medication of significance is heparin, which is used to counteract exposure of the blood to the surfaces of the CPB machines (Matthai, 2005).

Heparin-induced thrombocytopenia (HIT) is a prothrombotic disorder of coagulation caused by platelet-activating, heparin-dependent antibodies. The platelet activation effect leads to excessive thrombin generation, which evolves into the hypercoagulable state. HIT develops in approximately 1% of all inpatients receiving heparin, with its incidence depending on heparin type, length of treatment, and patient population (rates are higher in surgical patients than in medical patients) (Adams et al., 2007). Patients undergoing cardiac surgery are at a higher risk for HIT secondary to the large systemic dose and long exposure to unfractionated heparin required for intraoperative systemic anticoagulation (Selleng, Markentin, & Greinacher, 2007).

The diagnosis of HIT should be considered when the platelet count falls to less than 150,000 mm^3 or by greater than 50% of the baseline count between 5 and 14 days of exposure. Laboratory testing with platelet factor-4/heparin enzyme-linked immunosorbent assay (ELISA) antibody and serotonin release assay are necessary to identify whether the patient has acquired the antibodies (Levy & Hursting, 2007).

Clinical signs and symptoms may include venous or arterial thromboses and skin lesions. Correlation of laboratory data with clinical symptoms is important for an accurate diagnosis. When HIT is suspected, all heparin products must be discontinued, including use of heparin-coated vascular access catheters. A non-heparin anticoagulant—such as argatroban, a direct thrombin inhibitor to prevent new thrombosis—should be administered, even when confirmatory lab results are not yet available (Cypher, 2006). Careful and thorough assessment for areas of new thrombosis, evaluation of skin temperature, color, CRT, sensation, and peripheral pulses is essential. Ongoing evaluation for signs and symptoms of stroke, MI, pulmonary embolism, and renal impairment is equally necessary.

Postoperative care should include use of measures to prevent venous thrombotic

events, including early mobility (ambulation if possible), isometric exercises, sequential compression devices, graduated compression stockings, or combinations of these interventions (Mullen-Fortino & O'Brien, 2008).

RENAL COMPLICATIONS

The incidence of postoperative renal complications is reported as high as 30% (Mullen-Fortino & O'Brien, 2008; Silvestry, 2008; Talmor & Lisbon, 2005). Renal failure has been correlated with poor quality of life, a higher mortality rate, and a longer length of stay (LOS).

Preoperative risk factors for development of renal failure include poor cardiac function, type I diabetes, advanced atherosclerosis, increased age, moderate to advanced heart failure, prior revascularization or CPB procedure, preoperative hyperglycemia (greater than 300 mg/dL), decreased creatinine clearance, hypoalbuminemia, and elevated creatinine levels (1.4–2 mg/dL). Intraoperative risk factors include prolonged CPB time (3 hours or longer), use of contrast agents, and ventricular dysfunction (Mullen-Fortino & O'Brien, 2008; Rady et al., 1997; Silvestry, 2008; Talmor & Lisbon, 2005). Development of renal failure is believed to be related to renal artery vasoconstriction, intraoperative hypothermia, and loss of blood flow while on CPB (Silvestry, 2008). Fortunately, only a small percentage (1% to 5%) of patients with postoperative renal failure requires dialysis or other renal replacement therapy (Mehta et al., 2006; Silvestry, 2008; Talmor & Lisbon, 2005).

In one study, patients who required dialysis after cardiac surgery were older by 7 years than those who did not. Other risk factors identified in this study included history of diabetes, COPD, peripheral or cerebrovascular disease, heart failure, previous CPB or valve surgery, CPR, recent MI, and cardiogenic shock. The last two conditions are associated with decreased CO, which can decrease renal perfusion and subsequently lead to postoperative renal failure (Mehta et al., 2006).

Administration of renal dose dopamine remains controversial, as data do not support improved survival or prevention of renal failure with this therapy. Efficacy data on the use of dopaminergic-receptor agonists (e.g., fenoldopam [Cloropam®] and dopexamine [Dopacard®]) are inconsistent. Studies investigating the use of diuretics (loop or osmotic) have failed to demonstrate improved survival; indeed, use of these medications may place the patient at greater risk for renal failure development by exacerbating any existing reduction in preload (Talmor & Lisbon, 2005).

The ICU nurse plays a pivotal role in helping to prevent postoperative renal dysfunction. Prevention can best be accomplished by maintaining adequate renal perfusion through optimization of preload and CO (Talmor & Lisbon, 2005). Another preventive measure is avoiding administration of nephrotoxic agents whenever possible (Silvestry, 2008). Collaboration with the physician and critical care pharmacist to determine whether use of alternative therapies, such as angiotensin-converting enzyme (ACE) inhibitors or aminoglycosides, is feasible as indicated.

GASTROINTESTINAL COMPLICATIONS

Gastrointestinal (GI) complications are rare in cardiac surgery patients, occurring in only 2% of individuals who undergo such procedures (Andersson, Nilsson, Brandt, Hoglund, & Andersson, 2005; Talmor & Lisbon, 2005). The likelihood of a patient manifesting a GI complication in the ICU is small, as most early complications are reported to occur on postoperative days 6 and 7—well after the patient has likely been discharged from the ICU (Talmor & Lisbon, 2005).

Most GI complications are difficult to diagnose because of their atypical symptoms, the many different underlying diseases that cause them, and the inability of patients to accurately describe their symptoms. All of these factors may delay diagnosis or treatment. Prolonged mechanical ventilation, septic shock, and renal complications have been shown to be strong predictors of GI complications (D'Ancona et al., 2003).

Overall, the mortality rate is 50% among patients who experience serious GI complications, with the most likely cause of GI complications being perioperative visceral or splanchnic hypoperfusion. Hypoperfusion is likely to stem from low CO, which produces vasoconstriction, hypoxia, and hypoperfusion of the splanchnic bed with resultant intestinal ischemia. This complication is probably attributable to the increased age of cardiac surgery candidates, the longer and more complex procedures performed during cardiac surgery, and the increased prevalence of atherosclerosis in these surgical candidates (Filsoufi et al., 2007). Other GI complications include ileus, upper GI bleeding from gastritis or peptic ulcer disease, acute pancreatitis, cholecystitis, and acute hepatic failure (Lemmer et al., 2003; Mullen-Fortino & O'Brien, 2008).

The ICU nurse should monitor the patient for presence and changes in bowel sounds, abdominal pain or distention, nausea, and vomiting. Diagnostic lab tests (e.g., complete blood count, metabolic panel) and abdominal radiograph may be obtained if GI complications are suspected (Mullen-Fortino & O'Brien, 2008).

NEUROLOGIC COMPLICATIONS

Postoperative cardiac surgery patients may develop any of a wide range of neurologic complications. Neurocognitive insufficiency may occur in as many as 80% of these patients. Stroke, transient ischemic attack, decline in intellectual function, memory deficits, stupor, coma, and new onset of seizures are other reported complications following CPB. The incidence of neurologic complications has been reported to be between 2% and 16% in this patient population (Silvestry, 2008; Talmor & Lisbon, 2005). In one study, most patients with preoperative hypoalbuminemia were found to be at increased risk for development of postoperative neurologic complications (Rady et al., 1997). Neurologic complications following cardiac surgery are discussed in detail in Chapter 16.

SYSTEMIC INFLAMMATORY RESPONSE TO CARDIAC SURGERY

Cardiac surgery elicits a powerful inflammatory response that can have serious effects. Inflammation is the body's response to the disruption within the tissues and involves a series of controlled humoral and cellular reactions. Nonspecific activators of the immune response include trauma to the tissues during surgery, blood transfusions, and hypothermia. A specific activator of this response is CPB, which affects the immune system in several different ways. First, the surface of the CPB circuit causes activation of the immune system when blood comes in contact with the foreign surfaces of the circuit. Second, aortic cross-clamping causes reperfusion injury to the brain, kidneys, liver, heart, and lungs (Laffey, Boylan, & Cheng, 2002).

The systemic inflammatory response is characterized by the release of pro-inflammatory factors such as interleukins 6 and 8 (IL-6 and IL-8) and tumor necrosis factor (TNF). The amount released correlates with the development of postoperative complications (Chello et al., 2006). The inflammatory response seems to be most evident in the setting of postoperative pulmonary and cardiac dysfunction, where it causes acute lung injury and global myocardial dysfunction with subsequent peripheral vasodilation.

Current recommendations for treatment call for supportive care until the inflammatory response resolves. Much research is evolving in this area, with treatment modalities such as stress-dose steroids, aprotinin (Trasylol®), atorvastatin (Lipitor®), and improved circuit biocompatibility being explored (Laffey et al., 2002).

Aprotinin is the most potent anti-fibrinolytic agent that is used to decrease postoperative cardiac surgery bleeding; it also has anti-inflammatory properties. Specifically, aprotinin decreases neutrophils and macrophage activation, and it reduces release and activation of pro-inflammatory mediators (McEvoy, Reeves, Reves, & Spinale, 2007). Given the relationship between inflammation and hemostasis, aprotinin is indicated for use in postoperative cardiac surgery patients (Levy, Tanaka, & Bailey, 2008). Its use for this purpose is controversial, however (McEvoy et al., 2007).

Whenever blood is exposed to an artificial surface, thrombus development is likely. For this reason, anticoagulation is required during bypass procedures. To avoid additional anticoagulation, the use of heparin-coated circuits has been proposed. These devices are designed to decrease coagulation and diminish the associated inflammatory response, although their efficacy has not been consistently demonstrated (Taneja & Cheng, 2006).

INFECTIOUS COMPLICATIONS

Postoperative infections include bacteremia, pneumonia, urinary tract infections, and wound infections. Wound infections following cardiac surgery are classified as either superficial or deep (mediastinitis). Although wound infections are rare in cardiac surgery patients, their development results in increased morbidity, longer LOS, and higher costs. Identified risk factors for this complication include increased BMI, diabetes, COPD, prolonged surgery (greater than 90 minutes), and use of bilateral IMA grafts. In one study, most patients with preoperative hypoalbuminemia were found to be at increased risk for development of postoperative infectious complications (Rady et al., 1997). In another study, while no statistically significant differences among groups were found, patients with the highest BMI had a higher incidence of sternal wound infections. These manifestations occurred within 30 days of surgery (Maurer et al., 2002). Education of those patients with high BMI should include strict monitoring for signs of infection.

The ICU nurse should monitor the patient for purulent discharge from the wound, fever, increased pain or tenderness of the chest wall, or an unstable sternum. In addition to adhering to facility-specific wound care policies, administration of prophylactic antibiotic therapy for 48 hours and tight glycemic control help to minimize the likelihood of infections (Mullen-Fortino & O'Brien, 2008). Wound care is discussed in detail in Chapter 18.

SUMMARY

The initial 24 hours following cardiac surgery is a challenging and tenuous time. Patients have high levels of vulnerability and instability in the initial postoperative period. While there is some degree of predictability in terms of the postoperative trajectory, the trends toward increasing age and number of preoperative comorbidities in these patients have increased the level of complexity associated with this population. Most patients, despite their initial instability, are discharged from the ICU within 24 hours, often in 15–20 hours (St. Andre & DelRossi, 2005).

The role of the ICU nurse cannot be overemphasized in terms of these providers' influence in reducing the likelihood of critical events associated with postoperative complications. High levels of clinical judgment and caring practices are essential competencies. Prevention and prompt recognition of postoperative complications are pivotal to help ensure optimal patient outcomes.

CASE STUDY

An 82-year-old male is brought to the ED via ambulance after a brief loss of consciousness that caused him to fall. He is now alert and oriented × 3 with a negative neurological exam. The patient states that he has a 3-month history of chest pain that increases with activity and resolves with rest. He also reports shortness of breath (SOB) that has become progressively worse, to the point that he cannot make it to the top of the stairs at his home without feeling "winded." The patient also states he has been very healthy all of his life and does not seek medical care on a routine basis, although he takes a baby aspirin because heart attacks run in the family. The patient is divorced but has two grown children who live nearby and check in on him weekly. He reports a history of tobacco use for 30 years but has recently cut his smoking down to 1–2 cigarettes per day because of his increasing SOB.

Testing

CT scan of brain: Negative for bleed or stroke
12-Lead ECG: Sinus rhythm, HR 80, left ventricular hypertrophy
(The patient was admitted for observation and additional testing.)
Echocardiogram (TEE): Severe aortic stenosis with valve area 0.5 cm^2 (critical aortic stenosis) with severe left ventricular hypertrophy; moderate mitral regurgitation; no pulmonic stenosis or insufficiency; no tricuspid stenosis or insufficiency.
Cardiac catheterization: Left anterior descending artery with 99% occlusion; right coronary artery with 80% occlusion; obtuse marginal with 85% occlusion; severe triple vessel disease with critical aortic stenosis; EF 70%.

Because of the severity of symptoms, the critical aortic stenosis, and LAD occlusion, surgery is scheduled for the next morning. The intraoperative course is uneventful, and the patient receives a tissue aortic valve and a three-vessel coronary artery bypass. Postoperatively, the patient is transported to the ICU. Admission vital signs are as follows:

BP: 102/65 mm Hg (via arterial line)
HR: 102 bpm, sinus tachycardia, no ectopy
RR: 12 breaths/min (ventilator rate set at 12)
Temperature: 95.4 °F (35.2 °C)
PAP: 20/9 (11) mm Hg
CVP: 7 mm Hg
CI: 2.3 L/min (CO 4 L/min)
SVR: 1300 dyne/sec/cm^{-5}

The patient is receiving milrinone 0.375 mcg/kg/min, NTG 0.1 mcg/kg/min, and an insulin infusion at 4 units/hr.

Postoperative 15 Minutes

Urine output: 300 mL colorless urine
Chest tube outputs: Mediastinal: 50 mL sanguineous; pleural: 25 mL sanguineous

Laboratory Data

Hgb: 10.2 g/dL	PTT: 45.2 sec
Hct: 31.7%	PT: 16.8 sec
K: 3.5 mEq/L	INR: 1.4
Mg: 1.9 mg/dL	Platelets: 96,000/mm^3
Glucose: 270 mg/dL	

Critical Thinking Questions

1. What is a first priority for this patient?
   a. Replacement of electrolytes
   b. Pain medication
   c. Application of a warming blanket to correct the hypothermia
   d. Administer 0.9% NS to correct the low blood pressure
2. The patient is at higher risk for postoperative complications because of which of the following risk factors?
   a. Severely depressed left ventricular function
   b. Gender
   c. Presence of moderate mitral regurgitation
   d. Aspirin taken within 7 days prior to surgery

Postoperative 30 Minutes

BP: 90/43 mm Hg
PAP: 18/7 (11) mm Hg
CVP: 4 mm Hg
Chest tube outputs: mediastinal: 175 mL in container; pleural: 100 mL in container

Critical Thinking Questions

3. The ICU nurse is concerned about the increasing chest tube output and knows this patient is at risk for bleeding after cardiac surgery due to
   a. aspirin use.
   b. decreased platelet count.
   c. hypothermia.
   d. all of the above.
4. The cardiac surgeon is notified of the chest tube output. Protamine sulfate 25 mg is prescribed. Which of the following lab values should be evaluated to determine efficacy of this intervention?
   a. PT/INR
   b. Hemoglobin
   c. aPTT
   d. Magnesium

Postoperative 1 Hour

BP: 75/50 mm Hg
HR: 120 bpm, sinus tachycardia
PAP: 20/16 mm Hg

CVP: 18 mm Hg
CI: 1.5 L/min/m^2
Chest tube outputs: mediastinal: 180 mL in container; pleural: 100 mL in container

Critical Thinking Questions

5. Which of the following is the likely etiology of the patient's condition at this time?
   a. Tension pneumothorax
   b. Pleural effusion
   c. Cardiac tamponade
   d. Bleeding
6. Based on the patient's condition, which of the following interventions should the ICU nurse anticipate?
   a. Preparation for emergency resternotomy
   b. Replacement of electrolytes
   c. Administration of adenosine (Adenocard®)
   d. Insertion of an additional chest tube

The patient develops ventricular tachycardia with no pulse. The chest is immediately opened at bedside by the surgeon.

Critical Thinking Questions

7. Which of the following interventions is indicated at this time?
   a. Connection of epicardial pacing wires to external pacemaker box
   b. Internal defibrillation
   c. Administration of blood products
   d. Chest closure
8. How many joules should the nurse prepare to defibrillate with?
   a. 360 joules
   b. 5–20 joules
   c. 160 joules
   d. 200–300 joules

The patient returns to normal sinus rhythm. Post-resuscitation, 1 L of clot is evacuated from the chest. The patient's condition stabilizes, and he returns to the OR for washout and sternal closure. He receives intraoperative blood product replacement and returns to the ICU in stable condition. The remainder of his postoperative course is uneventful and he is discharged home after 10 days.

Answers to Critical Thinking Questions

1. c (Hypothermia in the cardiac surgery patient can cause increased myocardial oxygen demand, depression of ventricular function, and coagulopathy.)
2. d (Use of aspirin within 7 days of surgery and female gender are risk factors for postoperative complications. The patient did not have evidence of depressed left ventricular function based on his diagnostic test data.)
3. d (Decreased platelet count, use of aspirin, and hypothermia all can increase perioperative bleeding.)
4. c (Protamine sulfate reverses the effects of heparin. Large amounts of heparin are used for the cardiopulmonary bypass circuit and to systemically anticoagulate the patient.)

5. c (The nurse should be concerned about cardiac tamponade due to the sudden cessation of bleeding, tachycardia, narrow pulse pressure, and low cardiac index.)
6. a (Prepare for emergency sternotomy to relieve the tamponade. If time and the patient's hemodynamic profile allow, obtain an echocardiogram to confirm the diagnosis and guide the procedure. If time does not allow, emergency resternotomy is indicated.)
7. b (The nurse should obtain the paddles for internal defibrillation because the chest is open. Immediate defibrillation is indicated.)
8. b (Energy in the range of 5–20 joules is used for internal defibrillation. It takes less energy when the paddles are directly on the heart because the electricity does not have to permeate chest skin, bone, muscle, and fat.)

Clinical Inquiry Box

Question: Does the length of storage time for red blood cells affect patient outcomes post cardiac surgery?

Reference: Koch, C. G., Li, L., Sessler, D. I., Figueroa, P., Hoeltge, G. A., Mihaljevic, T., et al. (2008). Red-cell storage and complications of cardiac surgery. *New England Journal of Medicine, 358*(12), 1229–1239.

Objective: To evaluate the correlation of cardiac surgery complications and the length of time during which transfused cells are stored.

Method: A retrospective study was conducted to evaluate the complications in patients who had a coronary artery bypass grafting, heart valve surgery, or both over an 8-year period. Complications were correlated with the length of time during which blood had been stored prior to administration.

Results: Complications such as higher rates of in-hospital mortality, longer periods of intubation, renal failure, and sepsis were associated with patients who received blood that was stored for an average of 20 days.

Conclusion: Nurses should be aware that complication rates may be increased in patients who receive red blood cells that were stored for more than two weeks. Critical care nurses are in a position to review the latest research and propose policy changes such as limiting the age of stored blood for transfusion.

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WEB RESOURCES

Median sternotomy: http://www.youtube.com/watch?v=r7RsB0BA4EI

IABP complications: http://www.youtube.com/watch?v=X4gWT3u0FqQ

Heparin-induced thrombocytopenia: http://www.youtube.com/watch?v=_LVHEW8vH-E

Sepsis development and progression: http://www.youtube.com/watch?v=HoxoeP-l5Uw

Chapter 14

Pain Management

Susan Lynch

INTRODUCTION

Pain is one of the most significant problems for patients requiring surgery. Although it is recognized as a major healthcare issue for all patients, pain continues to be poorly managed. It is estimated that 50–70% of patients experience moderate to severe pain following surgery despite advances in care (Pogatzki-Zahn, Zahn, & Brennen, 2007).

Cardiac surgery patients are no different. In one study, patients who underwent cardiac or abdominal vascular surgery experienced moderate pain that did not lessen over the first few postoperative days (Puntillo & Weiss, 1994). It has been reported that more than 77% of cardiac surgery patients recall having postoperative pain (Gelinas, 2007a). Surveys of cardiac surgery patients at one academic medical center reflected lower than anticipated ratings; pain management specifically was identified as an area of concern. These findings included excellent clinical outcomes and high levels of technical expertise at the center (Cutshall et al., 2007). The data also corroborate other research findings of cardiac surgery patients' memories of postoperative pain while in the ICU (Maxam-Moore, Wilkie, & Woods, 1994; Meyerson, Thelin, Gordh, & Karlsten, 2001).

Although significant numbers of research studies support presence of postsurgical pain issues and advances in pain management, little to no progress has been made in reducing the incidence of postoperative cardiac surgery pain (Gelinas, 2007a). One possible contributor to inadequate pain management is the lack of reassessment following administration of analgesics. In one study, only 4.4% of pain activities entailed reassessment of efficacy of an analgesic intervention (Bucknall, Manias, & Botti, 2007).

Other data support that while there is a high prevalence of patients reporting moderate to severe levels of pain after cardiac surgery, those individuals receive only a small percentage of their prescribed/allotted analgesic dosage. Some patients receive less than half of the amount of analgesic that was prescribed (Maxam-Moore et al., 1994; Puntillo & Weiss, 1994; Watt-Watson & Stevens, 1998; Watt-Watson, Stevens, Garfinkel, Streiner, & Gallop, 2001; Watt-Watson et al., 2004).

Presence of pain following cardiac surgery has implications for optimal recovery. Patients have reported pain while turning, coughing and deep breathing, using the incentive spirometer, moving or turning in bed, and getting up (Gelinas, 2007a; Milgrom et al., 2004).

Pain management requires effective and efficient assessment, treatment, and evaluation by all members of the healthcare team, with nurses playing a critical role. Nurses are crucial in advocating for patients who are

experiencing pain, to help assure that those patients receive the best possible symptom management. To be an effective advocate, nurses must be able to recognize pain, be available, be ready to act, be empathetic instead of judgmental, and be willing to educate not only the patient but also the healthcare team (St. Marie, 2002).

WHAT IS PAIN?

Pain is defined by the International Association for the Study of Pain (IASP) as "an unpleasant and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP, 2008, p. 34). Pain was first defined by the subcommittee on Taxonomy of IASP in 1979, and this definition continues to be used in the literature (Gelinas, 2007b; IASP, 2008; Lome, 2005). Multiple factors—including previous pain experience, culture, mood, and coping skills—influence an individual's pain experience (Spacek, 2006). "The patient's experience of pain is seen as involving far more than a localized sensation; it encompasses what this sensation means to him" (McCaffery, 1972, p. 7).

Pain is subjective (IASP, 2008). The patient's self-report of pain is the most reliable indicator of its presence. Pain is described as "whatever the person says it is and exists whenever he says it does" (McCaffery, 1972, p. 8). However, it is important to note that the inability to communicate does not mean the patient is not experiencing pain. The use of the word "says" does not mean the patient must verbalize existence of pain. All patient behaviors—whether voluntary or involuntary, verbal or nonverbal—may indicate presence of pain (McCaffery, 1972). Measures must be taken to appropriately assess and treat patients who may not be able to verbally report pain (Gelinas, 2007b; IASP, 2008; Michaels, Hubbartt, Carroll, & Hudson-Barr, 2007; Spacek, 2006).

PAIN PHYSIOLOGY

Pain physiology can be described in relation to four distinct steps in the process of pain perception: transduction, transmission, perception, and modulation. Pain physiology related to the perception of pain begins with the initial tissue injury, whether real or perceived. *Transduction* occurs when the injury causes the release of mediators by stimulating the nociceptors or free nerve endings to cause the release of more mediators. *Transmission* occurs when the neurotransmitters activate the action potential, causing the information to travel along the primary neuron and to synapse with the secondary neuron in the dorsal horn of the spinal cord. *Perception* occurs as the brain processes the information. The spinal cord sends the information carried by the free nerve ending to the thalamus, and the information is then sent to the cortical areas of the brain where pain is perceived. *Modulation,* the final step, occurs when stimuli are either enhanced or inhibited by the hypothalamus, pons, and somatosensory cortex so as to process and transmit a pain sensation (Li, 2008; Lome, 2005). Figure 14–1 depicts the pain physiology process.

TYPES OF PAIN

There are two major classifications of pain: nociceptive and neuropathic.

Nociceptive pain occurs with direct stimulation of pain receptors. Examples of this type of pain include tissue injury or inflammation. Nociceptive pain can be further classified as either somatic or visceral. *Somatic pain* refers to the stimulation of pain in the cutaneous and deep layers of skin. Patients are able to localize this pain, which often comprises acute pain due to surgery. *Visceral pain* refers to pain resulting from infiltration or compression of the abdominal or thoracic viscera. It is often difficult for patients to localize visceral pain because the pain is referred to another part of the body (Goldstein & Morrison, 2005).

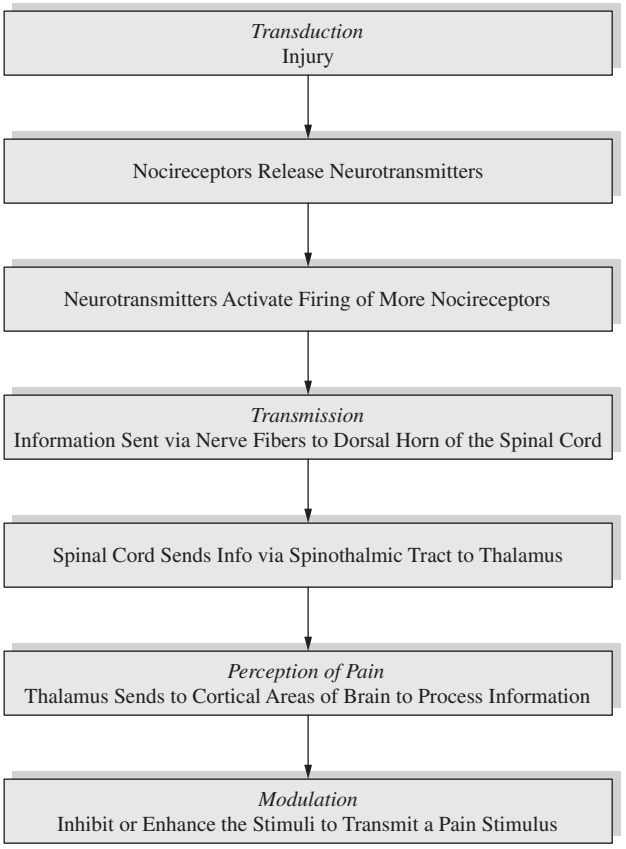

Figure 14–1 Pathophysiology of pain.

Postoperative cardiac surgery patients are likely to experience pain from a variety of sources. Incisions from sternotomy, thoracotomy and graft sites, required invasive procedures, tissue retraction and dissection, turning, and presence and removal of chest tubes are some of the identified etiologies (Cohen et al., 1993; Gelinas, 2007a; Heye, 1991; Mehta & Kumar, 2004; Meyerson et al., 2001; Mueller et al., 2000; Valdix & Puntillo, 1995; Watt-Watson & Stevens, 1998). In one study, patients who were having painful procedures (e.g., chest tube removal) were not premedicated for pain; these patients subsequently reported moderate to high levels of pain (Puntillo, 1994).

Neuropathic pain is primarily caused by a dysfunction in the nervous system that can occur either centrally or peripherally. It is caused by nerve damage. Examples of this type of pain include diabetic neuropathy and phantom pain (Chong & Brandner, 2006; Gelinas, 2007b).

Pain can also be categorized according to whether it is acute or chronic. *Acute pain* is described as having a recent onset or resulting from an ongoing injury, likely has a limited duration, and is often easier to treat. *Chronic pain* persists beyond the time of tissue healing and does not have a definable cause, as often there may be behavioral and emotional components (Hamill-Ruth & Marohn, 1999; Spacek, 2006). Acute and chronic pain can be visualized as a continuum instead of two distinct categories (Spacek, 2006). The duration of pain is considered when determining

whether the pain should be classified as acute or chronic. Acute pain typically lasts no more than 30 days, whereas chronic pain lasts 3–6 months (Gelinas, 2007b). For patients who undergo cardiac surgery, healthcare providers' focus remains on acute, nociceptive pain.

Unfortunately, cardiac surgery patients are also at risk for developing chronic pain. Over time, the patient's pain characteristics may evolve from acute, nociceptive pain to chronic, neuropathic pain in origin caused by the nerve damage associated with sternal incisions and retractions. Approximately 30–44% of patients who undergo thoracotomy exhibit chronic pain 6 months to a year after surgery has been reported (Cerfolio, Bryant, Bass, & Bartolucci, 2003; Jensen & Andersen, 2004). The risk of developing chronic pain seems to be reduced if postoperative pain is adequately managed (Markman & Philip, 2007; Pogatzki- Zahn et al., 2007), placing greater emphasis on the need for control of pain in acute periods.

In another study of cardiac surgery patients, persistent pain from any site (lasting at least 2 months postoperatively) was experienced by 29% of patients; 25% had persistent sternotomy pain. Other sites of pain included the shoulder, back, and neck. Pain intensity level in these patients was mild, with 7% of the study participants reporting that the pain interfered with activities of daily living. There was no significant difference in the incidence in those patients who received postoperative high thoracic epidural anesthesia and opioids (Ho, Royse, Royse, Penberthy, & McRae, 2002).

These findings were corroborated in another study of cardiac surgery patients. Patients who reported moderate to severe pain postoperatively were the same patients who reported chronic post-sternotomy pain more frequently (Lahtinen, Kokki, & Hynynen, 2006).

Individuals who experience uncontrolled acute pain for long periods of time are known to have a greater probability of developing chronic pain. One hypothesis for this phenomenon suggests that a neurophysiologic link results in glial activation following nerve or tissue injury. The signaling molecules involved in glial activation are ATP; CX3CL (fractalkine); CCL1 (monocyte chemotactic protein-1); the pro-inflammatory cytokines interleukin 1-beta (IL-1β), interleukin 6 (IL-6), tumor necrosis factor alpha (TNF-α), and substance P (SP); and glutamate (Marchand, Perretti, & McMahon, 2005). Nevertheless, individuals need early cessation of pain to prevent them from progressing to a state of chronic pain. Clearly, early recognition and treatment of acute pain is of prime importance.

■ PAIN ASSESSMENT

Pain assessment is a critical part of a nurse's total assessment of all patients. This statement was universally accepted and validated by the Joint Commission (2004) standard for pain assessment, which states, "A comprehensive pain assessment is conducted as appropriate to the individual's condition and the scope of care, treatment and services provided" (www.jointcommission.org). In the 2004 Joint Commission standards, PC.8.10 reads, "When pain is identified, the patient is assessed and treated by the organization or referred for treatment" (Alcenius, 2004, p. 12).

To meet the standards for pain assessment, organizations are expected to perform screening and intensive assessment. Screening should be conducted in the initial assessment to be able to determine the patient's need. Typically, screening will ask a question related to the patient's present pain state and history of pain. The progression to an intensive assessment is determined by the screening. Evaluation of pain needs to focus on several domains, including the pain's location, intensity, quality, and duration; relief measures; and the patient's perception of an acceptable pain level. Recommendations include using

pain assessment tools that are age and ability appropriate (Alcenius, 2004).

Preoperative Baseline Assessment

To optimally manage pain for surgical patients, nurses should obtain a thorough preoperative health history, encompassing topics related to the patient's current and past experiences with pain. Many factors can affect a patient's response and perception of pain—for example, fatigue, sleep deprivation, fear, anxiety, depression, anger, misinformation, altered mental status, educational level, cultural background, ethnic background, and pain experience (Hamill-Ruth & Marohn, 1999). Past experiences will alert the nurse to factors that may directly affect the patient's response to the current treatment, including medications that have been effective in the past, acceptable pain levels, concerns about pain, and educational needs. Discussions focusing on the patient's expectations can assist the nurse and the patient to jointly develop a plan of care related to pain that has desirable predicted outcomes (Dunwoody, Krenzischek, Pasero, Rathmell, & Polomano, 2008).

The pain assessment process includes multiple components, such as initial assessment, treatment, reassessment, and evaluation. Optimal goals include adequate pain management, manageable side effects, and assurance of safety (Dunwoody et al., 2008). Dimensions of the assessment should include the pain's location, description, intensity, duration, alleviating and aggravating factors, associative factors, and impact on life. To identify the location and obtain a description of the pain, the nurse should ask patients to describe the pain in their own words and to point to where the pain occurs (St. Marie, 2002).

Intensity or severity of pain is typically measured by one of the available assessment tools. The choice of pain assessment instrument typically depends on the characteristics of the individual patient. Options include a visual analog scale (VAS), numerical rating, verbal rating, faces rating, and behavioral observation scales (Dunwoody et al., 2008). Numeric rating scales are commonly used due to their ease of administration and understanding. With such tools, patients are asked to rate the severity of their pain on a scale of 0 (representing "no pain") to 10 (representing the "worst imaginable pain") (Bertagnolli, 2004; Dunwoody et al., 2008; Li, 2008). VASs, numeric rating scales, and verbal rating scales are all valid, reliable, and appropriate for clinical use (Dunwoody et al., 2008; Williamson & Hoggart, 2005). Identification of the best pain assessment tool for the patient preoperatively can assist the patient to provide the most reliable information using the scale postoperatively, thereby resulting is more effective pain management (Gelinas, 2007b).

Duration of pain includes questions concerning the timing of when the pain starts, how long it lasts, and which factors alleviate it. Patients are also asked about factors that aggravate the pain. Additionally, determining if any associated factors exist can assist the nurse in developing a more comprehensive treatment plan. For example, do nausea and vomiting accompany the pain? Treatment may need to include measures to control these associative factors. Determining how the pain affects the individual's activities of daily living provides supplemental information about how the pain interferes with normal functioning. Treatment strategies then can be aimed at specific challenges the patient faces related to daily life events (St. Marie, 2002).

Although a considerable amount of research on pain has been conducted over the past several decades, cardiac surgery patients have been the focus of only a small percentage of these studies. In such investigations, the focus has tended to be on pain intensity, with other important pain factors not being covered. Mueller and colleagues (2000) evaluated cardiac surgery patients' pain and found that pain intensity was highest during the first

2 postoperative days and lowest on days 3 and 7. Age was the only demographic variable that affected pain in this study; patients older than age 60 had lower pain intensity scores on the second postoperative day than did patients younger than age 60. Pain location varied, with patients reporting more shoulder pain on day 7. Pain distribution did not vary in this study.

Pain Assessment in Patients Who Are Unable to Communicate

Assessment of pain for patients who are unable to communicate can pose a challenge for nurses. It cannot be assumed that noncommunicative patients are not in pain. Instead, these patients must be assessed and treated appropriately for pain (Erdek & Pronovost, 2004; Gelinas, Fillion, Puntillo, Viens, & Fortier, 2006).

Utilization of a behavioral observation pain assessment tool may be necessary based on the patient's condition. Behavioral assessment tools have been created to measure those patient behaviors labeled as being indicative of the presence of pain. Examples of tools that have been tested in acute care settings with acute pain and that have the most reliable and valid data to date include the Checklist of Nonverbal Pain Indicators (CNPI), the Behavioral Pain Scale (BPS), and the Critical-Care Pain Observation Tool (CPOT). Typically, behaviors observed include facial expressions, body movements, ease of breathing, vocalization, and/or muscle tension. Points are given for observed behaviors in each category, resulting in a pain score that can be compared to the results obtained with more traditional pain assessment tools (Herr et al., 2006).

The American Society for Pain Management Nursing recommends that a hierarchical approach for assessment be used. First, all attempts must be made to obtain a patient self-report of pain. Explanation as to the difficulty of obtaining a self-report should be documented, and further investigation should ensue. A search for the potential cause of pain is conducted, with the provider assuming that pain is present. Observation of behaviors is the best approach when the patient is unable to make a self-report. Pain behaviors, however, do not always accurately depict pain intensity. Family reports of patient pain may also be considered, as family members can often identify subtle changes in the patient's status. However, discrepancies do exist between patient self-reports and reports given by other observers, such that severity of pain may not be accurately reported. Therefore, one must consider all aspects of an assessment and perhaps perform an analgesic trial, monitoring the patient's response to the medication (Herr et al., 2006).

MANAGEMENT OF PAIN

Adequate pain management is essential for the well-being of all patients and may even be considered a fundamental human right (Brennan, Carr, & Cousins, 2007). The consequences of inadequate pain management encompass physiologic, psychological, social, and economic ramifications. Physiologically, unrelieved pain results in many adverse effects, including increased heart rate, systemic vascular resistance, and circulating catecholamines. These effects place patients at greater risk for myocardial ischemia, stroke, and bleeding. Additionally, chronic pain results in decreased mobility, decreased sleep, immune dysfunction, dependence of medication, and codependence on family members. In terms of psychological effects, studies have shown that patients with chronic pain are four times more likely to suffer from depression and anxiety. Social and economic consid-

erations include the inability or decreased ability to work, which directly affects the individual's socioeconomic status and also has an impact on unemployment, disability, and use of government benefits (Brennan et al., 2007).

PHASES OF PAIN

The patient experiences pain through three phases: anticipation, presence, and aftermath. The nurse's responsibility is to intervene and assist the patient with each pain phase (McCaffery, 1972).

During the anticipation phase, interventions should focus on education and the reduction of anxiety. Anxiety has shown to increase the intensity of pain scores. Anxiety can often be relieved by knowing that pain may occur and developing a plan to manage it. This goal is best accomplished if these issues are discussed preoperatively prior to the occurrence of pain (Gallager & McKinley, 2007; McCaffery, 1972).

The presence of pain is the phase where interventions can directly affect the patient's level of comfort and pain intensity. Physiologically, the management of pain revolves around altering the source and perception of pain and blocking the transmission of pain impulses within the nervous system. Different pharmacologic and nonpharmacologic agents perform differently in relation to the pathology of pain. One strategy is to block or limit the effect of local mediators at the site of injury and decrease inflammation. Nonsteroidal anti-inflammatory drugs (NSAIDs) are able to block specific mediator production such as prostaglandin, thereby decreasing inflammation. Other medications such as clonidine (Catapres®) block the release of epinephrine from the nerve fibers. A second strategy involves limiting transmission to the secondary neurons in the dorsal horn. The action potential can be blocked or inhibited by local anesthetics and some anticonvulsants such as phenytoin (Dilantin®). Opioids also are used to inhibit both synapses at the dorsal horn. Additionally, nonpharmacologic interventions such as massage and application of heat or cold may inhibit transmission of pain-related messages. The final strategy in pain management is to enhance the inhibition of the pain sensation. Opioids are the agents of choice in such a case, as they will affect both the primary and secondary neurons. Tricyclic antidepressants can have the same effect by interfering with serotonin uptake and primary neuron transmission (Lome, 2005).

Opioids, non-opioids, and other analgesics used as adjuvant therapies are the mainstays in pain management (Cadden, 2007). Treatment modalities may differ, depending on whether the goal is treating acute versus chronic pain, or nociceptive versus neuropathic pain.

Traditionally, pain management strategies have applied the World Health Organization's (WHO) cancer pain treatment ladder in attempts to manage postoperative pain. This ladder suggests that the first step should include treatment options using non-opioids with or without adjuvants. Step two entails utilizing opioids for mild to moderate pain. Step three entails continuing opioid use for moderate to severe pain. Postoperative pain reaches its highest level initially after the surgery, but then rapidly improves (Li, 2008; Rosenquist & Rosenburg, 2003).

In one study, Reimer-Kent (2003) developed a pain management guideline using the WHO ladder to prevent pain following cardiac surgery. Under this guideline, most patients received acetaminophen around the clock, 89% of patients received an NSAID, and all patients received intermittent morphine. The morphine was converted from an intravenous medication to an oral preparation on the second postoperative day. The amount of morphine administered declined significantly

by the second postoperative day. Effective pain relief was reported in 95% of the patients in this study.

Evidence-based guidelines must direct efforts to manage postoperative pain related to site-specific surgeries. Rosenquist and Rosenburg (2003) gathered a multidisciplinary group to review and grade the evidence and then provide recommendations along these lines. An algorithmic approach to pain assessment was developed, with a flow diagram outlining the key considerations prior to, during, and after the treatment of pain. Site-specific pain management recommendations were provided regarding the use of pharmacologic and nonpharmacologic therapy. Table 14–1 summarizes these recommendations for pain management in conjunction with cardiothoracic surgery.

Intravenous opioids are the preferred method for managing patients who undergo coronary artery bypass grafting (CABG) surgery. These medications can be delivered via nurse- or patient-controlled methods. The use of patient-controlled analgesia (PCA) devices allows for small doses of opioids to be delivered by the patient. Patients are able to maintain a more stable blood concentration level with this approach, which may provide better pain control (Roediger, Larbuisson, & Lamy, 2006; Rosenquist & Rosenburg, 2003). In one study, most postoperative cardiac surgery

Table 14–1 Summary of Site-Specific Pain Treatment Recommendations

	Thoracotomy	**Coronary Artery Bypass Grafting**
Preferred treatment	**Pharmacologic** Epidural • Opioids Regional local anesthetics **Nonpharmacologic** Application of cold TENS Cognitive (patient dependent)	**Pharmacologic** Intravenous • Opioids • NSAIDs **Nonpharmacologic** None identified
Common usage	**Pharmacologic** PO, IM, IV opioids, and NSAIDs IV PCA opioids Intrathecal opioids Intrathecal local anesthetics **Nonpharmacologic** Cognitive (patient dependent)	**Pharmacologic** PO, IM opioids, and NSAIDs IV PCA opioids Intrathecal opioids **Nonpharmacologic** None identified
Comments	If there is a risk of or actual bleeding, avoid NSAIDs.	If there is a risk of or actual bleeding, avoid NSAIDs. If there is renal hypoperfusion, avoid all NSAIDs. **Rarely Used** Epidural Regional local anesthetics

NSAID = nonsteroidal anti-inflammatory drug; PCA = patient-controlled analgesia; TENS = transcutaneous electrical nerve stimulation.

Source: Rosenquist & Rosenburg, 2003.

patients received hourly intermittent dosages of morphine sulfate. Oral analgesics—primarily acetaminophen with oxycodone—were prescribed as well (Maxam-Moore et al., 1994). Morphine has been used effectively as opioid analgesia in other postoperative cardiac surgery patients (Coventry, Siffleet, & Williams, 2006).

NSAIDs delivered intravenously have also been identified as a preferred method of pain management for CABG patients (Rosenquist & Rosenburg, 2003). Their use in conjunction with opioids in the majority of other surgical procedures is well established. NSAIDs have been found to relieve pain more effectively and to decrease the use of opioids (Roediger et al., 2006). Data suggest that administering NSAIDs results in reduced pain scores, less opioid requirements, and no differences in mortality or incidence of serious side effects (Bainbridge, Cheng, Martin, & Novick, 2006).

However, NSAID use has been limited in cardiac surgery patients due to these medications' potential for troublesome side effects. The actual occurrence of side effects tends to depend on whether the medication inhibits cyclo-oxygenase 1 or 2 (COX-1 or COX-2), or both. Serious side effects, including sternal wound infections, myocardial ischemia, infarction, stroke, and pulmonary embolus, have been reported with the use of COX-2 inhibitors; as a consequence, these medications are not recommended for patients deemed to have an increased cardiac risk (Bainbridge et al., 2006; Roediger et al., 2006).

Epidural and local anesthetics are the preferred methods for managing pain in patients who are undergoing a thoracotomy (Rosenquist & Rosenburg, 2003). Agreement exists that epidural use in these patients provides an excellent method for pain control during the acute pain phase (Jensen & Andersen, 2004; Pennefather, Akrofi, Kendall, Russell, & Scawn, 2004; Tan, Guha, Scawn, Pennefather, & Russell, 2004). Other reported benefits of epidural analgesia include earlier extubation and enhanced pulmonary function (Stenseth et al., 1996).

Data are not consistent regarding the benefits of thoracic epidural anesthesia. In one study involving cardiac surgery patients, participants received either (1) thoracic epidural analgesia in combination with general anesthesia, which was followed by postoperative patient-controlled thoracic epidural analgesia, or (2) general anesthesia, followed by PCA with intravenous morphine. No differences between these two groups were observed in terms of pain relief, pulmonary function, ambulation, level of sedation, length of stay, or quality of recovery. The study authors did conclude, however, that thoracic epidural anesthesia decreases stress response and pain scores (Hansdottir et al., 2006). The major concern when using that method of pain management is the potential for development of an epidural hematoma. When this strategy is used, the ICU nurse must monitor for and report lower extremity motor weakness (Mehta & Kumar, 2004).

The use of intrathecal morphine has been reported to be effective in the management of postoperative cardiac surgery pain. In one study, patients undergoing on-pump bypass who received intrathecal morphine prior to induction of general anesthesia were extubated earlier and had a shorter ICU length of stay than did a comparison group of patients who did not receive the intrathecal injection prior to induction (Yapici et al., 2008).

PAIN SEQUELAE

If postoperative pain is not well controlled, complications such as pneumonia and chronic pain may occur. This possibility has encouraged the evaluation of other options to assist with the control of pain. Local anesthetic agents may prove effective if they are injected into the nerves prior to sternal wound closure (Barr, Tutungi, & Almeida, 2007; Markman & Philip, 2007). However, the

use of other types of local injections has not shown to be equally beneficial. One study examined the use of lidocaine injections in the skin prior to a thoracotomy; the results did not show any decrease in postoperative pain (Cerfolio et al., 2003). In another study, patients with shoulder pain following a thoracotomy procedure had bupivacine injected in the intrapleural space; no effective pain relief was noted (Pennefather et al., 2005). Epidural use has been limited in other cardiac surgery patients due to concerns about hypotension, decreased coronary perfusion, and potential hematoma formation after heparinization (Barr et al., 2007; Markman & Philip, 2007).

Nonpharmacologic interventions to assist with the pain control range from relaxation techniques, application of cold to the surgical site, and use of transcutaneous electrical nerve stimulation (TENS). The success of relaxation techniques typically is patient dependent, although results appear to be better if preoperative teaching has occurred (Rosenquist & Rosenburg, 2003). Unfortunately, to date nonpharmacologic methods of pain control have been only minimally addressed in research on cardiac surgery patients.

Use of music as a pain management approach has been studied and shows some promise. In a review of studies from 1995 through 2007, 42 studies were conducted to assess the efficacy of music as a means of pain control, with roughly half showing pain reduction might be achieved with this method. Of the 42 studies, 7 were conducted with cardiothoracic surgery patients (Nilsson, 2008). A study of postoperative cardiac surgery patients using music during rest in conjunction with medications showed a reduction in anxiety and pain as compared to rest and medications alone (Voss et al., 2004). In a later study, cardiac surgery patients who received music interventions also experienced a significant decrease in anxiety and pain compared to patients who rested. No difference was reported in opioid use between these two groups, however (Sendelbach, Halm, Doran, Miller, & Gaillard, 2006).

A study in patients who underwent CABG surgery revealed a correlation between use of slow, deep breathing with medications during painful procedures such as chest tube removal and a decrease in pain scores immediately after the procedure and 15 minutes later (Friesner, Curry, & Moddeman, 2006).

In a study of postoperative cardiac surgery patients, those who used TENS experienced less pain related to coughing and improved chest wall mechanics, tidal volume, and vital capacity (Cipriano, Carvalho, Bernardelli, & Peres, 2008).

Massage therapy has been implemented to promote comfort and reduce pain in cardiac surgery patients. It has been suggested providing massage helps decrease pain and anxiety in these patients and may be used as an alternative intervention when other nursing and medical treatments are not effective in helping with these symptoms (Anderson & Cutshall, 2007).

REASSESSMENT OF PAIN

The aftermath of the pain experience focuses on the nurse's evaluation of pain. The plan of care is reviewed, and assessment is refocused to determine if the interventions assisted with pain reduction. Reassessment of pain entails a comparison of the pain exhibited after the intervention versus the pain observed at the initial assessment (McCaffery, 1972). Attention must be given by nurses to reassess patients after any treatment to determine efficacy.

Little research has been conducted in the areas of reevaluation of pain and the measurement tools used to determine the patient's level of comfort. One study, however, found a

significant lack of reassessment by nurses. Its authors propose that knowledge, time, and workload may be factors that limit effective reassessment. Interestingly, these researchers found that nurses tended to be more focused on surgical incision pain and, therefore, did not consider other complaints of pain as a priority, resulting in a delay in treatment (Bucknall et al., 2007). It is crucial that nurses be aware of the potential gap in pain control measures that result from periods of increased and uncontrolled pain due to lack of timely reevaluation (Polomano, Dunwoody, Krenzischek, & Rathmell, 2008).

SPECIAL CONSIDERATIONS

Pain is a unique individualized experience. Nevertheless, some elements that affect the response to pain management techniques may apply across certain groups of people. Recognition of these special considerations may assist the nurse with managing an individual's pain. The special considerations for pain management discussed here are gender differences, cultural influences, and older age.

Gender

Research has shown that there are differences in the way males and females perceive and experience pain. Both biological and psychosocial factors are important to consider in this respect. Biological factors include hormone and cardiovascular status (Wise, Price, Myers, Heft, & Robinson, 2002). Other biological factors may relate to the type of stimulus and neural receptor differences (Giles & Walker, 2000). Psychosocial factors encompass individual expectations, emotions, and social learning (Giles & Walker, 2000; Wise et al., 2002). Societal norms in how children of both genders are raised, for example, play a part in their pain experience (Bernardes, Keogh, & Lima, 2008).

When studying pain differences reflective of gender, gender personality traits are measured in relation to pain. For example, persons identified with more feminine characteristics (women) report experiencing more pain than men. Additionally, it is reported that men—that is, those persons with more masculine traits—report a higher tolerance of pain (Bernardes et al., 2008). Gender-related differences exist among pain beliefs, expectations, and behaviors. Gender role expectations can account for males predominately underreporting pain and women being more apt to verbalize pain. Additionally, males demonstrate greater pain endurance, whereas women report a lower threshold and tolerance, resulting in their greater willingness to report pain (Wise et al., 2002). Similarly, studies suggest that women were more likely to experience severe pain and on a more frequent basis. In a study of cardiac surgery patients, female patients rated their pain on a VAS higher than did their male counterparts (Meehan, McRae, Rourke, Eisenring, & Imperial, 1995). Some differences in the reports of pain between males and females may be related to the issue of willingness to make a self-report: Males and females have differing behaviors regarding the expression and response to pain (Miller & Newton, 2006).

These data on gender differences have been corroborated through studies in other patients who have undergone cardiac surgery. Compared to males, females more frequently report less improvement in pain scores or higher pain intensity (Decker & Perry, 2003; Puntillo & Weiss, 1994; Valdix & Puntillo, 1995; Watt-Watson et al., 2004; Yorke, McLean, & Wallis, 2004), report lower health-related quality of life after cardiac surgery (Gjeilo, Wahba, Klepstad, Lydersen, & Stenseth, 2008), and experience a more difficult recovery (Vaccarino et al., 2003).

Conclusions regarding gender differences and recovery from cardiac surgery are not

consistent. King (2000), for example, reported fewer differences between males and females in terms of short-term recovery following cardiac surgery.

It has also been reported that gender-related biases may arise related to receipt of medical treatment. Women have been found to receive less pain medication. One reason for this difference may be related to the gender of the observer and perception of the person's pain (Bernardes et al., 2008; Miller & Newton, 2006). It has also been shown that women who present with pain in association with anxiety tend to receive less medical attention than when the pain is without emotional attachment (Bernardes et al., 2008).

Differences between males and females in the response to analgesia have been documented in the literature. Some studies show greater morphine potency but slower onset of pain relief in females. Additionally, it has been found that NSAIDs have refractory effects in women when doses exceed 800 mg. Pharmacokinetics may play a role in these differences, although studies to date have not shown any significance in clinical practice. One explanation may be that the differences are related to the specific drugs, rather than to whole categories of drugs (Giles & Walker, 2000).

Greenspan and colleagues (2007) suggest the need to address numerous variables in terms of how they influence gender-related pain response. These variables include comorbidities, culture, disability, medications, coping, and physical variables. To be able to appropriately respond to an individual's pain experience, nurses need to be sensitive to various societal norms and communication patterns related to gender and recognize the potential differences in reports of pain and their own potential biases (Miller & Newton, 2006).

Race, Ethnicity, and Culture

Distinguishing differences among the definitions of race, ethnicity, and culture has proved difficult in the literature, with many of the terms being used interchangeably. The term "race" in the literature has been debated as whether it is a biological or social concept. Typically, race is predominantly used to collect data regarding health disparities. There is no globally accepted definition of race; however, the National Institutes of Health has adopted the use of five racial categories to collect its data (Ezenwa, Ameringer, Ward, & Serlin, 2006).

By comparison, "ethnicity" refers to a group of people who share ancestry, social background, culture, and traditions that are sustained over a period of time and provide a sense of identity for group members. Typically, self-identification is the best approach to assigning individuals to a particular ethnic group (Lasch, 2002).

Finally, "culture" seems to be derived from behavioral and attitudinal norms in relation to belief systems. Culture is viewed as a factor influencing healthcare practices and illness beliefs. In terms of the pain experience, then, culture affects all areas related to pain, including expression, reporting, and management (Lasch, 2002).

Given that there are differences in the way these terms are used and studied, findings in this arena must be reviewed carefully. For example, a study of low back pain found that immigrated Latinos in New York showed pain responses more similar to the responses of a New England Latino group than to the responses of a group from Puerto Rico. It was concluded that the pain response is shaped by culture (Morris, 2001).

Questions often arise as to whether race, ethnicity, and culture affect how different groups biologically experience pain or how the factors influence the perception of pain. Additionally, culture can influence how the caregiver assesses and treats the pain of persons from different ethnic backgrounds.

Authors of several studies have reported that minorities are at higher risk for under-

treatment of pain (Lasch, 2002). Although not studying cardiac surgery patients specifically, significant differences have been reported in terms of the amount of narcotic analgesics administered to patients who were white as compared to those who were Hispanic or black (Bernabei et al., 1998; Bonham, 2001; Ng, Dimsdale, Rollnik, & Shapiro, 1996; Ng, Dimsdale, Shragg, & Deutsch, 1996; Todd, Deaton, D'Amato, & Goe, 2000; Todd, Samaroo, & Hoffman, 1993). In these cases, patients who were white received higher amounts of narcotics than did patients of other races. A systematic review of studies in this area reveals further disparities in pain management related to ethnicity and race in the United States (Cintron & Morrison, 2006). Consistent with these data, a relationship between race and ethnic background and prescriptions given for PCA has been identified (Ng, Dimsdale, Rollnik, & Shapiro, 1996; Salamonson & Everett, 2005). The lesson for critical care nurses is that care must be taken to acknowledge potential differences related to race, ethnicity, and culture in how pain is experienced, including the awareness that people are individuals with individual needs.

The Elderly Population

Pain management is complex with all patients—but it may be even more complex in the elderly population. This difference may be explained by elderly patients' tendency to underreport pain, difficulty communicating, and caregiver biases regarding the use of pain medication in the older patient (Goldstein & Morrison, 2005).

Although there may be no changes in the perception of pain in the older patient, some physiologic changes do occur with aging that need to be considered when utilizing pain medications. In particular, physiologic changes related to kidney and liver function may affect the way the older patient can metabolize and eliminate pain medication, resulting in a longer duration of action (Burgess & Burgess, 2008; Goldstein & Morrison, 2005). For this reason, care must be taken when prescribing, administering, and monitoring the effects of pain medications in the elderly population (Goldstein & Morrison, 2005).

Assessment is crucial for managing pain in the older patient. The pain scale chosen is important to the individual functioning of the older patient, and the same scale should be used consistently when assessing a particular patient to ensure reliability of the results (Goldstein & Morrison, 2005). Trying different scales to determine the best fit for individual patients is a good strategy. Often, older patients have more success using simple word scales, such as "none," "mild," "moderate," and "severe," than with a numeric scale or a VAS. Often, if the older patient has any cognitive impairment, rating pain may be difficult. The FACES scales, which seems to be easily used, often produces unreliable results with the elderly. The faces are often seen by these individuals as representing moods like sadness instead of pain, which results in understated pain intensity (Burgess & Burgess, 2008).

Additional challenges in using pain scales in the elderly population relate to vision and hearing loss, which are more prevalent in older individuals. It is critical that the nurse assess for these deficits and utilize assistive devices as appropriate.

For the patient with vision impairment, it might be best to use a verbal reporting scale; in contrast, hearing-impaired patients may prefer to use a printed scale that they can point or gesture toward. Depending on the severity of cognitive impairment, using a yes/no question with a behavioral observation scale may be the best option. Studies have found that using a family member's or nurse's judgment of the pain often leads to underestimation of the actual pain experienced (Goldstein & Morrison, 2005).

The older patient's view of pain and pain management is also considered a barrier to the effective assessment and treatment of pain. Many older patients may fear addiction to painkillers and, therefore, underreport their pain. Preparing patients for pain management following surgery provides nurses with a unique opportunity to educate patients and their families about pain, tolerance, dependence, addiction, the patient's disease process, and other pain control techniques (Goldstein & Morrison, 2005).

Treatment strategies for the older patient can include medication regimens in which the patient does not have to request treatment. Options to consider may include nerve blocks, epidural analgesia, and around-the-clock dosing of pain medications. Opioids are the most widely utilized therapy with surgical patients, though their dosing needs to be considered carefully. Typically, if communication deficits are present, nurses may be fearful of postoperative delirium and withhold opioids. Nevertheless, even though these medications may potentially contribute to postoperative delirium, recent studies suggest that patients with higher pain scores and uncontrolled pain are more likely to develop delirium (Burgess & Burgess, 2008).

The level of postoperative pain following cardiac surgery in relation to age has been investigated; results have been inconsistent, however. In one study, patients older than age 60 received less analgesic therapy than younger patients (Celia, 2000). Yorke, McLean, and Wallis (2004) found that older postoperative cardiac surgery patients received less analgesic therapy and were refused pain medication more often than were younger patients. In contrast, Decker and Perry (2003) reported higher levels of self-reported pain in older patients.

SUMMARY

Adequate pain assessment and management for cardiac surgery patients are pivotal in helping ensure a successful outcome. This complex process involves timely assessment, intervention, reassessment, and evaluation of pain management strategies. The relationship of traditional pain treatment strategies and their impact on pain physiology places the nurse in a better position for advocating for the best treatment strategy for patients. The care focus needs to address not only the acute postoperative period, but also the potential for development of chronic pain.

Nurses can best address their patients' pain by employing a thorough and systemic approach to pain assessment and treatment. Recognizing the importance of reassessment and evaluation can have a direct impact on the patient's experience and satisfaction regarding the nursing care provided. Knowledge that assessment techniques may need to be adjusted when providing care to nonverbal and elderly patients may allow for more optimal pain control. Acknowledging the differences in pain perception related to gender and culture will allow the nurse to recognize trends and avoid biases when dealing with different groups of people. Recognizing the individual and subjective nature of the pain experience will aid in maximizing positive outcomes for pain management.

CASE STUDY

A 79-year-old Hispanic female's admission status is post myocardial infarction, with preparations being made for CABG. The nurse takes the presurgical initial health history. During the course of this interaction, the nurse notes that the patient predominantly speaks Spanish, but can also speak English. Her daughter is available at the bedside. The patient reports a medical history of adult onset diabetes controlled with oral agents and chronic pain due

to arthritis. The physical assessment is unremarkable. The daughter adds that the patient has a history of anxiety. Current medications include aspirin daily, Diabeta® prior to meals, multivitamin daily, ibuprofen twice a day, Ambien® as needed at night for sleep, and Xanax® as needed every 12 hours for anxiety.

Critical Thinking Questions

1. Name the top priorities of your treatment plan regarding pain management?
2. Which factors may predispose the patient toward poor pain control postoperatively?
3. Which strategies could you implement to promote the best postoperative pain control?

The patient undergoes a CABG due to a left main artery blockage. She arrived to the ICU on a ventilator, with invasive hemodynamic monitoring, midsternal chest tubes, intravenous fluids, and vasopressor infusing. Morphine sulfate 1–2 mg/hr is ordered as needed for pain. The patient's vital signs are stable, with appropriate hemodynamic parameters. Weaning from the ventilator and vasopressors has commenced without difficulty. The surgeon and the anesthesiologist prefer to have the patient extubated as soon as possible. As the patient begins to wake up, you notice her grimacing and her respiratory rate increases. You decide you need to further assess her and treat her for pain.

Critical Thinking Questions

4. Which assessment technique provides the best evaluation of the patient's current pain?
5. Which factors should be considered when planning for treatment of this patient's pain?

You treat the patient with the lower range of the ordered dose of morphine sulfate. When reassessed after 30 minutes, the patient shakes her head "no" when asked if the pain is relieved.

Critical Thinking Question

6. What should be the nurse's next steps?

Answers to Critical Thinking Questions

1. The patient's pain will be adequately managed to a level below 4 on a 1–10 scale. The patient will be able to state preoperatively the plan of care regarding the treatment of pain and management of potential side effects.
2. Gender, age, history of chronic pain, culture, and history of anxiety.
3. Preoperative education regarding expectations related to pain and the strategies to treat pain is essential. Review of pain assessment scales to determine the best method for individual patients allows patients to best report their pain postoperatively.
4. All attempts should be made for the patient to self-report her pain. Use of visual or numeric analogue scale allows the patient to point to the number that corresponds with her pain level.

5. Attempt to identify the location of the pain by having the patient point to that area. You should ensure that the pain is surgical and not related to chronic pain of arthritis. Should the patient's NSAID medication be restarted? What is her history of medication response? Which doses previously had relieved the pain? The patient's experience with chronic pain may require a larger dose of pain medication now. Is the patient exhibiting any signs of anxiety? If so, this may contribute to her current pain experience. What is the time frame related to extubation? If extubation will occur in the near future, you may need to consider a lower opioid dose and other adjuvant strategies.
6. Consider re-treating the patient with the PRN morphine sulfate as prescribed. Consider seeking treatment for anxiety if the assessment suggests that anxiety may be contributing to her pain. If the patient's pain remains uncontrolled, consider a PCA. The lower basal dose may be more effective in controlling the patient's pain, plus it will allow the patient to remain awake and able to participate in her care. Additionally, assess the possibility of restarting NSAIDs for the patient's arthritis and new inflammatory processes as soon as deemed appropriate. NSAIDs are appropriate treatment options for CABG patients, though they are not often used immediately postoperatively due to their potential to enhance bleeding.

SELF-ASSESSMENT QUESTIONS

1. Which of the following is the most important assessment for the nurse to determine that a patient is experiencing pain?
   a. Measurement of increased blood pressure
   b. Observation of grimacing to touch
   c. Patient's report of pain
   d. Patient guarding the surgical area
2. Which of the following statements by the nurse best explains the concept of pain physiology?
   a. Pain physiology is a complex series of steps that includes tissue injury, which then triggers a reaction that results in the transmission of impulses to the spinal cord and brain, producing the perception of the pain.
   b. Pain is caused by actual events that cause injury to the tissues, which results in the brain perceiving the stimulus of pain.
   c. Pain physiology is truly a physiologic phenomenon that occurs as a result of injury to the skin, which is perceived by the person owing to triggers located in the brain.
   d. Pain physiology is subjective and categorized as acute or chronic, with the differentiation related to how long the patient has exhibited the symptoms.
3. A nurse is caring for a patient who is 5 days status post CABG surgery. Which of the following best describes the type of pain this patient is experiencing?
   a. Acute, neuropathic pain
   b. Acute, nociceptive pain
   c. Chronic, neuropathic pain
   d. Chronic, nociceptive pain
4. The nurse can best ascertain the severity of pain by asking the patient to
   a. describe the pain.
   b. discuss the timing and duration of the pain.
   c. rate the pain on a scale of 0 to 10.
   d. report relief of pain after treatment.

5. Which of the following is the best method to assess pain in a nonverbal patient?
   a. Family observations
   b. Physiologic changes
   c. FACES pain scale
   d. Behavioral assessment tool
6. Which of the following strategies would best suit the pain experience phase of anticipation?
   a. Education
   b. Continual reassessment
   c. Pharmacologic management
   d. Relaxation exercises
7. Which of the following pharmacologic combinations is the preferred treatment method for patients having a thoracotomy?
   a. Intravenous opioids and NSAIDs
   b. Intravenous PCA-delivered opioids and local anesthetics
   c. Epidural opioids and local anesthetics
   d. Epidural opioids and NSAIDs
8. Which of the following statements best explains why NSAIDs are limited in cardiac surgery patients?
   a. The potential for serious side effects with NSAID administration outweighs the benefits of these medications' use.
   b. There have been reports of neurological changes in elderly patients after NSAID administration.
   c. The inflammatory process is necessary for healing to occur.
   d. The amount of opioids used postoperatively is unchanged regardless of NSAID administration.
9. A nurse is caring for a male patient who requires cardiac surgery. Which of the following statements by the nurse indicates an understanding of gender differences related to pain?
   a. "Because males have a faster metabolism compared to females, males may require a higher dose of opioids to control their pain."
   b. "Observation of other indicators and behaviors of pain is warranted because males often underreport pain."
   c. "Caregiver perceptions regarding pain perception need to be recognized because males have a lower threshold for pain and complain more often than do females."
   d. "Gender differences have been linked primarily to physiologic processes, and men may require more aggressive treatment than females."
10. Which of the following issues is the most challenging in the treatment of pain in the elderly?
    a. Communication impairment may limit objective assessment data.
    b. Physiologic changes related to kidney and liver function may alter dosing guidelines.
    c. Fear of addiction may limit the patient's report of pain.
    d. Caregiver biases may lead to the under treatment of pain in elderly patients.

Answers to Self-Assessment Questions

1. c
2. a
3. b
4. c
5. d
6. a
7. c
8. a
9. b
10. a

Clinical Inquiry Box

Question: Do certain activities postoperatively induce more pain than other activities?

Reference: Milgrom, L., Brooks, J. A., Qi, R., Bunnell, K., Wuestefeld, S., & Beckman, D. (2004). Pain levels experienced with activities after cardiac surgery. *American Journal of Critical Care, 13*(2), 116–125.

Objective: To describe pain levels associated with five postoperative activities following cardiac surgery.

Methods: A secondary, descriptive analysis design was used for a subset of 711 patients undergoing cardiac surgery. The patients were divided into four groups: Group 1 consisted of those patients who had undergone single (aortic or mitral) valve replacement; group 2 patients underwent an on-pump CABG procedure; group 3 patients had an off-pump CABG procedure; and group 4 patients had undergone multiple cardiac surgeries. On postoperative days 1–6, the 711 patients were asked to rate their pain following five activities: rest while lying in bed, coughing, deep breathing or using an incentive spirometer, movement or turning in bed, and getting up to a chair or to walk. Analysis was focused on comparing pain levels for the postoperative day, activity, and type of surgery. Pain scores before and after chest tube removal and extubation were analyzed.

Results: Pain scores were higher on earlier postoperative days. The highest pain level was reported during coughing. As time progressed, pain lessened significantly. Changes in pain reported with coughing and deep breathing or using the incentive spirometer were significant over time between some surgery groups. The removal of chest tubes was a significant event in decreasing the pain associated with activities such as being at rest, coughing, and getting up.

Conclusion: Pain was present on postoperative day 6 in all patient groups, which indicates a need for nurses to critically evaluate pain and provide measures that will bring about relief. While nurses should certainly treat each patient individually, there is important information to be applied from this study. Although the level of pain did diminish over the 6-day period in all groups, nurses need to respond more aggressively to control pain. The removal of chest tubes resulted in a significant decrease in pain level with activities, and may be a point in the recovery trajectory at which to consider the timing of education.

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WEB RESOURCES

American Academy of Pain Management: www.aapainmanage.org/

Pain Management Nursing: http://www.painmanagementnursing.org/

American Pain Society: www.ampainsoc.org/

National Pain Foundation: www.nationalpainfoundation.org/default.asp

International Association for the Study of Pain (IASP): www.isap-pain.org/

American Society for Pain Management Nursing (ASPMN): www.aspmn.org/

National Pain Education Council: www.npecweb.org/

Daily Pain Diary: www.americangeriatrics.org/education/daily_pain_diary.pdf

Inside look at chronic pain: www.or-live.com/distributors/nlm-flash/chp_1867/rnh.cfm?id=704

Transcutaneous electrical nerve stimulation (TENS): www.intelihealth.com/IH/ihtIH/WSIHW000/8513/34968/363973.html?d=dmtContent

Chapter 15

Postoperative Dysrhythmias

Roberta Kaplow and Dawn B. Adams

INTRODUCTION

Patients may have dysrhythmias prior to surgery or develop them postoperatively. Indeed, dysrhythmias are a common complication following cardiac surgery. The origins of such dysrhythmias often include the atrium, atrioventricular (AV) node, and ventricle. This chapter discusses the most commonly encountered postoperative dysrhythmias, including their incidence, etiology, and suggested management.

Dysrhythmias may compromise cardiac output (CO) when they interfere with diastolic filling. It is essential that the nurse working in the ICU with postoperative cardiac surgery patients be proficient in identifying and possibly eradicating potential causes as well as promptly recognizing potentially life-threatening dysrhythmias. Assessment of the patient requires evaluation of cardiac rhythm, its effects on systemic perfusion, and etiologic factors.

ETIOLOGY

Several potential etiologic factors related to postoperative cardiac surgery dysrhythmias have been identified. Dysrhythmias in this patient population may result from cardiac problems (e.g., pericarditis, atrial infarction or ischemia, injury to the atrium during surgery, fluid overload-induced acute atrial enlargement), respiratory complications, electrolyte disturbances (e.g., hypokalemia, hyperkalemia, hypomagnesemia), surgical trauma (inadequate cardioprotection during bypass procedures), hypothermia, hyperadrenergic state, acid–base imbalance, anxiety, or pain (Bharucha & Marinchak, 2007).

The overall reported incidence of premature beats, tachydysrhythmias, and bradydysrhythmias is reported to be 30–50% in patients who have undergone coronary artery bypass grafting (CABG) procedures (Brister & Lenkei-Kerwin, 2005). The incidence is higher in patients who have undergone valve surgery or CABG in combination with valve surgery. In fact, patients in one study who had undergone CABG, valve and CABG, and valve-only procedures had a reported dysrhythmia incidence of approximately 90%, 100%, and 50%, respectively (Dewar, Rosengarten, Blundell, & Chiu, 1985).

ATRIAL DYSRHYTHMIAS

Atrial dysrhythmias are the most commonly encountered rhythm abnormalities encountered in the postoperative CABG patient. Their incidence is reported to be as high as 50% in patients undergoing valve replacements (Hogue, Creswell, Gutterman, & Fleisher, 2005). Atrial dysrhythmias that may develop in the postoperative cardiac surgery

patient may include sinus tachycardia, premature atrial contractions, atrial fibrillation (AF), and atrial flutter.

Sinus tachycardia is a common dysrhythmia following surgery in general, with cardiac surgery being no exception. It can be attributed to the normal stress response. As a general guideline, treatment of sinus tachycardia should focus on ameliorating its underlying cause. Etiology of sinus tachycardia may include pain, fever, anxiety, anemia, medications (e.g., catecholamines, pancuronium), hypermetabolic state (e.g., sepsis), or an increase in adrenergic tone (e.g., in a patient taking a beta blocker preoperatively). The presence of sinus tachycardia is not likely to cause adverse effects if the patient has normal left ventricular (LV) function, and treatment is usually not indicated (Brister & Lenkei-Kerwin, 2005).

Premature atrial contractions may also develop in the postoperative cardiac surgery patient. These abnormal beats are usually not clinically significant and rarely require treatment. However, they may signal the development of atrial tachydyshythmias in this patient population (Brister & Lenkei-Kerwin, 2005). Although not consistently defended in the literature, consideration may be given to administering magnesium sulfate in the immediate postoperative period to prevent or treat atrial tachydysrhythmias (Piotrowski & Kalus, 2004).

AF is a common dysrhythmia that may occur in the postoperative cardiac surgery patient. Its reported incidence ranges from 10% to 65% in this patient population. The incidence varies with type of procedure performed, with 20–40% for patients who have undergone CABG procedures developing AF and as many as 50% of patients who have undergone valve surgery experiencing this complication. The onset of occurrence is 1 to 3 days following surgery (Archbold & Zaman, 2000; Brister & Lenkei-Kerwin, 2005; Creswell, Schuessler, Rosenbloom, & Cox, 1993; Khalpey, Ganim, & Rawn, 2008; Maisel, Rawn, & Stevenson, 2001). The majority of patients with new-onset AF convert back to normal sinus rhythm (NSR) within 6 to 8 weeks postoperatively (Khalpey et al., 2008). It has been suggested that patients who develop AF following cardiac surgery are more likely to have other complications, including myocardial infarction (MI), heart failure, or respiratory failure (Almassi et al., 1997).

The pathophysiology of AF involves the rapid release of multiple impulses from the atrium to the AV node; however, the AV node can respond to only a few of these impulses. In AF, the patient's heart does not contract with maximum efficiency. The rapid quivering of the atria may result in hemodynamic compromise from decreased atrial filling and the atrial kick that can normally contribute as much as 20% of CO. In patients with normal LV function, however, AF is generally well tolerated (Brister & Lenkei-Kerwin, 2005). Because the blood lingers in the atria with AF, small clots may develop, which place the patient at risk for stroke. Development of AF may lead to increased length of hospital stay and greater use of resources (Aranski et al., 1996; Archbold & Zaman, 2000).

Treatment of AF focuses on control of rate and rhythm. Ultimately, intervention to bring about a conversion from AF to NSR is most desirable. Numerous approaches have been employed, including use of antiarrhythmic agents, synchronized cardioversion (SCV), and surgical interventions (e.g., Cox-Maze III, Ex-Maze procedure, or cryoablation). The Cox-Maze III procedure has a success rate of 75–97% and is often performed during the repair of a mitral valve (Ghavidel et al., 2008). In addition, a variety of ablative techniques that electrically separate the pulmonary veins from the atria have been used to ensure that impulses are not conducted. While ablative methods do not have as high a success rate as some other surgical approaches, they are simpler, are cost-effective, and have a 57–70% cor-

rection rate (Jais et al., 2004). Surgical treatment of AF is discussed in more detail in Chapters 3 and 6.

Risk Factors for Postoperative Atrial Dysrhythmias

Many studies have attempted to determine the etiology of AF in the postoperative cardiac surgery patient. A patient's demographic data and medical history provide insight into the probability of postoperative dysrhythmias. The primary predictor of postoperative AF is age. As the body ages, structural and size changes of the atria predispose the individual to develop atrial dysrhythmias (Sethares, Seifert, & Smith, 2008). Other risk factors include pericarditis, previous cardiac surgery, increased adrenergic tone, electrolyte depletion (e.g., potassium, magnesium), valvular heart disease, atrial enlargement, and preoperative atrial dysrhythmias (Creswell et al., 1993; Fuster & Ryden, 2001; Maisel et al., 2001).

A detailed preoperative history is important in preventing postoperative dysrhythmias. Analysis of the ECG will reveal the presence of preexisting conditions such as left ventricular hypertrophy, which is also believed to be a precursor to postoperative dysrhythmias (Hogue et al., 2005). The preoperative ECG also plays a role in later care, serving as a basis for comparison postoperatively. Chronic disorders such as those previously mentioned are thought to contribute to AF through their effects of remodeling of atrial tissue (Sethares et al., 2008). If AF is present, its rate should be controlled and its duration identified. If AF has been present for less than 6 months, it is likely that conversion to NSR can be obtained postoperatively (Piotrowski & Kalus, 2004).

Remodeling of the myocardium occurs in patients who experience long periods of volume or pressure elevation. The tension exerted on myocardial cells causes a reconfiguration of the muscle fibers. For patients with a previous history of chronic disease or congenital heart defects, changes in elasticity of the atrial wall lead to increased atrial excitability. Conduction pathways other than the heart's normal pathway develop in the refashioned tissue, with the ultimate result being AF or flutter (Furer, Gomes, Love, & Davendra, 2005). Knowing the factors that predispose the patient to developing AF, the nurse will attempt to anticipate the occurrence of this complication and will be prepared to quickly respond to the dysrhythmias and convert the patient to NSR.

Another factor thought to contribute to AF is hypomagnesemia (Archbold & Zaman, 2000; Miller et al., 2005). Hypomagnesemia may be attributed to the effects of hemodilution and beta-adrenergic-mediated mechanisms. Data remain inconsistent regarding the correlation between AF and low magnesium levels. Results of a meta-analysis suggest that prophylactic preoperative administration of magnesium may decrease the incidence of postoperative AF without increasing either morbidity or hospital length of stay (Miller et al., 2005). Prophylaxis for AF should include magnesium sulfate 2 g IV after cardiopulmonary bypass and on the first postoperative morning. In addition, metoprolol 25–50 mg PO or by nasogastric tube may be started 8 hours after surgery (Piotrowski & Kalus, 2004).

In another study, Baldwin and Heland (2000) noted the incidence of dysrhythmias to be approximately 20% in postoperative cardiac surgery patients. The dysrhythmias were reported to be related to removal of a pulmonary artery catheter. The most life-threatening of the dysrhythmias reported was nonsustained ventricular tachycardia (VT), which occurred in 2% of the cases.

Intraoperative Factors Contributing to Postoperative Atrial Dysrhythmias

Manipulation of the heart during surgery and changes in the fluid and electrolyte composition of the blood may also influence the incidence of postoperative dysrhythmias.

Techniques used to stop the heart from beating and to isolate the surgical area may be contributing factors to this complication. For on-pump procedures, the heart must be cooled and induced into arrest. Electrolyte-rich cardioplegic solutions are infused into the heart to allow the surgeon to work on a stabilized, nonbeating organ with this surgical approach (Guo-Wei, 1997).

Creswell, Alexander, Ferguson, Lisbon, and Fleisher (2005) reported a correlation between atrial ischemia and prolonged cardioplegia. Moreover, with the utilization of on-pump procedures, an inflammatory response that releases histamine during use of the aortic cross-clamp is associated with AF and flutter development (Fayaz, Pugh, Balachandran, Sudheer, & Hall, 2005).

During the intraoperative period, fluid balance and electrolyte composition in the blood are altered. Patients receive fluid boluses, and electrolyte shifts occur with loss of blood and the bypass procedure. Oftentimes, the electrolyte shifts result in decreased levels of potassium, magnesium, calcium, and pH, which are all essential to electrical conduction (Martin & Turkelson, 2006).

Data from another study suggest that the temperature of systemic cooling during bypass procedures contributes to the development of AF after CABG surgery. Data from this study also confirm that increasing age is a significant contributor to the incidence of AF in the postoperative cardiac surgery population (Adams et al., 2000). Other identified possible etiologic factors of AF that have been identified in the literature include atrial stretch from volume overload, increased sympathetic tone, ischemia, pericarditis, sympathomimetic agents (e.g., bronchodilators, inotropes), respiratory conditions (e.g., pneumonia, pulmonary embolism, atelectasis), metabolic/acid–base/electrolyte disturbances, and surgical manipulation (Khalpey et al., 2008; Stamou, Hill, & Sample, 2001).

Atrial Dysrhythmias Following Cardiac Transplant

Following cardiac transplantation, the ECG may reveal two P waves. In orthotopic heart transplantation, part of the right atrium of the diseased heart remains attached to the aorta and the donor heart is connected at this juncture. White-Williams and Grady (2008) delineated other sources to explain this phenomenon of the two P waves' appearance. If the donor P wave has the smaller height of the two waves, it indicates that the leftover sinus node of the removed heart is regulating the spread of the impulse onto the AV node and His bundle of the donor heart. The waveform may be misinterpreted as complete heart block.

In the cardiac transplant patient, postoperative AF incidence is the same as in other cardiac surgical procedures. Concern for electrolyte replacements related to volume resuscitation is also similar. Balance of potassium, magnesium, and calcium must be tightly controlled (Wade, Reith, Sikora, & Augustine, 2004).

Management of Postoperative Atrial Dysrhythmias

Several treatment strategies for the management of AF have been reported. When a postoperative cardiac surgery patient develops AF, hemodynamic stability status, possible underlying causes, and goals of treatment are all key considerations that need to be identified promptly.

Atrial fibrillation can decrease CO by 25–30%. Establishment of hemodynamic stability should be the principal goal of therapy. Signs of hemodynamic compromise may include hypotension, mental status changes, decreased urine output, impaired peripheral perfusion, chest pain, and signs of decreased CO or increased filling pressures (Khalpey et al., 2008).

Hemodynamic stability may need to be attained by controlling rate, rhythm, or both;

the former is usually all that is required and is the preferred method to treat AF (Fuster & Ryden, 2001). The pharmacologic agents used to prevent or treat postoperative dysrhythmias are discussed in detail in Chapter 12.

Agents that are used to control heart rate include beta blockers, calcium channel blockers, and digoxin. Digoxin may be effective in controlling the ventricular rate associated with AF, but is reportedly not as effective as beta-blocker therapy (Bharucha & Marinchak, 2007).

Metoprolol (Lopressor®) is recommended as first-line therapy for most patients. Diltiazem (Cardizem®), administered as an IV bolus and followed by an infusion, is the calcium channel blocker of choice. Calcium channel blockers are generally used if beta blockers are contraindicated. Digoxin may be contemplated for patients with contraindications for beta-blocker therapy (e.g., patients with low ejection fraction) (Khalpey et al., 2008). Data suggest that digoxin is less effective as a prophylactic measure (Thompson, Hirsch, & Pearson, 2002).

Antiarrhythmic agents used to convert the patient from AF include metoprolol, diltiazem, ibutilide (Corvert®), and amiodarone. Ibutilide is administered if SCV does not convert the rhythm. This medication is given as a bolus and may be repeated once. The ICU nurse must monitor the patient for development of torsade de pointes if ibutilide is used (Khalpey et al., 2008).

Prophylaxis for AF usually entails use of beta blockers, which should be resumed as soon as possible postoperatively. The efficacy of measures aimed at preventing postoperative AF is enhanced when beta blockers are initiated preoperatively (Maisel et al., 2001). Contraindications to beta blocker use include hemodynamic compromise, use of inotropes, and presence of heart block (i.e., first-degree heart block with a PR interval greater than 0.24 second, second- or third-degree heart block).

Although varying by procedure type, the overall decrease in risk for postoperative AF development with prophylactic beta-blocker administration has been reported to be 72% (Andrews, Reimold, Berlin, & Antman, 1991). Studies of patients who are undergoing CABG revealed that their risk for this complication decreased from 40% to 20% when prophylactic beta-blocker therapy was implemented. Similarly, patients having valve surgery had a decrease in risk from 60% to 30% (Fuster & Ryden, 2001; Maisel et al., 2001).

Amiodarone (Cordarone®) has been used prophylactically to prevent atrial dysrhythmias, though its efficacy in this indication is not as high as that for some other medications. Toxicities (e.g., pulmonary toxicity, thyroid, or liver dysfunction) should be considered if amiodarone is being used in this manner (Khalpey et al., 2008). Amiodarone prolongs both the duration and the refractory period of the myocardial cell action potential. It possesses mild alpha, beta, and calcium channel blocking effects, which have been shown to be effective in decreasing incidence of AF (Stamou et al., 2001). As noted in Chapter 12, amiodarone is generally recommended if a beta blocker or calcium channel blocker proves ineffective in the conversion of AF (Fuster et al., 2006).

Sotalol (Betapace®), another beta blocker, has also been evaluated for prophylaxis of AF in cardiac surgery patients. Data initially suggested that sotalol was as effective an agent as amiodarone (Wurdeman, Mooss, Mohiuddin, & Lenz, 2002). However, the 2004 ACC/AHA guidelines for postoperative CABG patients reflected a downgrading of the recommendation, as the efficacy of sotalol to prevent AF is not well established based on the most recent data (Bharucha & Marinchak, 2007).

In addition to the use of beta blockers to treat AF postoperatively, research supports the use of treating paroxysmal AF with carvedilol (Coreg®). Carvedilol blocks $alpha_1$ receptors and has a vasodilator effect, so that

its use results in little change in the hemodynamic profiles of patients. Postoperative paroxysmal AF, which is often seen in patients of advanced age, may be prevented with carvedilol (Tsuboi, Kawazoe, Izumoto, & Okabayashi, 2008). Carvedilol may be used to control the ventricular rate of AF.

Patients who developed AF during their postoperative cardiac surgery trajectory and remain in AF for more than 24 hours or have persistent incidents of AF should receive anticoagulant therapy. These individuals may be discharged home on oral anticoagulant therapy even if they convert to NSR prior to discharge. If patients have atrial enlargement, they remain at risk for development of recurrent paroxysmal AF (Brister & Lenkei-Kerwin, 2005; Khalpey et al., 2008). The patient who underwent CABG is also at increased risk for stroke development (Fuster & Ryden, 2001). In 2005, the American College of Chest Physicians released evidence-based clinical practice guidelines for the prevention and management of postoperative AF after cardiac surgery (McKeown & Gutterman, 2005). The use of atrial pacing for prevention of postoperative AF has been noted in the literature (Greenberg, Katz, Iuliano, Tempesta, & Solomon, 2000). In this study, the use of beta blockade and atrial pacing decreased length of hospital stay by 22%.

If the patient has hemodynamic instability accompanying rhythm disturbances, SCV should be performed. The clinician should follow advanced cardiac life support (ACLS) protocols for dysrhythmia management, as delineated by the American Heart Association.

If the patient develops supraventricular tachycardia (SVT) without symptoms of hemodynamic compromise following cardiac surgery, adenosine (Adenocard®) may be administered. Adenosine should not be used in patients with atrial flutter, those who underwent cardiac transplant, or those who have been partially revascularized (Khalpey et al., 2008). Adenosine is not effective in the treatment of atrial flutter, and this medication should be used with caution when the origin of the tachyarrhythmia is unclear, as it may produce ventricular fibrillation (VF) in patients with coronary artery disease and AF with a rapid ventricular response in the setting of pre-excited tachycardias. Adenosine should be avoided in patients with severe bronchial asthma (American College of Cardiology, American Heart Association, & European Society of Cardiology, 2003).

VENTRICULAR DYSRHYTHMIAS

Three dysrhythmias of ventricular origin have been reported in the postoperative cardiac surgery patient: premature ventricular contractions, VT, and VF. Premature ventricular contractions usually do not require intervention if the patient has normal LV function and electrolyte levels. Either or both of these etiologies require correction if LV or electrolyte abnormalities are present. For example, alterations in potassium or magnesium levels may be the underlying cause that would require optimization (Brister & Lenkei-Kerwin, 2005). In one study of postoperative cardiac surgery patients, administration of supplemental magnesium decreased the frequency of postoperative ventricular dysrhythmias and increased stroke volume in the early postoperative period (England, Gordon, Salem, & Chernow, 1992).

Ventricular dysrhythmias are less common than dysrhythmias of atrial origin in patients having undergone cardiac surgery and may be indicative of myocardial dysfunction. Paroxysmal VT occurs in 17% to 97% of patients; its development is believed to be related to reperfusion. Non-paroxysmal monomorphic or polymorphic VT or VF occurs in 1% to 3% of patients (Aranki, Cutlip, & Aroesty, 2008).

Predictors of postoperative VT or VF that have been identified include age younger than 65 years, female gender, body mass index less than 25 kg/m^2, unstable angina, ejection frac-

tion less than 50%, pulmonary hypertension, systemic hypertension, prolonged cardiopulmonary bypass (CPB), or need for inotropic agents, intra-aortic balloon pump (IABP), or both (Ascione, Reeves, Santo, Khan, & Angelini, 2004; Yeung-Lai-Wah et al., 2004).

Risk factors for development of monomorphic VT include a history of MI, heart failure, or decreased LV function. Development of polymorphic VT (i.e., torsade de pointes) is believed to be associated with perioperative MI and other risk factors (e.g., hemodynamic instability, increased sympathetic activity, metabolic derangements). In one study, patients who developed VT or VF had a significantly higher postoperative mortality rate as compared to patients who did not develop ventricular dysrhythmias (24.6% versus 1.5%) (Aranki et al., 2008).

Management of Postoperative Ventricular Dysrhythmias

Ventricular tachycardia associated with hemodynamic stability may be treated by ameliorating the underlying cause, including ruling out ischemia, replacing electrolytes, and avoiding inotropic therapy (Khalpey et al., 2008). In addition, a bolus of an antiarrhythmic agent (e.g., amiodarone, lidocaine [Xylocaine®], or sotalol) may be administered. Once converted, the patient may be maintained with a continuous infusion of the antiarrhythmic that was instrumental in the chemical conversion of the dysrhythmia. If VT persists despite administration of antiarrhythmic agents or if VT is associated with hemodynamic instability, concomitant SCV and antiarrhythmic administration may be indicated (Brister & Lenkei-Kerwin, 2005; Khalpey et al., 2008).

VF should be treated with immediate defibrillation per ACLS protocol, cardiopulmonary resuscitation, administration of epinephrine and antiarrhythmic agents, and eradication of the underlying cause. The amount of energy used for defibrillation depends on how the energy is delivered—monophasic or biphasic, although the latter is presently more common.

BRADYARRHYTHMIAS

Postoperatively, cardiac surgery patients may develop bradyarrhythmias such as sick sinus syndrome, Mobitz II, or complete heart block (Brister & Lenkei-Kerwin, 2005). AV block may occur in patients who have undergone surgery to repair an aortic, mitral, or tricuspid valve. Development of this complication is believed to be related to surgical injury and edema (Silvestry, 2008). Presence of a heart block may also be associated with an inferior wall MI. Etiologic factors that have been identified include medications (e.g., digoxin, amiodarone, calcium channel blockers, and beta blockers) and certain surgical approaches (Khalpey et al., 2008).

Management of Bradyarrhythmias

Management of bradyarrhythmias usually entails use of a temporary pacemaker. The epicardial wires that are oftentimes left in place following the initial cardiac surgery are used for this purpose (Zevola, Raffa, & Brown, 2002). Transcutaneous or transvenous pacing or a pulmonary artery catheter with a pacing port may also be used (Khalpey et al., 2008). Rarely do bradyarrhythmias persist. However, in the fewer than 5% of patients who do not experience resolution, permanent pacemaker implantation is required (Morris & St. Claire, 1999).

MANAGEMENT OF DYSRHYTHMIAS FOLLOWING CARDIAC TRANSPLANT

A significant difference exists in the pharmacological management of dysrhythmias following cardiac transplant and the approaches used in patients who undergo other open heart surgical procedures and develop postoperative

dysrhythmias. Digoxin is not as effective on the SA and AV node of the transplanted heart owing to the interruption in the nerve pathways from the incisions, which reduce the heart's response to the autonomic nervous system (Wade et al., 2004). Bradycardia may develop as a consequence of the incision made in the SA node during surgery. Atropine will not be effective in this instance because of the severing of the vagus nerve.

Management with a pacemaker should be the primary treatment (Wade et al., 2004). Pacemakers also give the patient the needed atrial kick and augment CO. Isoproterenol (Isuprel®), milrinone (Primacor®), and dobutamine (Dobutrex®) may be used to increase heart rate and improve the electrical stimulation of the newly implanted heart (Wade et al., 2004; White-Williams & Grady, 2008).

EPICARDIAL PACEMAKERS

During surgery for CPB or valvuloplasty, pacing electrodes are attached directly to the atria, the ventricles, or both. Wires are inserted in the event that the patient develops bradycardia, AF, or junctional rhythm, all of which require better conduction control to resolve. The wires are then secured to the epicardium and brought out through the skin. Depending on the surgeon's preference, the patient may have one or two sets of pacing wires emplaced. Generally, the ventricular and atrial wires exit the skin to the left and right of the sternum, respectively. It is essential for the ICU nurse to secure the leads to the patient's chest or abdomen, have a pacemaker generator with new batteries readily available, and ensure that all wiring and connections are tight and free of fraying. The ends of the pacer wires should be covered, insulated, and protected with a clean, dry dressing. Gloves should be worn when handling the wires, and other electrical appliances should be kept away from the ends to prevent electrical interference (Rushton & Kalpin, 2004).

SUMMARY

Nurses caring for patients who have undergone cardiac surgery must understand which patients are at risk for the development of dysrhythmias, know how to identify these dysrhythmias, be able to prevent their occurrence, and implement early treatment to correct any irregular rhythm that does arise. While it may be difficult to isolate the underlying cause of a postoperative dysrhythmia, correction of possible etiologies is an essential component of successful management. As part of the clinical patient assessment, the ICU nurse should note the duration of the dysrhythmia as well as any associated hemodynamic effects (Atlee, 1997).

CASE STUDY

A 53-year-old male is admitted to the ICU immediately following aortic valve repair. His history includes calcific aortic valve disease and dilatation of the ascending aorta due to Ehlers-Danlos syndrome. Ehlers-Danlos syndrome is a rare, inheritable, connective tissue disease in which patients usually have a history of bruising and hyperextendability of joints (history of being double jointed).

The patient underwent an aortic valve replacement with tissue (Starr) prosthesis and came off bypass without difficulty. On the third postoperative day, when he had left the intensive care unit and was making excellent progress mobilizing, he had further episodes of syncope and developed palpitations. The patient was noted to have atrial fibrillation with a rate of 124.

Critical Thinking Questions

1. Which factors might predispose this patient to new-onset atrial fibrillation?
2. Which prophylactic treatment could have been utilized to reduce the incidence of rhythm problems?
3. What is the best method to manage atrial fibrillation postoperatively in this patient?

Answers to Critical Thinking Questions

1. Aortic valve repair.
2. Statins.
3. Agents that are used to control rate include beta blockers, calcium channel blockers, and digoxin. Metoprolol is recommended as first-line therapy for most patients. Diltiazem is the calcium channel blocker of choice.

SELF-ASSESSMENT QUESTIONS

1. Treatment of premature atrial contractions is considered because this condition is associated with an increased risk of
   a. ventricular tachycardia.
   b. atrial tachyarrhythmias.
   c. bradycardia.
   d. sinus arrhythmia.
2. Which of the following postoperative cardiac surgery patients is most likely to develop atrial fibrillation?
   a. A 35-year-old male undergoing a heart transplant
   b. A 45-year-old male undergoing triple bypass surgery
   c. A 65-year-old female undergoing aneurysm repair
   d. An 88-year-male undergoing aortic valve surgery
3. Remodeling of the myocardium occurs in patients who experience long periods of
   a. volume or pressure elevation.
   b. intraoperative ischemia.
   c. postoperative arrhythmia.
   d. postoperative hypoxia.
4. In orthotopic heart transplantation, the ECG may reveal
   a. two P waves.
   b. a prolonged P-R interval.
   c. elevated T wave.
   d. an inverted QRS.
5. Which of the following patients with an arrhythmia should receive anticoagulant therapy?
   a. A patient who had six-beat runs of ventricular tachycardia
   b. A patient who has one episode of atrial fibrillation within 2 hours of surgery
   c. A patient who remained in atrial fibrillation for more than 24 hours
   d. A patient who had bradycardia preoperatively
6. If the patient develops supraventricular tachycardia following cardiac surgery, which medication should be administered?
   a. Metoprolol
   b. Epinephrine
   c. Adenosine
   d. Amiodarone
7. Premature ventricular contractions in postoperative cardiac surgery patients are most likely to occur due to alterations in levels of
   a. sodium.
   b. calcium.
   c. hemoglobin.
   d. magnesium.
8. The first line of treatment for ventricular fibrillation should be
   a. administration of epinephrine.
   b. immediate defibrillation.

c. cardiopulmonary resuscitation.
d. antiarrhythmic agents.

9. Symptomatic bradycardia may develop in the postoperative cardiac surgery patient. Which intervention should be expected?
   a. Atropine
   b. Epinephrine
   c. Pacemaker
   d. Ventilation
10. Epicardial pacing wires should be managed by ensuring that
    a. a transparent occlusive dressing is smoothly laid across the chest to secure the wires to the skin.
    b. the ends of the pacer wires are covered, insulated, and protected with a clean, dry dressing.
    c. the wires are removed immediately upon entering the ICU to prevent electrical injury.
    d. the wires are connected to a pacemaker generator at all times until their removal.

Answers to Self-Assessment Questions

1. b
2. d
3. a
4. a
5. c
6. c
7. d
8. b
9. c
10. b

Clinical Inquiry Box

Question: Which interventions can reduce the incidence of postoperative atrial fibrillation?

Reference: Patti, G., Chello, M., Candura, D., Pasceri, V., D'Ambrosio, A., & Covino, E., et al. (2006). Randomized trial of atorvastatin for reduction of postoperative atrial fibrillation in patients undergoing cardiac surgery: Results of the ARMYDA-3 (Atorvastatin for Reduction of MYocardial Dysrhythmia After cardiac surgery) study. *Circulation, 114*(14), 1455–1461.

Objective: To identify whether statin therapy as an intervention lowers the incidence of postoperative atrial fibrillation (AF) after cardiac surgery.

Method: A randomized controlled trial was conducted with 200 patients undergoing elective cardiac surgery with cardiopulmonary bypass. Subjects had no previous statin treatment or history of AF. Patients were randomized to atorvastatin 40 mg/day ($n = 101$) or placebo ($n = 99$) starting 7 days before cardiac surgery. Outcome variables measured were incidence of postoperative AF, length of stay, 30-day major adverse cardiac and cerebrovascular events, and postoperative C-reactive protein (CRP) variations.

Results: Atorvastatin significantly reduced the incidence of AF versus placebo. Length of stay was longer in the placebo versus atorvastatin arm. Peak CRP levels were lower in patients without AF, irrespective of their randomization assignment. Atorvastatin treatment conferred a 61% reduction in risk of AF, whereas high postoperative CRP levels were associated with increased risk of AF. The incidence of major adverse cardiac and cerebrovascular events at 30 days was similar in both arms of the study.

Conclusion: Treatment with atorvastatin 40 mg/day initiated 7 days before surgery significantly reduces the incidence of postoperative AF after elective cardiac surgery with cardiopulmonary bypass and shortens hospital stay. Nurses in the ICU caring for postoperative cardiac surgery patients should advocate for the use of a statin to improve patient outcomes.

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WEB RESOURCES

Anesoft rhythm recognition: www.anesoft.com/FreeStuff/rhythm/rhythm.html

A guide to reading and understanding EKG interpretation: http://students.med.nyu.edu/erclub/ekghome.html

EKG Library: www.ecglibrary.com/

ECG Learning Center: www.library.med.utah.edu/kw/ecg/

University of Wisconsin–Madison's self-study ECG manual: www.fammed.wisc.edu/medstudent/pcc/ecg/ecg.html

Chapter 16

Neurologic Complications

Myra F. Ellis

INTRODUCTION

Neurologic complications following cardiac surgery are considered severe because they affect mortality and, for survivors, quality of life. In addition, neurologic complications dramatically increase the length of hospitalization and costs associated with cardiac surgery (Eagle et al., 2004). They occur in an estimated 1% to 6% of postoperative cardiac surgery patients (Albert & Antman, 2003), with an increased incidence being noted in older patients (Silvestry, 2008). Neurologic complications are the second leading cause of morbidity and mortality in postoperative cardiac surgery patients; heart failure is the leading cause (McGarvey, Cheung, & Stecker, 2008).

Postoperative neurologic complications have been attributed to patient-specific factors, emboli, hypoperfusion, and metabolic derangements. Rates of neurologic complications are increasing, despite numerous advances in cardiac surgery (Newman et al., 2006). Estimates of cognitive impairment range from 20% to 70% during the first postoperative week, and approximately 10% to 40% of these patients continue to exhibit cognitive impairment 6 weeks after surgery (Bruce, Smith, Yelland, & Robinson, 2008).

Studies suggest that elderly patients with comorbidities and cardiovascular disease may benefit more from surgical treatment than from medical management. Technological advances of cardiopulmonary bypass (CPB) and other improvements, as well as increased life expectancy for the population as a whole, have allowed the benefits of cardiac surgery to be offered more frequently to older patients with more comorbidities (Ferguson, Hammill, Peterson, DeLong, & Grover, 2002). Unfortunately, elderly patients are at increased risk for pathophysiologic stress—including neurologic dysfunction—following cardiac surgery.

Postoperative stroke is a leading cause of death following CPB. The incidence of stroke has been reported to range from 0.4% to 14% in studies, with the variability being attributed to differences in patient populations, surgical procedure, and data collection methods. Increased rates of stroke and encephalopathy are attributed to increased numbers of high-risk patients undergoing cardiac surgery (McKhann, Grega, Borowicz, Baumgartner, & Selnes, 2006).

The incidence of negative sequelae from neurologic complications is reported to be approximately 8.4%. In one study, length of ICU stay for patients who experienced these complications increased from 3 days to 6–8 days; the length of hospital stay increased by 50% (Talmor & Lisbon, 2005). The death rate from neurologic complications in the 1970s was 7.2%, increased to 20% by the mid 1980s, and continues to rise (Newman et al., 2006).

This chapter discusses the incidence and extent of neurologic complications following cardiac surgery, offers evidence-based strategies for prevention of these undesirable sequelae, and describes nursing management of patients with adverse neurologic outcomes.

DESCRIPTION AND INCIDENCE OF NEUROLOGIC COMPLICATIONS

Neurologic complications cover a wide range of disorders, from debilitating stroke or coma to encephalopathy, delirium, and neurocognitive dysfunction. Adverse cerebral outcomes can be divided into two types: Type I and Type II. Type I deficits include stroke and major focal neurologic deficits, transient ischemic attacks (TIA), stupor, and coma. Type II deficits include new decline in intellectual function, confusion, agitation, disorientation, memory deficits, and seizure without evidence of focal deficit. The reported incidence of adverse cerebral outcomes varies widely, from 0.4% to 80%, depending on how the deficit is defined (Eagle et al., 2004; Silvestry, 2008; Talmor & Lisbon, 2005).

Other neurologic complications have also been reported following cardiac surgery, including injuries to the brachial plexus, phrenic nerve, cranial nerves, other peripheral nerves, and visual pathways. These injuries occur less frequently and are usually less serious, but nevertheless contribute to overall patient discomfort and morbidity (Grocott, Clark, Homi, & Sharma, 2004).

Type I Neurologic Deficits

Data from the largest prospective study of adverse cerebral outcomes following cardiac surgery reveal that Type I neurologic deficits occur in 3.1% of patients and are responsible for a 21% mortality rate in this population. In addition, Type I deficits contribute to the (relatively long) average stay of 11 days in the ICU and 25 days in the hospital, and are associated with a significant increase in the need for intermediate- and long-term care following hospital discharge (Roach et al., 1996). The financial cost of a perioperative stroke is estimated to be in the range of $90,000–$228,000, as a result of lost productivity and increased costs of care (Eagle et al., 2004).

Type II Neurologic Deficits

Type II neurologic deficits are more prevalent than Type I deficits, occurring in 3% of patients (Silvestry, 2008). Type II injuries also carry important implications for both long- and short-term disability and are associated with increased length of hospital stay, higher hospital costs, and an increased likelihood of discharge to rehabilitation or extended care facilities (Roach et al., 1996). A 53% incidence of abnormal neurocognitive function at discharge was reported in a study of patients undergoing coronary artery bypass grafting (CABG) with CPB. In this study, 24% of patients continued to have neurologic abnormalities at 6 months after their cardiac surgery, and 42% reported cognitive decline at 5 years following the surgery (Newman et al., 2001).

RISK FACTORS FOR NEUROLOGIC COMPLICATIONS

A number of risk factors have been implicated in adverse neurologic outcomes. These factors incorporate a combination of patient risk factors, intraoperative variables, and postoperative events. Importantly, the additive effect of variables significantly increases a patient's risk. Identification of risk factors has led to predictive models that allow stroke probability to be calculated for individual patients (McKhann et al., 2006). Risk factors are summarized in Table 16–1.

Predictors for Type I and II Deficits

Increasing age, especially greater than 70 years, is a strong predictor for both Type I and Type II

Table 16–1 Significant Risk Factors for Type I and Type II Neurologic Outcomes

	Risk Factors
Types I and II	Advanced age, especially greater than 70 years History of pulmonary disease History of hypertension Existing hypertension
Type I	Moderate to severe proximal aortic atherosclerosis History of neurologic deficit Diabetes mellitus History of unstable angina Use of left ventricular venting procedure Use of intra-aortic balloon pump
Type II	History of excessive alcohol consumption Postoperative dysrhythmias Prior cardiac surgery

Sources: Newman et al., 2006; Roach et al., 1996; Salenger, Gammie, & Vander Salm, 2003.

deficits. A history of significant hypertension has also been linked to both types of adverse neurologic outcomes following cardiac surgery (Roach et al., 1996). In addition, patients undergoing combined open chamber procedures and coronary artery surgery are at greatest risk for adverse neurologic outcomes; these complications are equally divided between Type I and Type II deficits (Newman et al., 2006).

Predictors for Type I Deficits

Moderate to severe proximal aortic atherosclerosis, as identified by intraoperative palpation, is the single greatest marker for a Type I neurologic deficit. The risk for Type I deficits increases fourfold in the presence of aortic lesions, with these kinds of complications being more prevalent in older patients (Roach et al., 1996). Atherosclerotic emboli are likely mobilized by manipulation of the aorta. Other significant risk factors that have been identified include history of neurologic abnormalities such as stroke or TIA, diabetes mellitus, history of unstable angina, and use of an intra-aortic balloon pump (IABP) (Roach et al., 1996). Perioperative hypoperfusion, nonpulsatile flow, intraoperative use of a left ventricular assist device, peripheral vascular disease (PVD), renal failure, left main coronary stenosis, emergent operation, number of aortic anastamoses, reoperations, and use of a non-membrane oxygenator have also been implicated as increasing the patient's risk of developing a Type I deficit in various studies (Salenger, Gammie, & Vander Salm, 2003).

Predictors for Type II Deficits

Factors that are associated with Type II deficits include preoperative substance abuse (e.g., alcohol consumption), dysrrhythmias, hypertension, prior CABG, PVD, metabolic conditions, and heart failure. Although a recent study linked aortic atherosclerosis to delirium (Rudolph et al., 2005), earlier studies did not show a strong correlation between the two, suggesting an etiology related to pathology of the microcirculation in the brain, rather than embolization (Hogue, Palin, & Arrowsmith, 2006). Other factors linked to Type II deficits include certain genetic factors (Mathew et al., 2007). Type II deficits are seen more commonly in patients who have experienced periods of hypoperfusion or hypotension (Grocott, Homi, & Puskas, 2005).

UNDERLYING PATHOPHYSIOLOGY

The precise mechanisms of cerebral injury following cardiac surgery are not fully understood. Numerous factors inherent to cardiac surgery play a role in adverse neurologic outcomes. These complications have been attributed primarily to the effects of CPB. Reported mechanisms are the embolization of gas and particulate matter, inadequate cerebral perfusion, and large fluctuations in hemodynamic parameters (Ganushchak, Fransen, Visser, de Jong, & Maessen, 2004).

Patients who undergo cardiac surgery experience a profound systemic inflammatory response, especially when CPB is used (Bhimji, Estabrooks, & Price, 2007). One of the effects of the systemic inflammatory response related to CPB is the development of clots. CPB activates the intrinsic and extrinsic pathways of the coagulation system secondary to factors including hypothermia, pumps propelling blood through the circuit, and exposure of blood to the artificial surfaces of the bypass circuit (Day & Taylor, 2005). These conditions may contribute to neurologic injury, although data on this point are inconclusive (Newman et al., 2006).

Cognitive decline has been linked to surgeries other than cardiac procedures, albeit at a lower rate. Notably, exposure to general anesthetic agents may contribute to cognitive decline. In addition, postoperative hyperthermia and cerebral edema have been linked to poor neurologic outcomes, although these complications may be an effect of processes that resulted in cerebral injury itself, rather than being directly responsible for the neurologic deficit (Grocott & Yoshitani, 2007).

STRATEGIES FOR NEUROPROTECTION DURING CARDIAC SURGERY

Careful preoperative screening can help identify patients who are at higher risk for developing neurologic complications. For example, a biomarker known as *N*-methyl-D-aspartate (NMDA) receptor antibody (NR2Ab) is predictive of severe neurologic adverse events after CPB. "Patients with a positive NR2Ab test (2.0 ng/mL) preoperatively were nearly 18 times more likely to experience a postoperative neurologic event than patients with a negative test" (Bokesch et al., 2006, p. 1432). A number of strategies have been suggested to minimize patients' risk of experiencing neurologic deficits following cardiac surgery.

Avoiding Injury

Many strategies to reduce complications during cardiac surgery focus on avoiding injury. As previously stated, atherosclerosis is an important predictor of stroke. The surgeon can identify high-risk patients intraoperatively with the use of epiaortic ultrasound or transesophageal echocardiogram (TEE) to modify cannulation sites and avoid atheroma (fatty deposits in arteries). Single cross-clamp technique and the use of an internal mammary artery–Y (IMA-Y) graft for proximal anastomosis to avoid aortic manipulation are also intraoperative strategies to minimize atheroembolism (Stamou, 2006). In patients with severe atherosclerosis, other options for surgery may be employed, such as off-pump coronary artery bypass grafting (OPCAB) or replacement of the ascending aorta under deep hypothermic circulatory arrest. Other strategies shown to reduce neurologic injury include emboli reduction with the use of cell saver to process shed mediastinal blood, post-pump arterial filters, and acid–base balance management with the Alpha-stat method (Hogue et al., 2006). Hyperglycemia, hypotension, and hyperthermia during rewarming have all been linked to adverse neurologic outcomes, and should be avoided (Grocott & Yoshitani, 2007).

OPCAB has been proposed as a means of decreasing the incidence of adverse neurologic outcomes following cardiac surgery. It

seems logical that avoiding CPB—the proposed etiology for much of the neurologic damage—would improve outcomes. However, conflicting results with this strategy have been reported from clinical trials, and the largest randomized controlled trial failed to show any significant reduction in stroke rate or incidence of neurocognitive dysfunction (Bucerius et al., 2003). The American College of Cardiology (ACC) does not find enough evidence to conclude that OPCAB is better for limiting neurologic deficits (Eagle et al., 2004; Barriero & Baumgartner, 2006). OPCAB is discussed in detail in Chapter 7.

Minimizing Injury

Other strategies to reduce neurologic complications focus on minimizing the extent of injury. These measures include rapid treatment of atrial fibrillation (AF) and early identification of and intervention for ischemic brain lesions. Some evidence suggests that increasing blood pressure to increase cerebral blood flow may help to minimize infarction size (McKhann et al., 2006).

DIAGNOSIS AND TREATMENT OF CENTRAL NERVOUS SYSTEM INJURY

Nurses who care for postoperative cardiac surgery patients should be able to recognize those patients who are at increased risk for central nervous system injury and differentiate between the various types of neurologic deficits. Care includes interval assessments of neurologic function and changes to the plan of care to enhance neurologic recovery.

Stroke

Stroke is a devastating complication following cardiac surgery. Its incidence ranges from 0.8% to 6% in this population (Arrowsmith, Grocett, & Newman, 2000; Ganushchak et al., 2004; McGarvey et al., 2005; McKhann et al., 2006; Stamou, 2006), with an estimated 5000–35,000 postoperative cardiac surgery patients developing a stroke annually (Albert & Antman, 2003; Stamou, 2006). The incidence appears to be increasing—a trend that is attributed to the higher acuity of surgical candidates (McKhann et al., 2006).

The majority of postoperative strokes are evident in the first 24 to 48 hours after surgery in patients who initially awaken without neurologic deficits (Henke & Eigsti, 2003; Hogue, Murphy, Schechtman, & Dávila-Román, 1999). Data from one study suggest that 42% of strokes are present on awakening from surgery and an additional 20% become evident on the first postoperative day (Likosky et al., 2003).

Most of the strokes following cardiac surgery are ischemic in nature (Henke & Eigsti, 2003). A small percentage (5%) of patients with ischemic strokes, however, experience hemorrhagic alteration or conversion. Intracranial hemorrhage with associated clinical significance is rare following cardiac surgery (McGarvey et al., 2006). Strokes that occur later in the postoperative period are more often associated with dysrhythmias, especially AF, valve surgery, or use of a ventricular assist device (McKhann et al., 2006).

An estimated 30% of patients who develop a stroke following cardiac surgery are believed to have had carotid artery disease present. Intraoperative cerebral microembolization during CABG has been suggested to be the most common etiology (Albert & Antman, 2003). In one study, 10 predictors that were present preoperatively were identified as increasing the risk of stroke: female gender, age, aortic surgery, previous stroke, critical preoperative state, poor ventricular function, diabetes, peripheral vascular disease, unstable angina, and pulmonary hypertension. Other risk factors that have been identified include redo surgery, valve surgery, calcified aorta, duration of CPB, renal failure, low cardiac output syndrome (LCOS), hypertension,

postoperative AF, recent myocardial infarction (MI), left ventricular dysfunction, and smoking (John et al., 2000; McGarvey et al., 2008; McKhann et al., 2006; Ricotta, Faggioli, Castilone, Hassett, & Brener, 1995). Patients who develop postoperative AF have a twofold to fivefold greater risk of having a stroke (Mullen-Fortino & O'Brien, 2008). Patients with LCOS are also at greater risk. The increased risk of stroke is believed to be related to blood pressure variability, which in turn increases the risk of thrombus formation and cerebral hypoperfusion (Hogue et al., 1999). As previously suggested, postoperative cardiac surgery patients who develop a stroke have higher mortality rates and longer hospital stays (Anyanwu, Filsoufi, Salzber, Bronster, & Adams, 2007).

Suspicion for stroke occurs when the patient fails to awaken, move extremities, follow commands after discontinuation of sedation, or any combination of these in the first 6 postoperative hours (McKhann et al., 2006). The onset of focal findings such as facial droop, hemiparesis, aphasia, visual disturbances, or pupil change may also indicate stroke. Assessment is often difficult in the immediate postoperative period, however, and is confounded by the patient's emergence from anesthesia and the effects of postoperative medications.

Diagnosis of stroke is made based on the presence of focal deficits in the physical exam and brain imaging results (Adams et al., 2007). Clinical signs of catastrophic stroke may include fixed and dilated pupils, posturing, Cushing's syndrome (hypertension with bradycardia), and persistent coma (Young, Bratina, Hickenbottom, Demchuk, & Wein, 1998). Patients suspected of having a stroke should be evaluated by a neurologist and undergo brain imaging.

Brain magnetic resonance imaging (MRI)—specifically, diffusion-weighted imaging—is the most accurate neuroimaging technique available; it is able to detect microemboli-related events (McKhann et al., 2006). Unfortunately, MRI is often impractical in postoperative cardiac surgery patients due to the presence of metallic implants such as valves, defibrillators, pacemakers, or surgical metal (e.g., epicardial pacing wires). An alternative imaging modality, head computed tomography (CT), can be used in such cases (McKhann et al., 2006).

Neurologic assessments of cardiac surgery patients should be conducted at regular intervals in the postoperative period. This review includes assessment of the patient's level of consciousness (LOC) and motor movement. As mentioned earlier, medications given during the intraoperative period can make accurate assessment more difficult. If deficits are suspected, a full neurologic assessment should be performed. The National Institutes of Health Stroke Scale (NIHSS) outlines a complete evaluation for stroke. The NIHSS assessment includes LOC, best gaze, visual fields, facial palsy, motor function of arms and legs, limb ataxia, sensory, best language, dysarthria, extinction, and inattention (NIH, 2003).

Treatment of Stroke

The treatment of stroke is primarily supportive in the immediate phase and aims to prevent secondary complications (Young et al., 1998). Monitoring the patient's neurologic, hematologic, and respiratory status is essential. Nursing care is guided by the specific neurologic deficits observed in the patient. More generally, the ACC and other groups have published evidence-based guidelines for the management of stroke patients (Adams et al., 2007).

OXYGENATION SUPPORT

Supporting adequate tissue oxygenation is important in patients who are experiencing cerebral ischemia. Patients with acute stroke may have abnormal breathing patterns and may need increased support to prevent

hypoxia, which can worsen brain injury. Hypoxia may be caused by airway obstruction, aspiration, pneumonia, or atelectasis. Endotracheal intubation may be necessary if concerns arise about airway protection or the patient's ability to maintain adequate oxygenation. Pneumonia is among the leading complications of stroke (Adams et al., 2007), so optimal nursing management includes measures to prevent ventilator-associated pneumonia. Oxygen saturation levels of at least 92% should be maintained (McGarvey et al., 2008).

TEMPERATURE MANAGEMENT

Increased body temperature is associated with increased morbidity and mortality in acute stroke patients (Adams et al., 2007). It is important to assess these patients for sources of hyperthermia, which may be related to brain injury or secondary infections. In such cases, patients may benefit from measures that lower their body temperature. Interventions to accomplish this goal may include the administration of antipyretics and application of cooling devices (McGarvey et al., 2008).

Hypothermia protects against cerebral ischemia. Data suggest that a 1 °C decrease in brain temperature is associated with a 7% decrease in cerebral metabolic rate. Conversely, even a mild increase in brain temperature (1–2 °C) can be harmful (Nussmeier, 2005). Although the neuroprotective benefits of hypothermia are well known, currently no evidence exists to support induced hypothermia as an intervention in the setting of acute stroke (Adams et al., 2007; Rees, Beranek-Stanley, Burke, & Ebrahim, 2007).

HEMODYNAMIC STATUS

Blood pressure (BP) control is crucial in stroke patients. Increased BP is common in the acute phase of stroke, with systolic BP (SBP) greater than 160 mm Hg being noted in 60% of these patients; elevation of SBP to more than 180 mm Hg is associated with adverse outcomes (Adams et al., 2007). Blood pressure should be maintained within a range that is adequate to maintain cerebral perfusion, yet does not exacerbate neurologic damage. Deleterious effects of hypertension may include vasogenic edema (permeation of intravascular fluid and proteins into the cerebral extracellular space), disruption of the blood–brain barrier, or an increase in myocardial oxygen consumption (Young et al., 1998).

Aggressive treatment of hypertension may disrupt autoregulation. Care should be taken to gradually decrease BP to a specified target, as the other extreme—hypotension—worsens neurologic outcome. In the majority of stroke patients, BP declines several hours after the onset of stroke symptoms without any medical intervention (Adams et al., 2007). Conversely, some evidence suggests that increased BP following embolic stroke can improve cerebral blood flow and limit ischemic effects (McKhann et al., 2006).

Periods of hypotension should be similarly avoided so as to maintain adequate cerebral perfusion. If necessary, this goal can be accomplished by administering fluid boluses. A vasopressor may be added if fluid therapy is not sufficient (McGarvey et al., 2008).

GLYCEMIC CONTROL

Hypoglycemia may mimic symptoms of stroke and may exacerbate brain injury. Initial assessment of the patient upon presentation of stroke symptoms should include measurement of serum glucose and correction of hypoglycemia.

Conversely, hyperglycemia is associated with poorer outcomes following stroke and should be avoided (McGarvey et al., 2008). Researchers hypothesize that hyperglycemia increases the infarct size associated with a stroke and elevates cerebral lactate levels, which results in acidosis of brain tissue and decreases the function of the mitochondria of the penumbra (an ischemic area that

is still viable, located adjacent to the area affected by the stroke). Hyperglycemia is also reported to disrupt the blood–brain barrier, which puts the patient at risk of developing cerebral edema; this complication, in turn, promotes brain cell death in the stroke-affected area. All of these factors influence morbidity and mortality following a stroke (Paolino & Garner, 2005).

The goal of glycemic control therapy should be to maintain serum glucose in the range of 80 to 140 mg/dL (Adams et al., 2007). Others suggest that serum glucose levels should not exceed 110 mg/dL (Capes, Hunt, Malmberg, Pathak, & Gerstein, 2001). Careful titration and control of serum glucose levels are key nursing measures, and are usually best accomplished with an insulin infusion.

ASPIRIN

Although studies have failed to confirm the efficacy of anticoagulation following CABG, and fibrinolysis is contraindicated in patients who have undergone this surgical procedure, administration of aspirin may decrease neurologic complications following CABG and improve outcomes following stroke. If not contraindicated, aspirin is recommended in postoperative cardiac surgery patients (McGarvey et al., 2008).

Prevention of Secondary Complications

Stroke following cardiac surgery increases both ICU and overall hospital length of stay. Patients who experience this neurologic complication are at increased risk for secondary complications and have a threefold to sixfold increased risk of death. Mortality rates in the range of 14% to 30% have been reported in hospitalized patients who follow this course (Bucerius et al., 2003; McGarvey et al., 2008). Anticipation of complications allows the ICU nurse to develop a plan of care to reduce the risk of stroke and its sequelae. Common secondary complications include aspiration, pneumonia, venous thrombotic events, urinary tract infections, and skin breakdown (Young et al., 1998).

Encephalopathy and Delirium

Postoperative cardiac surgery patients are at greater risk for not only a stroke, but also encephalopathy. "Encephalopathy" is a generic term that refers to several types of brain dysfunction. This term has been used synonymously with confusion, delirium, lethargy, depression, disorientation, hallucinations, transient ischemic attacks, mental status changes, combativeness, and agitation. The incidence of this complication has been reported to range from 8.4% to 32%. Patients who develop postoperative encephalopathy have worse outcomes, increased length of hospital stay (8 days versus 14 days in one study), and higher mortality rates—as much as threefold higher—than patients who do not develop this neurologic complication (Henke & Eigsti, 2003; McKhann et al., 2006). Postoperative encephalopathy may be related to the development of microemboli, cerebral edema secondary to the inflammatory response, inadequate temperature regulation, or cerebral hypoperfusion (Henke & Eigsti, 2003).

Risk factors that have been identified for the development of postoperative encephalopathy include age and history of stroke, hypertension, diabetes, and carotid bruit. Signs and symptoms typically manifest after extubation has taken place (McKhann et al., 2006).

Delirium is a "disturbance of consciousness with inattention that is accompanied by changes in cognition or perceptual disturbance and has an acute onset with a fluctuating course" (Chang, Tsai, Lin, Chen, & Liu, 2008, p. 568). It is the most common neurologic complication following cardiac surgery and may be the most difficult to manage (Sockalingam et al., 2005). This condition develops over the course of hours to days and

may be life-threatening. An estimated 3% to 73% of postoperative cardiac surgery patients develop delirium (Chang et al., 2008). The incidence is higher in those patients with preexisting psychological disorders, substantial prior alcohol use, left ventricular ejection fraction less than 30%, advanced age (greater than 65 years), or cerebral artery disease, and in those individuals who undergo an emergent procedure (Chang et al., 2008). Anesthesia, sedation, time in circulatory arrest greater than 30 minutes, and intraoperative hypothermia (less than 25 °C) are noted intraoperative causes. Etiologies of delirium in postoperative patients include sleep deprivation, renal or hepatic failure, cardiogenic shock, AF, massive blood transfusions (more than 1 L), bilirubin greater than 2 mg/dL, hypoalbuminemia, low hematocrit, acute infection, dehydration, and thyroid disorders (Chang et al., 2008; Khalpey, Ganim, & Rawn, 2008).

Symptoms of delirium include an inability to maintain attention, disturbance of consciousness, cognitive deficits, memory impairment, disorientation, inappropriate speech, and perceptual changes (Chang et al., 2008). Focal neurologic findings are not present in delirium. These changes are acute and not associated with preexisting psychiatric disorders. Early detection and treatment of delirium are important aspects of the nursing care of postoperative cardiac surgery patients and may limit this complication's severity or prevent it altogether.

Agitation often accompanies postoperative delirium. It is defined as extreme motor or vocal behavior that is disruptive, is unsafe for the patient and staff, or interferes with the delivery of patient care and medical therapies. Examples of agitation in hospitalized patients may include screaming, shouting, moaning, combativeness (e.g., biting, kicking, hitting, scratching), pulling out tubes and disconnecting monitoring devices, and getting out of bed. Delirium and agitation often present together and may be difficult to distinguish.

In one study, the signs and symptoms of delirium did not appear until the second or third postoperative day in the ICU. More than 70% of patients who developed delirium had their symptoms diminish within 24 hours of transfer from the ICU (Chang et al., 2008).

Treatment of Delirium

There are four main aspects of managing delirium: (1) identify and treat underlying causes; (2) provide environmental and support measures; (3) administer drug therapy aimed at treating symptoms; and (4) conduct regular evaluations of the effectiveness of treatment (Sockalingam et al., 2005).

IDENTIFICATION AND TREATMENT OF THE CAUSE

Delirium after cardiac surgery is common and multifactorial. As a consequence, it is important for ICU nurses to recognize patients who are at risk of developing this neurologic complication and adjust their care to reduce or prevent postoperative delirium. Recommended measures include creating an environment that promotes sleep so the patient's sleep cycle can be reestablished and avoiding the use of medications that may promote the development of delirium (e.g., benzodiazepines) (Khalpey et al., 2008).

In patients experiencing postoperative delirium, a thorough examination must be made to identify and allow for correction of possible causes. The underlying causes of delirium may be metabolic derangements including electrolyte disorders, drug or alcohol withdrawal, nutritional deficiencies, or medications. Several drugs frequently prescribed for cardiac patients are associated with neuropsychiatric changes, including beta blockers, calcium channel blockers, angiotensin-converting enzyme (ACE) inhibitors, diuretics, antiarrhythmics, and lipid-lowering agents (Sockalingam et al., 2005). Careful review of medications with emphasis on new medications is warranted

whenever delirium presents. Attempts should be made to avoid use of drugs that impair reality, such as benzodiazepines or barbiturates, in postoperative patients.

Analgesics are often associated with mental status changes. Undertreatment of pain, however, may contribute to increased stress and sleep deprivation, thereby exacerbating postoperative delirium. The ICU nurse should closely monitor patients to avoid adverse effects from analgesics. Special care should be given to administering narcotics in the elderly population, as glomerular filtration rate decreases with age (Demeure & Fain, 2006).

ENVIRONMENTAL AND SUPPORTIVE MEASURES

Environmental interventions include minimizing or eliminating factors that exacerbate delirium. In the ICU, patients are frequently exposed to interruptions in sleep patterns, noise, and excessive environmental stimulation (Vena, 2007). Transfer to a progressive care unit should be made as soon as medically feasible and often results in abatement of delirium-related symptoms. In addition, care should be taken to reduce sensory impairment and to return patients' glasses or hearing aids as soon as practical. In all cases, nurses should focus on providing patients with reorientation and reassurance.

In extreme cases of agitation, restraints may be needed to ensure the patient's safety. Attendance at the bedside by family members or sitters is preferable to the use of physical restraints. Sitters may participate in orienting activities by talking to the patient, engaging in frequent touch, and making eye contact.

DRUG THERAPY

Pharmacologic interventions are often necessary in patients with postoperative delirium, especially when agitation is present. Despite the prevalence of postoperative delirium, only a limited number of agents to treat this complication have been studied in postoperative patients.

The drugs most commonly used to treat delirium are antipsychotics. Haloperidol (Haldol®) is a first-generation, high-potency neuroleptic and is considered to be the drug of choice in the treatment of delirium in the ICU. The American Psychiatric Association (APA) recommends starting treatment with 1–2 mg every 2–4 hours as needed, with titration to higher doses in patients who continue to demonstrate agitation. In rare circumstances, a continuous infusion of haloperidol may be necessary (APA, 1999). Haloperidol has been linked to cardiac dysrhythmias, including torsade de pointes (Perrault, Denault, Carrier, Cartier, & Belisle, 2000).

Patients receiving antipsychotic therapy should have ECG monitoring, including measurement of the QT interval (see Box 16–1). A QT interval of greater than 450 milliseconds or more than 25% over baseline warrants close monitoring and possibly a reduction or discontinuation of haloperidol, although QT prolongation may not always precede dysrhythmias (Sockalingam et al., 2005).

Treatment of delirium with benzodiazepines is reserved for patients who are experiencing withdrawal from alcohol or sedative-hypnotics. Patients who are unable to tolerate high doses of antipsychotic medications may benefit from combined benzodiazepine therapy (APA, 1999). Multivitamin replacement should be implemented for patients with a deficiency in vitamin B. In extreme cases of delirium and agitation with hypercatabolic conditions, measures such as paralysis, sedation, intubation, and mechanical ventilation may be required (APA, 1999).

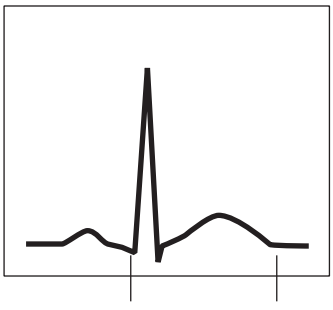
Newer antipsychotic agents such as risperidone (Risperdal®), olanzepine (Zyprexa®), and quetiapine (Seroquel®) may have fewer cardiac and extrapyramidal complications. To date, few studies have evaluated their use in cardiac surgery patients (Sockalingam et al., 2005).

Seizures

Seizures are a rare neurologic complication following cardiac surgery, occurring in 0.5% to 3.5% of patients following CABG (McGarvey et al., 2008). They most often accompany

Box 16–1 Measuring QT Intervals

$$\frac{QT}{\sqrt{RR}}$$ (the number of seconds between R waves)



Each small box in ECG paper equals 0.04 seconds.
Each large box equals 0.20 seconds.

cerebral insult from hypoxia or emboli (air or particulate). Seizures may also be caused by hypoxemia, hyponatremia, hypoglycemia, stroke, or medication overdoses, especially overdoses of lidocaine (Xylocaine®) or procainamide (Pronestyl®) (McGarvey et al., 2008). A thorough examination for contributing factors, evaluation by a neurologist, CT scan, and electroencephalogram (EEG) should be performed, and administration of anticonvulsant therapy should be considered (Young et al., 1998). An EEG is recommended for patients who are unresponsive 18 to 24 hours after surgery to determine if seizure activity is occurring without motor manifestations (McGarvey et al., 2008).

Cognitive Decline

Cognitive decline is commonplace following cardiac surgery, with its reported incidence ranging from 45% to 80% (Bernet, Grapow, & Zerkowski, 2004; Carrascal et al., 2005; Newman et al., 2001; Talmor & Lisbon, 2005). Typical cognitive disturbances include mild difficulty with memory, problem solving, attention, and ability to learn. Most patients report improvement of symptoms in 1 or 2 months.

Several studies evaluating postdischarge recovery in cardiac surgery patients show that patients need more information about what the effects of surgery are and what to expect during recovery (Jaarsma, Kastermans, Dassen, & Philipsen, 1995; Theobald & McMurray, 2004). To ensure that such education is provided, the ICU nurse should prepare patients and families for the possibility that the patient may experience some cognitive decline and reassure them that most patients experience improvement in these symptoms if they do occur. In addition, both patients and families should be taught about subtle changes and symptoms for which to observe.

Anxiety and Depression

Early studies of postoperative cardiac surgery patients suggest that the presence of anxiety and depression may have a negative effect on outcomes. Aside from their physiologic impact in the immediate postoperative period, these conditions are reported to have negative effects on long-term quality of life. Both the immediate and long-term effects may increase the risk of death following cardiac surgery (Pignay-Demaria, Lespérance, Demaria, Frasure-Smith, & Perrault, 2003). Data suggest a correlation between the presence of symptoms of depression and the chance of hospital readmission for cardiac

issues within 6 months of discharge following CABG (Saur et al., 2001). Other data indicate that unstable angina, MI, redo CABG, angioplasty, or death may result secondary to depressive symptoms (Baker, Andrew, Schrader, & Knight, 2001; Perski et al., 1998; Scheier et al., 1999). It has been further suggested that depressive symptoms have as significant an effect on cardiac surgery outcomes as having a low ejection fraction (less than 35%) or being of female gender (Connerney, Shapiro, McLaughlin, Bagiella, & Sloan, 2001).

Anxiety has variable effects on postoperative cardiac surgery outcomes. Differences that have been reported are typically attributed to anxiety type—that is, state or trait. In one small study, for example, patients with preoperative anxiety had higher associated morbidity and mortality rates, whereas patients with trait anxiety did not experience postoperative adverse events related to this disorder (Stengrevics, Sirois, Schwartz, Friedman, & Domar, 1996).

Depression occurs commonly after cardiac surgery and may last for 2 to 3 months. Although it may be severe in rare cases, depression is usually mild, disappears spontaneously, and is treated short term. Patients who reported a higher level of satisfaction with discharge teaching were less likely to experience postoperative depression (Davies, 2000).

In another study of patients admitted for CABG surgery, 92% had mild preoperative anxiety and 8% had major anxiety. Each of these patients was readmitted to the hospital within 6 months of discharge. In that same study, 72% of CABG surgery patients had depression preoperatively and were readmitted to the hospital. Depressive symptoms worsened in some of these patients, but most patients with preoperative depression experienced a resolution or reduction of their symptoms after CABG (Murphy et al., 2008).

Anxiety and depression occur more commonly in patients with a history of psychiatric disorders. Initial data suggest that psychological intervention with an antidepressant, psychotherapy, psychosocial management, or any combination of these measures should be employed, with the selection of a specific therapy being based on the patient's clinical manifestations. Implementation of any of these measures may result in decreased hospital length of stay, analgesic use, and postoperative morbidity. Administration of benzodiazepines should be done prudently, owing to the increased risk of delirium associated with use of these medications. Tricyclic antidepressants and monoamine oxidase inhibitors are contraindicated in patients with coronary artery disease because of their cardiovascular side-effect profiles. Caution should also be taken when administering serotonin reuptake inhibitors, as some of these agents may interact with cardiac medications (Pignay-Demaria et al., 2003). The ICU nurse should collaborate with members of the multidisciplinary team to help assure early recognition and prompt management of anxiety and depression in postoperative cardiac surgery patients.

OTHER NEUROLOGIC INJURIES

Although central neurologic complications receive more attention and have a greater impact on patient recovery, several other neurologic complications associated with cardiac surgery bear mentioning. These include, but are not limited to, injuries to the brachial plexus, phrenic nerve, and recurrent laryngeal nerve. The incidence of these injuries varies, and these conditions may be underrepresented in studies that examine postoperative complications (Grocott et al., 2004).

Brachial Plexus Injury

The brachial plexus includes divisions of the fifth and eighth cranial nerves and the first thoracic nerve; it forms the peripheral nerves that innervate structures of the upper extrem-

ities. The brachial plexus passes over the first rib and under the clavicle, with cords that pass downward into the axilla (Gray, 2000). The location and structure of the brachial plexus make it susceptible to injury by direct puncture, stretch, fractures, or displacement of the first rib.

Several prospective studies on cardiac surgery patients reported a 2% to 15% incidence of injuries to the brachial plexus and identified risk factors for this type of complication. Pertinent etiologic factors include sternal retraction, first rib fractures, use of IMA retractors, IMA dissection, positioning during surgery, central venous catheter placement, and advanced patient age (Grocott et al., 2004; McGarvey et al., 2008).

Sensory and motor symptoms associated with this type of injury will vary, depending on the site of the nerve damage. Brachial plexus injury often presents as paresthesia of the fourth and fifth digits on the affected side and discoordination of an upper extremity, but may also cause pain and weakness. The presence of pain is consistent with a peripheral injury; in contrast, the presence of confusion, cranial nerve involvement, or hemiparesis is typically consistent with a central injury (McGarvey et al., 2008). Patients often report symptoms several days postoperatively. These subtle injuries may be overlooked owing to the emphasis placed on more serious issues in the immediate postoperative period. Any patient complaints suggestive of brachial plexus damage should be reported to the surgeon, and a full assessment of motor and sensory function should be performed for the muscles affected by the brachial plexus.

The symptoms of brachial plexus injuries may persist for several months, but generally resolve without treatment (Grocott et al., 2004). In rare cases, prolonged recovery with residual symptoms has been reported. It is important to reassure patients that brachial plexus injury is generally transient—most patients are symptom-free at discharge. In any patient who experiences this type of injury, collaboration with a physical therapist is indicated to augment the patient's strength and flexibility (McGarvey et al., 2008).

Phrenic Nerve Neuropathy

Damage to one or both phrenic nerves may occur during cardiac surgery. This complication, which is usually related to the application of topical hypothermia or surgical trauma during left IMA dissection (Grocott et al., 2004), occurs in 1% to 30% of patients (McGarvey et al., 2008). The phrenic nerve traverses the thoracic cavity to provide deenervation to the diaphragm. The left phrenic nerve runs between the lung and mediastinal aspect of the pleura along the pericardium. The right phrenic nerve is deeper in the thoracic cavity, running lateral to the right subclavian vein (Gray, 2000).

Diagnosis of phrenic nerve damage may be made by chest radiograph, fluoroscopy, spirometry, ultrasound, or nerve conduction studies. The reported incidence of phrenic nerve dysfunction ranges from 26% to 70%, depending on the method used for diagnosis (Grocott et al., 2004). Postoperative atelectasis makes diagnosis more difficult.

Paralysis of the diaphragm results in immobility or paradoxical movement of the affected side. Unilateral phrenic nerve palsy is usually associated with minimal symptoms because of the recruitment of accessory muscles. The most common complaints include nocturnal orthopnea or dypsnea on exertion.

Phrenic nerve neuropathies generally resolve in 3 months to 1 year following cardiac surgery, but may take 2 years or longer to subside completely (McGarvey et al., 2008). In patients with underlying lung disease (e.g., COPD), the consequences may be more serious. Deterioration in lung function and extended hospital stays have been reported in these individuals (Grocott et al., 2004).

Bilateral phrenic nerve paralysis is a rare and serious complication of cardiac surgery

that carries a significant associated mortality. The first indication may be difficulty in weaning the patient with normal lung function from mechanical ventilation. Bilateral phrenic neuropathy has a much longer recovery time. These patients may compensate with accessory muscle use during the day, but experience respiratory insufficiency at night. Prolonged ventilatory support may be necessary (Grocott et al., 2004; McGarvey et al., 2008).

Recurrent Laryngeal Nerve Neuropathy

The left recurrent laryngeal nerve lies in close proximity to the parietal pleura as it encircles the aortic arch (Gray, 2000). Vocal cord paralysis as a result of injury to this nerve is less common than injury to the brachial plexus or phrenic nerve, with a reported incidence in the range of 1% to 2% (Hamdan, Moukarbel, Farhat, & Obeid, 2002).

The left recurrent laryngeal nerve may be injured during cardiac surgery if the pleura is opened and large amounts of ice slush are placed in the pleural cavity. Other sources of injury to the left recurrent laryngeal nerve include tracheal intubation, central line placement, surgical dissection, and trauma from the TEE probe (Grocott et al., 2004).

For the nurse caring for postoperative cardiac surgery patients, it is important to observe patients with a weak or ineffective cough, respiratory insufficiency, or hoarseness following extubation, as these may be indications of recurrent laryngeal nerve neuropathy and not laryngeal edema. Dysphagia, change in voice quality, inefficient cough, and throat clearing are often associated with vocal cord paralysis. Patients in whom this complication is suspected should remain NPO until further evaluation is performed, as these patients are at risk for aspiration and pneumonia (Hamdan et al., 2002).

Patients with unilateral vocal cord paralysis may demonstrate respiratory insufficiency, stridor, and signs of airway obstruction. In postoperative cardiac surgery patients, these symptoms are often attributed—erroneously—to cardiac or respiratory dysfunction. It is essential that the ICU nurse identify these symptoms both correctly and promptly to avoid patient decompensation and reintubation (Hamdan et al., 2002).

A definitive diagnosis is made by performing laryngoscopy in a spontaneously breathing patient. Recovery following unilateral vocal cord paralysis usually takes 8 to 12 months. Most patients recover with conservative treatment, but occasionally patients may require reintubation, tracheostomy, vocal cord medialization (an implant to provide bulk to the vocal cord), or any combination of these measures (Grocott et al., 2004).

Laryngeal Nerve Injury

IMA harvesting may cause injury to the anterior intercostal nerves. Symptoms may include numbness, tenderness, pain with light touch, or persistent burning pain over the sternum or left anterolateral aspect of the chest wall. Although symptoms typically resolve within 4 months, in some patients symptoms may last as long as 28 months (McGarvey et al., 2008).

Other Peripheral Neuropathies

Other, less common peripheral neuropathies have also been reported. Horner's syndrome, (characterized by miosis, ptosis, and anhydrosis [inability to sweat]) is thought to result from damage to the cervical sympathetic chain. Such damage occurs from a first rib fracture. Injury to the saphenous nerve during saphenous vein harvest may result in neuralgia that presents as anesthesia, hyperesthesia, and pain along the medial side of the operative leg and foot. Endoscopic vein harvesting has reduced the incidence of this injury (Grocott et al., 2004).

SUMMARY

Despite advances in cardiac surgery, neurologic complication rates are increasing. ICU nurses caring for postoperative cardiac surgery patients should be able to recognize which patients are at increased risk for these complications and plan their care so as to prevent these complications from occurring, minimize the associated detrimental effects, and help ensure effective symptom management. Preventive strategies may include maintaining adequate blood pressure, avoiding development of shock, preventing infection, and administering albumin (Chang et al., 2008). Although management of neurologic complications is primarily supportive in nature, early recognition and prompt intervention may minimize complications (Silvestry, 2008).

CASE STUDY

Mrs. S. is a 78-year-old patient who was admitted for elective three-vessel CABG and mitral valve replacement for mitral regurgitation. Her medical history was significant for intermittent atrial fibrillation, diabetes, hypertension, and transient ischemic attacks. Her intraoperative course was uneventful, and Mrs. S. was extubated and weaned off inotropic therapy on the day of surgery. She was transferred to the progressive care unit on postoperative day 1. Her medications included metoprolol 25 mg PO q12h (hold for systolic BP < 90 or HR < 50), furosemide 40 mg PO q12h, and atorvastatin 20 mg qhs.

On postoperative day 2, Mrs. S. reported feeling "funny." The nurse detected several subtle neurologic changes from the morning assessment. She was less responsive, had some difficulty with speech, and demonstrated a slight right facial droop. Her BP and serum glucose were checked to rule out hypotension or hypoglycemia as causes; both were found to be within the patient's normal limits. The nurse alerted the house officer.

The nurse initiated a "stroke code." The members of the team quickly responded to offer rapid assessment and recommendations for care. Mrs. S.'s neurologic status was assessed utilizing the NIHSS. She was alert and oriented, and had normal pupil function, normal motor and sensory function, a slight right facial droop with normal sensation, and dysarthria. She was transferred to the ICU for further evaluation and treatment.

Mrs. S. was hemodynamically stable and experiencing no respiratory difficulty. She was transported to the radiology department for a noncontrast CT scan. The CT scan showed no evidence of hemorrhage. Her recent surgery made her ineligible for fibrinolytic therapy.

Mrs. S. returned to the ICU, and a dopamine infusion was started to increase her blood pressure and cerebral perfusion. The patient had a BP of 110/70 mm Hg, which was lower than her admission BP of 170/80 mm Hg. A goal BP of 160 mm Hg was achieved with a dopamine infusion rate of 5 mcg/kg/min. Mrs. S.'s neurologic changes resolved after initiation of the dopamine. An NPO restriction was maintained until the patient's ability to swallow could be assessed, and a small-bore feeding tube was inserted for enteral feedings. Mrs. S.'s serum glucose was 210 mg/dL; an insulin infusion was started and the cardiac surgery insulin protocol initiated.

On postoperative day 5, Mrs. S. was weaned off the dopamine infusion without any changes in neurologic function. She was subsequently transferred to the progressive care unit. Her clinical status continued to improve, and she was discharged home on postoperative day 8.

Critical Thinking Questions

1. Why was a dopamine infusion initiated in this patient when her blood pressure was recorded at 110/70 mm Hg?
2. Which risk factors did Mrs. S. have that put her at risk for a postoperative stroke?
3. Which factors contributed to Mrs. S.'s outcome?
4. Why was an insulin infusion an important aspect in Mrs. S.'s plan of care?

Answers to Critical Thinking Questions

1. In addition to avoiding hypertension in patients who experience a stroke, periods of hypotension should be avoided to maintain cerebral perfusion.
2. Mrs. S. underwent a CABG procedure. Intraoperative cerebral microembolization during CABG is believed to be the most common etiology for postoperative stroke. Other factors that put this patient at higher risk include her female gender, age greater than 65 years, and history of atrial fibrillation, and diabetes.
3. Mrs. S. recognized a change in her clinical status and alerted the nurse, who immediately summoned the stroke team. A stroke team is a multidisciplinary group with specialized training and experience in stroke care. The physicians on a stroke team have typically received additional preparation to care for patients with a stroke. Patients who are managed by a stroke team often experience better outcomes.
4. Hypoglycemia may mimic symptoms of stroke and may exacerbate brain injury. Initial assessment at the presentation of stroke symptoms should include measurement of serum glucose and correction of hypoglycemia. In addition, hyperglycemia is associated with poorer outcomes following stroke and should be avoided (McGarvey et al., 2008). It has been suggested that hyperglycemia increases the infarct size associated with a stroke and elevates cerebral lactate levels, which results in acidosis of brain tissue and decreases the function of the mitochondria of the penumbra (an ischemic area that is still viable, located adjacent to the area affected by the stroke). Hyperglycemia is also reported to disrupt the blood–brain barrier, which puts the patient at greater risk of developing cerebral edema; this complication, in turn, promotes brain cell death in the stroke-affected area. All of these factors affect morbidity and mortality following a stroke.

SELF-ASSESSMENT QUESTIONS

1. Which of the following is *not* a risk factor for neurologic complications following cardiac surgery?
   a. Increased age
   b. Hypertension
   c. Previous stroke
   d. Previous myocardial infarction
2. The rate of stroke is increasing in cardiac surgery patients. This trend most likely reflects the
   a. increased age of patients.
   b. increased number of high-risk patients.
   c. increased number of transfusions during surgery.
   d. both a and b.
3. Haloperidol is the drug of choice for management of postoperative delirium not related to substance abuse. The nurse should monitor the ECG to detect which side effect?
   a. QTc prolongation
   b. First-degree AV block
   c. Ventricular dysrhythmias
   d. Ischemic changes

4. Neurologic complications in cardiac surgery patients result in
   a. increased cost of hospitalization.
   b. increased length of stay.
   c. increased mortality.
   d. all of the above.
5. An example of a Type II neurologic deficit is
   a. delirium.
   b. agitation.
   c. transient ischemic attack.
   d. cognitive decline.
6. Which of the following peripheral nerve deficits is *not* associated with cardiac surgery?
   a. Brachial plexus injury
   b. Left recurrent laryngeal neuropathy
   c. Phrenic nerve dysfunction
   d. Sciatic nerve injury
7. Atherosclerotic aorta, which increases risk for Type I neurologic deficits, may be detected intraoperatively by
   a. epiaortic scanning.
   b. TEE.
   c. palpation of the aorta.
   d. all of the above.
8. Management of postoperative stroke includes
   a. systemic hypothermia.
   b. glycemic control.
   c. maintaining SBP > 180 mm Hg.
   d. thrombolytic therapy.
9. Patients report a lower incidence of postoperative depression with which of the following interventions?
   a. Antidepressant therapy with serotonin inhibitors
   b. Discharge teaching that includes realistic expectations of recovery
   c. Discharge to a long-term facility
   d. Preoperative administration of haloperidol
10. Which of the following is *not* a risk factor for both Type I and Type II neurologic deficits following cardiac surgery?
    a. Combined open chamber procedures and CABG
    b. Age
    c. Diabetes mellitus
    d. Hypertension

Answers to Self-Assessment Questions

1. d
2. d
3. a
4. d
5. b
6. d
7. d
8. b
9. b
10. c

Clinical Inquiry Box

Question: Does the timing of a postoperative stroke after a CABG determine the patient's rehabilitation needs?

Reference: Lisle, T. C., Barrett, K. M., Gazoni, L. M., Swenson, B. R., Scott, C. D., Kazemi, A., et al. (2008). Timing of stroke after cardiopulmonary bypass determines mortality. *Annals of Thoracic Surgery, 85*(5), 1556–1563.

Objective: The purpose of this study was to evaluate the mortality and rehabilitation needs of individuals having a stroke subsequent to CABG.

Methods: Consecutive cardiac surgery charts were reviewed. Among the 7201 patients, 202 patients had developed stroke postoperatively. Stroke was classified as early or late. An early stroke occurred within 24 hours after the surgery; a late stroke occurred more than 24 hours after the surgery. Data were collected on patient characteristics, intraoperative variables and outcomes, postoperative course, stroke severity, and discharge status. The relationship between the timing of stroke and discharge status was analyzed with logistic regression.

Results: In this study, 2.8% of the patients had a stroke. Incidence of early stroke (within 24 hours after surgery) was 22.8% (46 of 202 patients); incidence of late stroke (more than 24 hours after surgery) was 77.2% (156 of 202 patients). Factors found to be independently associated with stroke-related death included early stroke within 24 hours postoperatively and preoperative chronic renal insufficiency. Those individuals surviving an early stroke had greater rehabilitation needs.

Discussion: An early stroke is associated with a higher risk of death and rehabilitation needs. Strategies to prevent a postoperative stroke are needed. Meticulous assessment by the ICU nurse is essential for rapid identification and treatment of stroke.

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Chapter 17

Fluid and Electrolyte Imbalances Following Cardiac Surgery

Carol Isaac MacKusick

INTRODUCTION

Numerous factors increase the cardiac surgery patient's predisposition for postoperative fluid and electrolyte imbalances, including anesthesia, induced hypothermia, physiologic effects of cardiopulmonary bypass (CPB) techniques, shock resulting in renal insult, cardioplegia, rapid fluid and electrolyte shifts across fluid compartments following CPB, stress associated with surgery, intraoperative volume repletion, hemodilution, the rewarming process that follows hypothermia, or other comorbidities (Margereson, 2003; Pezzella, Ferraris, & Lancey, 2004). This chapter provides an overview of some of the common acid–base and fluid and electrolyte imbalances, treatments for these alterations, and the ICU nurse's role in caring for these patients in the immediate postoperative period. The chapter concludes with a brief look at acute renal failure (ARF) and its treatment implications as they relate to the patient who has undergone cardiac surgery.

FLUID AND ELECTROLYTE DISTRIBUTION

Slightly more than half of the average adult's body weight is made up of fluid—55% to 60% of body weight in men, 50% to 55% of body weight in women, and slightly less in older adults (Holte, Sharrock, & Kehlet, 2002; Matfin & Porth, 2005). The term "fluid" refers to both water and electrolytes found in the body. Electrolytes are substances that develop a positive (cation) or negative (anion) electrical charge when dissolved in water (Matfin & Porth, 2005).

Fluids are found in both the intracellular and extracellular compartments of the body. Intracellular fluid (ICF) accounts for approximately two-thirds of all body fluids. It is located primarily in skeletal muscle mass and provides nutrients for daily cellular metabolism. ICF contains high levels of potassium and phosphorus, and has a moderate amount of magnesium and proteins (Margereson, 2003). Extracellular fluid (ECF) is further divided into intravascular fluid (plasma) and interstitial fluid (between the cells). ECF is more easily lost than ICF because of its location. Electrolyte values that are reported reflect plasma levels and are generally considered representative of ECF status (Speakman & Weldy, 2001).

FACTORS AFFECTING FLUID VOLUME DISTRIBUTION

Fluid balance and homeostasis are maintained by several body systems, including the heart, lungs, endocrine system, and renal system. Additionally, the pituitary, adrenal, and

parathyroid glands all play important roles in maintaining fluid balance and composition. Without a properly functioning cardiovascular system, blood could not be pumped to the kidneys. The renal system requires approximately 25% of cardiac output (CO) for adequate function to occur. A mean arterial pressure (MAP) less than 70 mm Hg results in shunting of the blood supply away from the kidneys (Parker, 2006).

Without proper lung function, blood is inadequately oxygenated, carbon dioxide is not removed through exhalation, and insensible water loss does not occur. The lungs act as the first line of defense against acid–base imbalances. Without all three body systems functioning in harmony, acid–base, fluid, and electrolyte disturbances will occur.

Fluid exchange takes place between the intracellular and extracellular compartments according to differences in hydrostatic pressure and colloid osmotic pressure (COP). Surgery causes a decrease in COP by causing increased capillary permeability, which results in fluid shifts from the vasculature to the interstitium (Holte et al., 2002).

The endocrine system causes sodium and water retention and potassium excretion by stimulating production of antidiuretic hormone (ADH) in response to surgical trauma. ADH secretion causes the kidneys to reabsorb water with a subsequent decrease in diuresis and serum sodium concentration in the postoperative period. Increased production of renin and aldosterone leads to sodium retention and potassium excretion (Holte et al., 2002). Cortisol, which is secreted in response to stress, contributes to postoperative fluid homeostasis by maintaining capillary integrity and by inhibiting the production of stress-related inflammatory mediators, thereby decreasing postoperative fluid shifts (Holte et al., 2002).

Following CPB, the body experiences an increase in ECF volume (by 20% to 30%), sodium retention, and potassium excretion. The estimated amount of fluid buildup is 800 mL for each hour the patient is on CPB. COP decreases by an estimated 50% over this same period (Pezzella et al., 2004).

■ ACID–BASE IMBALANCES

Acid–base balance is determined by the arterial blood pH (hydrogen ion concentration; normal range 7.35–7.45), arterial carbon dioxide (pCO_2; normal range 35–45 mm Hg), partial pressure of oxygen (pO_2) in arterial blood (normal range 80–100 mm Hg), and bicarbonate (HCO_3) value (normal range 22–26 mEq/L). Table 17–1 provides a brief overview of arterial blood gas (ABG) values and their interpretive implications.

The human body desires to maintain a state of homeostasis at all times. When

Table 17–1 Arterial Blood Gas Values and Interpretation

Lab Parameter	Normal Value	Results and Implications
pH	7.35–7.45	< 7.35 = acidosis > 7.45 = alkalosis If compensation is suspected and the pH is within normal limits, look at the "end" where the pH falls: Is it closer to the acidosis side or the alkalosis side?
HCO_3	22–26 mEq/L	< 22 = metabolic acidosis > 26 = metabolic alkalosis
pCO_2	35–45 mm Hg	< 35 = respiratory alkalosis > 45 = respiratory acidosis
pO_2	80–100 mm Hg	< 80 = possible hypoxemia

changes in pH occur, buffer systems are activated to assist the body to normalize pH. As changes in pH occur, cellular responses are stimulated immediately. When the cellular responses are inadequate to handle the resultant change in pH, the respiratory system will provide compensation; if needed, the renal system will activate its compensatory mechanisms as well. ABG changes that are primarily driven by the kidneys may take days to appear, whereas changes caused by the respiratory system will occur in a matter of minutes (MacKusick, 2007). ABG interpretation is discussed in detail in Chapter 11.

Acidosis

Respiratory Acidosis

Respiratory acidosis may occur in the immediate postoperative period following cardiac surgery and is a direct result of inadequate ventilation or sedation (Pezzella et al., 2004; Sinclair, 2006). Table 17–2 lists common causes of respiratory acidosis in the postoperative cardiac surgery patient. Evaluation of ABG results and observation for signs and symptoms are essential roles of the ICU nurse. Signs and symptoms of respiratory acidosis may include dizziness, confusion, weakness,

Table 17–2 Common Causes of Respiratory Acidosis in the Postoperative Cardiac Surgery Patient

Central respiratory depression
- Cardiac arrest with resultant cerebral hypoxia
- Obesity
- Use of opiates, sedatives, or anesthesia

Pulmonary issues
- Acute respiratory distress syndrome
- Aspiration, pneumonia, and/or airway obstruction
- Asthma
- Atelectasis
- Bronchospasm or laryngospasm
- Pneumothorax
- Pulmonary edema
- Pulmonary embolism
- Restrictive lung diseases

Increased CO_2 production
- Shivering
- Sepsis

Hypoventilation secondary to the following conditions:
- Pain
- Sternal incision
- Residual anesthesia
- Awakening with inadequate analgesia and impaired respiratory mechanics

Side effects of opiates

Other
- Inadequate mechanical ventilation (user error)
- Inadequate ventilation/perfusion ratio (decreased ventilation)
- Neuromuscular blocking agents

Sources: Chikwe, Beddow, & Glenville, 2006; Gerhardt, 2007; Gothard, Kelleher, & Haxby, 2003.

palpitations, tetany, convulsions, or ventricular fibrillation. Of note, ventricular fibrillation is more likely to occur during the intraoperative or immediate postoperative period (Wilkins & Wheeler, 2003).

Treatment of respiratory acidosis will vary according to the cause, but generally focuses on improving the patient's ventilation/perfusion (V/Q) status. Conventional interventions performed by the ICU nurse include frequent pulmonary hygiene and encouraging turning, coughing, and deep breathing. Titration of sedation may be indicated if it will not cause excessive patient discomfort. If the patient is on mechanical ventilation, respiratory acidosis can be corrected by increasing the patient's minute ventilation; this goal can be accomplished by increased the preset rate or tidal volume. If the patient is not on mechanical ventilation and conventional interventions are not successful in correcting the respiratory acidosis, depending on the patient's clinical status and ABG results, intubation and mechanical support may be required (Lemmer, Richenbacher, Vlahakes, & Behrendt, 2003).

Metabolic Acidosis

Because a state of electrical neutrality must be maintained within the body at all times, patients with a metabolic acidosis must retain a positive (cation) ion to adjust for the increasing bicarbonate. This goal is accomplished by the renal system, which accumulates positively charged potassium ions. Hyperkalemia frequently accompanies a metabolic acidosis (unless the metabolic acidosis is caused by lactic acidosis or diarrhea). It has been suggested that when acid (hydrogen ion) levels are high in the blood, the body attempts to compensate by causing muscles to take up the excess hydrogen. In order to maintain neutrality, in exchange for the hydrogen ions, potassium is transferred into the blood. Signs and symptoms of metabolic acidosis may include headache, confusion, drowsiness, nausea and/or vomiting, and warm, flushed skin from peripheral vasodilation. CO may decrease, and myocardial contractility is depressed (Porth, 2005a). Because of the contractility issues commonly associated with metabolic acidosis and the potential for hyperkalemia, the ICU nurse should monitor for dysrhythmias in patients who develop this imbalance. Table 17–3 lists common causes of metabolic acidosis seen in the postoperative cardiac surgery patient.

Metabolic acidosis is generally classified as having either a high or normal anion gap. Bicarbonate and chloride are considered the major anions in the body. In cases where a metabolic acidosis is accompanied by a loss of bicarbonate, a normal anion gap metabolic acidosis state is present. The most common causes of normal anion gap acidosis include renal tubular acidosis, excessive administration of isotonic solutions, and diarrhea (Parker, 2006). In cases where the concentration of anions increases (thereby destroying the electrical neutrality of the body), a high anion gap acidosis is said to exist (Hertford, McKenna, & Chamovitz, 1989). Lactic acidosis, renal failure, and diabetic ketoacidosis (DKA) are the most common causes of high anion gap acidosis.

Alkalosis

Respiratory Alkalosis

Some form of hyperventilation is typically the cause of a respiratory alkalosis (Adrogué & Madias, 1998). Table 17–4 lists common causes of respiratory alkalosis seen in the postoperative cardiac surgery patient. Respiratory alkalosis is usually seen as a later complication in the postoperative cardiac surgery patient, arising as a compensatory mechanism (e.g., in response to diuretic therapy) (Pezzella et al., 2004).

Signs and symptoms of respiratory alkalosis may include lightheadedness, inability to concentrate, headache, numbness and tingling of the extremities, tinnitus, palpitations,

Table 17–3 Common Causes of Metabolic Acidosis in the Postoperative Cardiac Surgery Patient

Hemodynamics
- Decreased cardiac output
- Inadequate systemic perfusion
- Decreased cardiac function
- Decreased peripheral perfusion
- Hypotension
- Hypovolemia
- Vasoconstriction from hypothermia

Physiologic conditions (increasing acids)
- Sepsis
- Renal failure
- Renal tubular acidosis
- Regional ischemia
- Diabetic ketoacidosis
- Splanchnic ischemia
- Anaerobic metabolism

Sources: Chikwe, Beddow, & Glenville, 2006; Gerhardt, 2007; Gothard, Kelleher, & Haxby, 2003; Pezzella, Ferraris, & Lancey, 2004.

Table 17–4 Common Causes of Respiratory Alkalosis in the Postoperative Cardiac Surgery Patient

Hypoventilation secondary to the following conditions:
- Anxiety or fear
- Pain or generalized discomfort

Increased oxygen demand as a result of the following conditions:
- Fever
- Bacteremia (especially with gram-negative organisms)
- Sepsis

Pulmonary disorders
- Pneumonia
- Pulmonary edema
- Pulmonary embolism
- V/Q mismatch (increased ventilation, decreased perfusion)

Medications: Respiratory stimulants

User error
- Inappropriate ventilator settings
- Hyperventilation during transfer from OR

Sources: Chikwe, Beddow, & Glenville, 2006; Gerhardt, 2007; Gothard, Kelleher, & Haxby, 2003; Rimailho, Goldstein, & Vincent, 1985.

Table 17–5 Common Causes of Metabolic Alkalosis in the Postoperative Cardiac Surgery Patient

Adrenal disorders: hyperaldosteronism
Hypokalemia
Hypochloremia
Excessive diuretic administration
Nasogastric suctioning
Overuse of potassium wasting drugs (e.g., increased use of thiazide diuretics)
Vomiting
Massive transfusions (from citrate)

Sources: Chikwe, Beddow, & Glenville, 2006; Gothard, Kelleher, & Haxby, 2003.

dry mouth, sweating, chest pain, or nausea and vomiting. Late stage signs and symptoms may include loss of consciousness or seizures (Adrogué & Madias, 1998). The neurologic symptoms may be caused by a hypocalcemic state, which is commonly seen with a respiratory alkalosis. This acid–base disturbance can cause an increase in protein binding of ionized calcium (the amount of calcium not bound to protein and available for use by the body). Treatment is aimed at correcting the underlying cause.

Metabolic Alkalosis

Common causes of metabolic alkalosis in the postoperative cardiac surgery patient are presented in Table 17–5. Like respiratory alkalosis, metabolic alkalosis is usually seen later in the postoperative cardiac surgery patient, acting as a compensatory mechanism (Pezzella et al., 2004). Patients often have concomitant hypokalemia and hypocalcemia; these underlying conditions must be simultaneously corrected. The relationship between potassium and hydrogen ions was explained in the section on metabolic acidosis. In the case of a metabolic alkalosis, while the underlying physiologic principles remain the same, the opposite effect occurs. With a metabolic alkalosis, as the hydrogen ion concentration increases in the blood, potassium ions move into the cells to maintain neutrality. This results in a hypokalemic state. Signs and symptoms of metabolic alkalosis include poor skin turgor (from fluid loss). Treatment is aimed at restoring fluid balance and correcting the underlying disorder.

ELECTROLYTE IMBALANCES

Electrolyte imbalances are frequently seen in postoperative cardiac surgery patients. The ICU nurse should recognize normal values, signs, and symptoms associated with these imbalances, and implement appropriate interventions to correct the imbalances. Table 17–6 lists the common electrolytes and their associated normal values.

Potassium Imbalances

Fluctuations in potassium levels are common following cardiac surgery and can affect cardiac automaticity and conduction (Khalpey, Ganim, & Rawn, 2008; Pezzella et al., 2004). Potassium works with sodium to help maintain fluid balance within the body, with kidney regulation being the mechanism that governs the balance (Flanagan, Devereaux, Abdallah, & Remington, 2007). The potassium found in the extracellular fluid is responsible for neuromuscular function and plays a major role in myocardial contractility, function, and rhythm (Rose & Post, 2000a).

Table 17–6 Electrolyte Reference Values

Electrolyte	Normal Value*
Potassium	3.5–5.0 mEq/L
Sodium	135–145 mEq/L
Magnesium	1.8–2.4 mg/dL†
Phosphorus	2.5–4.5 mg/dL
Calcium	8.5–10.5 mg/dL‡

*Normal value markers may vary according to facility. Always check with your local laboratory if unsure of the normal values for any laboratory finding.

†Serum magnesium may also be reported in millimoles per liter (mmol/L). In these cases, normal values would be in the range of 0.65–1.1 mmol/L.

‡Serum calcium can be reported as total calcium, ionized calcium, or non-ionized calcium. The value provided in the table is for the total calcium. A normal ionized calcium value is in the range of 4.4–5.3 mg/dL.

Hyperkalemia

The major causes of hyperkalemia in the postoperative cardiac surgery patient are decreased urinary output, cardioplegia, decreased insulin levels, metabolic acidosis, diabetes, and hemolysis of red blood cells (Khalpey et al., 2008; Margereson, 2003; Pezzella et al., 2004). Oliguric or anuric renal failure, or failure to excrete and metabolize potassium through the kidneys may occur postoperatively as well (Parker, 2006). Many cardiac medications can cause hyperkalemia (e.g., angiotensin-converting enzyme [ACE] inhibitors, potassium-sparing diuretics, beta blockers, unfractionated heparin, and digoxin). Massive blood transfusions are also associated with higher levels of potassium.

Evidence of hyperkalemia may be noted in the electrocardiogram (ECG). Peaked T waves (see Figure 17–1), a widening QRS complex, and a prolonged PR interval may be noted (Montague, Ouellette, & Buller, 2008). Cardiac arrest may occur at any point if potassium levels continue to increase (Roth & Patel, 2003).

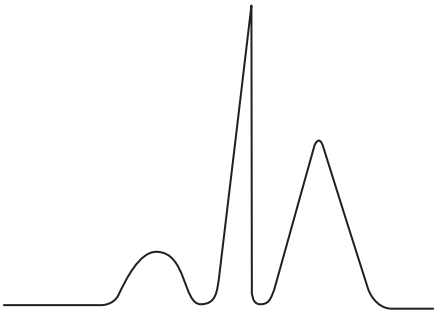

Figure 17–1 Hyperkalemia—peaked T wave.

Source: Illustrated by James R. Perron

Patients with progressive hyperkalemia will present with ventricular dysrhythmias and may develop nausea, paresthesias, muscle weakness, cramps, or paralysis (muscle weakness first appears in the larger muscles and the myocardium). These signs and symptoms are directly related to the effect of the elevated potassium on the cellular membrane potential. Respiratory failure may also occur as a result of hyperkalemia (Margereson, 2003).

Treatment of moderately elevated serum potassium levels may include sodium polystyrene sulfonate (Kayexalate®). Kayexalate acts by exchanging sodium ions for potassium ions in the gastrointestinal (GI) tract, thereby allowing for elimination of excess potassium in the stool. Before Kayexalate is administered, however, it must be known if a patient can tolerate an increase in serum sodium (Kozar & Moore, 2006).

Emergent renal replacement therapy is an option to lower serum potassium levels in those patients who do not respond to conservative therapy. With severe hyperkalemia, 10 units of regular insulin with one ampule of $D_{50}W$, calcium gluconate (if no cardiac symptoms related to the hyperkalemic state are present), a beta agonist, or sodium bicarbonate may be administered. These interventions are temporary in nature, but will provide almost immediate lowering of potassium levels and allow time for the patient to be prepared for dialysis therapy (Margereson, 2003).

Hypokalemia

The major causes of hypokalemia in cardiac surgery patients include brisk diuresis, rapid correction of hyperglycemia with insulin, adrenal hyperreactivity, vomiting, alkalosis, and hypothermia (Margereson, 2003; Pezzella et al., 2004). Patients may also present with a dilutional hypokalemia (Leier, Dei Cas, & Metra, 1994).

Hypokalemia is associated with increased ventricular dysrhythmias and hypertension (Whelton et al., 1997), a prolonged PR interval, U-wave development as the T wave flattens, and ST-segment depression (Diercks, Shumaik, Harrigan, Brady, & Chan, 2004). Figure 17–2 illustrates the development of U waves in the hypokalemic patient.

Hypokalemia will typically manifest as muscle weakness, fatigue, hypotension, and absent or diminished bowel sounds (Margereson, 2003). It is frequently accompanied by metabolic alkalosis and hypomagnesemia. Ventricular dysrhythmias and syncope are likely to develop as well (Leier et al., 1994).

Treatment for hypokalemia involves replacement of potassium, either orally or intravenously. If concomitant hypomagnesemia exists, initial correction of magnesium levels is required (Leier et al., 1994). Typically, serum potassium levels will increase by 0.1 mEq/L for each 2 mEq of potassium replacement administered (Pezzella et al., 2004).

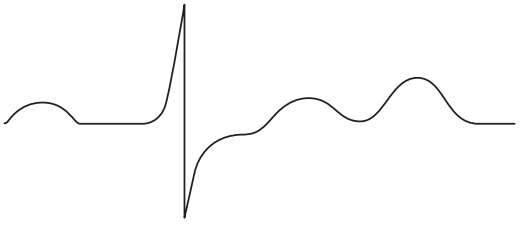

Figure 17–2 Hypokalemia—U wave.
Source: Illustrated by James R. Perron

Sodium Imbalances

Sodium is the major extracellular ion found in the body; its concentration normally ranges between 135 and 145 mEq/L. Sodium is directly responsible (in conjunction with potassium and the sodium potassium ATPase pump) for maintaining the fluid balance in the body, ensuring appropriate water distribution, and maintaining the ECF volume status (Rose & Post, 2000b).

Hypernatremia

Hypernatremia is a relatively uncommon phenomenon, but is associated with a 40–60% (or greater) mortality rate. This imbalance occurs when there is a gain of sodium in excess of water or a loss of water in excess of sodium. In the postoperative cardiac surgery patient, it is most commonly seen in conjunction with hyperventilation. Hypernatremia may also develop secondary to dehydration from fever, diabetes, or use of osmotic diuretics (Margereson, 2003).

Signs and symptoms of hypernatremia may include thirst, fever, restlessness, weakness, dry oral mucosa or tongue, poor skin turgor, and disorientation with possible progression to lethargy, stupor, or coma (Howanitz & Howanitz, 2007). Depending on the cause, treatment focuses on either increasing water within the body or removing sodium from it (Kang, Kim, & Oh, 2002; Margereson, 2003).

Postoperative cardiac surgery patients with a severe hypernatremia (greater than 150 mEq/L) may experience an acid–base imbalance that is difficult to correct. In this situation, use of tromethamine (Tham®) to treat metabolic acidosis is recommended instead of sodium bicarbonate, as the latter therapy may increase sodium levels further and cause central nervous system effects (Pezzella et al., 2004).

Hyponatremia

Hyponatremia commonly arises when cells swell as water enters them. This swelling can

progress to the point that it eventually leads to cellular rupture. Common causes of hyponatremia include use of certain medications (e.g., thiazide diuretics, nonsteroidal anti-inflammatory drugs [NSAIDs]), pneumonia, and acute respiratory failure. Signs and symptoms may include headache, nausea and vomiting, generalized muscle weakness, and fatigue. CO may be decreased as well (Leier et al., 1994). Cheyne-Stokes respirations and respiratory failure may accompany severe hyponatremia (Rai, Whaley-Connell, McFarlane, & Sowers, 2006). Only rarely will a postoperative cardiac surgery patient develop hyponatremia in the absence of hyperglycemia, however (Pezzella et al., 2004).

Hyponatremia may be seen in patients who are either hypovolemic, normovolemic, or hypervolemic. Most often, this type of sodium imbalance is seen in patients with severe heart failure, in whom a decrease in CO triggers the release of ADH, which in turn causes hypervolemia. Patients who are hypovolemic may develop hyponatremia secondary to brisk diuresis, excess insensible loss from the skin or GI tract, or glucocorticoid deficiency. Those who are normovolemic may develop hyponatremia secondary to hypokalemia, medications, or hypothyroidism. Patients with hypervolemia may develop hyponatremia secondary to renal failure or heart failure (Margereson, 2003).

The patient with hypervolemia-associated hyponatremia will present with changes in mental status, restlessness, anxiety, decreased urinary output, weight gain, peripheral and dependent edema (including pitting edema; see Figure 17–3), hypertension, jugular vein distention, shortness of breath, diffuse crackles, and muffled heart sounds (Flanagan et al., 2007). Because of their fluid volume status, these patients also present with low hemoglobin and hematocrit levels (Flanagan et al., 2007).

Treatment of hyponatremia includes replacement of fluid in patients with hypovolemic hyponatremia, water restriction in hypervolemic or normovolemic hyponatremia, and management of associated adrenal and ADH imbalances as appropriate. For those patients with volume overload, treatment includes a loop diuretic agent, fluid restriction to less than 1000 mL/day, and positive inotropic agents (Leier et al., 2004). During treatment, close monitoring and accurate intake and output records should be maintained, and the nurse should monitor the patient's vital signs closely to assess for rapid fluid changes (Flanagan et al., 2007). The level of sodium replacement depends on the extent of loss; infusions of 3% or 5% sodium must be closely monitored. Rapid changes in level of consciousness indicate worsening cerebral edema.

Magnesium Imbalances

Magnesium is an electrolyte that plays a key role in cellular function. Some researchers have suggested that magnesium is equally as responsible for ensuring appropriate electrical conduction in the heart as is potassium (Henke & Eigsti, 2003). Magnesium helps maintain cellular permeability and neuromuscular excitability, and it is intrinsically involved in the appropriate utilization of ATP (Pezzella et al., 2004). Levels of this ion are regulated by GI absorption and renal excretion; the normal range is 1.8–2.4 mg/dL.

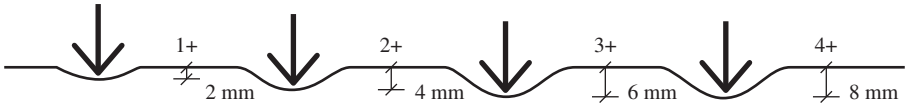

Figure 17–3 Pitting edema.

Source: Illustrated by James R. Perron

Hypermagnesemia

Hypermagnesemia is most likely to occur in patients with decreased renal function. The patient with an elevated magnesium level will present with lethargy, muscle weakness, dilated pupils, nausea, vomiting, diarrhea, anorexia, muscle weakness, decreased or absent bowel sounds, and hypotension (Margereson, 2003). Initially the patient will present with hypotension and shallow respirations, followed by periods of apnea (Hoffman, 2002). Ventricular dysrhythmias, bradycardia, a prolonged PR interval, complete heart block, and cardiac arrest are not uncommon in patients whose magnesium levels exceed 2.5 mg/dL (Henke & Eigsti, 2003; Margereson, 2003).

Magnesium levels greater than 10 mg/dL (particularly 15 mg/dL) are usually fatal. Treatment for symptomatic hypermagnesemia includes an infusion of insulin and glucose as well as intravenous calcium gluconate, which acts as a magnesium antagonist. Calcium gluconate rapidly reverses cardiac dysrhythmias or respiratory depression directly related to hypermagnesemia. The ICU nurse should prepare to administer 10–20 mEq of calcium gluconate over 10 minutes or follow facility policy in cases of life-threatening hypermagnesemia. Patients who develop this electrolyte imbalance will also require fluid resuscitation and loop diuretics. Mechanical ventilation may be required for those individuals with severe respiratory depression, and a temporary pacemaker may be required in patients who experience severe bradycardia. Hemodialysis may be required if the patient's renal function is inadequate (Hoffman, 2002).

Hypomagnesemia

Hypomagnesemia is a common clinical problem in postoperative cardiac surgery patients, especially in those individuals who develop hemodilution following CPB or who receive diuretics (Margereson, 2003; Pezzella et al., 2004). This electrolyte imbalance is associated with atrial and ventricular dysrhythmias. Affected patients may present with depression, muscle weakness, coronary spasm, confusion, and irritability. Tetany, delirium, and seizures are also possible (Margereson, 2003).

Hypomagnesemia is often accompanied by hypophosphatemia, hypocalcemia, and hypokalemia. The action of the parathyroid gland and hormone release will be inhibited in cases where the serum magnesium falls below 1.0 mg/dL (Hoffman, 2002). Changes will appear on the ECG tracing, including nonspecific T wave changes, appearance of U waves, prolonged QT intervals, widened QRS complex, ST-segment depression, peaked T waves, and torsade de pointes. Ventricular ectopy, paroxysmal supraventricular tachycardia, premature ventricular contractions, and atrial and ventricular fibrillation are likely to occur as well (Margereson, 2003; Pezzella et al., 2004). Finally, insulin resistance may occur in patients with severe hypomagnesemia, making serum glucose levels hard to control (Rodriguez-Hernández, Gonzalez, Rodriguez-Morán, & Guerrero-Romero, 2005).

Treatment entails magnesium repletion. If magnesium is to be given intravenously, the patient's renal function should be determined prior to its administration to help avoid refractory hypermagnesemia (Phillips, 2004). Additionally, during infusions of magnesium, urinary output should be closely monitored. If urinary output decreases to less than 100 mL over 4 hours, the infusion of magnesium should be discontinued and the surgeon notified. The actions of magnesium as a calcium channel blocker, as a regulator of intracellular potassium, and in the activation of ATP explain the antiarrhythmic effects of this electrolyte (Pezzella et al., 2004).

Protection of the patient's overall condition remains a high priority in a hypomagnesemic patient. Seizure precautions should be implemented, the airway and respiratory status should be continually monitored, and

falls precautions implemented for those individuals who have an altered mental status.

Calcium Imbalances

The majority (greater than 99%) of the body's calcium is found in the skeletal system. Most of the remaining calcium is found inside cells, with only 0.1–0.2% of this remaining 1% being found in the extracellular fluid (Matfin & Porth, 2005). A normal serum calcium level is in the range of 8.5–10.5 mg/dL in individuals with a normal pH and normal serum albumin levels. For every 1 g/dL decrease in serum albumin, there is an approximate 0.75–1.0 mg/dL decrease in total calcium. For every 0.1 unit rise in pH, there is an approximate 0.16 mg/dL decrease in total calcium (Matfin & Porth, 2005). Assessment of total calcium level requires pH and serum albumin evaluation.

In cases of protein malnutrition or other issues affecting serum albumin, an ionized calcium level is a more accurate indicator of calcium status than total calcium level. Ionized calcium is the calcium that is not bound to protein; its normal range is from 4.4 to 5.3 mg/dL (Fukagawa, Kurokawa, & Papadakis, 2008). Adequate levels of ionized calcium are essential for cardiac performance (Khalpey et al., 2008).

Hypercalcemia

Three basic causes exist for hypercalcemia: increased intestinal absorption, increased bone resorption, and decreased elimination (Matfin & Porth, 2005). Decreased elimination of calcium is seen generally with medication use, or when decreased availability of physiologic calcium is present (such as with acidosis). Medications that increase serum calcium levels include thiazide diuretics and lithium carbonate, both of which decrease renal calcium excretion. Some estrogens also increase calcium levels.

Patients with hypercalcemia will present with altered mental status, fatigue, weakness, lethargy, anorexia, nausea, vomiting, constipation, decreased renal function or ARF, polyuria, polydipsia, shortened QT segments, and depressed T waves. Nonspecific dysrhythmias, bradycardia, and first-, second-, or third-degree heart block may develop. Bundle branch blocks may also be seen. If left untreated, the patient with severe hypercalcemia may develop psychosis or lethargy that leads to coma (Ziegler, 2001).

Calcitonin may be given intravenously as a treatment for hypercalcemia; it enhances urinary calcium excretion and decreases bone resorption of this electrolyte (Matfin & Porth, 2005). Glucocorticoids have also been used successfully in cases of hypercalcemia; however, results will not be seen for 5 to 7 days with this therapy, and patients may develop increased risks for hyperglycemia and sodium and water retention (Jacobs & Bilezikian, 2005).

Hypocalcemia

Hypocalcemia occurs when serum calcium is less than 8.5 mg/dL. At a minimum, measurement of ionized calcium level is needed to confirm a diagnosis of hypocalcemia, and these data should always be reviewed in conjunction with the acid–base status of the patient. A patient with a low serum calcium but normal ionized calcium is typically asymptomatic, and is referred to as having pseudohypocalcemia.

Development of hypocalcemia is expected following CPB, hemodilution, low CO, or administration of citrated blood (Khalpey et al., 2008). Packed red blood cells, for example, are conditioned with citrate to prevent their coagulation. When citrate combines with calcium, hypocalcemia can occur. This effect generally does not occur during normal blood transfusions because citrate has adequate time to metabolize in the liver; only in cases of faster than normal blood transfusions or cases of liver dysfunction does this citrate–calcium binding become a potential problem. Cardiac surgery patients who develop sepsis are also predisposed to hypocalcemia (Morgan, Mikhail, & Murray, 2005).

Patients with hypocalcemia may report numbness or tingling of the fingers and toes. Muscle cramps, spasms, tremors, twitching, and abdominal and intestinal cramps are common. Bowel sounds are hyperactive. Because of the increased neuromuscular activity, hypocalcemic patients who are left untreated may develop seizures, laryngospasm, and bronchospasm. These spasms may lead to laryngeal stridor, which will eventually necessitate intubation if the calcium level is not adequately treated (Litwack, 2003). Auscultation of breath sounds may reveal inspiratory and expiratory wheezing. In approximately 70% of patients with hypocalcemia, positive Trousseau's and Chvostek's signs are present. Trousseau's sign is considered positive when an inflated blood pressure cuff elicits a carpopedal spasm (see Figure 17–4). Chvostek's sign is considered positive when tapping of the facial nerve elicits facial muscle movement (Parker, 2006).

Cardiac complications associated with hypocalcemia include a decrease in myocardial contractility and CO. Symptoms of hypocalcemia will likely include hypotension, a prolonged QT interval, shortness of breath, and dysrhythmias ranging from bradycardia to asystole. Heart sounds may be muffled (Khalpey et al., 2008).

Acute hypocalcemia should be promptly corrected with administration of 10% calcium gluconate. This medication may be given either as an intravenous push over 5 to 10 minutes or mixed in 0.9% normal saline for infusion according to facility policy.

Phosphorus Imbalances

Phosphorus is mainly an intracellular anion, accounting for approximately 1% of the total body weight of adults (Matfin & Porth, 2005). These ions play integral roles in the repair of cells and tissues, and are crucial ions in the production of ATP (Parker, 2006). Phosphorus is excreted through the kidneys; as kidney function declines, phosphorus levels are likely to increase (Porth, 2005b). As is the case with calcium, hormonal regulation is provided through the parathyroid gland (Weinman et al., 2007). A normal serum phosphorus is in the range of 2.5–4.5 mg/dL.

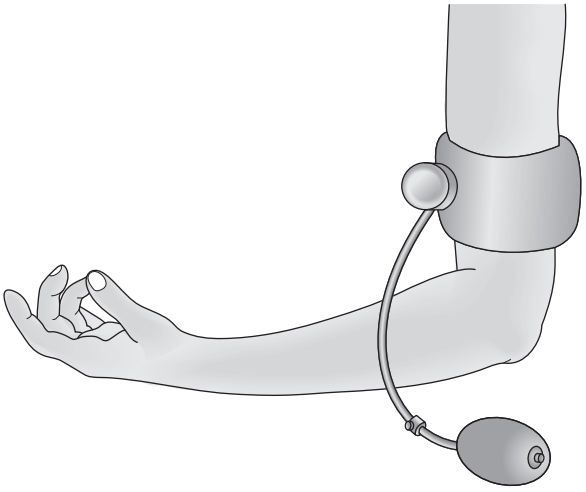

Figure 17–4 Test for hypocalcemia.
Source: Illustrated by James R. Perron

Hyperphosphatemia

Hyperphosphatemia is defined as a serum phosphorus level greater than 4.5 mg/dL, but becomes clinically significant when phosphorus levels exceed 5.0 mg/dL (Patterson, 2008). Almost all cases of hyperphosphatemia are a direct result of decreased renal function. When the glomerular filtration rate (GFR) falls below 50 mL/min, the kidneys are no longer able to adequately metabolize phosphorus (Parker, 2006). Respiratory acidosis and DKA may also lead to hyperphosphatemia. It is suggested that the relationship between a respiratory acidosis and hyperphosphatemia is twofold. First, a sudden rise in carbon dioxide levels can lead to an elevation in phosphorus levels. Second, presence of a respiratory acidosis causes phosphorus to move from the intracellular to extracellular fluid compartment. Presence of a metabolic acidosis, as seen in DKA, is associated with hyperphosphatemia (Patterson, 2008).

Signs and symptoms of hyperphosphatemia may include altered mental status, delirium, seizures, paresthesias (especially around the mouth or in the fingers and toes), and tetany. Positive Trousseau's and Chvostek's signs, hypotension, and cardiac dysrhythmias may also be present. Heart sounds may be muffled, and a pericardial friction rub may be present, indicative of potential heart failure. The QT interval is often prolonged.

If kidney function is adequate, normal saline infusions may help return the serum phosphorus to baseline. If the patient is symptomatic, emergent renal replacement therapy may be indicated (Parker, 2006).

Hypophosphatemia

Patients with a serum phosphorus level less than 1.0 mg/dL are considered severely compromised. Because of the key role that phosphorus plays with ATP, a sharp decrease in phosphorus levels results in cell energy depletion (Mailhot & Richardson, 2006). The most common cause of hypophosphatemia is increased renal elimination of this ion, as is seen with respiratory alkalosis or postoperative stress (Margereson, 2003).

Signs and symptoms of severe hypophosphatemia include paresthesias; severe, profound, and progressive muscle weakness; tremors; muscle pain and tenderness; lethargy; confusion; anxiety; and apprehension. If this condition is left untreated, the patient will develop hypoxia and bradycardia. Hypotension will be present, and stroke volume will be decreased. Muscle weakness will eventually lead to acute respiratory failure from decreased contraction of the diaphragm (Margereson, 2003). Seizures and coma may also be present. Hemolytic anemia as well as leukocyte and platelet dysfunction will be noted. Respiratory rate decreases as phosphorus levels decrease. However, if the hypophosphatemia is related to presence of respiratory alkalosis, tachypnea will be present. Hypomagnesemia and hypercalcemia are oftentimes present in conjunction with hypophosphatemia (Mailhot & Richardson, 2006; Matfin & Porth, 2005).

Treatment for severe hypophosphatemia requires intravenous replacement of phosphorus. The precise therapy employed depends on the patient's renal status, as one phosphorus preparation is built on sodium and the other relies on potassium. For the patient with adequate renal function, potassium phosphate may be administered; sodium phosphate should be administered to those patients with decreased renal function.

Should a heart block or flaccid paralysis develop, the infusion of phosphorus should be immediately discontinued, as these symptoms indicate rebound hyperphosphatemia. A patient with severe hypophosphatemia may also be more prone to infection. Consequently, a complete blood count should be

performed on the postoperative cardiac surgery patient to provide information about the presence of bleeding and possible infection.

FLUID BALANCE AND VOLUME MANAGEMENT

Fluids shift on an as-needed basis between compartments to maintain homeostasis. This fluid exchange is partly affected by osmolarity, and hence by electrolyte concentrations (Margereson, 2003). Shifts between compartments occur as the body seeks to maintain an appropriate cation and anion distribution as well as optimal fluid levels in each compartment. Frequently, alterations in fluid volume status accompany electrolyte imbalances. For the nurse caring for the postoperative cardiac surgery patient, either hypovolemia or hypervolemia may represent worsening of a preexisting medical condition or may be related to the surgical procedure and associated interventions. In either case, it is important to recognize the implications of alterations in fluid volume status and to determine appropriate courses of treatment.

Hypovolemia

Hypovolemia, which is also known as fluid volume deficit (FVD), results when both water and electrolytes are lost together. This condition is not the same as dehydration, which results from water loss alone (and, therefore, leads to hypernatremia). An isotonic fluid volume deficit indicates that electrolyte levels remain essentially unchanged (Matfin & Porth, 2005).

Common causes of fluid losses in the postoperative cardiac surgery patient include blood loss, fever, and third spacing of fluid (Parker, 2006). Hypovolemic shock results when circulating blood volume falls to such a low level that vital organs are not perfused adequately, causing irreversible damage to these organs (Wilkins & Wheeler, 2006).

Assessment

Hypovolemia is common postoperatively, but often proves difficult to assess. FVD is generally gauged as mild, moderate, or severe. Mild FVD represents a loss of approximately 2% of total body weight; moderate FVD entails an approximately 2–5% body weight loss; and severe FVD involves a greater than 8% body weight loss (Matfin & Porth, 2005).

Signs and symptoms of FVD and hypovolemia include decreased capillary refill time, central venous pressure (CVP), and urinary output; dizziness; increased osmolality, specific gravity, thirst sensation in conscious patients, hemoglobin and hematocrit, and blood urea nitrogen (BUN) to serum creatinine ratio (usually greater than 30:1); postural or prolonged hypotension; tachycardia; weak and thready pulse; and decreased vein filling. A urinary output rate that is less than 0.5 mL/kg/hr is indicative of severe FVD and inadequate renal perfusion. Assessment of skin turgor reveals skin that does not "spring" back, but rather remains in the tented position; dry oral membranes; and a tongue that appears shrunken, with fissures. Severe FVD is also accompanied by confusion, upper body weakness, and speech difficulties.

Shock develops when FVD is left untreated. In such cases, hypotension becomes severe and perfusion to vital organs is compromised (Matfin & Porth, 2005; Wilkins & Wheeler, 2006).

Treatment

Treatment of hypovolemia depends on the cause of the FVD. Hypoxia is likely to develop in cases of shock; oxygen should be administered to maintain adequate saturation (Wilkins & Wheeler, 2006). The goal of FVD treatment is to expand plasma volume until a desired MAP has been attained and sustained. When planning for delivery of replacement fluids, daily fluid losses and intraoperative fluid loss must also be accounted for and

added into the replacement. A fever greater than 101 °F (38.3 °C) increases the daily fluid requirement by approximately 500 mL. If the patient is not severely hypotensive, the fluid replacement plan may be based on an assumption that 50% to 80% of the fluid loss will be replaced over 12 to 24 hours; in cases where severe hypotension or shock exists, volume repletion must take place much more quickly (Sue & Bongard, 2008). The vasodilation that occurs with rewarming following CPB may necessitate administration of additional fluid to maintain adequate cardiac output (Margereson, 2003).

Fluid Challenge

For the patient who has developed oliguria and who has a urinary output of less than 0.5 mL/kg/hr, a fluid challenge should be anticipated. The goal of a fluid challenge is to replenish the intravascular volume. A supplemental dose of fluid (e.g., 250 mL) is administered over a short period of time (e.g., 15 minutes). Administration of fluid challenges may avert use of inotropic agents, which are associated with tachycardia and increased myocardial oxygen consumption (Margereson, 2003).

The fluid needs will differ for patients based on their history, comorbidities, and the surgical procedure performed. In addition, patients' hemodynamic profiles and tolerance to fluid will vary. For example, as discussed in Chapter 5, postoperative patients who underwent valve repair for aortic stenosis will initially continue to have left ventricular hypertrophy following surgery. This condition may result in outflow obstruction and subsequent postoperative hemodynamic instability from preload reduction. Treatment will include volume repletion. Conversely, postoperative patients who underwent repair for aortic regurgitation will likely require vasodilator therapy. Patients who underwent repair for mitral stenosis will likely need prudent fluid administration in combination with inotropic support to augment CO. Finally, patients who underwent repair for mitral regurgitation may develop postoperative right ventricular failure and, therefore, require inotropic administration (Khalpey et al., 2008).

Ongoing debate surrounds the use of crystalloids versus colloids for fluid resuscitation. As their name connotes, isotonic crystalloid solutions contain the same concentration of electrolytes as interstitial fluid, along with a high percentage of water (Margereson, 2003). Commonly used isotonic replacement fluids include 0.9% normal saline and lactated Ringer's (LR) solution.

Factors driving the use of crystalloid therapy include their ready availability in the ICU and their low cost. Other considerations with crystalloid administration for volume repletion include their tendency to cause decreased blood viscosity, increased urinary output with associated sodium and potassium excretion, and increased peripheral blood flow, thereby improving tissue perfusion (Margereson, 2003).

A negative aspect of isotonic crystalloid administration is that approximately 75% of the volume moves out of the vascular space, with half being lost to the circulating volume shortly after crystalloid administration. Further, one of the components of crystalloids used for fluid repletion in postoperative cardiac surgery patients is sodium. If excessive amounts of sodium are administered, the patient's osmolarity may become elevated and water may be drawn from cells, resulting in cellular dehydration. Some providers prefer to alternate administration of 0.9% normal saline with administration of LR in an effort to avoid this excessive sodium load. Administration of LR, however, can result in hyperkalemia, especially in patients with renal dysfunction (Margereson, 2003).

Administration of hypotonic crystalloids may occasionally result in cerebral edema or seizures. Administration of glucose-containing solutions for volume repletion (e.g., D_5W) can

cause dilutional hyponatremia or hyperglycemia with hyperosmolarity and osmotic diuresis, as such solutions do not contain any electrolytes (Margereson, 2003).

Following CPB, patients develop a low COP secondary to a systemic inflammatory response. Colloids such as albumin, plasma protein fraction, or fresh frozen plasma can raise COP. Unlike crystalloids, colloids remain in the intravascular space for an extended period of time, allowing for the osmotic force to promote movement of water back into the intravascular space from the interstitium (Margereson, 2003).

Although they are more expensive than crystalloids, colloids may be preferred following cardiac surgery, as crystalloid therapy may decrease COP and increase the risk of pulmonary edema. In some patients, colloid administration may improve the patient's hemodynamic profile and improve balance between oxygen supply and demand (Margereson, 2003).

Healthcare providers should remember that most colloids do not contain clotting factors or contribute to oxygen-carrying capacity (Margereson, 2003). In addition, some of the protein molecules do eventually leak into the interstitium. When this phenomenon occurs, the oncotic pull may promote third spacing of fluid.

The efficacy of fluid challenges is traditionally assessed based on improvements in the patient's hemodynamic profile and physical assessment findings. It is suggested that evaluation for respiratory changes associated with CVP and blood pressure be made, as these changes reflect changes in filling pressures secondary to pleural pressure (Holte et al., 2002).

Third Spacing

Third spacing refers to the movement of fluids from the vascular space to a part of the body where exchange with the rest of ECF is decreased, resulting in alterations of capillary membrane permeability (Khalpey et al., 2008). Symptoms of third spacing will mimic those associated with FVD, except that either weight gain may occur or weight may remain stable in conjunction with third spacing.

Third spacing occurs in two phases. The first phase mimics FVD (except for the weight loss), and the second (recovery) phase mimics hypervolemia. In the postoperative cardiac surgery patient, third spacing is most likely to arise as a result of vasodilation, hypothermia, or hyperemia (increased amount of blood) to the tissue bed (Khalpey et al., 2008). Treatment is aimed at moving the fluid from the third space to the cellular compartments as well as forcing diuresis. During the initial stage, treatment with LR is generally considered appropriate unless other alterations in electrolyte balance are present. The goal remains to provide adequate circulating volume to maintain an optimal blood pressure and urinary output until the recovery phase begins (Hammon, 2008).

Hypervolemia

Fluid weight gain with subsequent diuresis should be anticipated following CPB. Hypervolemia, which is also known as fluid volume excess (FVE), occurs when water and serum sodium are proportionately increased in the body. Common causes of FVE include excessive intake of fluids that cannot be removed (e.g., as occurs in renal failure or heart failure, or following administration of fluids at an excessive rate), excessive sodium intake, or inadequate sodium and water elimination (e.g., secondary to heart, renal, or liver failure). In the postoperative cardiac surgery patient, hypervolemia is most commonly related to excessive fluid administration intraoperatively, most notably in patients with either preexisting renal dysfunction, heart failure, or hypoalbuminemia (Kerns, 2006).

Assessment

Patients with FVE will manifest weight gain; peripheral edema; distended peripheral veins; jugular venous distention; increased CVP; crackles; decreased dilutional BUN, hemoglo-

bin, and hematocrit; and bounding pulses. In cases of severe FVE, pulmonary edema, ascites, or pleural effusion may develop (Matfin & Porth, 2005). Patients may also develop hypertension, cough, and dependent edema. Some patients may report visual blurring or headache, and an S_3 heart sound is a common finding (Parker, 2006).

Treatment

For patients who do not have preexisting renal dysfunction, diuresis is attempted to normalize volume status when hypervolemia occurs. Electrolyte balance must be carefully monitored during this time to avoid potentially life-threatening complications of rapid diuretic therapy. For the patient with ARF, renal replacement therapy (usually through hemodialysis or continuous renal replacement therapy [CRRT]) will be necessary to maintain an appropriate fluid volume state. Fluid restriction to less than 1000 mL/day is typically implemented as well.

Glycemic Issues

Postoperative cardiac surgery patients may have comorbidities that include either type I or II diabetes mellitus. During times of increased stress, serum glucose levels become more labile, and the patient with or without a history of diabetes is more likely to exhibit hyperglycemia or hypoglycemia. The fluctuations in blood glucose levels will also result in alterations in fluid and electrolyte status. Tight glycemic control is essential to help ensure improved patient outcomes (Talmor & Lisbon, 2005).

■ ACUTE RENAL FAILURE AND RENAL INSUFFICIENCY

Unfortunately, some postoperative cardiac surgery patients may have sustained renal damage from ischemia or decreased blood flow. CPB causes an increased secretion of catecholamines, renin, angiotensin II, aldosterone, vasopressin, atrial natriuretic peptide, and pro-inflammatory mediators. Release of these substances leads to decreases in renal blood flow and glomerular filtration rate (Khalpey et al., 2008). During CPB, attempts to protect the kidneys focus on ensuring hemodilution, returning to a pulsatile flow as soon as possible, and reestablishing a normal body temperature as quickly as possible (Henke & Eigsti, 2003).

The incidence of ARF in patients undergoing cardiac surgery is approximately 30%. Of these patients, 1% to 5% may require dialysis therapy (Silvestry, 2008; Talmor & Lisbon, 2005). Patients who had a myocardial infarction may have resultant renal impairment or acute tubular necrosis (ATN) from the ischemia. Patients who are older, have diabetes mellitus, or who have a history of heart failure are more likely to develop ARF following a cardiovascular event (Campbell, 2003; Henke & Eigsti, 2003). Other individuals at higher risk include those with poor underlying cardiac performance, advanced atherosclerosis, and preexisting decreased GFR. The amount of time spent on CPB and intraoperative instability are also predictors of the development of postoperative renal impairment (Silvestry, 2008).

Renal perfusion must be maintained in all patients. Urinary output should be at least 0.5 mL/kg/hr. For these goals to be met, satisfactory CO and blood pressure are essential. Maintaining them at appropriate levels can be accomplished by delivering volume repletion to keep up with urinary output, which is typically 200–300 mL/hr following CPB. If urinary output is maintained with use of diuretics, renal perfusion is considered adequate (Khalpey et al., 2008).

Azotemia

Azotemia is the buildup of nitrogenous waste products from protein metabolism; these wastes are normally eliminated by urination (Broscious & Castagnola, 2006; Dirkes & Kozlowski, 2003). The patient with azotemia

will demonstrate increasing serum creatinine and BUN levels, and GFR will decrease. As GFR continues to decline, FVE will develop. Uric acid levels may also rise, resulting in symptoms of gouty arthritis or joint and soft tissue pain (Dirkes & Kozlowski, 2003).

Acute Renal Failure

Acute renal failure may be recognized by a sudden, rapid deterioration in renal function. Despite new treatment strategies and improved surveillance methods, the number of patients who develop ARF is increasing, and mortality rates remain greater than 50% (Ronco, Kellum, Mehta, Bellomo, & Palevsky, 2002). Elderly patients and individuals with a history of previous CPB procedures, type I diabetes mellitus, renal disease, or heart failure prior to admission seem to be most predisposed to development of ARF (Campbell, 2003; Khalpey et al., 2008; Mullen-Fortino & O'Brien, 2008; Silvestry, 2008).

One study evaluated patients who underwent CPB procedures for development of ARF. Patients who were identified to be at greater risk for postoperative renal dysfunction included those with heart failure, type I diabetes mellitus, preoperative hyperglycemia (greater than 300 mg/dL), preoperative serum creatinine in the range of 1.4–2.0 mg/dL, and aged 70–95 years. Perioperative factors that increased patient risk included CPB time of at least 3 hours and ventricular dysfunction (Talmor & Lisbon, 2005). These factors result in either renal artery vasoconstriction, hypothermia, atheroembolic disease, or loss of pulsative blood flow (Silvestry, 2008).

Three types of ARF exist, and diagnosis is based on the point of initial renal insult:

- Prerenal: injury occurring before the kidney
- Intrarenal: intrinsic to the kidney
- Postrenal: injury occurring after the kidney

The most common cause of ARF is ATN, a form of intrarenal ARF (Richard, 2001).

ARF results in alterations in electrolyte balance, acid–base and fluid volume status, nitrogenous waste accumulation, and decreased production of erythropoietin. In the majority of cases, an insult occurs, resulting in multiple organ damage and affecting the ability of the kidneys to function appropriately. Management of ARF will vary based on the etiology and the degree of renal injury (MacKusick, 2007).

The predominant cause of ARF in postoperative cardiac surgery patients is ATN. The majority of cases of ATN result in suppression of bone marrow, endocrine disturbance, coagulopathy, and cardiovascular dysfunction as normal homeostasis can no longer be maintained (MacKusick, 2007). Prolonged hypotension and hypovolemic shock are the most common causes of ATN. Renal cellular death begins to occur when MAP falls below 75 mm Hg (Richard, 2001). The extent of the renal damage may be estimated by determining the length of time of renal ischemia, with ischemia of 25 minutes or less generally causing reversible mild injury, ischemia of 40–60 minutes causing damage that will take the kidneys 2 to 3 weeks to recover from, and ischemia lasting longer than 1–1.5 hours causing irreversible damage (Richard, 2001). As ischemia progresses, the renal tubular cells swell and become necrotic (MacKusick, 2007).

Assessment

The patient with ATN will present with oliguria or anuria, elevated BUN and serum creatinine, and isosthenuria (a condition in which urinary osmolality approximates plasma osmolality). Oliguria is generally defined as urinary output less than 400 mL over a period of 24 hours; anuria is defined as urinary output less than 100 mL in 24 hours. Patients should be closely monitored for life-threatening alterations in electrolyte levels.

Frequent laboratory testing will be necessary to monitor serum electrolytes and complete blood count. FVE will develop, and the patient will present with its associated signs and symptoms.

The patient with ATN will progress through the stages of AFR in a relatively predictable pattern. Initiation is the first stage; it is followed by oliguria, diuresis, and then recovery. The last two stages will typically not be managed in the ICU and, therefore, are not within the scope of this chapter. The total length of time from onset of renal damage to recovery can last from months to 1 year.

Initiation Stage

The initiation stage of ARF begins when the renal insult occurs and lasts from a few hours to a few days. Initial signs and symptoms of renal impairment are noticed, and the cause of ARF is investigated. Initial signs and symptoms generally include a decrease in urinary output, crackles, muffled heart sounds, development of a new heart murmur or S_3 gallop, and an increase in body weight indicating FVE (MacKusick, 2007).

Oliguric Stage

Oliguria is a decrease in urinary volume to less than 400 mL/24 hours. The diminished urinary output seen with ATN occurs when shock or dehydration leads to inadequate perfusion of the kidneys. The oliguric stage generally lasts from 1 to 2 weeks (Richard, 2001).

Laboratory data will indicate a decrease in GFR, an increase in serum creatinine and BUN, and an elevation in the electrolytes excreted by the renal system (potassium, sodium, and phosphorus). Laboratory data must be closely monitored, because a frequent cause of death during the oliguric stage is cardiac arrest secondary to hyperkalemia (Richard, 2001).

As azotemia progresses, the patient is at increased risk for developing infection and GI bleeding (Campbell, 2003; Dirkes & Kozlowski, 2003). The mortality rate during the oliguric stage is greater than 50% (Ronco et al., 2002). Approximately half of all patients with ARF do not present with oliguria (MacKusick, 2007).

During the oliguric/anuric stage, the patient needs to be closely monitored for alterations in electrolyte status and prepared for renal replacement therapy to remove waste products and excess fluid, and to return electrolytes to near normal levels.

Treatment

Morbidity and mortality rates are significantly increased in postoperative cardiac surgery patients who develop renal dysfunction (Khalpey et al., 2008). The most essential prevention measure and treatment intervention for ARF is maintaining adequate renal perfusion (Talmor & Lisbon, 2005). Nursing interventions focus on maintaining strict intake and output and monitoring oxygen saturation, vital signs, and fluid volume status. Prevention of further ischemia is necessary to prevent additional renal damage from occurring. Because third spacing and significant diuresis are common following CPB, a fluid challenge will likely be initiated. Urinary output should be maintained at a rate of at least 0.5 mL/kg/hr. The patient's hemodynamic profile must be optimized, and use of nephrotoxic agents should be avoided (Khalpey et al., 2008). If the patient progresses to oliguria or anuria, renal replacement therapy will be required.

Both hemodialysis and CRRT act via the principles of osmosis, diffusion, and filtration. CRRT has the added advantage of being able to slowly and safely provide for ultrafiltration and thereby help remove excess fluid over a slower period of time. This gradual action is beneficial when cardiac performance is compromised and the patient cannot tolerate rapid fluctuations in fluid volume status (Jaski & Miller, 2007). Cardiac failure

intrinsically leads to hemodynamic instability, making CRRT an optimal choice for the postoperative cardiac surgery patient in ARF (DiMuzio, 2008).

While receiving CRRT, the patient must be closely monitored for alterations in fluid and electrolyte status, as well as cardiac, respiratory, GI, and neurologic function. Successful CRRT results in removal of fluid and toxins, clearer breath sounds, improved CO, and stabilization in vital signs (DiMuzio, 2008).

SUMMARY

Caring for postoperative cardiac surgery patients requires extensive knowledge, skill, and sound critical thinking that allow the critical care nurse to perform patient assessment and management in a rapidly changing environment. Life-threatening alterations in fluid and electrolyte balances may be present from previous surgery, previous comorbid conditions, or a combination of these factors.

Nurses caring for postoperative cardiac surgery patients should also be aware of the manifestations of acute renal failure and its treatment options. The primary methods of prevention and treatment of ARF for cardiac surgery patients entail interventions that optimize filling pressures and cardiac output (Talmor & Lisbon, 2005). Patients usually receive diuretic therapy starting on the first postoperative day. This therapy typically continues until the patient's preoperative weight has been reestablished (Mullen-Fortino & O'Brien, 2008). The ICU nurse plays a pivotal role in attaining and maintaining fluid and electrolyte balance and optimizing patient outcomes.

CASE STUDY

A patient with a history of diabetes mellitus, acute coronary syndrome, tobacco use (one pack per day for 25 years), and hypertension with renal failure is admitted with chest pain. An echocardiogram and cardiac catheterization are performed. Their results indicate left ventricular hypokinesis and an ejection fraction of 22% on echocardiogram and three-vessel disease on cardiac catheterization. The patient undergoes three-vessel CABG surgery, with grafts being taken from the left internal mammary artery and saphenous vein. Cardiopulmonary bypass and aortic cross-clamp times are 180 minutes and 124 minutes, respectively. Postoperative ICU admission vital signs are 88/56, HR 112, RR 14, temperature 101.2 °F, CVP 1 mm Hg, and PAOP 6 mm Hg.

Critical Thinking Questions

1. Based on these clinical data, how would you classify the patient's fluid status?
2. Which risk factors are present in association with this patient's condition?
3. What should the initial management include?
4. The patient receives 2 L of crystalloid solution over the next 2 hours with no change being noted in the patient's status. An orientee questions you why the patient's hemodynamic status has not changed. How should you respond?
5. Given this patient's history and condition, which electrolyte imbalance is he at risk for developing?
6. If the electrolyte imbalance identified in Question 5 develops, which ECG tracing findings should the ICU nurse anticipate?

Answers to Critical Thinking Questions

1. This patient's clinical picture is consistent with fluid volume deficit.
2. Risk factors in this patient include fever, intraoperative blood loss, and third spacing of fluid.
3. Management of fluid volume deficit includes expansion of intravascular volume to prevent prolonged hypotension, shock, and their sequelae. There remains ongoing debate regarding use of crystalloids versus colloids for volume repletion. Because crystalloids are typically readily available in the ICU, initial management will likely begin with 0.9% normal saline or lactated Ringer's solution. Because the patient is vasodilated, concomitant administration of norepinephrine may be indicated to avoid the negative sequelae of prolonged hypotension.
4. Approximately 75% of crystalloid volume moves out of the circulating volume/vascular space.
5. This patient is at risk for developing hyperkalemia secondary to hemolysis of red blood cells during bypass, cardioplegia, metabolic acidosis from the history of renal failure, and diabetes.
6. The patient with hyperkalemia may develop peaked T waves. This will be followed by a prolonged PR interval, absent P waves, and eventual degradation of the QRS complex.

SELF-ASSESSMENT QUESTIONS

1. Which of the following is an effect of surgery that can cause alterations in fluid volume in the postoperative cardiac surgery patient?
   a. Increase in colloid osmotic pressure
   b. Decrease in hydrostatic pressure
   c. Impaired renin production
   d. Secretion of cortisol
2. Which of the following is a potential cause of respiratory acidosis in the postoperative cardiac surgery patient?
   a. Reversal of neuromuscular blocking agents
   b. Shivering
   c. Diabetes
   d. Hypotension
3. A patient has the following arterial blood gas results following cardiac surgery: pH 7.54/pCO_2 44/pO_2 83/SaO_2 94%/HCO_3 30. Which of the following electrolyte abnormalities should the ICU nurse anticipate?
   a. Hypokalemia
   b. Hyperchloremia
   c. Hypophosphatemia
   d. Hypermagnesemia
4. A patient who underwent cardiac surgery had excessive intraoperative bleeding, requiring rapid transfusion with multiple units of packed red blood cells. Which of the following acid–base disturbances should the ICU nurse anticipate?
   a. Respiratory alkalosis
   b. Metabolic acidosis
   c. Respiratory alkalosis
   d. Metabolic alkalosis

5. Which of the following ECG changes is associated with a hypomagnesemic state?
   a. Narrowing QRS complex
   b. Progressive lengthening of PR interval
   c. Flattened T wave
   d. Presence of U wave
6. Your postoperative cardiac surgery patient has the following lab results: Na 142 mEq/L; K 4.7 mEq/L; Mg 12.4 mg/dL; Phos 2.8 mg/dL; Ca 9.2 mg/dL. Administration of which of the following is indicated?
   a. 250 mL 5% albumin
   b. 1 mEq/kg sodium bicarbonate
   c. 20 mEq calcium gluconate
   d. Albuterol via nebulizer
7. The patient's wife requests an explanation for the intervention described in Question 6. Your best response is
   a. "To prevent dehydration that can occur with his current condition."
   b. "To correct an associated acid–base imbalance he developed from the results of his blood work."
   c. "To treat any respiratory depression associated with his electrolyte imbalance."
   d. "To prevent wheezing from developing in association with his lab values."
8. A patient's QT intervals are shortening and the T waves are depressed. Which of the following lab values should the ICU nurse anticipate?
   a. K 3.0 mEq/L
   b. Mg 1.4 mg/dL
   c. Phos 5.2 mg/dL
   d. Ca 12.6 mg/dL
9. Which of the following values, if sustained, is indicative of fluid volume deficit?
   a. MAP 55–60 mm Hg
   b. U/O 0.3–0.4/kg/hr
   c. CVP 0–1 mm Hg
   d. Heart rate 110–120 beats/min
10. Which of the following lab results should the ICU nurse anticipate when caring for a patient with hypokalemia?
    a. Phos 4.8 mg/dL
    b. Ca 7.8 mg/dL
    c. Na 151 mEq/L
    d. Mg 1.3 mg/dL

Answers to Self-Assessment Questions

1. d
2. b
3. a
4. d
5. d
6. c
7. c
8. d
9. b
10. d

Clinical Inquiry Box

Question: When should a nurse expect to see postoperative atrial fibrillation in the cardiac surgery patient?

Reference: Scherr, K., Jensen, L., Smith, H., & Kozak, C. (2007). Atrial fibrillation following cardiac surgery: A retrospective cohort series. *Progress in Cardiovascular Nursing, 21*(1), 7–13.

Objective: This study was designed to identify the incidence and time of onset of atrial fibrillation (AF) in the postoperative cardiac surgery patient.

Method: Data were obtained on demographic, preoperative, perioperative, and postoperative risk factors for postoperative AF, documented episodes of AF, and clinical outcomes through a retrospective chart review of 1078 adults who underwent cardiac surgery.

Results: Researchers found that 39.6% of the patients in the study had postoperative atrial fibrillation. Of these individuals, more than half had undergone valve surgery. The peak onset of AF occurred on the second postoperative day. Predictors for AF included greater age, history of AF, combined cardiac valve and coronary artery bypass grafting surgery, and high magnesium levels on the third postoperative day.

Conclusion: Although multiple factors are associated with atrial fibrillation, the nurse should be vigilant in monitoring magnesium levels in cardiac surgery patients and should be aware that AF is most likely to occur on the second postoperative day.

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WEB RESOURCE

Water and electrolyte balance: http://www.youtube.com/watch?v=vvGyHBWcQQU

Chapter 18

Wound Care

Mary Zellinger and Vicki Morelock

INTRODUCTION

Assessment and care of postoperative surgical sites will have a profound impact on patient outcomes after cardiac surgery. Surgical site infections (SSIs) of the sternum and underlying mediastinum occur in 0.4% to 4% of patients who undergo such procedures (Eagle et al., 2004; Engelman et al., 2007). Infections can lead to significant morbidity, warranting an increased length of hospitalization and higher financial costs at best, and patient mortality at worst. Observant practitioners must routinely assess for factors that may potentially slow surgical wound healing, and follow strict and consistent protocols in caring for these incisions. This chapter describes the wound care that is required for the postoperative cardiac surgery patient and explores the pivotal role the ICU nurse plays in preventing potentially fatal complications associated with SSIs.

INCISION SITES

Surgical access options in cardiac surgery patients have greatly increased in the past several years. Midline sternotomy access is still the most common access and is used for patients who are operated on with or without the aid of cardiopulmonary bypass (CPB). The lengths of these incisions can range from 6 to 10 inches. Mini-thoracotomy incisions of approximately 2 inches are also used in cardiac surgery. Port-access and robotic surgeries approach the heart through the left chest wall in the case of coronary artery bypass and through the right chest wall for mitral valve repair or replacement.

In minimally invasive bypass, ports for the left internal mammary artery (LIMA) harvest are placed in the third, fifth, and seventh intercostal spaces (ICSs), with a fourth slightly larger working port (2 to 3 inches) for the anastomosis located in the fourth or fifth ICS. Minimally invasive valve procedures may utilize a mini-right thoracotomy (third ICS for aortic and fourth ICS for mitral valve) or a hemisternotomy (upper for aortic valve procedures and lower for mitral valve procedures) (Rosengart et al., 2008). Each of these procedures has the potential to result in the complication of infection.

CONDUITS

The internal mammary artery (IMA) is an ideal conduit to use for bypass grafting, although other conduits may be used as well. The IMA does not have valves as the vein grafts do, so there is no obstruction to flow. In addition, arteries are more vasoresponsive than veins. The IMA is taken down from the chest wall during on-pump, off-pump, and minimally invasive surgery and does not require a separate incision for removal.

The radial artery is another frequently used conduit. It can be removed without fear of diminishing blood flow to the hand if the ulnar artery is functioning adequately. Removal of the radial artery typically requires a 2- to 4-inch incision. Because of its visibility, it may be easily monitored in the postoperative period.

The gastroepiploic artery is rarely used as a conduit during cardiac surgery because of the high chance of contamination that may occur when the abdominal cavity is open at the same time as the sternum.

The saphenous vein is often removed from the leg when the other arteries are not available or when additional grafts are needed. The saphenous vein may be removed via a 3- to 6-inch incision for quick use in an emergency, or it may be removed using two or three small (1.5 to 2.5 cm) incisions via endoscope (Crouch et al., 1999). The saphenous vein is the most commonly used graft, even though the grafts become occluded in 12% to 27% of patients in the first year, with half of those occlusions occurring within the first month. Annual occlusive rates for saphenous veins range between 2% and 4%, and only 69% of patients will experience a 10- to 12-year period free of reoperations or coronary angioplasty (Vorp, Maul, & Nieponice, 2005).

While saphenous veins are the most commonly utilized grafts, arterial grafts are the preferred conduits. Arterial grafts have better long-term patency, but are short in length, have a small diameter, and have limited availability, resulting in the need for multiple grafts.

RISK FACTORS FOR WOUND COMPLICATIONS

Several factors put patients at greater risk for developing postoperative wound complications. These risk factors can be categorized as preoperative, intraoperative, or postoperative.

Preoperative Risk Factors

Preoperative assessment of risk factors for wound complications is imperative, and early and sustained attention to these risk factors is mandatory. Table 18–1 lists the most common preoperative risk factors.

The presence of diabetes may impede wound healing by leading to a compromised immune system. Both chemotaxis and phagocytosis play a role in the development of a wound infection. If serum glucose levels remain elevated, both processes will be compromised (Turina, Fry, & Polk, 2005). Chemotaxis is the oriented movement toward or away from a chemical stimulus—in this case, the process by which white cells are attracted to the site of an infection. Phagocytosis is the

Table 18–1 Preoperative Risk Factors for Wound Complications

Diabetes
Advanced age
Obesity
Large breast size
COPD (i.e., emphysema)
Urgent or emergent CABG repeat operations
Steroids
Preoperative hospital stay of greater than 5 days
Poor nutrition
Venous impairment
Renal failure
Certain medications
Jaundice
Decreased mobility/activity
Dehydration
Respiratory disease
Infection
Anemia
Smoking
Pain
Decreased immunity

CABG = coronary artery bypass grafting.

Sources: DeBaun, 2007; Paul et al., 2007.

ingestion of bacteria by these white cells. Delayed macrophage introduction and diminished leukocyte migration, which cause a prolonged inflammatory phase, interfere in the wound healing process (Streeter, 2006). Unfortunately, a large number of patients presenting for surgery are unaware of their diabetic status and, therefore, may have an uncontrolled serum glucose level preoperatively.

Advanced age may also diminish wound healing. Evidence suggests that inherent differences in cellular structure and function, for example, may impair tissue repair and regeneration in older patients (Eagle et al., 2004; Pittman, 2007). Comorbidities are more common among the elderly as well, and any of these may affect wound healing (Pittman, 2007).

Obesity is a risk for sternal infections because of the increased force applied to the line of closure in these patients, which affects the quantity, aggregation, and orientation of collagen fibers. In addition, undue pressure on the wound may lead to ischemia of the surrounding tissues (Ridderstolpe, Gill, Granfeldt, Åhlfeldt, & Rutberg, 2001).

COPD and emphysema may present problems because effective wound healing requires adequate oxygenation, hemoglobin for oxygen transport, and adequate tissue perfusion (Ragheb & Buggy, 2004). These conditions may not be present with COPD or emphysema.

Protein-calorie malnutrition and the resultant body composition changes are additional considerations that may delay wound healing. The local ability to supply oxygen to the healing wound process is inhibited by peripheral vascular disease, previous radiation, chronic inflammation, or any combination of these conditions (Streeter, 2006).

Prior VAD insertion and preoperative inotropic support have been identified as risk factors for orthotopic heart transplants (Filsoufi et al., 2007).

Intraoperative Risk Factors

Intraoperative risk factors also affect the potential for postoperative problems related to wound healing. Use of both IMAs is associated with increased chance of infection because these arteries provide the major source of blood supply to the sternum. The removal of the IMA significantly devascularizes the sternal half from which it is taken. Surgical technique and adherence to sterile technique certainly have critical implications for incision and mediastinum status. Other potential offending factors include the number of bypass grafts used, excessive use of electrocautery or bone wax, prolonged operative time, and the need for blood transfusions. The last factor increases the risk of infection incrementally based on the number of units transfused (Banbury, Brizzio, Rajeswaran, Lytle, & Blackstone, 2006; Eagle et al., 2004; Keib & Pelham, 2006).

An increase in the number of coronary artery grafts, which can prolong surgical time, increases the likelihood of infection. Long cardiopulmonary pump runs, long surgical procedure times (greater than 4 hours), and any infractions in sterile technique are all known to increase the risk for infection (Eagle et al., 2004; Haycock, Laser, Keuth, & Montefour, 2005; Keib & Pelham, 2006; Mangram et al., 1999). Hypothermia, which is used for cardiac protection during surgery, needs to be corrected quickly in the immediate postoperative period, as prolonged hypothermia increases the risk for infection. Rewarming can be carried out using warming blankets, fluid warmers, and radiant heat lamps (Streeter, 2006).

In rare cases, periods of ischemia may lead to the development of myocardial or pulmonary edema (Marshall & Barash, 2004). The edematous organ may prohibit closure of the chest wall without causing a pressure tamponade and, subsequently, a significant decrease in cardiac index (the amount of

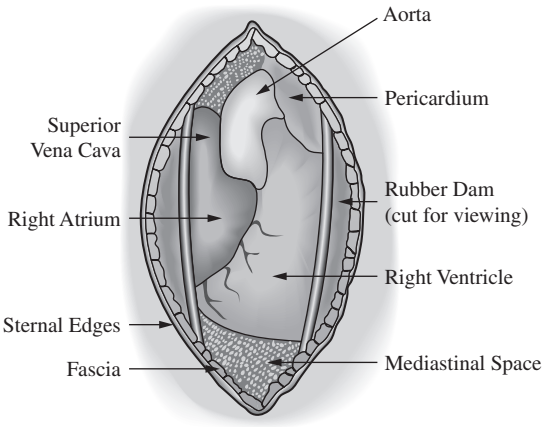

Figure 18–1 Rubber dam.

Source: Adapted from Zellinger, M. & Lienberger, T. (1991). Use of the rubber dam after open heart surgery. *Critical Care Nurse, 11*(8), 24–27.

blood ejected by the heart ÷ body surface area). This complication may warrant leaving the chest cavity open after the procedure for a period of several days to allow for cardiac recovery. During this time, the sternal opening is covered by an impermeable piece of rubber latex called a "rubber dam" (Zellinger & Leinberger, 1991) (see Figure 18–1).

While the mediastinum is left open in such cases, it does not remain exposed. The rubber dam is securely sutured to the skin edges, covered with gauze that has been soaked in povidone-iodine, and then covered with a sterile dressing. The initial dressing change should be performed with the surgeon in attendance so that the site can be assessed and evaluated together with the ICU nurse, thereby preventing unnecessary additional site exposures. All dressing changes are done with strict aseptic technique. The need for and presence of this rubber dam does not increase the risk of sternal infection (Pokorny, Koldjeski, & Swanson, 2003).

Postoperative Risk Factors

In the immediate postoperative period, risk factors for wound infection include early chest reexploration, need for transfusion of more than five units of blood, prolonged mechanical ventilation time, and prolonged periods of cardiopulmonary resuscitation (CPR) (Keib & Pelham, 2006). Other postoperative risk factors include autotransfusion of mediastinal blood, low cardiac output (CO; the amount of blood ejected by the heart each minute), sternal instability, and infections arising from sites other than the sternal incision. For example, a patient with a tracheostomy is at greater risk for poor wound healing because of the close proximity of the surgical incision to the tracheal stoma, which is colonized by bacteria. In such a case, the sternal wound should be protected from the tracheostomy by dressings.

WOUND INFECTIONS CLASSIFICATION

An infection that occurs within 30 days of a surgical procedure is considered an SSI, according to the definition established by the Centers for Disease Control and Prevention (see Box 18–1) (Mangram et al., 1999). Numerous classifications of SSIs have been developed. Most often, they are classified as either superficial or deep, although a more structured approach utilizing numerous classification types has been proposed in the literature (Vlajcic, Zic, Stanec, & Stanec, 2007).

Box 18–1 Criteria for Defining Surgical Site Infections

Superficial Incisional SSI

- Infection occurs within 30 days after surgery
- Involvement of only the skin and subcutaneous tissue of the incision
- Dehiscence of superficial incision
- Stable sternum
- At least one of the following:
  - Purulent drainage from the incision
  - Organisms isolated from an aseptically obtained culture of incision's tissue or fluid
  - Presence of signs and symptoms of infection of incision (e.g., pain, tenderness, swelling, redness, heat)
  - Diagnosis of superficial SSI is made by the physician or mid-level provider

Deep Incisional SSI

- Infection occurs within 30 days after surgery (if no implant was left in place) or 1 year (if an implant was left in place and infection appears to be related to surgery)
- Exposed fascia and muscle of incision
- Exposed bone with a stable or unstable wired sternum
- Exposed necrotic or fractured bone, unstable, heart exposed
- Exposed bone with a stable or unstable wired sternum or exposed necrotic or fractured bone, unstable, heart exposed with the presence of septicemia
- Inflammation of the sternum
- Presence of at least one of the following:
  - Purulent drainage from the deep incision (e.g., mediastinum)
  - Dehiscence of deep incision
  - Deep incision opened by surgeon because of presence of either fever > 38° C, localized pain, or tenderness
  - Presence of an abscess or other signs of deep incision infection
  - An abscess or other sign of infection of the deep incision is discovered
  - Diagnosis of deep SSI is made by the physician or mid-level provider

Notes:

1. If there is presence of both superficial and deep SSIs, it should be reported as a deep incisional SSI.
2. If there is an infection with drainage to an organ/space related to the surgery, it should be reported as a deep incisional SSI.

Organ/Space SSI

- Infection occurs within 30 days after surgery (if no implant was left in place) or 1 year (if an implant was left in place and infection appears to be related to surgery)
- Infection entails any anatomical structure (e.g., organ, space) aside from the incision
- Presence of at least one of the following:
  - Purulent drainage from a drain in the organ/space
  - Organisms isolated from an aseptically obtained culture of incision's tissue or fluid

Sources: Jones et al., 1997; Mangram et al., 1999; Vlajcic, Zic, Stanec, & Stanec, 2007

Superficial Wound Infections

Superficial wound infections are classified as Type 1. These wound infections involve only the skin or subcutaneous tissue around the incision and occur within 30 days of surgery. They may be identified by purulent drainage, isolated organisms upon culture; signs and symptoms of purulent infection such as pain, tenderness, swelling, redness or heat, or purulent drainage; diagnosis of an SSI by the healthcare provider; or any combination of these (Sweene, Lindholm, Borowiec, & Carlsson, 2004).

Sternal Wound Dehiscence

Sternal wound dehiscence is associated with a Type 2 infection. A Type 2 infection is further classified into one of three subcategories:

- Type 2A: a sterile viable bone
- Type 2B: a nonviable bone in the presence of sternal osteitis (inflammation) in the upper two-thirds of the sternum
- Type 2C: a nonviable bone in the presence of sternal osteitis in the lower third of the sternum

A Type 2 sternal wound infection could require any number of interventions, ranging from debridement and rewiring to flap surgery (Rand et al., 1998; Vlajcic et al., 2007).

Mediastinitis

Deep wound infections that occur within 30 days of a procedure and involve the deep soft tissue (i.e., the fascia and muscle) warrant a diagnosis of mediastinitis, which is considered a Type 3 sternal wound infection. Typically, purulent drainage, dehiscence of the surgical site, fever, pain, tenderness at the site, and evidence of infection will be noted. A diagnosis of a deep incisional infection will be made (Horan, Gaynes, Martone, Jarvis, & Emori, 1992). Treatment of a Type 3 sternal wound infection often requires a total sternectomy and an omentum pedicled flap, along with an advanced flap of the pectoralis major (Athanassiadi, Theakos, Benakis, Kakaris, & Skottis, 2007; Vlajcic et al., 2007).

Septicemia

A Type 4 infection is characterized by the presence of a Type 2 or 3 wound infection along with septicemia. Treatment of such an infection involves radical debridement, delayed closure, and aggressive intravenous antibiotic therapy (Vlajcic et al., 2007).

PREVENTION OF SURGICAL SITE INFECTION

Appropriate incisional care must be initiated in the preoperative phase and includes a variety of necessary interventions. Preoperative prevention of SSIs begins with a meticulous handwashing campaign (Haycock et al., 2005; Mangram et al., 1999). In addition, a multitude of preventive strategies (listed in Box 18–2) are incorporated into any cardiac surgical program to decrease the risk of postoperative complications.

Local Collagen-Gentamicin

The use of local collagen-gentamicin has been found to reduce the incidence of sternal wound infections (SWIs) caused by all major clinically important microbiological agents, including coagulase-negative *Staphylococcus* (CoNS). Given the cost of this intervention, centers may want to limit its use to adult cardiac surgery patients who have diabetes, a BMI greater than 25 kg/m^2, or both (Friberg, 2007). If a patient has a suspected infection prior to an elective operation, the infection source and site need to be identified and treated before the patient undergoes surgery. Elective surgery should be postponed until the infection has resolved.

Box 18–2 Strategies for Preventing Surgical Site Infections

- Identify and treat infections before the patient undergoes an elective operation.
- Minimize hair removal at or around the incision site. Use clipping instead of shaving when hair removal is needed.
- Bathe the patient with an antiseptic solution at least the night before surgery.
- Clean the incision site of any gross contaminants and then prep the skin with chlorhexidine gluconate, povidone-iodine, or alcohol-containing products prior to making the incision.
- Use local collagen-gentamicin.
- Use additional fixation wires at the lower sternum.

Sources: Eagle et al., 2004; Friberg, 2007; Friberg, Dahlin, Soderquist, Kallman, & Svedjeholm, 2006; Mangram et al., 1999.

Hair Removal

It is recommended to not remove hair preoperatively unless the hair at or around the incision site might interfere with the surgical procedure. If hair removal is necessary, hair should be removed with electrical clippers rather than the traditional shaving method. Clipping is recommended to be performed immediately prior to the operation (Mangram et al., 1999).

Preoperative Skin Cleansing

Cleansing the patient's skin at least the night before surgery is imperative. In particular, removing gross contaminants by showering or bathing, and then cleansing the skin with a preparation that lowers microbial skin burden, has proven to be effective in lowering the incidence of SSIs (Eagle et al., 2004; Edmiston, Seabrook, Johnson, Paulson, & Beausoleil, 2007; Haycock et al., 2005; Mangram et al., 1999).

Several antimicrobial preparations are currently available: chlorhexidine gluconate (CHG), povidone-iodine, alcohol, and triclosan (an antibacterial chemical). Recent reports in the literature suggest that CHG is the superior product in terms of its ability to remove the microbial burden on the skin and to maintain a greater residual activity hours after the skin is prepared (Mangram et al., 1999). In addition, some promising results have been obtained in using CHG to reduce drug-resistant *Acinetobacter* and methicillin-resistant *Staphylococcus aureus* (MRSA) counts (DeBaun, 2007).

Two methods are currently used to prepare patients' skin:

- The rinse-off method, which uses a CHG 4% solution or presoaked scrub packets
- The no-rinse method, which uses CHG 2% presoaked preparation cloths

To lower the microbial count, it is recommended to apply the antiseptic several times. Studies comparing these two methods have shown some positive results with the no-rinse method (Edmiston et al., 2007; Ryder, 2007). With this method, the patient takes an initial shower to wash any gross contaminants off the skin and to enhance comfort. After a minimum of 1 hour, the no-rinse method cloth is used to prepare the skin. It is recommended that the no-rinse method be used at least once the evening before and again the morning of surgery. It is then followed by the skin preparation in the operating room (DeBaun, 2007).

MRSA Prophylaxis

Staphylococcus aureus is a frequent offender in sternal wound infections. The nares are known to be colonized with *S. aureus* in 20–30% of the healthy population (Mangram et al., 1999). Mupirocin (Bactroban®) ointment, a topical antibiotic, is effective in treating nasal colonization, including some resistant strains of this pathogen. Mupirocin, given intranasally, is recommended preoperatively for all cardiac surgical patients (Engelman et al., 2007). It is administered by the patient for a period up to 5 days to reduce any nasal colonization involving *S. aureus.* Treatment should begin at least 1 day preoperatively and may extend into the postoperative period until the treatment is complete (Cimochowski et al., 2001).

Preoperative Antibiotic Administration

Preoperative antibiotic administration should be performed for all cardiac surgical patients to reduce their risk of postoperative infection (Eagle et al., 2004). This measure reduces the incidence of infection fivefold; *S. aureus* has been identified as the infective organism in more than 50% of all SSIs (Engelman et al., 2007). A cephalosporin is the preferred prophylactic agent of choice for cardiac surgery procedures in populations who do not have a high incidence of MRSA. Data suggest that prophylaxis with glycopeptides, such as vancomycin (Vancocin®), is more effective in preventing infection by methicillin-resistant organisms, but is less effective in countering methicillin-sensitive organisms (Coskun & Aytac, 2006). One or two doses of a glycopeptide in combination with a cephalosporin are reasonable when a healthcare facility has a "high incidence" of MRSA, the patient is especially susceptible to colonization, or the patient is having a prosthetic valve or vascular graft inserted (Engelman et al., 2007).

If a patient is allergic to cephalosporins, a glycopeptide should not be used as a sole agent, as it does not provide any coverage for gram-negative organisms. In such a case, it is recommended that an aminoglycoside be used in addition to the glycopeptide.

Prophylaxis is accomplished by administering one preoperative dose and one postoperative dose (Engelman et al., 2007). Correctly timing the preoperative antibiotic dose is essential so that a bactericidal concentration of the drug is present in the patient's serum and tissues by the time the skin incision is made (Haycock et al., 2005). With the cephalosporins, the dose needs to be administered within 30 minutes of the time the incision is made. It is best accomplished by the anesthesiologist after induction of anesthesia. The surgeon should confirm that the antibiotic dosing has occurred prior to the scalpel being in hand. If the length of the operative procedure exceeds 3 hours, redosing is mandated based on cephalosporin pharmacokinetics. If the combination of a glycopeptide and an aminoglycoside is used, the medications are usually administered over a 60- to 90-minute period (dependent on dosage) (Eagle et al., 2004; Engelman et al., 2007; Mangram et al., 1999).

Glycemic Control

According to a 2004 consensus statement by the American College of Endocrinology (ACE), strict glycemic control entails maintaining a critically ill (e.g., immediate postoperative cardiac surgery) patient's serum glucose at a level up to 110 mg/dL. For patients who are not critically ill (e.g., cardiac surgery patients who are preparing for discharge and are healing at home), the recommendation is that preprandial serum glucose should be 110 mg/dL, up to a maximum of 180 mg/dL (ACE, 2004). Attaining these goals improves healing potential and lessens the risk of mediastinitis (Eagle et al., 2004; Haycock et al., 2005). Appropriate glycemic control is accomplished by assessing the patient preoperatively for elevated HbA_{1c} levels, noting any history of diabetes, and checking for any elevated serum glucose. If a

patient's HbA_{1c} level is greater than 8%, the risk for morbidity and mortality is known to be significantly increased (Peter, Cox, & Evans, 2008). Ideally, achieving adequate glycemic control before the patient's admission for surgery will lower the risk for infection (Furnary et al., 2003).

If patients have elevated serum glucose levels, they should be treated with a continuous intravenous insulin infusion immediately prior to, during, and immediately following surgery in the postanesthesia care unit and the ICU. The literature does not include any studies that have compared protocols for insulin infusions specifically in postoperative cardiac surgery patients. It is suggested that published protocols in the literature be reviewed, and that one be selected that is appropriate for the respective facility. Regardless of which protocol is followed, serum glucose levels should be checked frequently (e.g., every 1 to 2 hours during this time frame) to adjust the insulin infusion. No evidence-based guidelines have been established regarding the ideal frequency for serum glucose testing (Buonocore, 2008). Box 18–3 provides sample orders for ensuring glycemic control.

Once the patient is being advanced on a diet, serum glucose targets should be adjusted to 150 mg/dL as a mean target (110 mg/dL preprandial; 180 mg/dL maximum serum glucose) (Buonocore, 2008; Kazlauskaite & Fogelfeld, 2003). The patient is then transitioned from the intravenous insulin back to the oral agent, subcutaneous injections, or both (Streeter, 2006). If serum glucose levels

Box 18–3 Sample Postoperative Orders to Ensure Adequate Glycemic Control

1. **Treatment for Elevated Blood Glucose (BG > 140 mg/dL):**
   Begin insulin infusion.
   Insulin infusion: 125 units of regular insulin in 250 mL 0.9% normal saline (1 mL of solution = 0.5 unit of insulin).
   Bedside BG monitoring hourly until the patient is within the target range for two consecutive readings; then obtain BG every two hours. If the BG is above or below the targeted range, resume hourly readings. (If using an arterial line specimen, do so consistently while the patient is on the insulin infusion). **When a nondiabetic patient has BG < 100 mg/dL for two consecutive measurements, discontinue insulin infusion.**
   Target BG range on insulin infusion: 80 mg/dL to 110 mg/dL.
   Step 1: Calculate insulin infusion rate: (BG – 60) × 0.04 (multiplier) = units of insulin per hour (× 2 to determine milliliters per hour).
   Adjusting the multiplier:
   BG greater than target range: Increase multiplier by 0.01.
   BG within target range: No change in multiplier.
   BG less than target range: Decrease multiplier by 0.01.
2. **Treatment for Low Blood Glucose (BG < 80 mg/dL):**
   (a) BG = 60–79 mg/dL: Give $D_{50}W$ using this formula: **(100 – BG) × 0.3 = mL $D_{50}W$ IV push.** Adjust the multiplier per protocol.
   (b) BG < 60 mg/dL: Give $D_{50}W$ using this formula: **(100 – BG) × 0.3 = mL $D_{50}W$ IV push.**
   Decrease insulin infusion to 50% of current infusion rate. Recheck BG in 30 minutes:
   BG > 80 mg/dL: Decrease multiplier by 0.01; then return to step 1 of the formula. For example, if you have been using the **Step 1 formula** (BG – 60) × 0.04, you are now going to use the multiplier 0.03 instead of 0.04 (you have dropped the multiplier by 0.01).
   BG = 60–80 mg/dL: Repeat step 2a.
   BG < 60 mg/dL: Notify physician and repeat step 2b.

Source: Emory University Hospital, Atlanta, GA. Cardiac Surgery Postoperative (ICU/PACU) Orders. Used with permission.

remain elevated, consultation with an endocrinologist to attain tighter glycemic control is advisable.

Avoiding Potentially Contaminated Sources

Reestablishing the skin barrier can prevent the onset of superficial infections. Diabetes, obesity, and renal failure are all conditions that can delay wound closure. Dressings are not the only way to protect the incision site, however. ECG wires are known to be a source of infection; disposable leadware is now available and should be considered for SSI prevention (Jancin, 2004). Blood pressure cuffs have also been noted to be infection sources, and disposable cuffs are now available. Individual stethoscopes should be cleansed with alcohol after each individual patient assessment. It is imperative that the nurse assess the incision regularly and protect the incision as it heals.

Postoperative Dressings

Postoperative incisional dressing assessment should be performed upon admission and every 4 hours thereafter until the patient is transferred out of the ICU. To minimize the risk of infection, most cardiac surgical centers opt to keep the initial dressing on the incision for 24 to 48 hours, as per CDC recommendations (Mangram et al., 1999).

If the patient is experiencing a coagulopathy, a small amount of blood or blood-tinged fluid may drain from the incisional site. The dressing may be reinforced unless it becomes saturated, at which time it should be changed using sterile technique.

Evaluation of the Incision Site

Phases of Incision Healing

An incision goes through three major phases as it heals. During the inflammatory phase, after the incision is made, a cascade of clotting and immune responses produces inflammation at the incision site. After incisional closure, neutrophils migrate toward the fibrin clot at the margins of the incision and fill in the incisional space. During the proliferative phase, new capillaries are formed, and fibrin, collagen, and growth factors spread across the wound bed. Basophils (white blood cells) migrate to the incision borders and multiply. During the maturation phase, collagen matrix development furthers wound closure. As keratinization occurs, the skin thickness returns to normal (Streeter, 2006). Recent studies have found that placement of additional fixation wires at the lower sternum, along with prophylaxis with a local collagen-gentamicin, decreases the incidence of deep sternal wound infections (Friberg et al., 2006).

After waiting the designated time stated in the protocol (see Box 18–4 for an example), performing appropriate hand hygiene, and donning clean gloves, the nurse should remove and discard the dressing. Initially, the incision site may appear slightly red around the edges. The edges should be well approximated, with minimal tension evident. The surrounding tissue should display no inflammation, hematoma, swelling, erythema, skin discoloration, or warmth, and it should not cause pain when palpated. Several variables, if present, need to be documented and brought to the attention of the surgeon; the variables indicative of an infection are listed in Table 18–2.

Table 18–2 Variables Indicative of Infection

A nonapproximated incision
Excessive pain and tenderness
Redness, odor, or swelling
Wound breakdown
Exudate (Note the amount—none, minimal, moderate, heavy leakage through the bandage—and type—serous/straw-colored fluid or serosanginous/red fluid, as well as any frank blood or pus [creamy yellow or green].)

Box 18–4 Procedure: Incision Care Following Cardiac Surgery

Purpose: To reduce the potential for nosocomial infections while patients are in the hospital having cardiac surgery.

Equipment

- Chlorhexidine (CHG) 2% preop no rinse cloth
- Towels
- Disposable linen savers or bed pad if scrubs are used
- 4 × 4 sterile dressings
- Tape
- Sterile gloves for dressing change (if incision is less than 48 hours old and the dressing is saturated)
- Sterile water or NS (if incision is less than 48 hours old and the dressing is saturated)

After Surgery

1. Dressings are to remain on for 48 hours. When heavily stained with blood, they are to be changed using sterile technique. **(This procedure is to be done by RNs and LPNs only.)**
   a. Don clean gloves and a mask, remove the old incision dressing, and dispose of it into the proper receptacle; remove the dirty gloves.
   b. Open the 4 × 4 packages and retain them in a sterile manner.
   c. Put on sterile gloves using the proper procedure.
   d. Use chlorhexidine (CHG) 2% preoperative, no-rinse cloth to cleanse the area around the incision. Cleanse the incision gently.
   e. Cover incision with sterile 4 × 4 pads and cover with paper tape.
   f. Write the date, time, and initials on the tape.
2. After 48 hours, dressings are to be removed.
3. If the patient is unable to shower, bathe the incision daily with one package of CHG 2% preoperative, no-rinse disposable cloths. Cleanse the incision gently.
4. If the wound still has some drainage, reapply a dressing using clean technique; the patient may require only spot dressings in areas where oozing is occurring. Change these PRN.
5. Give the patient a CHG shower daily if there is no chest tube (traditional or flexible silastic drain [e.g., Blake]).Use CHG 2% preoperative, no-rinse disposable cloths in the shower.

Source: V. Morelock & M. Zellinger, Emory University Hospital, Atlanta, GA. Procedure: Incision Care, Cardiac Surgery. Used with permission.

Saphenous vein graft infections occur more frequently in obese patients. Drainage of noninfected serosanginous fluid from leg incisions is common. Oftentimes, the drainage results from an underlying hematoma that has liquefied and is draining out of the neighboring skin incision. The presence of erythema, induration, and undue tenderness to palpation indicate infection. In such cases, the patient may require a dilation and curettage procedure, followed by open packing of the wound and administration of antibiotics. If unresolved, these infections can lead to the need for further interventions, including skin grafts, vascular procedures, or even amputations (Alam, Kowalski, & Sample, 1999).

NURSING RESPONSIBILITIES TO ENHANCE WOUND HEALING

The radial artery site is easy to assess because of its visibility. Assessment should always include color, capillary refill time, temperature, and presence of an ulnar artery pulse.

Assessment for an underlying incision hematoma is imperative, as its presence may impede blood flow to the hand and lead to loss of function. Elevation of the arm and affected hand on pillows will help decrease any edema.

The multitude of factors that may potentially affect wound healing should be examined for each individual patient. Providing for optimal wound healing, eliminating any underlying causative or contributory factors, and stimulating positive physiologic factors required for the healing process are essential ICU nursing interventions. Optimizing the patient's nutritional status, including assuring that the patient is consuming a diet with adequate protein and caloric intake, trace metals, and vitamins, is equally essential. Collaboration with a dietitian is recommended.

The ICU nurse should also assess the patient's emotional and psychosocial status. Depression makes it difficult for a patient to be fully compliant with treatment regimens, which may potentially inhibit wound healing. Assuring adequate pain control helps enhance the patient's willingness to be active and ability to adhere to treatment regimens. If the patient has an elevated serum glucose level, close monitoring to maintain tight glycemic control to promote wound healing is required. Renal or liver insufficiency also requires correction, as both of these conditions will impede healing. Finally, ICU nurses must implement measures to optimize perfusion and oxygenation and promote early ambulation, as feasible.

WOUND INFECTION SEQUELAE

When wound infections occur, they can be devastating. Most SSIs are identified on a post-hospitalization basis. They can require frequent outpatient and emergency department (ED) visits, radiology services, lab work, home health services, hospital readmissions for treatment, and possibly, further surgery. In addition, the patient and family will deal with the consequences of a loss in the patient's productivity. In particular, the patient may be out of work for an extended period of time. The patient's functional status may be decreased, such that the individual requires assistance to complete activities of daily living and with transportation to and from the many visits to physicians, clinics, or EDs (Kirkland, Briggs, Trivette, Wilkinson, & Sexton, 1999). The rate of ICU admissions is 60% higher among infected patients than among uninfected patients (Kirkland et al., 1999).

Impact of Postoperative Infection

In an attempt to quantify the impact of postoperative infection, the Agency for Healthcare Research and Quality (AHRQ) reviewed patient safety indicators to identify injuries among patients from 994 hospitals in 28 states. In this study, a total of 7.45 million hospital discharge abstracts were reviewed. The researchers found that failure in the process of care can precipitate postoperative infection, and that infection is associated with an overall increase of 9.58 extra hospital days, $38,000 to $40,000 in excess charges, and a 4.31% mortality rate (Zhan & Miller, 2003).

Reducing the potential for any SSI to occur is a paramount concern with any surgical procedure. Approximately 500,000 SSIs occur in conjunction with the estimated 27 million surgical procedures performed in the United States annually (Barnett, 2007). The Deficit Reduction Act, passed in 2005, allows the Centers for Medicare and Medicaid Services (CMS) to adjust payments downward for patients experiencing hospital-acquired infections; this provision took effect in October 2008. As of October 1, 2008, the Centers for Medicare and Medicaid Services no longer reimburses for hospital-acquired conditions such as SSIs—specifically, mediastinitis after CABG surgery (CMS, 2008) or flap surgery due to an SSI. Hospitals are now paid at the "without compli-

cations rate" when such SSI-related events occur, instead of the "with complications" higher rate that they had been receiving in the past (Barnett, 2007). The impact of this change in billing practice on hospitals' financial status could be considerable.

SSIs affect numerous parties: the patient, insurance companies, medical caregivers, and hospitals. There has been increasing focus on preventing SSIs as one element of the Institute for Healthcare Improvement's (IHI) initiatives in the "Protect 5 Million Lives from Harm" campaign (McCannon, Hackbarth, & Griffin, 2007).

The general trend is toward surgical patients who are increasingly sicker and have more complex comorbidities. Many of these patients are elderly (older than 80 years). When these patients get SSIs, increasing numbers of them are infected with resistant strains of microbes (e.g., MRSA and vancomycin-resistant enterococcus [VRE]).

WOUND INFECTION PREDICTION

The CDC's National Nosocomial Infection Surveillance (NNIS) system predicts the risk of SSI based on three factors: length of surgery, wound class, and the patient's American Society of Anesthesiology (ASA) score (Hollenbeak et al., 2000). This system has not been adapted specifically for cardiac surgery, however.

The first scale to predict surgical wound infections in CABG patients was developed in 1998 (Hussey, Leeper, & Hynan, 1998; Troutman, Hussey, Hynan, & Lucisano, 2001). This scale, which is known as the Sternal Wound Infection Predictor Scale (SWIPS), consists of weighted predictors related to the preoperative, intraoperative, and postoperative phases of care (see Table 18–3).

Validation of other risk score assessment tools for mortality such as the EuroSCORE (Nashef et al., 1999) and the Society of Thoracic Surgeons risk scores (Fowler et al., 2005) has suggested that these two newer scoring systems outperform the NNIS risk index (Paul et al., 2007). The Society of Thoracic Surgeons (STS) has recently developed a score that predicts the risk for infection following CABG, which includes both preoperative and intraoperative scores and takes several variables into account.

Table 18–3 Sternal Wound Infection Predictor Scale (SWIPS)

Variable	Weight
Preoperative	
Smoking	9
Diabetes mellitus	
IDDM	7
NIDDM	5
COPD	8
Preoperative ICU stay	4
Obesity (> 30 kg/m^2)	4
Advanced age (> 70 years)	3
Sex (male)	1
Impaired immune response	8
Intraoperative	
Bilateral IMA	6
Single IMA	3
Long operative time (> 4 hr)	7
Reexploration for bleeding	6
Long cardiopulmonary bypass time (> 2 hr)	6
Postoperative	
Hypoperfusion/hypotension	8
Ventilator support (> 48 hr)	6
Pharmacologic support	
Dopamine/dobutamine only	2
All others	6
Postoperative CPR	7
Hypoxemia	5
Banked blood transfusions	3

CPR = cardiopulmonary resuscitation; IDDM = insulin-dependent diabetes mellitus; IMA = internal mammary artery; NIDDM = non-insulin-dependent diabetes mellitus.

Source: Hussey, Leeper, & Hynan, 1998.

Currently, mediastinitis occurs in 0.04% to 4.0% of cardiac surgical patients, with more than 50% of these infections involving *Staphylococcus aureus* and *epidermidis*. The 1-year mortality rate of those patients with deep sternal infections is as high as 22%—far higher than the 0.6% mortality rate in patients who do not develop infections (Hollenbeak et al., 2000).

MANAGEMENT OF WOUND INFECTIONS

Sternal Wound Infections

Sternal wound infections may be superficial, involving only the skin and subcutaneous fat, or they may be deep, involving the sternum and underlying structures. Superficial infections are characterized by drainage from the wound and local inflammation, even as the underlying sternum remains stable (see Box 18–1). In this instance, removal of the overlying skin sutures, culture of the drainage, administration of antibiotics, and local dressings are often effective interventions. These wounds respond well to vacuum-assisted closure therapy and may heal without any further surgery (Agarwal et al., 2005). Reconstructive surgery can be avoided in clinically stable patients with the use of vacuum-assisted closure (Chen et al., 2008).

Mediastinitis

Bacterial mediastinitis starts when the invasion of a pathogen causes an inflammatory response. The invading bacteria proliferate, and the body forms a thick layer of fibrin in an attempt to encapsulate the foreign agents. An area of dead space forms underneath the sternum as the infection expands through sinus tracts that have formed. The patient develops fever, and the systemic inflammatory response causes production of leukocytes and proinflammatory mediators. In recent years, CoNS has been identified as the most common causative agent of SWI (Friberg, 2007).

Mediastinitis can develop as early as 7 to 10 days following a cardiac surgical procedure. Patients have often been discharged home before any sign of this infection occurs. Oftentimes, the first sign is significant serous drainage that appears 4 to 5 days postoperatively. Patients experience fever, chills, pain, and leukocytosis within 2 to 5 days after the onset of infection. Erythema may form on the outside borders of the incision and is often first seen at the xiphoid process. Occasionally, a section of the incision may dehisce and purulent drainage will exude from the site.

More commonly, mediastinitis becomes evident later in the postoperative course, usually within 30 days after surgery. Patients often develop sternal pain, become lethargic, and demonstrate unwillingness to do many activities that they were doing previously. The incision then begins to drain purulent fluid and will separate. Upon assessment, the nurse often finds that the sternum is unstable, with the borders rubbing against each other. Pain will be worse with respiration. Fever, chills, and leukocytosis are evident.

Treatment of mediastinitis depends on the stage of the infectious process at the time of diagnosis. If identified early, the sternum is not destroyed—success may be achieved with prompt surgical intervention, debridement of the sternal edges, copious irrigation of the mediastinum, placement of retrosternal irrigation and drainage catheters, rewiring of the sternum, and closure of the fascia and skin. Appropriate intravenous antibiotic therapy is given for a minimum of 7 days. The results of a gram stain are utilized to identify the appropriate antibiotic to infuse through an irrigating catheter, with the fluid being directed to exit via drainage catheters. The irri-

gation continues for 3 to 5 days, until the drainage is sterile as confirmed by culture. Although frequently successful, this treatment method can have serious complications—for example, erosion of the catheters into mediastinal structures and systemic toxicity from absorption of the irrigating antibiotic. For these reasons, this procedure is reserved for specific groups of patients (Lemmer, Richenbacher, & Vlahakes, 2003).

More longstanding, advanced infections are associated with large amounts of suppurative fluid in the mediastinum, loss of integrity of the sternum, and diffuse cellulitis of the skin and subcutaneous tissue. Patients with such infections may require opening of the sternum and debridement of necrotic tissue, exposure and draining of the mediastinum, and packing of the wound with moist gauze. Vacuum-assisted closure therapy can be used as a bridge between debridement and closure of the wound. It can assist in decreasing overall wound edema, reduce bacterial counts in the wound, and reduce the time to closing the wound (Agrawal et al., 2005). After control of the infection is achieved and a healthy-appearing bed of granulation tissue forms, secondary closure is performed with or without a muscle flap (Vlajcic et al., 2007).

The most frequently used approach to treating serious mediastinitis is a single-stage procedure in which radical debridement of the sternum and cartilage is performed with advanced muscle flaps, using the pectoralis major and/or rectus muscles (Eagle et al., 2004). Depending on the degree of sternal resection required, the remaining bone tissue may or may not be approximated. Soft silastic drains are placed beneath the muscle flaps and connected to gentle suction. Often performed by a plastic surgeon, this procedure may be associated with decreased morbidity and mortality and a decreased length of hospital stay. The patient may be discharged home with the drains in place. The drains remain in place until the daily drainage volume becomes small; they may then be removed in the physician's office. Early aggressive use of muscle flaps in serious mediastinitis is considered the optimal approach (Vlajcic et al., 2007).

Several long-term complications are associated with mediastinitis. Notably, patients have a significant increase in mortality during the first year and subsequent 4 years. The potential for other nosocomial infections, including systemic infections, is increased as well. Patients may develop sepsis and organ system failure. Identifying mediastinitis early allows for earlier treatment and is associated with a better prognosis (Vlajcic et al., 2007).

For patients who have a relatively uneventful postoperative course, discharge may occur on the third or fourth postoperative day. Many infections do not become evident until after the patient has been discharged, which makes early diagnosis of sternal infection and mediastinitis after cardiac surgery difficult. In some patients, fever, leukocytosis, and a positive blood culture will be the first manifestations of a hidden infection that will become obvious only later. The most common early sign is fluid drainage from the wound; sternal instability usually develops subsequently.

Clear and thorough patient education reviewing the appearance of a normally healing incision is of utmost importance in recognizing postoperative wound infections. The patient and family members must be instructed to frequently observe the incision for any changes in status and to call the surgeon's office if changes or questions arise (see Box 18–5). Given the trend toward earlier hospital discharge following cardiac surgery, fewer SSIs will be detected prior to patient discharge. Without careful supervision and intervention, the physical and financial costs of these infections will increase.

Box 18–5 Patient/Family Discharge Education Regarding Incision Care

Emphasize the following points when explaining wound care at home:

- Shower daily with soap and water.
- Avoid sitting in bathtub.
- A dressing is needed only if drainage is present.
- Remove any dressings applied during hospitalization on the day after going home.
- Inspect the wound daily using a mirror. If you see any redness, irritation, swelling, tenderness, or unusual drainage, contact the surgeon or surgeon on call.

Optimal Patient Outcomes

- Patient can perform appropriate incision care.
- Patient has no signs and symptoms of infection.
- Patient can list signs and symptoms of infection.
- Patient modifies lifestyle to reduce risk factors that may impede wound healing.

SUMMARY

Sternal wound infections occur in a small percentage of patients who undergo cardiac surgery. There is a high associated cost in terms of morbidity, mortality, and length of hospitalization, and financial costs if they develop. A number of predictive variables have been identified that put the patient at greater risk for development of a sternal wound infection, and a number of preventive strategies must be implemented to avoid development of this potentially catastrophic complication. Although a sternal wound infection is not likely to develop while the patient is in the ICU postoperatively, initiation of preventive measures must begin while the patient is in the early phase of recovery. The ICU nurse has a pivotal role in preventing sternal wound infections and beginning the essential patient and family education that must be accomplished to help ensure optimal postoperative outcomes are attained.

CASE STUDY

A 79-year-old patient with a history of smoking one pack of cigarettes per day, diabetes, obesity, and inactivity due to peripheral vascular disease was admitted to the emergency department with chest pain. The patient was taken for a cardiac catheterization, where it was determined that emergency cardiac surgery was necessary.

The surgeon performed a quadruple coronary artery bypass. Because of the patient's peripheral vascular disease, both internal mammary arteries were used as conduits. The surgery lasted 4 hours, after which time the patient was admitted to the ICU on epinephrine and milrinone (Primacor®) infusions to augment cardiac output. Initial arterial blood gas results were as follows: pH 7.23, pCO_2 51 mm Hg, pO_2 89 mm Hg. Copious endotracheal secretions were noted. The initial chest tube drainage was 350 mL for the first hour and 300 mL for the second hour.

Critical Thinking Questions

1. What were some of the preoperative risk factors present for development of a sternal wound infection?
2. Which predictive factors for the development of wound infection are present postoperatively?
3. How frequently should the incision dressing be changed in the postoperative period?
4. What are four preventive strategies that the ICU nurse should incorporate into the care plan to prevent postoperative wound contamination?

Answers to Critical Thinking Questions

1. Diabetes, advanced age, obesity, tobacco (possibility of COPD) and emergent CABG procedure.
2. Low cardiac output and use of both internal mammary arteries. Use of both IMAs is associated with an increased risk of infection because these arteries provide the major source of blood supply to the sternum.
3. If a rubber dam is present, the initial dressing change should be performed with the surgeon in attendance so that the site can be assessed and evaluated by both physician and nurse simultaneously, thereby avoiding unnecessary site exposures. For all open chest wounds sterile dressing changes are performed every 24 hours. If the patient has a closed chest incision, the initial dressing change occurs 48 hours postoperatively. If needed the dressing is reapplied and changed every 24 hours. All dressing changes must be done with strict aseptic technique.
4. (1) Postoperative incisional dressing assessments should be performed upon the patient's admission to the ICU and every 4 hours thereafter until the patient transfers out of the ICU. (2) To minimize the risk of infection, keep the initial dressing on the incision for 24 to 48 hours, as per CDC recommendations. (3) The multitude of factors affecting wound healing should be examined for each individual patient. Providing for optimal wound healing, eliminating any underlying causative and/or contributory factors, and stimulating the positive physiologic factors required for the healing process are essential nursing interventions. (4) Optimizing the patient's nutritional status, assuring that the patient is ingesting a diet with adequate protein and caloric intake, trace metals, and vitamins is equally essential.

SELF-ASSESSMENT QUESTIONS

1. Preoperative risk factors for mediastinitis include
   a. Asian race, Marfan syndrome, and advanced age.
   b. advanced age, chest pain, and emphysema.
   c. emphysema, advanced age, and male gender.
   d. advanced age, emphysema, and infection.
2. The most common infectious organism found in mediastinitis is
   a. *Staphylococcus aureus.*
   b. *Staphylococcus epidermidis.*
   c. *Enterococcus.*
   d. gram-negative *Serratia.*
3. Postoperatively, a sterile dressing should be kept on
   a. 72 to 96 hours.
   b. 8 to 12 hours.
   c. 12 to 24 hours.
   d. 24 to 48 hours.

4. Discharge education should incorporate
   a. characteristics of a normal and abnormal healing incision.
   b. self-treatment procedures for infection.
   c. normal lab results from hematological lab studies.
   d. referral to online resources.
5. An initial indication of infection may include
   a. serous drainage, increased chest tube drainage, and bradycardia.
   b. serous drainage, fever, and pain.
   c. leukocytosis, bradycardia, and hypertension.
   d. hypertension, decreased chest tube drainage, and serous drainage.
6. A prophylactic antibiotic regimen should consider which of the following measures?
   a. Adding a glycopeptide to the cephalosporin, such as vancomycin, for two to three doses postoperatively is appropriate in patients who have received a prosthetic valve or vascular graft.
   b. Routine postoperative antibiotic regimen should be no longer than 72 hours.
   c. A glycopeptide, such as vancomycin, provides adequate broad-spectrum coverage and can be used as a solo prophylactic agent.
   d. The initial dose of antibiotic should be timed to provide a bactericidal concentration in the tissues and the serum before the incision is made, and is usually given 30 to 60 minutes prior to incision.
7. All of the following factors promote wound healing *except*
   a. renal failure or liver failure.
   b. adequate pain control to promote ambulation and respiratory exercises.
   c. a diet that contains adequate protein, caloric intake, trace metals, and vitamins.
   d. strict glycemic control.
8. Continuous intravenous insulin infusions should be used perioperatively
   a. to maintain serum glucose less than 180mg/dL.
   b. in all patients who are hyperglycemic, whether they have a history of diabetes or not.
   c. while adjusting the infusion by checking serum glucoses every 3 to 4 hours and following the established protocol of the institution.
   d. but lower doses may be needed because patients experience an insulin sensitivity.
9. All of the following statements about mediastinitis are true *except*
   a. it may begin with serous drainage a few days postoperatively and can progress to purulent drainage, and a portion of the wound may dehisce.
   b. bilateral IMAs, prolonged ICU stay, decreased cardiac output, and autotransfusion are all factors that lessen the patient's risk for infection.
   c. mediastinitis is a deep sternal incisional infection that affects fascia and bone, occurring within 5 to 30 days postoperatively.
   d. diabetes, renal failure, increased age, large breast size, and obesity all increase a patient's risk of developing mediastinitis.

Answers to Self-Assessment Questions

1. d
2. a
3. d
4. a
5. b
6. d
7. a
8. b
9. b

Clinical Inquiry Box

Question: What is the incidence of sternal wound infections?

Reference: Strecker, T., Rösch, J., Horch, R. E., Weyand, M., & Kneser, U. (2007). Sternal wound infections following cardiac surgery: Risk factor analysis and interdisciplinary treatment. *Heart Surgery Forum, 10*(5), E366–E371.

Objective: To evaluate the outcomes of interventions utilized in the treatment of complicated median sternotomy wounds.

Methods: In a retrospective review covering a three-year period, 3016 consecutive open-heart surgery patients were evaluated. The majority of patients (65.6%) underwent coronary artery bypass grafting (CABG). The remainder received surgery for artificial heart implantation, cardiac transplantation, aorta reconstruction or replacement, cardiac valve replacement, or combined CABG and valve replacement.

Results: Of the 3016 subjects, only 2.1% developed sternal wound infections. Treatment of the infections consisted of debridement, irrigation, and rewiring in 56 cases. Vacuum-assisted closure therapy was utilized in 34 patients, and 19 patients eventually required reconstructive surgery with either rectus abdominis or pectoralis major flaps. Significant risk factors for the development of a surgical site infection included diabetes mellitus, rethoracotomy, duration of operation, and, interestingly, the time of operation (morning versus afternoon).

Discussion: In this study, a decreased risk of infection was associated with first position on the OR schedule. Although vacuum-assisted closure therapy is useful in preventing reconstructive surgery, a small percentage of patients will require flap surgery. The type of flap will depend on the location of the infection and the availability of muscle for the closure of the wound. Nurses should be aware that hospitals in the future might decide to schedule cardiac surgery only as the first case, especially for those patients at the highest risk for surgical site infection.

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WEB RESOURCES

Centers for Medicare and Medicaid Services (CMS): http://www.cms.hhs.gov/

Deficit Reduction Act: http://www.cbo.gov/ftpdocs/70xx/doc7028/s1932conf.pdf

Institute for Healthcare Improvement: http://www.ihi.org/ihi

Guideline for the Prevention of Surgical Site Infection, 1999: http://www.cdc.gov/ncidod/dhqp/gl_surgicalsite.html

Chapter 19

Bridge to Transplant and Cardiac Transplantation

Rachel S. Ball, Kathy Halabicky, Laurie Hartman, Erin Lindstrom, and Tracey Romans

INTRODUCTION

The American Heart Association (AHA) estimates that nearly 5 million Americans are currently living with heart failure (HF); approximately 550,000 new cases are diagnosed in the United States each year (AHA, 2007) and more than 287,000 people die from HF annually (AHA, 2008). Over the past 10 years, medical advances have greatly increased treatment options for people living with heart failure. In particular, mechanical circulatory support technology has emerged as a life-saving option for patients with acute and chronic heart failure that is not amenable to maximal therapy.

In the United States, 257 facilities currently have the ability to perform heart transplants. The Organ Procurement and Transplantation Network (OPTN) reported that 2684 patients were awaiting such transplants in March 2008. In 2007, 2030 heart transplants were performed in this country. As of December 5, 2008, there were 2705 waitlisted candidates for a heart transplant (OPTN, 2008). An estimated 10% to 15% of these patients will die each year while awaiting a heart transplant (McCalmont & Ohler, 2008).

This chapter reviews the management of heart failure as the patient moves through the trajectory of illness toward transplantation. The role of the critical care nurse is discussed during the various phases of illness through the transplant process and beyond.

MANAGEMENT OF HEART FAILURE

When patients are diagnosed with HF, they typically are started on diuretics or ultrafiltration techniques to assist with volume overload. When their clinical status declines and these measures are no longer effective, these patients may be admitted to the ICU in cardiogenic shock requiring inotropic (e.g., dobutamine [Dobutrex®], dopamine [Intropin®], milrinone [Primacor®], inamrinone [Inocor®]) or mechanical support (e.g., ventricular assist device [VAD], intra-aortic balloon pump [IABP]). If the patient requires ongoing hospitalizations for HF and management strategies are not beneficial, consideration should be given for heart transplantation (McCalmont & Ohler, 2008). IABP therapy and pharmacologic interventions for HF are discussed in Chapters 10 and 12, respectively.

CRITERIA FOR HEART TRANSPLANTATION

A patient may become a candidate for a heart transplant based on specific criteria. The patient may have terminal HF that has not responded to medical therapy or cardiomyopathy (ischemic, nonischemic, idiopathic, or valvular). In addition, the predicted 1-year survival rate for the patient should be 50%. Another proposed physiologic criterion

is a maximum oxygen consumption of less than 14 mL/kg/min with associated restrictions on activities of daily living (if the patient is not receiving beta-blocker therapy) or less than 12 mL/kg/min if the patient is receiving beta-blocker therapy (Colucci & Piña, 2008). Other criteria include labile fluid balance and renal function that are not related to lack of adherence with the medical regimen. The potential candidate must be emotionally stable and have sources of social support (McCalmont & Ohler, 2008).

As their condition continues to deteriorate, patients with HF will be evaluated for a match with the criteria for heart transplantation. Both absolute and relative contraindications to heart transplant have been reported in the literature.

Relative contraindications to heart transplant include age greater than 65 years, reversible pulmonary hypertension (pulmonary vascular resistance [PVR] less than 400 dyne/sec/cm^{-5} while receiving vasodilator therapy), reversible hepatic or renal dysfunction, HIV positive status, asymptomatic peripheral vascular disease (PVD) or cerebrovascular disease (CVD), insulin-dependent diabetes mellitus requiring high doses of insulin, receiving therapy for a psychiatric disorder, body mass index (BMI) greater than 35 kg/m^2, and cachexia (BMI < 20 kg/m^2). These last two conditions are associated with poor postoperative healing and recovery (McCalmont & Ohler, 2008). HIV positive patients are evaluated based on their overall health status, anticipated long-term survival, and presence of infectious disease, and must have a stable $CD4^+$ count to be considered viable candidates for heart transplant (Pelletier et al., 2004).

Absolute contraindications to heart transplant include advancing age, severe pulmonary hypertension (PVR ≥ 400 dyne/sec/cm^{-5}), irreversible hepatic dysfunction (i.e., cirrhosis, bilirubin > 2.5 mg/dL, or transaminase levels higher than twice the normal values), irreversible renal dysfunction (i.e., creatinine > 3 mg/dL, glomerular filtration rate < 40 mL/min), severe PVD or CVD, uncontrolled diabetes/poor glycemic control (i.e., HbA_{1c} > 7.5%), active peptic ulcer disease, amyloidosis, current or recurrent diverticulitis, severe mental illness or psychosocial instability, demonstrated noncompliance with medical regimens, no social support, and active use of alcohol, drugs, or tobacco (McCalmont & Ohler, 2008).

Patients must be evaluated for their degree of renal dysfunction, as anti-rejection medications (especially calcineurin inhibitors [CNIs], discussed later in this chapter) are nephrotoxic. Individuals with severe pulmonary hypertension may develop right heart failure, which is associated with immediate death following transplant because the donor heart will not pump effectively. Peptic ulcer disease is a contraindication because the steroid therapy required as part of the transplant process may impair postoperative healing. Patients with hepatic failure are not candidates for transplant because many of the post-transplant medications are hepatotoxic and these individuals have a higher risk of developing coagulopathies. Patients with amyloidosis are at greater risk for post-transplant recurrence, as their disease may spread to other organs. Persons with severe mental illness or a history of noncompliance with medical regimens are at risk for rejection if they do not consistently follow the required anti-rejection therapy regimen (McCalmont & Ohler, 2008).

MECHANICAL CIRCULATORY SUPPORT

Because the number of transplant candidates exceeds the number of available organs, leading to protracted times spent on the waitlist, a patient's clinical status will likely decline while awaiting a new heart, and medications may become ineffective. Patients will then be evaluated for mechanical circulatory support as the wait continues; this care is termed "bridge to transplant."

Mechanical circulatory support has three primary functions: bridge to transplant, bridge to recovery, and destination therapy. Short-term, temporary devices are often used as a bridge to recovery in the setting of acute cardiogenic shock or cardiopulmonary arrest. Under these circumstances, circulatory assistance provides immediate hemodynamic support, restoring blood flow to vital organs while decompressing the heart, thereby helping the patient avoid pulmonary edema and minimizing cardiac workload so as to maximize the individual's chances of recovery.

Types of Short-Term Assist Devices

Examples of short-term assist devices commonly used in emergent situations include Abiomed pumps (Danvers, Massachusetts), TandemHeart PTVA® (CardiacAssist, Inc.), and extracorporeal membrane oxygenation (ECMO). TandemHeart PTVA® and ECMO are particularly useful in the setting of acute cardiogenic failure because of the relative ease of their placement if experienced personnel are available. All of these short-term devices require anticoagulation, generally with heparin. Figures 19–1, 19–2, and 19–3 show a left ventricular assist device (LVAD), right ventricular assist device (RVAD), and a biventricular assist device (BiVAD), respectively.

Abiomed Pumps

Abiomed produces two blood pumps, called the BVS 5000 and the AB 5000, that are

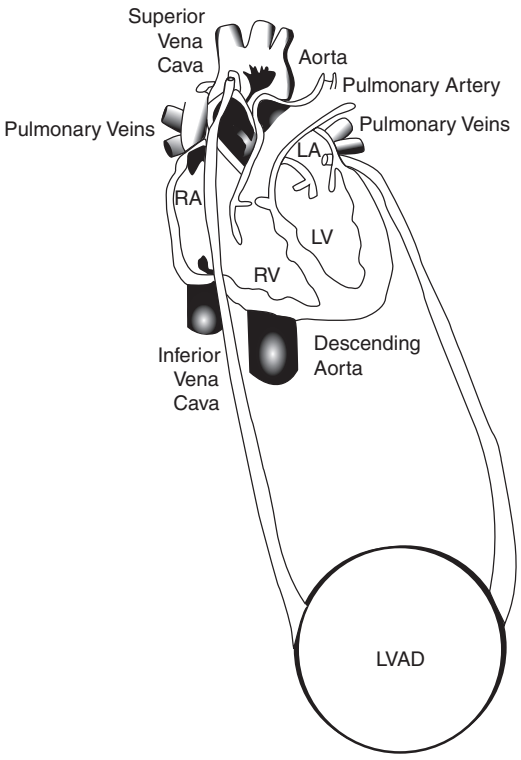


Figure 19–1 Left ventricular assist device.

Source: Illustrated by Lydia Lemmond

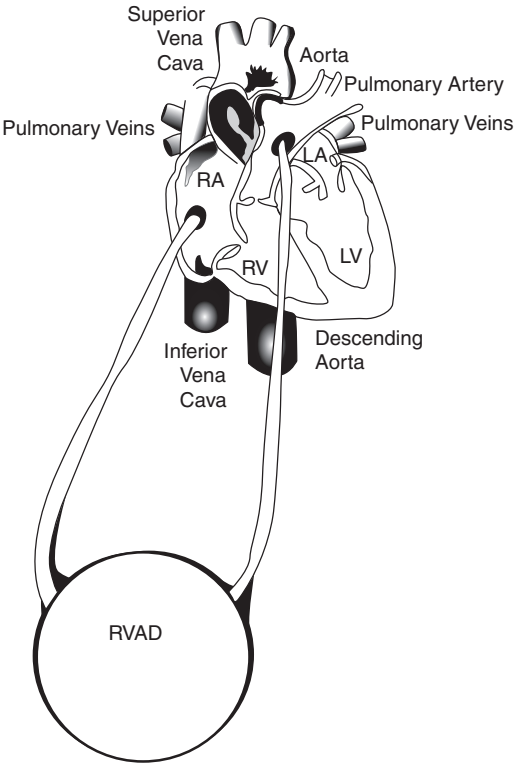


Figure 19–2 Right ventricular assist device.

Source: Illustrated by Lydia Lemmond

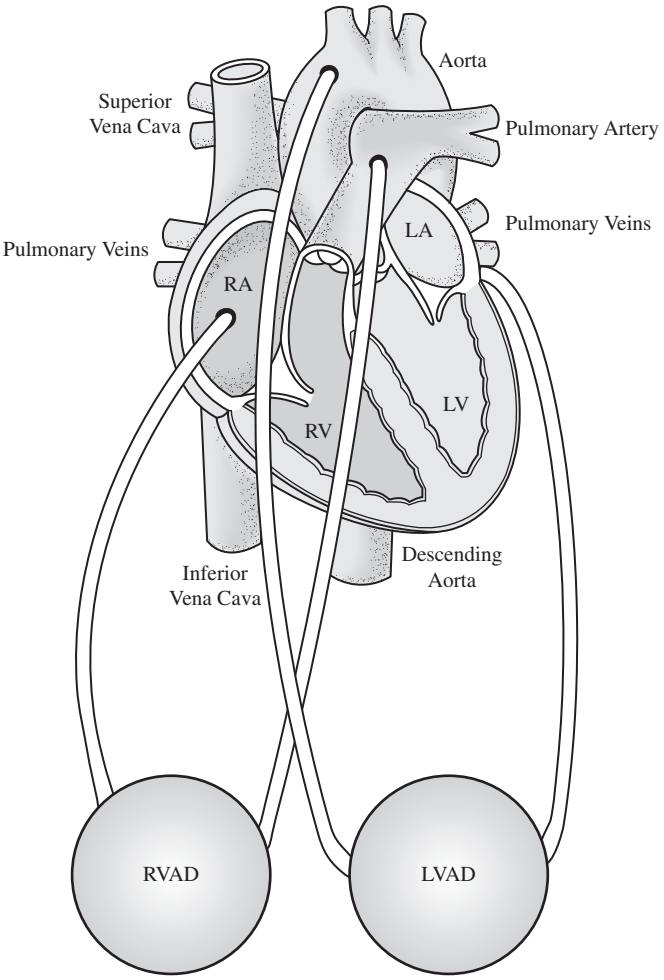
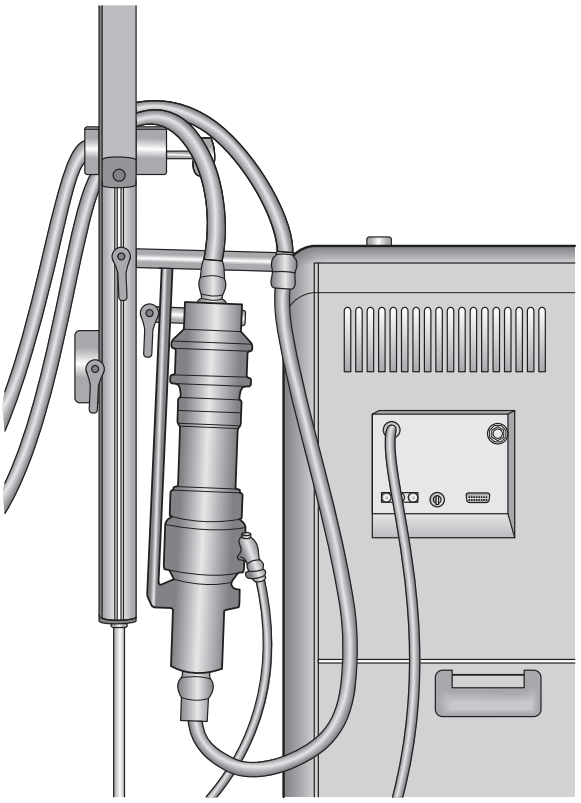


Figure 19–3 Biventricular assist device.
Source: Illustrated by Lydia Lemmond

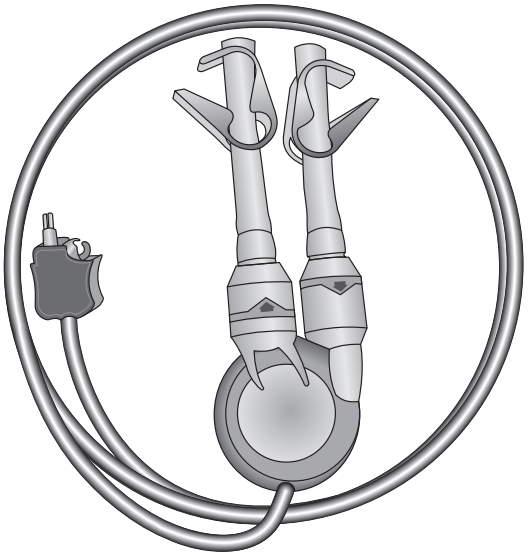
designed for temporary mechanical circulatory support (see Figure 19–4). These pumps can be used for left, right, or biventricular support. Both models require sternotomy for direct access to and cannulation of the heart and great vessels. The BVS 5000 consists of a pump with an atrial chamber that fills with blood through gravity-assisted drainage, and a ventricular chamber that returns blood to the body pneumatically (utilizing movement of compressed air). The chambers are separated by two trileaflet valves. This pump can produce blood flow ("cardiac output") at a rate as high as 5 L/min. Abiomed's newer pump, the AB 5000, has improved durability and shorter tubing, which improves patient mobility, allowing for rehabilitation and ambulation.

TandemHeart PTVA

The TandemHeart PTVA also offers temporary circulatory assistance. In contrast to the Abiomed pumps, this device utilizes a centrifugal pump and is inserted percutaneously (see Figure 19–5). Under fluoroscopic guidance in the cardiac catheterization laboratory,



(A)



(B)

Figure 19–4 Two Abiomed blood pumps—(A) BVS 5000 and (B) AB 5000.

Source: Illustrated by James R. Perron

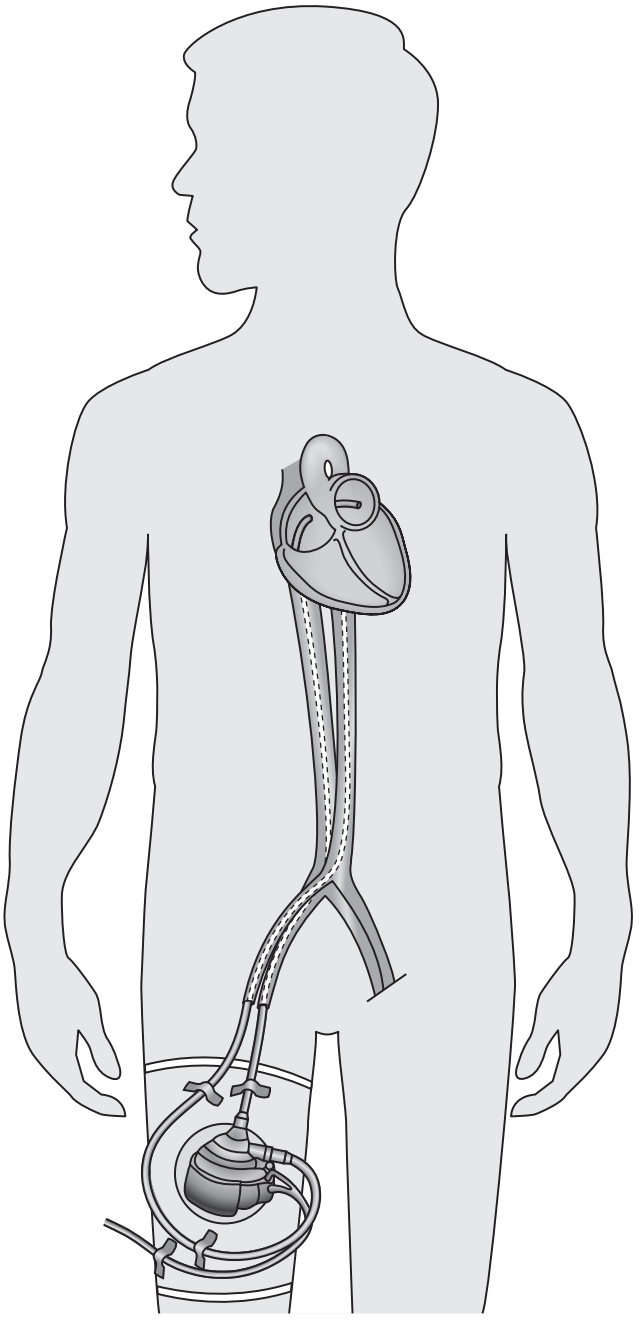


Figure 19–5 The TandemHeart PTVA—this device utilizes a centrifugal pump and is inserted percutaneously.

Source: Illustrated by James R. Perron

a drainage cannula is inserted into the femoral vein up the inferior vena cava, into the right atrium, across the inter-atrial septum, and into the left atrium. The pump withdraws oxygenated blood from the left atrium, propels it via a magnetically driven, six-bladed impeller through the outflow port, and returns it to the femoral artery via an arterial cannula. The pump is capable of delivering blood flow at a rate as high as 4 L/min. The TandemHeart support has been used successfully in postcardiotomy cardiogenic shock patients (those who have developed HF as a result of heart surgery or a heart attack) and as a bridge to a definitive therapy. It can be readily removed if native function returns.

Extracorporeal Membrane Oxygenation

ECMO is a technique of partial cardiopulmonary bypass (CPB) that was initially developed to treat reversible neonatal respiratory failure. The equipment typically used for standard CPB in open heart surgery has been modified to reduce hemolysis, thrombus formation, and risk of air embolus. ECMO can provide support for days or weeks. With this technique, blood is continuously withdrawn from any large central vein and pumped into a gas exchanger that oxygenates hemoglobin and removes carbon dioxide. The oxygenated blood is then pumped into any large artery (see Figure 19–6). The heart and lungs are bypassed, providing both hemodynamic and respiratory support. The blood oxygenation provided by this method is a distinct advantage of ECMO.

Short-Term Mechanical Circulatory Support

Following placement of a short-term circulatory assist device (e.g., a VAD) and a period of hemodynamic stability, the patient's heart function and need for continued support are assessed. Some patients will regain some or most of their previous cardiac function, thus allowing for the removal of the assist device. Patients with acute viral myocarditis, for example, often experience improved cardiac function once the initial inflammatory processes within the cardiac muscle have resolved. Many patients, however, have chronic irreversible HF such that long-term mechanical support, transplant, or both will be necessary.

Recovery of heart function is assessed by briefly decreasing the amount of support provided by the device (e.g., decreasing device blood flow to 2 L/min) and monitoring the patient's hemodynamic parameters. If this brief trial is well tolerated, a surface echocardiogram is obtained during a trial of decreased support to more accurately assess valve and ventricular function. Depending on the results of these trials, plans are made for device removal followed by appropriate medical therapy if there has been adequate recovery of cardiac function, or for transition to a longer-term implanted device, heart transplant, or both if poor cardiac function persists.

Long-Term Mechanical Circulatory Support

Longer-term mechanical circulatory support is accomplished with the class of devices generally termed VADs. VADs are implanted mechanical pumps that are used in patients with end-stage heart disease. VADs assist the weakened heart by pumping blood throughout the body. They were originally designed to stabilize HF patients until a donor heart became available, a strategy referred to as "bridge to transplant." Alternatively, they may be used in patients who are not candidates for or have declined heart transplant, in which case their use is termed "destination therapy."

Research supports the use of VADs as destination therapy. In 2001, the Randomization Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure

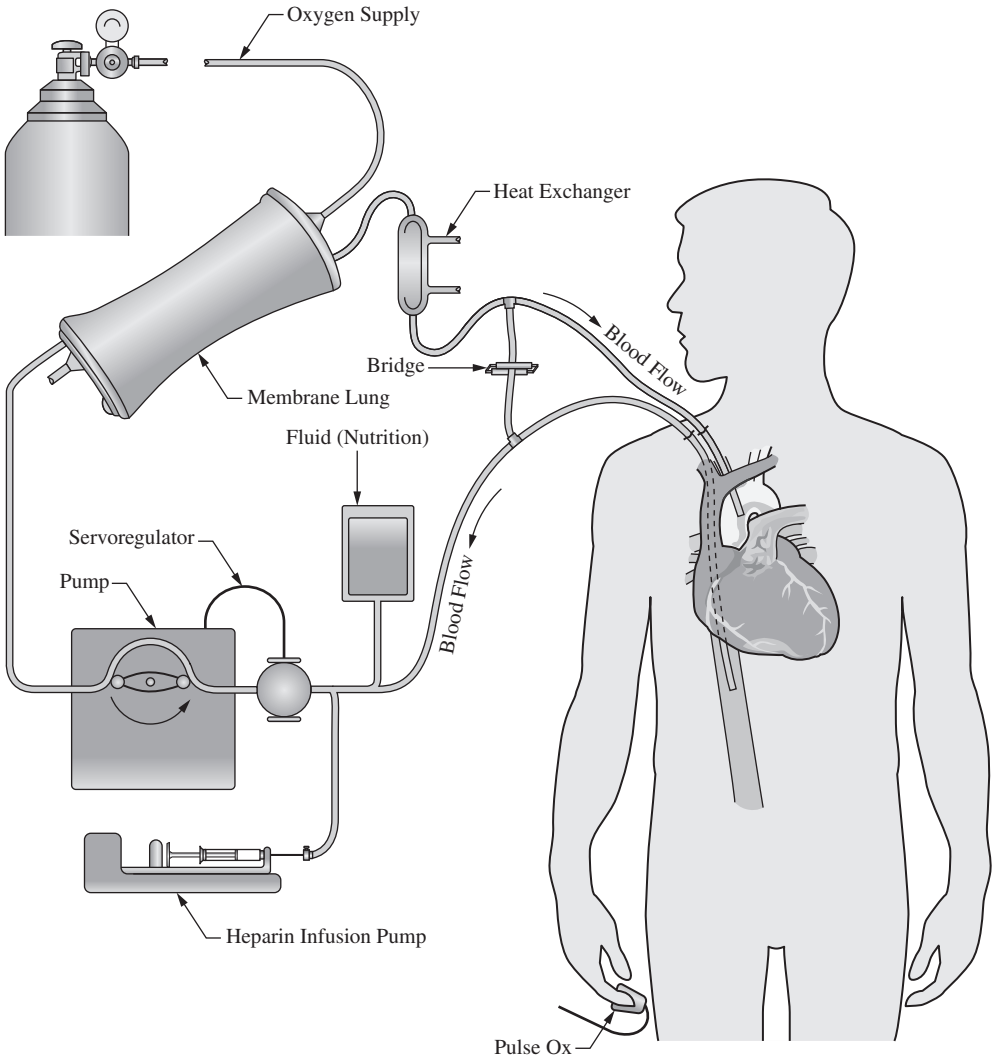


Figure 19–6 ECMO Pulse Ox.

Source: Illustrated by James R. Perron

(REMATCH) trial compared the outcomes with LVAD devices for destination therapy and the outcomes with medical therapy. Rose and colleagues (2001) noted that survival rates at 1 year were better in the LVAD patients than in the medical therapy patients (52% versus 25%, respectively). In addition to realizing a survival benefit, patients on LVAD gained energy and vitality as a result of improved organ perfusion, and they reported overall increased quality of life.

VENTRICULAR ASSIST DEVICES

VADs can provide left, right, or biventricular support. The most common scenario involves the use of LVAD to counteract left ventricular dysfunction. Mechanisms for movement of blood vary among the different types of pumps. In general, blood is drained from the apex of the left ventricle to the pump via the inflow cannula. It is returned to the body via an outflow cannula, which is attached to the

aorta. The pump is housed in the pre-peritoneal space of the abdomen, near the stomach. A percutaneous drive line that is tunneled across the pre-peritoneal space to the left side of the body carries the electrical cable and air vent to the electrical controller outside the patient's body. The risk of pump infection is reduced by tunneling the cable across the abdomen, thereby increasing the distance from the pump to the exit site and allowing the body to form a natural seal around the cable.

Table 19–1 compares the various types of VADs.

VAD Selection Criteria

Selection criteria for patients requiring VAD therapy are rigorous. As part of patient evaluation, a battery of lab and diagnostic tests are performed to determine eligibility for the device. Cardiologists, social workers, nurses, VAD coordinators, and the cardiac surgeon collaborate to determine the appropriateness of the device for the patient and decide on the plan of care. The surgeon determines the type of support required (i.e., LVAD, RVAD, or BiVAD). LVAD recipients must have adequate right ventricular (RV) function to achieve a successful outcome because the LVAD is dependent on blood flow from the right ventricle. If RV failure is present, patients' length of stay, post-implantation morbidity/mortality, and costs are increased (Decoene et al., 2004). In such cases, temporary biventricular support may be initiated, with discontinuation of the RVAD if the right ventricle has recovered.

Types of VADs

Biventricular Support

The Thoratec BiVAD can be used for biventricular support. In the patient with biventricular failure, this pump affords a chance for recovery to transplant that an LVAD cannot. Unfortunately, the portable mechanical driver that powers the device is large and noisy, so transport of the device is tedious. Use of this BiVAD requires initial anticoagulation with heparin, followed by warfarin (Coumadin®) and antiplatelet therapy, which means that bleeding complications are increased when this type of pump is used. One important advantage is the Thoratec BiVAD's innovative design, which places the blood chambers external to the body, allowing for placement of this device in smaller patients.

Heartmate XVE

The Heartmate XVE is another type of implanted, long-term pump with pulsatile blood flow. It is used for left ventricular (LV) support and is FDA approved for both destination therapy and bridge to transplant. Because this model is a larger pump, patient selection criteria include the requirement that the patient's body surface area (BSA) be greater than 1.5 m^2. As a consequence, the Heartmate XVE device is used more frequently in males. The surface of the device is coated with a unique texture, allowing antiplatelet therapy to consist of aspirin alone. This is an important advantage because it means there is a decreased risk of thromboembolism with this device (Bojar, 2005). Bleeding risks are also minimized because warfarin therapy is not required.

Heartmate II

The Heartmate II is an axial (continuous) flow LVAD device that is more compact than the Heartmate XVE, so it can be used in patients who have smaller frames (BSA of 1.3–2.3 m^2). This device is FDA approved for use as a bridge to transplant; approval for destination therapy is expected. Clinical trials of this device by the manufacturer have demonstrated its superior durability in comparison with pulsatile pumps (Haft et al., 2007). In addition, the Heartmate II generates less noise and has a smaller percutaneous lead than the Heartmate XVE. These features are

Table 19–1 Ventricular Assist Devices

	Heartmate II	Heartmate XVE	Thoratec	Novacor	Total Artificial Heart (TAH)	Abiomed (AB 5000)	TandemHeart
Type of blood flow	Pulsatile flow	Continuous flow (axial)	Pulsatile flow	Pulsatile flow	Pulsatile flow	Pulsatile flow	Continuous flow (axial)
BSA limitation (m^2)	1.5	1.3	1.2	1.5	1.7	1.2	1.2
Anticoagulation	ASA only	Antiplatelet therapy and initial heparin followed by warfarin	Antiplatelet therapy and initial heparin followed by warfarin	Antiplatelet therapy and initial heparin followed by warfarin	Antiplatelet therapy and initial heparin followed by warfarin	Antiplatelet therapy and initial heparin followed by warfarin	Antiplatelet therapy and heparin
Indications for use	Destination and bridge to transplant	Destination and bridge to transplant	Bridge to transplant	Bridge to transplant	Bridge to transplant	Temporary support and bridge to recovery	Temporary support and bridge to recovery
Positive aspects	Permits nontethered ambulation Approved for patient discharge No warfarin required	Permits nontethered ambulation Approved for patient discharge Quiet operation Small percutaneous lead	Flexible design for biventricular support	Three-year durability	Biventricular support	Flexible design for biventricular support	Percutaneous placement by cardiology
Negative aspects	Limited durability Large device	Fixed motor speed with risk of LV suction events	Large portable driver system impairs active lifestyle	Large size limits application to small people	Large console limits mobility System not designed for hospital discharge	Cannot be discharged Extracorporeal design limits mobility	Patients must remain in bed with no mobility Partial support
Support design	LVAD	LVAD	LVAD, RVAD, or BiVAD	LVAD	Total orthotopic heart	LVAD, RVAD, or BiVAD	LVAD

ASA = aspirin; BiVAD = biventricular assist device; BSA = body surface area; LV = left ventricular; LVAD = left ventricular assist device; RVAD = right ventricular assist device.

appreciated by patients. The Heartmate II pump requires anticoagulation with heparin/warfarin as well as antiplatelet therapy, so the risk of postoperative bleeding is increased when this device is used.

Total Artificial Heart

A total artificial heart (TAH) is a treatment alternative for patients with biventricular failure who are hospitalized candidates for heart transplant. An example of such a device is the SynCardia CardioWest. It replaces the function of both ventricles and the four heart valves. The CardioWest device is implanted in the patient's chest and attached to the atria. Tubes from the CardioWest ventricles continue from the patient's chest to a power-generating console. The TAH can deliver cardiac output (CO) as high as 9.5 L/min. It reportedly augments renal and hepatic blood flow and improves survival of heart transplant patients with preoperative biventricular failure (McCalmont & Ohler, 2008).

Postoperative Complications

VAD placement is a major surgical procedure with significant risk for postoperative complications. The most common complications are bleeding, hypovolemia, tamponade, organ failure (e.g., kidney, liver), stroke, air embolism, need for inotropic support for RV failure, sepsis, driveline infection, and device failure (McCalmont & Ohler, 2008; Piccione, 2000). Infusion of inotropes may be required until the patient becomes hemodynamically stable following insertion of the device (McCalmont & Ohler, 2008). Dobutamine at 3–5 mcg/kg/min or milrinone at 0.125–0.375 mcg/kg/min are commonly used in the postoperative period to provide RV support.

Table 19–2 provides general infection control guidelines for VADs. Bleeding in the immediate post-insertion period is common owing to the frequent use of aspirin and warfarin in HF patients preoperatively for severely depressed LV function, coronary artery disease, dysrhythmias, or any combination of these conditions.

Because LVAD function depends on adequate flow from the right ventricle, patients are monitored closely for signs of right heart failure. Pulmonary artery catheters (PACs) are inserted preoperatively for close monitoring

Table 19–2 General Infection Control Guidelines

Dressings over drive-line exit sites must be kept clean and dry at all times.
Sterile dressing changes to the drive-line exit site must be performed at least daily. Dressing should be changed more frequently when increased drainage is observed.
Immobilize the drive-line or exit cannulas with abdominal binders continuously. This prevents trauma to the exit site and helps to develop tissue ingrowth around the drive line, which promotes formation of a skin barrier. Trauma to exit sites significantly increases the risk of infection.
Remove monitoring lines as soon as possible to decrease the risk of infection.
Notify the physician of a change in the patient's temperature (<36 °C [96.9 °F] or >38.5 °C [101.4 °F]) or other signs and symptoms of drive-line infection (e.g., redness, increase in drainage, foul odor, or skin separation from drive line).
Ensure adequate nutrition (maintain albumin > 2.5 g/dL). This is essential for the healing process to occur.

Source: Adapted with permission from Kaplow & Hardin, 2007, p. 216.

of right-sided function (right atrial pressure, central venous pressure).

Nitric oxide (NO) has been shown to improve RV function by selective pulmonary vasodilation, which in turn improves LV filling, CO, and systemic arterial pressure (Idrees et al., 2008). NO delivered postoperatively can be continued until the chest is closed and RV stability is achieved (McCalmont & Ohler, 2008).

Respiratory therapy can help promote pulmonary vasodilation by making ventilator adjustments that allow for permissive hypercarbia. Prevention of hypoxia is also important to avoid pulmonary vasoconstriction (Hoskote et al., 2004).

The patient who has undergone LVAD placement is at risk for the development of renal failure (Topkara et al., 2006). Baseline renal dysfunction is common in the HF population, and renal function may transiently worsen in the postoperative period. Because renal failure or severe renal insufficiency is generally a contraindication to heart transplant, care must be taken to minimize injury to the kidneys in this patient population. Diligent intake and output surveillance and medication administration are necessary so that the kidneys can recover for the impending heart transplant.

Transient hepatic dysfunction due to congestion associated with HF and intraoperative transfusions may occur. Supportive care including maintenance of appropriate fluid balance is usually sufficient to correct this problem (McBride et al., 2001).

Requirements for Being Discharged with an LVAD

Patients who return home with an LVAD require extra education to ensure the safety of this therapy. A grounded, three-pronged plug outlet near the patient's sleeping site will be required for the patient to switch to the power source during sleep. In the event of battery or generator failure, ongoing functioning of the device can be ensured through hand pumping, switching power sources, emergency interventions, or a combination of these. Extra batteries should always be available (Mason & Konicki, 2003). Table 19–3 lists the safety precautions required of the patient who is discharged to home with an LVAD.

Table 19–3 Safety Precautions at Discharge

Maintain a method to obtain a backup generator in case of power failure.
Notify the local electricity provider of the use of life support equipment.
Use a transport power base unit to and from the hospital.
Avoid immersion in water (e.g., do not sit in a tub of water).
Avoid static electricity (e.g., touching a computer screen).
Never disconnect both batteries simultaneously.
Protect the vent filter from water.
Cardiopulmonary resuscitation varies depending on the model of LVAD used.
Do not engage in excessive jumping or contact sports.
Do not let children sit on the patient's chest.
No exposure to MRI is allowed.
Pregnancy is not permitted.

LVAD = left ventricular assist device; MRI = magnetic resonance imaging.

HEART TRANSPLANTATION

Although many patients can be supported with either medical management or a VAD, another cohort of patients would benefit from receiving a heart transplant. As mentioned previously, however, the number of potential transplant recipients far exceeds the number of available donors.

There are two approaches to performing a heart transplant. With the orthotopic approach, the recipient's heart is replaced with a donor heart. With the heterotopic approach, the recipient's heart is left in place and the donor heart is "piggybacked" to the right side of the recipient's heart. This latter approach is rarely used.

Donor hearts are typically placed either with the Lower and Shumway method (the donor heart is anastomosed to the left atrium, right atrium, pulmonary artery, and aorta), atrial cuff technique (anastamoses are made at the inferior and superior vena cavae), or the heterotopic (end-to-end anastamoses of the donor superior vena cava, pulmonary artery, and aorta). Regardless of which technique is employed, denervation of the donor heart results in a higher heart rate, orthostatic hypotension, and the inability to experience angina in the transplant recipient. The patient has a median sternotomy incision and is placed on CPB during the transplant procedure (Wade, Reith, Sikora, & Augustine, 2004).

Once the transplant procedure is completed, pacing wires are secured. The patient may require inotropic support to be removed from CPB. If the patient has elevated pulmonary artery pressures, nitric oxide may be used to decrease PVR. The patient's systemic vascular resistance will not be affected by the use of NO; hypotension should not ensue. Once the patient is successfully removed from CPB and the donor heart is functioning, the patient is transferred to the ICU for recovery (Wade et al., 2004).

CARE IN THE IMMEDIATE POSTOPERATIVE PERIOD

Factors Influencing Patient Recovery

After the transplantation procedure is complete, the heart recipient's postoperative care and course will vary depending on many factors. Comorbidities such as renal or pulmonary dysfunction will affect both patient progress and decision making. Heart failure patients who have been bridged to transplant with an LVAD may benefit from enhanced end-organ perfusion preoperatively and, therefore, better tolerate the stress of the heart transplant procedure and its aftermath (Goldstein, Smego, & Michler, 2006).

Donor and harvest factors may also influence postoperative course. For example, the efficacy of the strategies utilized to maintain hemodynamic stability in the donor after brain death will affect the donor heart's function (Poston & Griffith, 2004).

Duration of the cold ischemia of the donated heart will also be relevant to the recipient's recovery. Cold ischemic time refers to the amount of time from cross-clamping of the donor with subsequent removal and immersion of the heart in iced saline until removal of the cross-clamp after it has been implanted into the patient (Anderson, 2008a). The maximum cold ischemic time is 6 hours, and preferably should be less than 4 hours. Younger organs tolerate relatively longer ischemic times; older organs tolerate shorter ischemic times (Russo et al., 2007).

The surgical technique used may also influence the patient's outcome. For example, the bicaval technique (attachment of the donor heart with anastamoses in the superior and inferior vena cavae) has been found to improve patient survival, atrial geometry (non-fluoroscopic imaging system), and hemodynamics as well as to decrease valvular insufficiency, dysrhythmias, pacing requirements, vasopressor requirements and hospital stay when compared with biatrial technique (attachment of donated heart to recipient atrial "cuffs") (Morgan & Edwards, 2005). Patients who receive transplants via the bicaval technique are less likely to develop mitral or tricuspid regurgitation, atrial thrombus, or tachydysrhythmias because atrial anatomy is maintained with this technique (Wade et al., 2004).

Postoperative Care

Once admitted to the ICU, heart transplant patients typically remain on mechanical ventilation for 12–48 hours and remain in the ICU for 2–3 days. The overall reported ICU mortality is less than 5% in this population. The most common morbidities (and their incidence) are infection, especially pulmonary (10%); pulmonary hypertension with right heart dysfunction (10%); nodal dysrhythmias (5%); bleeding (24%); hyperacute rejection (less than 1%); and pain (8–10%) (Jaffe & Samuels, 2003; Wade et al., 2004).

The immediate postoperative period can be quite challenging for both patients and nurses. While the immediate postoperative recovery after a heart transplant generally progresses without complication, patients may demonstrate high levels of vulnerability and low levels of stability. Clinical issues and implications specific to the recovery from anesthesia and CPB are discussed in detail in Chapter 8. Hemodynamic monitoring and care for the patient on mechanical ventilation, including weaning and extubation, are described in Chapters 9 and 11, respectively. Early weaning and extubation is the goal for all heart transplant patients (Wade et al., 2004). Postoperative complications related to CPB are discussed in detail in Chapter 13; those specific to heart transplantation are discussed next.

Bleeding

Postoperative bleeding is a common problem in all cardiac surgery patients, including those who have undergone heart transplant. Postoperative bleeding may be related to hypothermia, administration of heparin for CPB, preexisting hepatic dysfunction from HF and associated low CO, surgical-site tissue trauma, and platelet destruction from CPB (Wade et al., 2004). For this reason, the sternotomy wound is often left open, covered with a sterile, occlusive, transparent dressing to permit visualization of accumulating blood and rapid mediastinal exploration, should it be required.

The ICU nurse should observe the patient for tachycardia, chest tube drainage greater than 100 mL/hr, cardiac index less than 3 L/min/m^2, falling hemoglobin and hematocrit levels, decreased mixed venous saturation (SvO_2), and increasing oxygen requirements. Results of coagulation profiles and platelet count should be evaluated as well. Hypotension and decreasing pulmonary artery and central venous pressures are late signs of bleeding. If the patient is to be transfused with blood or blood products, cytomegalovirus (CMV)-negative patients must receive CMV-negative products (Wade et al., 2004).

Strategies employed for bleeding are the same as those used in other cardiac surgery patients. These interventions include aggressive transfusion of blood products (e.g., fresh frozen plasma, cryoprecipitate) as indicated; use of plasminogen inhibitors (e.g., aminocaproic acid [Amicar®]) and factor VII; administration of additional protamine sulfate, desmopressin (DDAVP®), or aprotinin (Trasylol®); and early reexploration (Wade et al., 2004).

Hypovolemia

Post-transplant hypovolemia has the same etiology in heart transplant recipients as it does in other cardiac surgery patients who have undergone CPB. CPB causes increased capillary permeability, with resultant third spacing of fluid. Intraoperative use of diuretics and initial high post-CPB urinary output further contribute to the postoperative heart transplant patient's hypovolemic state. Hypovolemia, in turn, results in a decrease in preload and cardiac function (Wade et al., 2004).

The ICU nurse must observe heart transplant recipients for hypotension; decreased pulmonary artery, pulmonary artery occlusive, and central venous pressures; decreased urinary output; and decreased cardiac output/index.

Hematocrit levels may be elevated as a consequence of hemoconcentration. Patients may manifest hypotension more so than might be expected after receiving narcotics or sedation (Wade et al., 2004).

Treatment of hypovolemia focuses on volume repletion. This goal may be accomplished through administration of either a crystalloid or a colloid, depending on facility protocol. As discussed in Chapter 17, the debate over the efficacy of crystalloid versus colloid therapy continues. The ICU nurse must carefully monitor the patient's vital signs, urinary output, and hemodynamic profile both during and following volume resuscitation. Development of fluid overload can cause dilation of the right ventricle and pulmonary edema, which can be life-threatening (Cohn, 1997; Wade et al., 2004). Fluid resuscitation is discussed in detail in Chapter 17.

Right Heart Failure

Right heart failure is a major cause of morbidity and mortality in the post-heart transplant period. The left ventricle is generally able to tolerate increased afterload (the amount of work the heart must do to eject blood). However, the implanted right ventricle is not physiologically adapted to overcome high afterload as would be encountered with pulmonary hypertension, which develops frequently in HF patients. Opposed by high pulmonary pressures or pulmonary vascular resistance, the implanted right ventricle dilates readily and fails. This chain of events is difficult to reverse once it begins. Further, right heart dilation alters septal position and function, which in turn interferes with LV function (Stobierska-Dzierzek & Brook, 2001). High preoperative pulmonary artery pressure (PAP) may predict increased risk for development of this syndrome; however, normal PAP does not preclude it. Right heart failure is characterized by an elevated central venous pressure, edema, and hepatomegaly (Taegtmeyer, 2006).

Hypertension

Prevention and prompt detection and management of hypertension following heart transplantation are essential to prevent surgical site dehiscence. Administration of nitroprusside may be indicated (Wade et al., 2004).

Pulmonary Hypertension

Pre-emptive protective maneuvers are frequently successful in avoiding right heart failure. Pulmonary artery catheters are placed in all heart transplant patients. NO is frequently utilized to selectively dilate the pulmonary vasculature without decreasing systemic blood pressure (Wolfgang, Bauer, & Podesser, 2006); its administration has been shown to decrease the incidence of RV dysfunction in patients with pulmonary hypertension (Ardehali, Laks, et al., 2001). Other agents that dilate the pulmonary vasculature (e.g., nitroglycerin [Tridil®], sodium nitroprusside [Nipride®], prostaglandin E-1 [PGE1], and prostacyclin [PGH2]) may cause hypotension and, therefore, are used less often in post-heart transplant patients.

Patients are generally weaned from NO on the first postoperative day or later, depending on the overall clinical picture and their degree of hemodynamic stability. NO has a very short half-life, and patients are weaned from it very slowly (over hours). Rebound pulmonary hypertension and acute RV dysfunction are likely if use of NO is abruptly discontinued or weaning proceeds too quickly. Acidosis, hypercarbia, and hypoxemia increase pulmonary vascular resistance. Patients are, therefore, hyperventilated to achieve a pH in the range of 7.45–7.49 and a pCO_2 in the range of 30–35 mm Hg. Hypoxemia must be avoided (Ardehali, Hughes, et al., 2001).

Chronotropy (Rate) Issues

Tachycardia, which decreases ventricular filling time and hence the risk of RV dilation, is achieved with an atrial pacer set to the AAI

mode (atria paced, atria sensed, inhibited) and a rate of 100–120 beats per minute via epicardial pacing wires. The AAI mode is selected because transplanted hearts generally have intact conduction systems (unlike in valve surgery patients, who are susceptible to temporary heart blocks). Utilization of the heart's intrinsic conduction system promotes ventricular synchrony, which stabilizes the septum in a manner that facilitates LV and RV function. Tachycardia is also achieved pharmacologically with isoproterenol (Isuprel®), which also dilates pulmonary vasculature; dobutamine; and dopamine.

Contractility (Inotropy, Force of Contractions) Issues

Inotropic agents such as dopamine, dobutamine, and milrinone are used to support RV and LV function. Milrinone is frequently given at a dose of 0.125–0.375 mcg/kg/min and has the added benefit of promoting pulmonary vascular dilation. If these pharmacological interventions (which are described in detail in Chapter 12) are not successful in preventing or ameliorating right heart failure, insertion of an intra-aortic balloon pump (see Chapter 10) or right ventricular assist device may be necessary.

Hypotension

As in other cardiac surgery populations, blood pressure variability is common in patients who have received heart transplants. Hypotension decreases coronary artery perfusion, which is undesirable in all cardiac surgery patients and may contribute to right heart failure in the heart transplant patient. Treatment is directed at the underlying mechanism.

Vasodilation may be exacerbated by medications such as milrinone. Decreasing the dose of vasodilator medications, if feasible, may mitigate hypotension. Vasopressors such as norepinephrine (Levophed®) and vasopressin (antidiuretic hormone [ADH]) are often required to maintain a mean arterial pressure greater than 65 mm Hg. Vasopressin is thought to cause less constriction of the pulmonary vasculature, is associated with lower arrhythmogenicity, and increases glomerular filtration rate. For these reasons, it is used preferentially either alone or with norepinephrine (Kee, 2003).

Allograft Dysfunction

Post-transplant cardiac function depends on a number of factors, including donor and harvest factors, cold ischemic time, surgical technique, condition of the donor heart, and preservation techniques (McGiffin, Kirklin, Naftel, & Bourge, 1997; Morgan & Edwards, 2005; Poston & Griffith, 2004; Russo et al., 2007; Wade et al., 2004). Poor cardiac function is an early cause of allograft failure (Anderson, 2008a).

LV systolic dysfunction is categorized as either early or late. Early LV dysfunction occurs either intraoperatively or in the immediate postoperative period. Late LV dysfunction develops weeks to years following a heart transplant. The most common cause of both early and late LV dysfunction is allograft rejection. If LV dysfunction develops within days of the transplant, it typically occurs secondary to one of three etiologies: hyperacute rejection, reperfusion injury, or a suboptimal donor heart (Anderson, 2008a).

REPERFUSION INJURY DURING SURGERY

Allograft dysfunction may be caused by reperfusion ischemia, prolonged cold ischemic time (greater than 5 hours), or both. This condition may be only a temporary complication (myocardial stunning), and resolve after 12 to 24 hours after heart transplant. The heart does sustain ischemic injury when such dysfunction occurs, despite the short duration (Anderson, 2008a).

SUBOPTIMAL DONOR HEART

As is well known, the number of heart transplant candidates far exceeds the number of

donor hearts available. Consequently, some transplant programs have been accepting "suboptimal" donors (e.g., patients older than 63 years of age, hearts with mild left ventricular hypertrophy). These hearts have typically been treated with higher doses of inotropic agents or vasopressors or have LV dysfunction (Anderson, 2008a).

LV systolic dysfunction is treated with inotropic support and appropriate fluid administration. Dopamine, dobutamine, or milrinone may be used to increase CO without associated increases in systemic vascular resistance (SVR). Each of these inotropic agents is discussed in detail in Chapter 12. IABP therapy may be initiated if high doses of inotropes are required to maintain LV function; IABP therapy is discussed in detail in Chapter 10. Monitoring of the patient's vital signs and hemodynamic profile is essential while any of these therapies is being used (Wade et al., 2004).

If the patient's SVR is elevated, an associated decrease in CO may ensue. An elevated SVR may be managed with administration of a vasodilator such as nitroglycerin or nitroprusside.

On rare occasions, a patient's SVR may be low following heart transplant. This condition is believed to be related to use of angiotensin-converting enzyme (ACE) inhibitors for heart failure, release of proinflammatory mediators as occurs with CPB, or decreased levels of vasopressin. Low SVR may be treated with a vasopressor such as norepinephrine or epinephrine (Adrenaline®). If neither of these medications is effective in reversing the deficit, vasopressin may be given (Landry et al., 1997; Wade et al., 2004). All of these agents are discussed in detail in Chapter 12.

RV dysfunction following heart transplantation is likely due to pulmonary hypertension and can be difficult to manage. Patients with HF have chronic high left atrial pressure. Following the transplant, pulmonary hypertension and RV failure can occur because the donor heart may not be accustomed to pumping against such a high pulmonary artery pressure. Management of pulmonary hypertension may include administration of a vasodilator (e.g., nitroglycerin) with an inotrope (e.g., dobutamine or milrinone). If the patient's pulmonary artery systolic pressure exceeds 50 mm Hg, administration of intravenous prostaglandin E_1 and prostacyclin may be considered (Wade et al., 2004). Inhaled nitric oxide is another agent that may be used to decrease pulmonary vascular resistance without diminishing SVR (Kieler-Jenson, Lundin, & Ricksten, 1995; Wade et al., 2004).

Rhythm Disturbance and Electrocardiograph Changes

Development of cardiac dysrhythmias is common following heart transplant. For patients who undergo transplants, the likelihood of such abnormalities in heart rhythm is higher in the immediate postoperative period. Such dysrhythmias may result from surgical trauma to the sinoatrial (SA) and atrioventricular (AV) nodes, ischemia, suture lines, rejection, and transplant vasculopathy (TV) (Rothman & Eisen, 2008).

BRADYCARDIA

Bradycardia may occur in the immediate postoperative period. This complication affects as many as 50% of orthotopic heart transplant patients, typically taking the form of SA node dysfunction. SA node dysfunction occurs as a result of ischemia, intraoperative manipulation of the SA node, perinodal atrial tissue, SA artery trauma, or pretransplant use of amiodarone (Cordarone®) (Rothman & Eisen, 2008). Whereas bradycardia immediately following surgery has little prognostic significance, late-onset bradycardia may be an indicator of organ rejection or TV (Rothman & Eisen, 2008).

Atropine sulfate is not effective in denervation of the sympathetic and parasympathetic nerves. Bradycardia may be treated with

administration of a $beta_1$-receptor agonist (e.g., dobutamine or isoproterenol). It has also been suggested that administration of theophylline or terbutaline (Brethine®) may increase heart rate in heart transplant patients who develop bradycardia (Rothman & Eisen, 2008; Wade et al., 2004).

Clinically significant bradycardia will typically be treated with epicardial pacing. Dual-chamber pacing is preferred so that the patient's cardiac output can be augmented from the atrial kick (Rothman & Eisen, 2008; Stecker, Strelich, Chugh, Crispell, & McAnulty, 2005; Wade et al., 2004).

ATRIAL DYSRHYTHMIAS

The incidence of atrial fibrillation (AF) in heart transplant patients ranges from 0.33% to 24% (Ahmari et al., 2006; Khan et al., 2006; Rothman & Eisen, 2008). AF usually occurs within the first 2 postoperative weeks; later development is associated with higher mortality rates (Rothman & Eisen, 2008). It has been suggested that AF is attributable to surgical insult in this patient population (Cui, Tung, Kobashigawa, Laks, & Sen, 2001; Khan et al., 2006).

The incidence of atrial flutter ranges from 2.8% to 15% in heart transplant recipients (Ahmari et al., 2006; Khan et al., 2006; Rothman & Eisen, 2008). Atrial flutter usually develops late in a patient's post-transplant course (Rothman & Eisen, 2008). It is believed to result from rejection, which causes increased cardiac fibrosis (Cui et al., 2001; Khan et al., 2006), but may occur if rejection has not occurred (Rothman & Eisen, 2008). Rejection-associated atrial flutter may be caused by impairment in atrial conduction and refractoriness, alteration in atrial hemodynamics, and decreased ventricular function, all of which occur with rejection (Rothman & Eisen, 2008).

Atrial dysrhythmias may be ameliorated by maintenance of normal electrolytes (potassium > 4 mEq/L, magnesium > 2 mg/dL) and dose reduction of inciting medications as appropriate.

Treatment of atrial dysrhythmias is discussed in detail in Chapter 15. Of note, digoxin (Lanoxin®) is not effective in the denervated heart. Caution should be exercised when treating atrial dysrhythmias with beta blockers or calcium channel blockers (Wade et al., 2004). Radiofrequency ablation has been used to treat atrial flutter in heart transplant patients (Rothman & Eisen, 2008).

SUPRAVENTRICULAR TACHYCARDIA

Types of supraventricular dysrhythmias that have been reported in orthotopic transplant patients include AV reentrant tachycardia, Wolff-Parkinson-White syndrome, and non-paroxysmal atrial tachycardia (Rothman & Eisen, 2008). The incidence of supraventricular tachycardia (SVT) is reported to be approximately 1.3% (Khan et al., 2006). SVT has been treated successfully with radiofrequency ablation (Rothman & Eisen, 2008).

VENTRICULAR DYSRHYTHMIAS

Premature ventricular contractions may occur in as many as 100% of orthotopic heart transplant patients early in the postoperative period. The incidence decreases over time, however (Rothman & Eisen, 2008).

Paroxysmal ventricular tachycardia (VT) decreases in incidence after the initial postoperative period. There may be a correlation between VT, rejection, and TV (Rothman & Eisen, 2008). Non-paroxysmal ventricular dysrhythmias are rare in the postoperative heart transplant patient. Development is usually related to severe TV or allograft rejection (Rothman & Eisen, 2008).

CONDUCTION DELAYS

Right bundle branch block (RBBB) is a common ECG finding post-transplant, but has no clinical or prognostic significance (Golshayan et al., 1998). This condition may be encountered in as many as 70% of orthotopic trans-

plant patients (Rothman & Eisen, 2008). Development of a RBBB may be associated with prolonged donor ischemic time and several rejection episodes. Etiologic factors may include RV hypertrophy from elevated pulmonary artery pressures or right bundle damage during endomyocardial biopsy procedures (Rothman & Eisen, 2008).

AV node function remains intact following a heart transplant. As a consequence, high-degree AV block is rare in post-transplant patients, especially in the early postoperative period. Late development carries an increased mortality rate (Rothman & Eisen, 2008).

Cardiac Tamponade

Cardiac tamponade may develop either gradually or suddenly in patients who have undergone heart transplantation. It results from fluid accumulating in the pericardial sac, which causes compression of the atria, restriction of venous return to the heart and ventricular filling, and results in a decrease or cessation of preload and a potential precipitous decline in CO (Massé & Antonacci, 2005). Early tamponade may also result from persistent mediastinal bleeding not being evacuated by chest tubes or clot formation.

The diagnosis of cardiac tamponade may be difficult in heart transplant recipients because hypotension and tachycardia are common scenarios in the immediate postoperative period. When caring for these patients, the ICU nurse should maintain patency of chest tubes, monitor vital signs and hemodynamic profiles, and observe for signs and symptoms including those listed in Table 19–4.

If cardiac tamponade develops, initial management should include volume resuscitation to optimize filling pressures (being careful not to overload the right ventricle), and initiation and titration of inotropes (if blood pressure is not responsive to fluid resuscitation). Diagnosis is made by echocardiogram. Definitive treatment entails surgical intervention or emergent bedside sternotomy (Wade et al., 2004). The required equipment and nursing care for the latter procedure are discussed in Chapter 13.

Renal Dysfunction

In the immediate post-transplant period and following CPB, the patient's urinary output will be increased. Once these effects have worn off, it is important that urinary output be maintained at a rate of at least 0.5 mL/kg/hr. Renal function requires preservation because of the nephrotoxic immunosuppression agents that will be administered to prevent rejection of the donated heart. The ICU nurse must maintain adequate renal perfusion with

Table 19–4 Signs and Symptoms of Cardiac Tamponade

Sudden decrease or cessation of chest tube drainage
Dyspnea
Low cardiac output with hypotension
Narrowing pulse pressure
Inappropriately fluctuating mean arterial pressure
Increased central venous pressure
Low cardiac output/index
Sudden oliguria
Altered mental status
Diaphoresis
Dysrhythmias, including tachycardia
Cyanosis or pallor
Anxiety
Restlessness
Low-voltage QRS on ECG
Electrical alternans on ECG
"Water bottle heart" and cardiac enlargement on chest radiograph
Hepatomegaly

Sources: Kaplow & Reid, 2006; St. Andre & DelRossi, 2005; Talmor & Lisbon, 2005; Wade, Reith, Sikora, & Augustine, 2004.

fluids, vasoactive agents, or combinations of these to maintain a mean arterial pressure in the range of 60–80 mm Hg. Monitoring of hourly urinary output and all renal function tests should be performed by the ICU nurse as well (Wade et al., 2004).

Psychosocial Conditions

In addition to all of the physiologic stressors associated with heart transplantation, a variety of psychosocial conditions may surface in the postoperative period. Patients are typically and predictably euphoric and relieved that they were recipients of a long-awaited organ. However, depression, anxiety, confusion, and delirium may develop in a few postoperative days. It is common for the patient to inquire about the donor and to experience difficulty coping with the knowledge that a death was associated with the organ procurement. Patients may manifest violent outbursts, attempt to climb out of bed, inappropriately yell at staff, or have hallucinations. Maintaining patient safety during this time is essential (Wade et al., 2004). The etiology and specific management of these neurocognitive disorders are discussed in detail in Chapter 16. Maintaining a calm, reassuring environment and demonstrating a high level of caring practices are essential for optimal psychological patient outcomes.

■ PROGRESSION OF CARE

Transfer to a progressive care unit occurs when the patient is extubated, hemodynamically stable, and no longer receiving any vasoactive medications. The expectation throughout the hospitalization period and beyond is that patients will participate actively and fully in their own recovery.

Pain Management

Transplant patients' requirements for pain medications vary. Back, shoulder, and chest discomfort are common. While the patient is intubated, a continuous infusion of either morphine or fentanyl is typically administered. The narcotic should be titrated so that the patient is easily arousable to verbal stimuli and able to follow simple commands. Once the patient has been extubated, use of acetaminophen and oral narcotics (e.g., oxycodone) is preferred. Nonsteroidal anti-inflammatory drugs (NSAIDs) are avoided given the compounded risk of nephrotoxicity when these medications are utilized in conjunction with CNIs (e.g., tacrolimus [Prograf®], cyclosporine [CsA, Sandimmune®]) to prevent rejection (Wade et al., 2004).

Nutrition

A clear liquid diet is initiated at the time of extubation and advanced as tolerated. Because many heart transplant recipients are malnourished preoperatively with little nutritional reserve, caloric intake is followed closely and enteral feeding is initiated readily for patients who are not meeting caloric requirements. All transplant patients are followed by a registered dietitian.

Activity

Coughing, deep breathing, and early ambulation are important in heart transplant recipients, as in all postoperative patients. Physical and occupational therapy services are consulted for all transplant patients, and a program of progressive activity is undertaken as soon the patient's status permits. Patients are encouraged to assume gradually increasing responsibility for their self-care needs.

Rejection

Rejection occurs when T cells recognize the implanted heart tissue as foreign and mount an immune response targeted at eliminating it. The immune response leads to inflammation, cell damage, and death. If left unchecked, progressive decline in organ function ensues.

Three types of rejection are distinguished: hyperacute, acute, and chronic. Hyperacute rejection is rare, but is caused by an antigen–antibody reaction. It may occur within minutes to hours after a transplant and is often fatal. Acute rejection occurs when surface cell antigens of the donor heart are recognized as being "non-self." This type of rejection usually occurs within the first few weeks after a transplant, but may occur years later. Chronic rejection is manifested by accelerated graft vasculopathy and typically does not occur within the first year of a transplant (Anderson, 2008a; Wade et al., 2004).

Despite use of immunosuppressive medications, rejection is a relatively common occurrence. In one study, 35–45% of recipients had at least one episode of acute rejection in the first year after transplant. Rejection rates were higher in those receiving CsA plus mycophenolate mofetil (MMF) than tacrolimus plus MMF. Rates of rejection were higher in women than in men, and decreased with advancing recipient age (Taylor et al., 2007). Risk factors for rejection include previous episodes of rejection, young patient age, female gender, female donor, positive CMV serology, prior infections, OKT3 induction, and hemodynamic compromise (Kubo et al., 1995; Michaels et al., 2003).

Patients experiencing rejection may be asymptomatic, or they may demonstrate dysrhythmias, a ventricular gallop, or increased central venous pressure (Snell, Randolph, & Artig-Brown, 2007). A myocardial biopsy is needed for definitive diagnosis. Complications of myocardial biopsy include pneumothorax, cardiac perforation, and tricuspid valve injury. Surveillance biopsies are obtained weekly for the first month after transplant, every 2 weeks for the second and third months, monthly through the ninth month, at the one-year anniversary, and then every 6 months. Biopsies are obtained more frequently if episodes of rejection occur. Rejection is treated with an intensification of immunosuppression, which is usually accomplished with a steroid pulse or OKT3 (Lindenfeld et al., 2004a).

Rejection is prevented by ongoing administration of immunosuppressive agents. Protocols for immunosuppression are facility specific. The components of standard immunosuppression are a corticosteroid, a CNI, and an antiproliferative agent. Some facilities may also prefer induction therapy with antilymphocyte antibody for the first few weeks following a transplant, reflecting the increased risk of acute rejection during this time (Wade et al., 2004).

The treatment of rejection depends on the histologic grade and the clinical situation. Mild rejections (grade 1A, grade 1B, or low grade 2) are treated by optimizing the immunosuppression regimen in current use. Moderate rejections (advanced grade 2, grade 3A, and low grade 3B) are usually treated with augmented immunosuppression. Severe rejection (grade 4) is typically treated by administering high doses of intravenous corticosteroids, anti-3 globulin (ATG), or polyclonal antibodies (e.g., OKT3) and by optimizing the initial immunosuppression regimen (Taylor, 2007).

Immunosuppression

The term "immunosuppression" is used in the organ transplant setting to describe methods by which the transplant recipient's immune system is prevented from "attacking" the newly implanted organ. Given that rejection is mediated almost entirely by T cells, with B cells playing a lesser role, immunosuppressants target T-cell function in a variety of ways (Oka & Yoshimura, 1996).

Immunosuppression regimens generally consist of two phases—induction and maintenance—with intensification of treatment or addition of new agents for episodes of rejection. The particular strategy employed varies by surgeon preference, facility practice, and patient factors, but is uniformly initiated

at the time of transplant. A discussion of the most commonly used immunosuppressant medications follows.

Corticosteroids

Corticosteroids (e.g., methylprednisolone [Medrol®]) are useful in all phases of immunosuppression (induction, maintenance, and management of acute rejection), as these agents suppress nearly all mechanisms of immunity. Corticosteroids act directly on cell DNA to influence transcriptional regulation, which alters the expression of genes involved in immune and inflammatory responses (Lindenfeld et al., 2004b). Through this mechanism, corticosteroids affect the number and distribution of leukocytes, their ability to signal other immune cells, and their functional ability (e.g., decreased phagocytosis, inhibition of secretion of inflammatory substances) (Smith, 2002).

Short-term use of these drugs is associated with hyperglycemia and lability of mood; otherwise, corticosteroids are generally well tolerated. Long-term use is associated with multiple toxicities, including osteoporosis, chronic adrenal suppression, infection, fluid and sodium retention, cataract formation, peptic ulcer development, and cosmetic effects. Side effects that are detrimental to cardiovascular health include hypertension, dyslipidemia, and diabetes. For this reason, research related to immunosuppression in heart transplant recipients has focused on regimens that minimize corticosteroid exposure (Smith, 2002). Administration of a prophylactic H_2 blocker may prevent gastrointestinal bleeding. Patients should have bedside blood glucose testing at least every 6 hours to monitor for hyperglycemia (Wade et al., 2004).

Calcineurin Inhibitors

The CNI category of anti-rejection agents includes tacrolimus and CsA. These drugs prevent T-cell proliferation. Specifically, a T cell becomes activated when it encounters an antigen (something "other than self," such as transplanted heart tissue). Clonal expansion of that specific T cell then ensues as the body attempts to eradicate the foreign substance. The signaling process that initiates replication of this cell is mediated by cellular calcineurin and extracellular interleukin 2 (IL-2). CNIs block this pathway, thereby inhibiting clonal expansion of T cells. This type of immunosuppression is highly effective, but significant toxicities—most notably nephrotoxicity—limit their use in the post-transplant setting. Other significant toxicities include new-onset diabetes mellitus, hyperkalemia, hypomagnesemia, and dyslipidemia. The incidence of hypertension and hyperlipidemia is reportedly lower with tacrolimus than with CsA; a higher incidence of diabetes is noted with tacrolimus, however (Wade et al., 2004).

Because a significant proportion of heart transplant patients have baseline renal dysfunction (and possibly additional insult following CPB), CNIs are more often included in maintenance therapy rather than being used as induction therapy. The nurse should monitor renal function tests and intake and output. Levels (troughs) are followed daily until stable, and then periodically. Because CNIs are metabolized by the cytochrome P450 3A enzyme system, drug interactions are common and can significantly alter drug levels. Ketoconazole, diltiazem, fluconazole, erythromycin, and itraconazole may increase CNI levels; isoniazid, phenobarbital, phenytoin, and rifampin may decrease CNI levels (Kobashigawa, 1999; Wade et al., 2004).

Monoclonal Antibodies

Muromonab-CD3 (OKT3) is a murine (mouse-derived) monoclonal antibody targeted at a specific "marker" on the surface of T cells that (1) transiently activates and eliminates nearly all T cells from peripheral circulation, and (2) renders remaining or subsequent T cells incapable of activation (Smith, 2002). OKT3 may be used as induction therapy, par-

ticularly in patients with renal dysfunction who would not tolerate CNIs. It is administered intravenously during the first 14 post-transplant days (Wade et al., 2004). Response to therapy can be assessed by measurement of CD3-expressing lymphocytes.

Because OKT3 initially activates T cells through binding of the monoclonal antibody with the CD3 surface protein, side effects are those associated with cytokine release. These side effects may range from common, minor flu-type symptoms (e.g., fever, chills, and minor pulmonary or gastrointestinal [GI] symptoms) to life-threatening symptoms (e.g., bronchospasm, tachycardia, bradycardia, encephalopathy, seizures, renal insufficiency, and graft thrombosis). Symptoms from a "first dose response" are termed cytokine release syndrome and include decreased myocardial contractility, hypotension, coronary vasospasm, increased capillary permeability, chest pain, alterations in bronchial and GI smooth muscle control, dyspnea, wheezing, myalgias, arthralgias, headache, and weakness. A life-threatening infection (especially CMV), and Epstein-Barr virus (EBV)-related lymphoproliferative disorder may also develop in some patients (Sayegh, 2008; Sevmis et al., 2005; Smith, 2002).

OKT3 significantly decreases the patient's lymphocyte count, so there is an increased risk of lymphoma and vascular rejection when this medication is given to heart transplant patients (Wade et al., 2004). Interestingly, the 22nd report of the Registry of the International Society for Heart and Lung Transplantation reported that patients who received OKT3 as part of an induction regimen had higher rejection rates during the first year after transplant than both patients who received polyclonal antibody or IL-2 induction and patients who received no antibody induction (Taylor et al., 2005). These data were later corroborated by Stehlik and colleagues (2006), who reported that heart transplant patients who received OKT3 for more than 7 days experienced a higher incidence of antibody-mediated rejection. As a result, OKT3 has been replaced by other agents for early rejection prophylaxis in many centers.

Daclizumab (Zenapax®) and basiliximab (Simulect®) are other monoclonal antibodies used to suppress the immune system. These two agents target only the IL-2 receptor; as a consequence, they inhibit only activated T cells, rather than all T cells. IL-2 receptor antagonists (IL-2Ras) are being used to a greater extent for induction therapy. These monoclonal antibodies are also administered for the first 14 days post-transplant (Wade et al., 2004).

In a comparison of basiliximab and OKT3 as induction agents after heart transplant, researchers found that both agents had similar efficacy. However, basiliximab was associated with shorter ICU length of stay post-transplant (Vaqueriza et al., 2006).

In another comparison study, heart transplant patients received MMF, CsA, and prednisone with or without basiliximab. In this study, survival and renal function were not affected by the addition of basiliximab. The 1- and 3-year rates of acute rejection, however, were improved with basiliximab therapy (Rosenbaum et al., 2006).

More recently, a systematic review of clinical trials evaluating IL-2Ras failed to demonstrate that these agents are effective in improving survival of heart transplant patients or in decreasing the incidence of cardiac allograft rejection. The authors of this meta-analysis concluded that use of these agents remains uncorroborated (Møller, Gustafsson, Gluud, & Steinbrüchel, 2008).

Mycophenolate Mofetil (Cellcept®)

MMF is an antiproliferative agent that inhibits an enzyme that is necessary for DNA synthesis. All cells other than B cells and T cells can use alternative pathways that do not require this enzyme for DNA production; consequently, they are not affected by the

drug. MMF is used concomitantly with CNIs, corticosteroids, or both. Side effects are largely gastrointestinal in nature, although leukopenia may occur as well (Meiser et al., 1999).

Azatriaprine (Imuran®)

Azatriaprine (AZA) is another antiproliferative agent that may be used to prevent rejection following a heart transplant. A large randomized comparative study was conducted between MMF and AZA. Data from this study initially revealed no differences in terms of rejection prevention. However, 72 patients in the study were unable to take oral medications (MMF was not available in intravenous form at the time); 75% of these patients had not previously received any study drug and were given AZA. These 72 patients had a high (56%) mortality or retransplant rate. The MMF-treated group had an 11% reduction in treated rejection episodes and a 34% reduction in biopsy-proven rejection associated with hemodynamic compromise. In addition, the MMF-treated group had a statistically lower 12-month post-transplant mortality rate (6.2% versus 11.4%). Further, during the first 12 months post-transplant, among the remaining 578 patients enrolled in the study, there were no deaths in the MMF-treated patients as compared to 12 deaths (32%) in the AZA-treated patients. Hemodynamic compromise was experienced by both groups of patients. The researchers concluded that MMF was more effective in preventing and successfully treating rejection. The patients receiving MMF experienced more diarrhea, esophagitis, and opportunistic infections than did the AZA-treated patients; the AZA-treated patients experienced more leukopenia than the MMF recipients (Kobashigawa et al., 1998).

In a more recent study, heart transplant patients received induction therapy with rabbit ATG followed by steroids, CsA, and either MMF or AZA. One-year survival rates and LVEF were similar for the two groups. However, CsA levels were lower in the MMF-treated group at the 3-month mark. The 1-year survivors who received MMF were less likely to develop rejection, experienced fewer cases of steroid-resistant rejection requiring cytolytic therapy, and had more patients weaned off steroids by 1 year. Renal function was better for the MMF-treated group; they had higher creatinine clearance and lower serum creatinine levels (Hamour, Lyster, Burke, Rose, & Banner, 2006).

Regardless of which antiproliferative agent is used, the nurse should monitor complete blood counts to evaluate for presence of associated myelosuppression (Wade et al., 2004).

Polyclonal Antibodies

Polyclonal antibodies used for immunosuppression in heart transplant patients include thymoglobulin (Sangstat®; rabbit ATG) and lymphocyte immune globulin (Atgam®; ATG, antithymocyte globulin [horse]). Whereas OKT3 is a monoclonal antibody directed at a specific protein found on T cells, thymoglobulin contains antibodies that bind with multiple T-cell antigens. It is produced by collecting and purifying the sera from rabbits or horses that have produced antithymocyte antibodies in response to immunization with human thymocytes. Proposed mechanisms of action include depletion of circulating T cells, modulation of cell surface receptor molecules, and induction of apoptosis (programmed cell death) of activated T cells (Smith, 2002). It has been suggested that these polyclonal antibodies' lymphocytotoxicity may also have a role in preventing organ rejection. Both thymoglobulin and lymphocyte immune globulin are administered for the first 14 days post-transplant (Wade et al., 2004).

Side effects unique to thymoglobulin include anti-antibody reactions that can render the drug ineffective; serum sickness, which results from anti-antibody–drug complexes; and increased risk of malignancy, possibly related to profound inhibition of T cells (Smith, 2002). Patients are also at risk for ana-

phylaxis; acetaminophen (Tylenol®) and diphenhydramine (Benadryl®) are administered as premedications to prevent this allergic reaction. The nurse should also monitor the complete blood count and subsets of T cells during therapy for any patient who receives these polyclonal antibodies (Wade et al., 2004).

Targets of Rapamycin Inhibitors

Sirolimus (Rapamune®) and everolimus (Certican®, currently in clinical trials) are relatively new agents that block T-cell proliferation through a mechanism similar to, but distinct from, the mechanism underlying CNIs' activity. These agents block the signaling necessary for T-cell growth and clonal expansion via inhibition of serine-threonine kinase mammalian TOR (mTOR) (Lindenfeld et al., 2004a). They are powerful inhibitors of growth factor-induced proliferation of lymphocytes (Formica et al., 2004).

Targets of rapamycin (TOR) inhibitors are the focus of intense research. Data comparing everolimus or sirolimus and AZA suggest similar survival rates among patients. However, studies have shown that incidence of CMV or cardiac allograft vasculopathy is higher in AZA-treated patients than in everolimus-treated patients (Eisen et al., 2003; Keogh, 2002). In a later study, everolimus was more effective than AZA in preventing rejection in heart transplant patients. In the everolimus-treated patients, there was a lower incidence and severity of cardiac allograft vasculopathy and major adverse cardiac events 4 years after transplant than in the AZA-treated patients (Eisen, 2006).

The ultimate role of TOR inhibitors in the immunosuppressant armamentarium and their impact on long-term outcomes remain to be determined. Currently, these agents are typically used as adjuncts to other medications or as a rescue therapy if toxicities limit the use of other classes of immunosuppressants in heart transplant patients. It has also been suggested that these medications might be administered early in the post-transplant period as a means to delay initiation of CNIs, thereby avoiding the nephrotoxicity concerns associated with the latter medications. While fluid retention is clinically demanding when TOR inhibitors are given, renal and cardiac function may be maintained using this approach (Griffith, Augustine, & Wade, 2003). Sirolimus has significant renal toxicity and also causes dyslipidemia. Impaired wound healing has been documented in some patients (Knight et al., 2007).

Nursing monitoring of patients receiving TOR inhibitors includes assessment of complete blood count data to evaluate for development of myelosuppression and to monitor sirolimus levels (Wade et al., 2004).

Infectious Disease Following Heart Transplant

Infection is a primary complication following a heart transplant and is a common cause of morbidity and mortality in this patient population (Bethea, Yuh, Conte, & Baumgartner, 2003). Interventions to prevent development of infection are essential.

Heart transplant patients are at increased risk for development of infection for a number of reasons. For example, anti-rejection medications, especially when used in combination, uniformly increase the risk of infection and malignancy. Transplant recipients are also at risk for common hospital-acquired infections as well as opportunistic infections (e.g., *Pneumocystis carinii*, yeast, fungus). Reactivation of old infections, such as CMV, toxoplasmosis, herpes simplex (HSV), varicella zoster (VZV), or EBV poses another threat: Primary infection by these organisms is not "cured," but rather is controlled by a competent immune system. Reactivation of infection does not occur in the immunocompetent patient because T and B cells are constantly circulating and immediately respond to any renewed activity of these latent organisms.

T and B cell function in the immunocompromised patient, however, is diminished; thus reactivation of these infections will not be curtailed by a weakened immune system (Bethea et al., 2003).

Other common sources of nosocomial infection include invasive lines, catheters, and devices; surgical incisions; and mechanical ventilation equipment. An infectious organism may also be present in the allograft (Wade et al., 2004).

Meticulous nursing care is essential to prevent this potentially lethal complication. Preventive measures are crucial in this regard; they include meticulous handwashing; administration of antibiotic, antiviral, and antifungal agents (the choices will be facility specific); and discouraging visitation by persons with colds or flu, or by persons who recently received a live vaccine. Invasive lines, catheters, and devices should be managed using aseptic technique and removed as early as possible. When feasible, early ambulation should be encouraged. Data do not support use of protective isolation garb when caring for post-transplant patients (Walsh et al., 1989). As discussed in Chapter 18, strict glycemic control decreases the risk of deep sternal wound infection. While the patient is intubated and on mechanical ventilation, evidence-based interventions should be implemented to prevent ventilator-associated pneumonia (VAP). Guidelines to prevent VAP may be found on the American Association of Critical-Care Nursing's Web site in the "Practice Alerts" section.

Assessment for and prompt recognition of presence of signs and symptoms is equally important. These may include fever, hypotension, tachycardia, increased cardiac output/cardiac index, decreased systemic vascular resistance, increased oxygen requirements, mental status changes, elevated white blood cell count, and changes in the incision (e.g., purulent drainage, redness, swelling) (Wade et al., 2004).

The most common pneumonia-causing bacteria found in heart transplant patients in the immediate postoperative period (the first month) are gram-negative bacilli (GNB). *Staphylococcus epidermidus, Staphylococcus aureus,* and GNB are the organisms most commonly linked to sternal wound infections and mediastinitis. Urinary tract infections in this patient population are often caused by GNB, enterococcus, or *Candida albicans.*

After the first month, pneumonia may develop secondary to CMV, HSV, or *P. carinii* pneumonia (PCP). Cutaneous infections with HSV, VZV, atypical *Mycobacterium* species, or *Cryptococcus* may develop as well (Bethea et al., 2003).

Infection Prophylaxis

Several well-established procedures to minimize infectious risks exist. First, the heart donor is screened for HIV, hepatitis B virus (HBV), hepatitis C virus (HCV), CMV, and *Toxoplasma gondii.* Likewise, the heart recipient is screened before transplant for antibodies to CMV, toxoplasmosis, HIV, HSV, VZV, HBV, HCV, *T. gondii,* endemic fungi, and EBV. Presence of antibodies indicates previous infection and confirms the risk for reactivation of these infections in the setting of immunosuppression. The patient also receives a tuberculin skin test (Bethea et al., 2003). Prophylactic medications are used to prevent reactivation of a latent infection when appropriate.

To prevent de novo (new infection in recipient) infections with PCP, patients receive trimethoprim-sulfamethoxazole (Bactrim®), dapsone, or inhaled pentamadine (Nebupent®). Nystatin (Mycostatin®) or clotrimazole (Mycelex®) is administered to prevent *Candida* infection. Ganciclovir (Cytovene®) is administered to prevent CMV infection; the patient will be discharged home on acyclovir (Zovirax®). Patients are also placed on routine endocarditis precautions (Bethea et al., 2003). Although these regimens are highly effective in preventing infectious complications, infec-

tion remains a significant cause of mortality through the 1-year mark following transplant (Taylor et al., 2007).

Transplant Vasculopathy

Transplant vasculopathy (TV) refers to allograft coronary artery disease (ACAD) in the transplanted heart. It is the leading cause of death in patients after the first 5 years following a heart transplant (Bethea et al., 2003). TV in transplant recipients is distinguished from CAD in other populations in that it results in diffuse luminal narrowing rather than discrete lesions. Therefore, it is often not amenable to percutaneous or surgical intervention. Another difference is the absence of collateral circulation (Bethea et al., 2003). TV remains the predominant barrier to long-term survival following a heart transplant (Aranda & Hill, 2000).

The etiology of TV may be either immunologic or non-immunologic. Examples of non-immunologic factors leading to development of TV include the age of the donor, hyperlipidemia, and CMV infection (Bethea et al., 2003). Development of TV may begin weeks following transplant and evolve at a subtle, yet rapid pace, resulting in complete obliteration of the lumen of coronary vessels and allograft failure due to ischemia (Bethea et al., 2003).

At present, the best treatment for TV is retransplantation, because the coronary luminal damage tends to be diffuse. The need for a prudent lifestyle and preventive measures are emphasized to include smoking cessation, control of hypertension, and cholesterol reduction (with diet and medications). Statins and calcium channel blockers have been shown to decrease the incidence of TV (Bethea et al., 2003; Wenke et al., 2003).

Several medications—sirolimus, everolimus, and MMF—have been shown to decrease the incidence of both vasculopathy and rejection (Patel & Kobashigawa, 2004). More research is needed in this area, however.

Post-transplant Surveillance

Because the transplanted heart is denervated (the nerve supply has been cut), the transplanted heart will not receive autonomic nervous system or vagal nerve stimulation. As a result, the patient's heart rate will be higher than normal. Transplant recipients will also have physiologically altered responses to stress; the denervated heart is unable to compensate with an increase in heart rate to maintain cardiac output. This lack of response makes orthostatic hypotension difficult to manage. Further, angina is not experienced in response to ischemia. Dyspnea on exertion may increase, although early signs of rejection or TV may be absent.

Surveillance is implemented to screen for the development of any or all of these problems. An ECG, surface or transesophageal echocardiogram (TEE), stress test, and left heart catheterization are performed at regular intervals in all patients who undergo a heart transplant (Bethea et al., 2003; Wade et al., 2004).

OUTCOMES AFTER HEART TRANSPLANT

The Registry of the International Society for Heart and Lung Transplantation's *24th Official Adult Heart Transplantation Report* provided detailed data on outcomes following transplant; these data are summarized in Table 19–5. Currently, graft half-life (the time at which 50% of all transplant recipients remain alive) is 10 years.

Kaplan-Meier curves show a steep decline in survival during the first 6 months following heart transplant, then a linear decline. Survival during the first 6 to 12 months after transplant is improving, but has not changed beyond that point in patient recovery. Causes of death vary by time after transplant. In the first 30 days post-transplant, in order of decreasing frequency, graft failure, multi-organ failure, and non-CMV infection caused 67% of deaths. From 31 to 365 days post-transplant, non-CMV

Table 19–5 Outcomes Within One Year Following Transplant, 2000–2003, and Within Ten Years Following Transplant, 1994–2006

	Within 1 Year	Within 10 Years
Hypertension	76.8%	98%
Renal dysfunction		
All	31.7%	14%
Abnormal creatinine < 2.5 mg/dL	22.1%	
Creatinine > 2.5 mg/dL	7.8%	8%
Long-term dialysis	1.5%	5%
Renal transplant	0.3%	1%
Hyperlipidemia	68.7%	93%
Diabetes	30.2%	37%
Coronary artery vasculopathy	7.0%	53%

Sources: Anderson, 2008b; Taylor et al., 2005, 2007.

infection (33%), graft failure (18%), and acute rejection (12%) are the leading causes of death. After 5 years, TV and late graft failure explain 30% of deaths, followed by malignancies (22%) and non-CMV infections (10%). In summary, non-CMV infection is the leading single cause of death from 6 months through 10 years postoperatively. Transplant vasculopathy (confirmed) and "graft failure" collectively cause more deaths than infection in this patient population (Taylor et al., 2007).

PATIENT TEACHING

The complexity of transplant care and the consequences of noncompliance make effective patient teaching critically important. Adherence to mandated care regimens is more likely if patients understand why these practices are important. For this reason, several educational strategies are used to relay information and confirm understanding. Methods of teaching include provision of printed information, verbal instruction from multidisciplinary team members, opportunities to practice skills, demonstration, and return demonstration. Patients are also provided with contact information so they can call with questions.

Infection Avoidance

Because infection poses one of the greatest threats to the transplant patient, avoidance and recognition of infection constitute a major focus of teaching. Common signs of infection are reviewed and patients are instructed to call if they develop any of these signs. Response to infection will be blunted in the immunosuppressed patient, so signs of infection may be nonspecific (e.g., malaise, "don't feel right"). Patients are advised to avoid people who have received live vaccines, and to check with their transplant provider before receiving any immunization. Other routine practices include proper handwashing, avoiding contact with people who are ill, observing standard food hygiene procedures, and avoiding stagnant water, gardening, and digging. Patients frequently assume that the risk of gardening can be avoided if their hands are protected with gloves; in reality, the risk with gardening relates to inhalation of spores mobilized by manipulation of dirt. Finally, patients are encouraged to avoid any unnecessary medical procedures in the first 6 months after transplant.

Activity

As part of patient teaching, the nurse must review the anticipated schedule of return visits. Clearance to return to work or school or to resume driving will depend on each individual's progress. Physical restrictions include no lifting, pulling, or pushing of any item weighing more than 10 pounds for the first 6 weeks post-transplant. This is followed by orders not to lift, pull, or push anything weighing more than 25 pounds for at least 12 weeks. Patients are also provided with a MedicAlert® bracelet.

Nutrition

Nutritional counseling is provided as patient education. Patients should consume a diet low in sodium, potassium, cholesterol, and saturated fat. They are instructed to avoid nutritional supplements except as directed by their transplant team.

An increase in acute (grade 3A) rejection has been noted in late post-transplant patients (more than 1 year post-transplant) who started taking nutritional supplements that contained echinacea, zinc, or coenzyme Q10. In a study examining this phenomenon, the patients had no hemodynamic compromise, subtherapeutic levels of antirejection medication, or rejection episodes in the 6 months preceding the acute rejection. The rejection persisted despite steroid pulse therapy, suggesting that the rejection was resistant to steroids. It was noted that each of the supplements involved stimulates immunologic activity by destroying levels of IL-2, IL-6, tumor necrosis factor, interferon, and natural killer cells and by altering cytochrome P450 pathways. It was suggested that nurses amend their screening procedures to include nutritional supplements such as "power bars," which may contain at least one of these supplements (Boguszewski et al., 2006).

Exercise

Transplant patients are prescribed a specific exercise program by physical therapists. This program is implemented during hospitalization and continued upon discharge. Patients are also referred for cardiac rehabilitation. Teaching includes information regarding transplanted heart physiology.

The transplanted heart responds differently to exercise than does a native heart. For instance, the resting heart rate is higher because of the lack of vagal innervation. Heart rate is slower to increase with exercise, and slower to return to baseline following physical exertion. In addition, the maximal heart rate is lower in a transplanted heart.

When engaging in exercise, transplant patients are advised to warm up and cool down for 7 to 10 minutes, and to self-monitor perceived exertion and degree of dyspnea. They are taught to monitor for other subjective symptoms such as dizziness, unusually heavy perspiration, nausea, and a general sense of fatigue.

Long-term physical activity is important to ensure favorable results following heart transplantation. Studies suggest that hypertension, hyperlipidemia, doubts about expected benefits, side effects of immunosuppressant therapy, perceived negative sense of well-being, poor self-efficacy, continued cigarette smoking, and obesity are associated with a lower level of physical activity among women who receive heart transplants (Evanelista, Dracup, Doering, Moser, & Kobashingawa, 2005; Franklin, Swaim, & Shepard, 2003; Kobashingawa et al., 1999; Yates, Price-Fowlkes, & Agrawal, 2003). Exercise training in heart transplant patients has also been found to increase capacity for physical work (Kobashigawa et al., 1999). Further, data suggest that endurance and strength training by heart transplant patients may improve muscle function and aerobic performance, decrease side effects of immunosuppression agents,

and control risk factors associated with cardiac allograft vasculopathy (Marconi & Marzorati, 2003).

Sexual Activity

Issues related to sexuality are also included in discharge teaching; specific instructions are provided, as well as opportunity to discuss concerns. Sexual activity may be resumed as soon as the patient feels ready. For 6 weeks, patients should avoid putting weight on their arms and chest. Sexual activity may be aerobic and should be approached as such, with appropriate warm-up (e.g., foreplay) and cool-down (e.g., cuddling) periods being employed.

Patients should be given the opportunity to discuss other sexuality-related issues, such as anxiety, lack of desire, and body image concerns, and should be reassured that these concerns are not unusual. They should be invited to speak openly about their concerns and ask questions as they arise. Finally, all patients are strongly encouraged to avoid pregnancy due to the theoretical and documented risk to the fetus prenatally and later in life (Skotzko, Stowe, Wright, Kendall, & Dew, 2001; Subramaniam & Robson, 2008).

Other Instructions

Other instructions included in discharge teaching include recommendations regarding pet care, travel, and procedure for contacting the donor family. Sunscreen use is encouraged due to risk of photosensitivity and increased risk of skin cancer associated with immunosuppressive medications. Skin squamous cell carcinoma is a significant cause of morbidity and mortality in organ transplant patients (Ulrich, Degen, Patel, & Stockfleth, 2008).

Medications

Transplant patients are prescribed multiple medications that must be taken exactly as prescribed. The consequences of noncompliance (whether intentional or inadvertent) may be devastating. Every effort is made to ensure that the patient understands the dosing and administration regimen for all medications to be taken. Printed information should be provided to patients listing each medication prescribed along with its mechanism of action, side effects, interactions, dosing schedule, and procedures to be followed for missed doses. Due to the risk of interactions or compounded toxicities, patients are instructed not to take any over-the-counter medication without clearance from the transplant provider. Exceptions to this rule include acetaminophen, acetylsalicylic acid, docusate sodium (Colace®), senna, loperamide (Immodium®), and bismuth subsalicylate (Kaopectate®).

New prescriptions from non-transplant-care providers should also be cleared by the transplant team. Patients are instructed to avoid all NSAIDs. Herbal teas, medications, and other nutritional supplements should also be cleared with the transplant team.

SUMMARY

The ICU nurse plays a pivotal role in optimizing outcomes of patients and families during HF exacerbations and the wait for a heart transplant. High levels of critical thinking are required to care for the patient on inotropic or mechanical support. Evaluating the patient's candidacy for transplant and supporting the patient and family as they await availability of a suitable donor heart require high levels of clinical inquiry and caring practices. The ICU nurse also has a role as a facilitator of learning as the patient and family learn about management strategies for HF and about the transplant process. Bridge to transplant therapy requires the nurse to provide realistic information to both patients and families about the percentage of patients (20–40% [Leeper,

2006]) who may die while receiving VAD therapy either due to complications or while awaiting a heart transplant.

Post-transplant patients have a high degree of vulnerability in the immediate postoperative period. The ICU nurse must monitor for, promptly recognize, and treat the significant complications that can affect short-, intermediate-, and long-term survival following heart transplantation.

CASE STUDY

A 19-year-old college football player presented to the ED with chest pain, dyspnea, and fatigue. Six months ago, he was diagnosed with non-ischemic cardiomyopathy, thought to be related to a viral illness. At the time of his diagnosis, an echocardiogram revealed an ejection fraction of 13% with severe mitral regurgitation (MR), tricuspid regurgitation (TR), and moderate pulmonary hypertension. The patient was started on Coumadin® for his heart wall motion abnormalities and discharged home on metoprolol (Lopressor®), furosemide (Lasix®), and lisinopril (Prinivil®). Several days after his discharge, he developed aphasia. MRI confirmed a stroke. The patient's speech returned to normal within 24 hours with no other deficits. Aside from an embolic stroke and heart failure, the patient has no other significant past medical history.

At the time of his hospital admission, the patient reported that he had been doing well at home up until 1 week ago, when he began to experience poor appetite, fatigue, and worsening dyspnea. The night prior to admission, he began having nonradiating, constant chest pain with no aggravating or alleviating factors. He denied associated lightheadedness, palpitations, or lower extremity edema. He was admitted to the hospital with the concern of worsening heart failure.

Examination

Physical findings at the time of the patient's admission showed a normal neurologic exam. Vital signs were within normal limits, with the exception of a heart rate of 120. Jugular vein distention (JVD) was noted. Breath sounds were clear throughout. Examination revealed S_1 and S_2 heart sounds, with a holosystolic murmur best heard at the apex. The patient's abdomen was soft with normal bowel sounds; the liver edge was not palpable. The patient had moderate lower extremity edema. Bilateral lower extremities were cool to the touch, with 1+ pedal pulses.

Pertinent Laboratory Results

Na = 135 mEq/L, K = 4.3 mEq/L, BUN = 17 mg/dL, Cr = 1.3 mg/dL
WBC = 7.9 mm^3, Hgb = 13.2 g/dL
TCK = 215 units/L, CKMB = 2.1 ng/mL, troponin = 0.06 ng/mL
BNP = 275 pmol/L

Diagnostics

An ECG revealed sinus tachycardia, normal axis, normal intervals, left ventricular hypertrophy by voltage criteria, and no ST or T wave changes. A chest radiograph revealed an

enlarged cardiopericardial silhouette. There was demonstration of diffuse airspace opacities, likely representing mild pulmonary edema. No effusions or pneumothorax was identified. Cardiac MRI revealed marked cardiomyopathy of both the left and right sides of the heart secondary to cardiomyopathy. The patient had global hypokinesis and a markedly reduced ejection fraction of 13.6%. Right heart catheterization results were as follows: RA 27, RV 57/27, PA 57/33, PA mean 43, PAOP 25, CO 3.3 L/min, and CI 1.3 L/min/m^2.

Impression/Diagnosis

The right heart catheterization and echocardiogram results were consistent with worsening heart failure, biventricular fluid overload, and worsening cardiac output.

Decision Making

The patient was transferred to the ICU for closer hemodynamic monitoring. A pulmonary artery catheter was inserted, and the patient was started on milrinone at 0.25 mcg/kg/min for low cardiac indices. The infusion was titrated up to 0.5 mcg/kg/min. The patient was placed on a fluid restriction, diuresis with intravenous furosemide was begun, and a heparin infusion was initiated.

This patient is a 19-year-old male with non-ischemic cardiomyopathy of 6 months' duration, who had an acute exacerbation of his heart failure symptoms and became inotrope dependent with unsatisfactory hemodynamics and resting symptoms. His cardiac indices remained less than 2 L/min/m^2 on high-dose milrinone therapy. In addition, he had a significantly elevated resting heart rate at 120 beats per minute. Based on these findings, the patient was at risk for death from progressive heart failure and ventricular dysrhythmias. It was recommended that he undergo evaluation for heart transplantation, with left ventricular assist device (LVAD) therapy serving as a bridge to heart transplant.

Critical Thinking Questions

1. How are patients selected for VAD therapy?
2. What are potential complications when using an LVAD?
3. Why was milrinone utilized for this patient?

Answers to Critical Thinking Questions

1. Selection criteria for patients requiring VAD therapy are rigorous; a battery of labs and diagnostic tests are performed to determine eligibility for this therapy. Cardiologists, social workers, nurses, VAD coordinators, and the cardiac surgeon collaborate to determine the patient's eligibility and develop a plan of care. The surgeon determines the type of assist device support required. LVAD recipients must have adequate right ventricular function to have a successful outcome because the LVAD is dependent on blood flow from the right ventricle. If right ventricular failure is present, then length of hospital stay, post-implantation morbidity/mortality, and cost are all increased (Decoene et al., 2004). In such cases, temporary biventricular support may be initiated, with discontinuation of the RVAD if right ventricular recovery occurs.
2. LVAD placement is a major surgical procedure that carries a significant risk for postoperative complications. The most common complications are bleeding, hypo-

volemia, tamponade, organ damage (i.e., renal failure, liver failure), stroke, device failure, need for inotropic support for right ventricular failure, sepsis, and drive-line infection (Piccione, 2000). Bleeding in the immediate postoperative period is common due to the frequent use of aspirin and Coumadin® in HF patients preoperatively for severely depressed left ventricular function, coronary artery disease, or dysrhythmias.

3. Inotropic agents such as dopamine, dobutamine, and milrinone are used to support right and left ventricular function. Milrinone is frequently administered at a dose of 0.125–0.375 mcg/kg/min and has the added benefit of promoting pulmonary vascular dilation.

SELF-ASSESSMENT QUESTIONS

1. What are the possible postoperative complications following LVAD implantation?
   a. Bleeding and hypervolemia
   b. Stroke and reflex tachycardia
   c. Left ventricle failure and bleeding
   d. Tamponade and left ventricular depression
2. Your patient has a history of right ventricular failure and requires support from a ventricular assist device. Which of the following devices will most likely be utilized?
   a. Heartmate VE
   b. TandemHeart
   c. Thoratec
   d. Heartmate XVE
3. For which of the following patients would a heart transplant be contraindicated?
   a. A 47-year-old patient with early-stage Alzheimer's disease
   b. A 68-year-old patient with unstable diabetes mellitus
   c. A 28-year-old patient with protein calorie malnutrition
   d. A 32-year-old patient who received the hepatitis B vaccine
4. Which statement by a heart transplant candidate indicates a need for additional education?
   a. "I am looking forward to living without coronary artery disease following the transplant."
   b. "My HbA_{1c} is 7% this week; I'm doing well."
   c. "I am surprised I will have to stay on Lipitor® (atorvastatin calcium) for the rest of my life."
   d. "My wife and I will have to find another hobby to share because I can no longer garden."
5. The patient with which preoperative hemodynamic profile is at risk for development of right heart failure following transplant?

	B/P	PAP	CVP	PAOP
a.	163/71	25/10	3	4
b.	132/74	42/26	10	12
c.	88/50	30/20	16	15
d.	140/82	34/15	4	14

6. Your postoperative heart transplant patient develops atrial fibrillation. Which of the following should *not* be used?
   a. Amiodarone
   b. Digoxin
   c. Calcium channel blocker
   d. Cardioversion

7. Your postoperative heart transplant patient develops sinus bradycardia with mental status changes. Which of the following medications is *least* likely to be used initially?
   a. Isoproterenol (Isuprel®)
   b. Atropine sulfate (Atropine®)
   c. Epinephrine (Adrenalin®)
   d. Dopamine (Intropin®)
8. Which of the following increases pulmonary vascular resistance?
   a. Alkalosis
   b. Nitric oxide
   c. Nitroglycerin
   d. Hypoxemia
9. Which of the following patients is most likely to have a more challenging immediate postoperative heart transplant recovery?
   a. A patient who received a heart with a cold ischemic time of 4 hours
   b. A patient who underwent heart transplantation using the biatrial technique
   c. A patient who was supported on a left ventricular assist device preoperatively
   d. A patient who had preoperative left ventricular dysfunction
10. A postoperative cardiac transplant patient will be unable to manifest which of the following conditions?
    a. Angina
    b. Reflex tachycardia
    c. Vasovagal response
    d. Decreased cardiac output

Answers to Self-Assessment Questions

1. d
2. c
3. a
4. a
5. b
6. b
7. b
8. d
9. b
10. d

Clinical Inquiry Box

Question: What is the incidence of ventricular dysrhythmia in VAD therapy?

Reference: Bedi, M., Kormos, R., Winowich, S., McNamara, D., Mathier, M., & Murali, S. (2007). Ventricular arrhythmias during left ventricular assist device support. *American Journal of Cardiology*, *99*(8), 1151–1153.

Objective: To identify the incidence, risk factors, and clinical significance of ventricular dysrhythmias with ventricular assist device therapy.

Method: A descriptive study was conducted in 111 patients who received LVAD support as a bridge to cardiac transplant. Data were collected on the frequency of ventricular dysrhythmia (ventricular fibrillation, sustained ventricular tachycardia, or nonsustained ventricular tachycardia). Risk factors for the development of ventricular dysthythmia were analyzed. The mortality rate was compared for those individuals who had ventricular dysrhythmias and those patients who were dysrhythmia free.

Results: Ventricular dysrhythmias occurred in 24 patients (22%) during device support. Ischemic heart disease was the cause of heart failure in 71% of patients with ventricular dysrhythmias, compared to 45% of patients without dysrhythmias. The mortality rate was significantly higher during LVAD support in the group with dysrhythmias (33%) in contrast to the mortality rate in those patients without dysrhythmias (18%). The earlier the dysrhythmias occurred, the higher the potential for mortality.

Conclusion: Early occurrence of ventricular dysrhythmias after VAD therapy initiation predicts a higher mortality rate. Nurses need to be vigilant in assessing for ventricular dysrhythmias for timely interventions.

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WEB RESOURCES

Cardiac surgery: http://www.youtube.com/watch?v=qVYiGdQKP4s

Ventricular assist device: http://www.youtube.com/watch?v=DLV6kIfvSDA

Life on the transplant list: http://www.youtube.com/watch?v=xS7v4M-VmGw

Innovations in heart and lung transplantation: http://www.youtube.com/watch?v=BxwQxI6n0bE

Deciding whether to have transplant surgery: http://www.youtube.com/watch?v=Q0qQX6Ps79c

Orthotopic heart transplant: www.youtube.com/watch?v=N7etGEtdCCk

ECMO: www.youtube.com/watch?v=Psci-wZKN_s

Glossary

ACORN cardiac support device: A polyester mesh fabric that is wrapped snugly around the ventricles. It provides passive support to the ventricles, which should reduce wall stress and prevent further remodeling.

Afterload: The resistance against which the left ventricle must pump to move blood forward. The pressure of the arterial systemic circulation produces afterload. Smooth muscle tone in the arterioles can increase the resistance to blood flow and increase afterload. Medications can alter the amount of resistance that arteriolar smooth muscle generates.

Allograft: The transfer of an organ from one person to another. The donor is not a twin, but is of the same species.

Allograft coronary artery disease (ACAD): Development of coronary artery disease in heart transplant patients. It can be described based on the degree of stenosis of the affected vessel(s). ACAD is often associated with graft failure.

Alveolar-arterial oxygen gradient (A-a gradient): A method of measuring intrapulmonary shunt. The calculation is the difference between the concentration of alveolar oxygen entering the alveoli and the concentration of oxygen diffused into the arterial blood.

Ankle-brachial index: An assessment used to evaluate arterial blood flow to the lower extremities. Results of this calculation are used to rate the degree of peripheral artery disease and to determine if the saphenous vein is suitable for use during cardiac surgery.

Annuloplasty: Surgical repair of an ineffectual heart valve.

Aortic regurgitation: Incomplete closure of the aortic valve leaflets, resulting in a backflow of blood. There is a reflux of blood from the aorta into the left ventricle (LV) during diastole because the valve leaflets fail to close completely and remain tightly closed during diastole. Acute aortic regurgitation imposes a large volume load on the LV, which a normal heart cannot accommodate. The sudden increase in end-diastolic volume (preload) will result in increased left ventricular end-diastolic pressure (LVEDP) and decreased cardiac output. Aortic regurgitation is identified by the presence of an early diastolic murmur that can be heard at the second and third intercostal spaces at the right sternal border and the second and fourth intercostal spaces at the left sternal border. The murmur of aortic regurgitation usually decreases in intensity and disappears before S_1.

Aortic stenosis: Narrowing or constriction of the aortic valve that creates a pressure gradient. The aortic valve does not open completely, which creates a left ventricular outflow tract obstruction and increases both

the workload and afterload of the left ventricle. The calcification of aortic stenosis is regarded as a proliferative and inflammatory process, similar to atherosclerosis.

Arterial pulse contour continuous cardiac output monitoring: A method that estimates cardiac output by use of pulse contour analysis; it is an indirect method based on analysis of the arterial pressure pulsation waveform. The key underlying concept is that the contour of the arterial pressure waveform is proportional to stroke volume. The arterial pressure waveform is used to calculate cardiac output, stroke volume variance, intrathoracic volumes, and extravascular lung water. These data may predict response to fluid therapy.

Assisted aortic end-diastolic pressure: The pressure in the aorta at the end of diastole when counterpulsation has assisted the cardiac cycle. It is usually lower than the unassisted end-diastolic pressure.

Assisted systole: The systolic aortic pressure when counterpulsation has assisted the cardiac cycle. It is usually lower than the unassisted systole due to the action of balloon deflation.

Atrial cuff technique (bicaval technique): A method used during heart transplantation in which the donated heart is attached to the recipient's atrial "cuffs."

Atrial septal defect (ASD): An opening between the right and left atria. Oxygenated blood leaves the left atrium and returns to the right atrium through this opening rather than continuing forward to deliver oxygen to cells, organs, muscles, and tissues throughout the body.

Balloon valvotomy/valvuloplasty: Use of a balloon to stretch open a narrowed heart valve or to break adhesions in a scarred valve.

Beating heart surgery: See *off-pump coronary artery bypass.*

Bicaval technique: A method used during heart transplantation in which the anastamoses are made in the superior and inferior vena cavae.

Biologic valves: Valves that are constructed from bovine, porcine, and human cardiac tissue.

Biventricular assist device (BiVAD): A type of mechanical support for the heart. It is used when both the right and left ventricles are failing. Blood is drained from each ventricle through cannulae to centrifugal pumps, which provide circulatory support in severely decompensated heart failure patients until a heart transplant can be performed. A BiVAD is typically used when a left ventricular assist device does not provide sufficient circulatory support.

Bridge to recovery: Use of a mechanical circulatory device (ventricular assist device [VAD]) to support circulation in patients with heart failure. If myocyte damage is not permanent, myocardial cells may regain their ability to function. The VAD supports the patient until heart function improves and is adequate without mechanical support.

Bridge to transplantation: Use of a mechanical circulatory device (ventricular assist device [VAD]) to support circulation in patients with severe heart failure until a donor heart becomes available and a transplant can be performed.

Cardiac allograft vasculopathy: See *coronary artery vasculopathy.*

Cardiac catheterization: An invasive diagnostic test whereby a catheter is inserted and advanced into the heart chambers or coronary arteries. It reveals information about the blood pressure of the heart and the heart's ability to pump, blood flow in the heart chambers, presence and degree of narrowing of the coronary arteries, and valve function.

Cardiac output (CO): A measure of the amount of blood that is ejected by the heart each minute. It is affected by the individual's preload, afterload, and contractility.

Cardioplegia: A method of intentionally arresting the heart's motion with infusion of a solution to facilitate performance of cardiac surgery. The solution contains potassium (to decrease myocardial oxygen consumption and the rate of anaerobic metabolism while the heart is ischemic), magnesium (to decrease myocardial oxygen consumption), calcium (to decrease the chance of reperfusion injury), procaine (vasodilator and antiarrhythmic; may decrease dysrhythmias following aortic cross-clamping), bicarbonate (to counter the metabolic acidosis that occurs secondary to anaerobic metabolism while the heart is in arrest), hypothermia (decreases myocardial oxygen consumption and increases the heart's tolerance to ischemia), mannitol (to decrease edema related to hypothermia and ischemia, and may minimize reperfusion injury), dextrose (to counter edema due to hypothermia and ischemia, and for continued energy production), amino acids (for energy production, may minimize reperfusion injury, and has a role as a scavenger for oxygen free radicals), and oxygenated blood (to optimize the heart's metabolic environment and minimize reperfusion injury). The patient's circulation is diverted to a heart–lung machine that takes over the function of these two organs. The heart is isolated from the body with cross-clamping of the aorta. A cold cardioplegic solution is then instilled to decrease myocardial oxygen consumption and increase the heart's tolerance to ischemia, thereby preventing heart damage during the procedure.

Cardiopulmonary bypass (CPB): The temporary rerouting of blood from the right atrium to the aorta via an oxygenator (bypass machine), thereby bypassing the heart and lungs during the surgical procedure.

Carotid bruit: A sound associated with turbulent blood flow that may indicate arterial stenosis.

Central venous oxygen saturation ($ScvO_2$): A method used to determine how much oxygen the tissues are extracting. It entails analysis of a blood sample from a central venous catheter.

Cold ischemia time: The time from cross-clamping of the donor heart, with subsequent excision and immersion of the heart in iced saline, to removal of the cross-clamp after the donor heart's implantation in the recipient.

Commissurotomy: A procedure that opens commissures (the contact area for the valve leaflets), which have developed scarring and do not open to allow blood to flow.

Contractility: The rate and ability of the myocardial muscle to shorten itself, or the amount of strength evidenced by the myocardium when it ejects blood. It is influenced by heart rate, neural factors, and certain metabolic states.

Coronary artery bypass grafting (CABG): See *surgical revascularization.*

Coronary artery vasculopathy (CAV): A type of stenosis caused by plaque in the coronary arteries. The lesions contain inflammatory cells (including T cells). CAV is a major cause of long-term morbidity and mortality in heart transplant patients who survive past the first year. Innate and adaptive immune responses result in development of vascular lesions.

Cox/Maze III procedure: A modification of a procedure that interrupts the reentrant pathways required for atrial fibrillation using surgical incisions. The Cox/Maze III procedure remains the standard surgical therapy for atrial fibrillation. It entails a number of incisions being made on the right and left atria. "Maze" refers to the pattern of incisions made in the atrium. The incisions cause scarring, which does not conduct electricity, stops irregular electrical activity, and eradicates atrial fibrillation. The scarring also prevents future irregular electrical signals from developing.

Cox/Maze IV procedure: A procedure that uses radiofrequency ablation to eradicate atrial fibrillation.

Deep sternal wound infection: Infection of the sternum and underlying structures.

Destination therapy: Use of a ventricular assist device in patients with severe heart failure who are not candidates for or have declined heart transplant.

Diastolic augmentation: The increase in pressure in the aorta above the balloon catheter that results with balloon inflation during diastole. This phenomenon increases perfusion in the coronary arteries and myocardial oxygen supply.

Dicrotic notch: When referring to an intra-aortic balloon pump or intra-arterial pressure monitoring, an area on the downstroke of the arterial waveform that results from the slight pressure increase created by closure of the aortic valve.

Dor procedure: Also known as endoventricular circular patch plasty repair. A procedure whereby the left ventricle is reconstructed using a purse-string suture to isolate nonfunctional segments of myocardium (rather than excising them) and a circular patch to control the shape of the ventricle. The Dor procedure is usually performed concomitantly with a coronary artery bypass graft.

Drug-eluting stent (DES): A metal tube or "scaffold" inserted into a coronary artery following dilation of the vessel with a balloon (balloon angioplasty). The tube is coated with a drug to prevent reblockage (restenosis) of the vessel.

Dynamic cardiomyoplasty (DCMP): An innovative technique whereby the latissimus dorsi muscle is wrapped around the heart. An implanted stimulator is then used to stimulate the muscle to contract in synchrony with ventricular contraction. Due to the borderline clinical improvement associated with this technique, DCMP is rarely used in the United States, though it remains in use in other areas.

Dynamic response test: See *square wave test.*

Ejection fraction (EF): The percentage of blood volume of the left ventricle that is ejected with each contraction. A normal ejection fraction is approximately 65–70%.

Electrical bioimpedence: A noninvasive method to determine cardiac output. Using this technology, cardiac output is identified by changes in impedance that take place as blood is ejected from the left ventricle into the aorta and is calculated from changes in thoracic impedance. Change in thoracic blood volume during the cardiac cycle can be used to calculate cardiac output.

Endoaneurysmorrhaphy: A procedure that involves excising an aneurysm and reapproximating the wall edges using a Dacron patch to control the shape and size of the ventricle. It is used to treat ventricular tachycardia. Although this approach attempts to restore more normal ventricular geography, data indicate that it does not improve LV function.

Endoscopic atraumatic coronary artery bypass grafting (EndoACAB): A combination of both two methods to perform off-pump coronary artery bypass grafting. The internal mammary artery is harvested using an endoscopic approach, and the anastomosis is performed with direct visualization through a small thoracotomy incision.

Endovascular/"keyhole" procedure: A type of minimally invasive procedure. It entails use of a small (5-mm or 3-mm) endoscope to access the heart through the intercostal space. The 5-mm scope makes it easy to maneuver between ribs, increasing visibility, and has been used to close a patent ductus arteriosus, thereby eliminating the need for a thoracotomy. The digital camera and processing make pictures from the 5-mm scope better

than the pictures provided by the traditional surgical 10-mm scope. The 3-mm scopes are designed to feel and work like standard instruments used by cardiac surgeons.

Endoventricular circular patch plasty repair: See *Dor procedure.*

Ethanol septal ablation: A procedure for relieving outflow obstruction symptoms that is accomplished by infusing ethanol into the first septal branch of the left anterior descending coronary artery via an angioplasty catheter. This technique reduces outflow tract obstruction, increases exercise capacity, and improves symptoms.

Ex-Maze procedure: A cutting-edge technique for the Maze procedure. It is performed endoscopically on the outside of a beating heart. The ablation device uses unipolar radiofrequency energy with vacuum-maintained contact and suction-controlled saline perfusion to ensure uniform energy transmission and transmural lesion development. Because the procedure is performed on a beating heart, atrial function can be monitored during treatment. Patients can convert to normal sinus rhythm during the procedure or within 6 weeks. The Ex-Maze procedure is less invasive and does not require cardiopulmonary bypass. It is safer, associated with less postoperative pain, and has fewer complications.

Extracorporeal membrane oxygenation (ECMO): Use of a machine that can provide oxygen to the blood while it is circulating outside of the body. Blood is removed from the body via a catheter, pumped through a machine to be oxygenated by an artificial lung, and returned to the body through another catheter. The heart and lungs are bypassed, providing both hemodynamic and respiratory support. Blood oxygenation is a distinct advantage with this technique.

Fast flush: See *square wave test.*

Geometric mitral reconstruction (GMR): A procedure in which an annuloplasty ring is used to restore a more normal mitral valve anatomy that has achieved favorable outcomes. GMR has consistently resulted in significant improvements in ejection fraction. It is indicated for patients with cardiomyopathy and mitral regurgitation.

Graft closure: Failure of the harvested vein (graft) to maintain patency, usually due to platelet aggregation. Antithrombotic therapy is initiated to prevent this complication of coronary artery bypass surgery.

Heterotopic method: A heart transplantation technique utilizing end-to-end anastamoses of the donor to the superior vena cava, pulmonary artery, and aorta. The donor heart is placed "piggyback" to the recipient heart. This method may be used in patients with severe pulmonary hypertension or if there is a mismatch between the donor and recipient heart size. It is rarely used.

Hypertrophic cardiomyopathy (HCM): A common genetic cardiovascular disease characterized by abnormal myocytes leading to hypertrophy without dilatation and preserved systolic function. Hypertrophy is most severe in the ventricular septum. It is asymmetrical and usually occurs at the level of the LV outflow tract, leading to subaortic stenosis or asymmetrical HCM. In addition, abnormal systolic anterior motion of the mitral valve contributes to the outflow obstruction.

Hypothermia: A decrease in internal (core) body temperature below normal values. Often, a temperature less than 95 °F (35 °C) is considered hypothermia.

Infective endocarditis: A condition that occurs when bacteria attach to and destroy the surface of a valve leaflet or chordae. If a valve is damaged, immune cells, platelets, and fibrin migrate to the site to initiate healing of the valve. If bacteria become trapped under layers of these cells, "clumps" of tissue (called vegetations) can develop on the valves and within the heart muscle (endocarditis). Vegetations may break off and become emboli.

Internal mammary artery (IMA): An artery in the chest located adjacent to the left anterior descending coronary artery. There is one IMA on either side of the sternum. This artery is resistant to cholesterol buildup (atherosclerosis), which makes it a good choice for use as a graft in coronary artery bypass surgical procedures.

Intra-aortic balloon pump (IABP): A mechanical device that is used to improve cardiac function on a temporary basis. It increases blood flow, oxygen delivery to the heart, and cardiac output, and decreases the amount of work the heart must do to eject blood through a process called counterpulsation.

Intrapulmonary shunt (IPS): The percentage of cardiac output that does not participate in gas exchange. This portion of blood passes through the lungs but is not exposed to ventilated alveoli, so gas exchange does not take place and the blood leaves the lungs desaturated.

International Society of Heart and Lung Transplantation (ISHLT) grading system: The standardized cardiac biopsy system that is used to grade acute heart rejection.

Keyhole procedures: See *endovascular/"keyhole" procedures.*

Left-to-right shunting: Diversion of blood from the left heart to the right heart, rather than forward into the systemic circulation. Oxygenated blood from the arterial circulation mixes with deoxygenated blood from the venous system. Chronic left-to-right shunting may cause right ventricular failure, tricuspid regurgitation, atrial arrhythmias, paradoxical embolization, and cerebral abscesses.

Left ventricular assist device (LVAD): A type of mechanical support for the left ventricle. Blood is drained from the apex of the left ventricle to a pump via an inflow cannula. It is returned to the body via an outflow cannula, which is attached to the aorta. The pump is housed in the pre-peritoneal space of the abdomen, near the stomach. A percutaneous drive line that is tunneled across the pre-peritoneal space to the left side of the body carries an electrical cable and air vent to the electrical controller outside the patient's body.

Lower and Shumway method: A method used during heart transplantation in which the donor heart is anastomosed to the left atrium, right atrium, pulmonary artery, and aorta.

Maze procedure: See *Cox/Maze III procedure.*

Mean arterial pressure (MAP): The driving force for peripheral blood flow and the preferred pressure to be evaluated in unstable patients. It is measured electronically by first integrating the area under the arterial pressure waveform and then dividing by the duration of the cardiac cycle.

Mechanical assist device: A device used to support cardiac function over the short or long term. Mechanical circulatory support has three primary functions: bridge to transplantation, bridge to recovery, and destination therapy. Short-term, temporary devices are often used as bridge to recovery in the setting of acute cardiogenic shock or cardiopulmonary arrest. Under these circumstances, circulatory assistance provides immediate hemodynamic support, restoring blood flow to vital organs while decompressing the heart, avoiding pulmonary edema, and minimizing cardiac workload to maximize the patient's chances of recovery.

Mediastinitis: Inflammation of the mediastinum; an uncommon but severe complication following cardiac surgery. Its incidence is reportedly higher in patients who have undergone grafting with bilateral internal mammary arteries.

Minimally invasive cardiac surgery (MICS): An alternative approach to coronary artery bypass graft surgery that entails use of a laparoscopic procedure to perform cardiac

surgery. MICS has also been defined as cardiac surgery without the use of cardiopulmonary bypass or sternotomy; rather, smaller incisions are made. MICS also refers to various procedures used to bypass blocked coronary arteries.

Minimally invasive direct coronary artery bypass (MIDCAB): An alternative approach to traditional coronary artery bypass grafting (CABG). Differences between the two approaches are threefold. First, the incision size is much smaller for MIDCAB; several 3-inch to 5-inch incisions are made between the ribs as compared to a 10-inch to 12-inch median sternotomy incision in conventional CABG procedures. Second, because MIDCAB is a beating heart procedure, no cardioplegia is instilled to stop the heart. Third, because MIDCAB is a beating heart surgery and no cardioplegia is instilled, cardiopulmonary bypass (CBP) is not required for MIDCAB procedures. MIDCAB procedures are performed on patients with one or two blockages to the right coronary artery, left anterior descending coronary artery, or its branches on the front of the heart.

Minimally invasive direct view: Techniques that were developed to repair or replace the mitral valve and repair or replace the aortic valve. The main benefit of minimally invasive direct view valve surgery is the avoidance of a median sternotomy. An 8-cm incision is made and cartilage removed to allow for direct visualization of the valves.

Minute ventilation: The volume of gas exchange (inhaled and exhaled) in one minute. It is measured by multiplying respiratory rate and tidal volume.

Mitral regurgitation: Incomplete closure of the mitral valve leaflets, resulting in a backflow of blood into the left atrium during ventricular systole.

Mitral stenosis: Narrowing or constriction of the mitral valve that creates a pressure gradient. The narrowing creates resistance to the forward flow of blood into the left ventricle during diastole.

Mitral valve annuloplasty ring: A three-dimensional ring that improves mitral valve function and left ventricular shape.

Myocardial revascularization: Restoration of blood supply to the myocardium. It may be accomplished by either percutaneous intervention or surgery.

Myectomy: A procedure that involves excision of a section of sub-aortic septal muscle approximately 3–7 cm long and 3–12 g in weight, with or without mitral valve replacement. Left ventricular myectomy is recommended for patients with drug-refractory symptomatic outflow obstruction (peak gradient > 50 mm Hg under resting conditions and/or gradient > 50 mm Hg measured). Surgery may also be considered in symptomatic patients with documented outflow obstruction under physiologic exercise but with absent or very mild resting obstruction. One additional subset of patients may benefit from LV myectomy: young, asymptomatic patients with documented severe outflow tract obstruction (gradient 75–100 mm Hg).

Myxoma: A benign cardiac tumor. It causes obstruction of blood flow, which leads to the clinical presentation of heart failure, signs of central nervous system (CNS) embolization, and/or constitutional symptoms such as fever, weight loss, fatigue, weakness, arthralgia, and myalgia. Tumor resection is the only effective treatment.

Negative inspiratory pressure: Also referred to as negative inspiratory force. The amount of negative pressure that the patient generates during a forced inspiration when working against an obstruction to flow. It is a reflection of a patient's ability to take a deep breath and generate a cough that is strong enough to clear secretions.

Nitric oxide (NO): A product that is released by endothelial cells. It produces vasodilation and increased vascular permeability.

Off-pump coronary artery bypass (OPCAB): Also known as a beating heart procedure. This type of minimally invasive cardiac surgery entails a median sternotomy or thoracotomy incision; no bypass machine is required. The surgeon sews the grafts onto the beating heart using specialized instruments to stabilize the myocardial tissue. OPCAB may be performed on patients needing four or five vessels repaired, as compared with minimally invasive direct coronary artery bypass (MIDCAB), where only one or two vessels can be repaired. With OPCAB, an artery or vein from the lower extremities is used to make the bypass.

Orthotopic heart transplant: A heart transplant approach that entails replacing the recipient heart with the donor heart.

Overdamped waveform: A situation in which a pressure waveform is sluggish and has an exaggerated or falsely widened and blunt tracing. It will cause the patient's systolic pressure to be recorded as falsely low and the diastolic pressure to be recorded as falsely high.

Oxygen consumption: The amount of oxygen used by the body's tissues.

Oxygen delivery: The amount of oxygen that is carried to the body's tissues each minute.

PaO_2/FiO_2 ratio: An index of oxygenation. A PaO_2/FiO_2 ratio of less than 200 is associated with a significant intrapulmonary shunt.

Papillary fibroelastoma: A benign cardiac tumor that occurs on the heart valves and may cause obstruction or central nervous system (CNS) embolization.

Paroxysmal nocturnal dyspnea (PND): A feeling of shortness of breath that awakens the patient. It is usually relieved when the patient assumes an upright position.

Partial left ventriculectomy: A procedure to restore the proper mass-to-diameter ratio for the left ventricle. In this procedure, a section of the left ventricular wall from the apex to the mitral annulus is removed, and the edges are reapproximated. Improvements in signs of heart failure and ejection fraction have been achieved with this technique. Because other surgical procedures have achieved results superior to those produced with the partial left ventriculectomy, this procedure is no longer in use in most of North America; however, it is still used in other areas where cardiac transplantation is less readily available.

Percutaneous mitral balloon valvotomy (PMBV): See *commissurotomy.* This technique has very successfully reduced left atrial gradient, increased mitral valve area, and improved symptoms of mitral stenosis.

Percutaneous transluminal coronary angioplasty (PCTA): A technique that uses an arterial catheter and various mechanical means to increase the diameter of diseased coronary arteries, thereby improving blood flow.

Pericardiectomy: Surgical removal of part of the membrane that surrounds the heart (pericardium). It is usually performed to treat inflammation and prevent collection of fluid in the pericardial sac (between the pericardium and heart), which can cause hemodynamic compromise from poor cardiac filling and emptying.

Phlebostatic axis: An anatomic landmark located at the fourth intercostal space, midpoint of the anterior–posterior diameter. Leveling at the phlebostatic axis is performed to eradicate the effects of hydrostatic forces on the hemodynamic pressures.

Phrenic nerve injury: A complication following cardiac surgery with cardiopulmonary bypass. The extent of injury can range from neuropathy to paralysis of the diaphragm. It is often reported to be attributed to the use of hypothermia, application of ice slush around the heart, and harvesting of the internal mammary artery that occur during surgery.

Postcardiotomy cardiogenic shock: Heart failure that develops as a result of heart surgery or a heart attack.

Preload: The pressure found in the left ventricle at the end of diastole. It is sometimes referred to as left ventricular end-diastolic pressure. Right-sided preload is the pressure found in the right atrium at the end of diastole.

Prosthetic valves (mechanical valves): Valves that are manufactured from man-made materials such as metal alloys, pyrolite carbon, and Dacron. Mechanical prosthetic valves are more durable and last longer than biologic valves.

Pulmonary hypertension: High blood pressure in the arteries that supply the lungs and right side of the heart. It develops when these vessels become constricted or obstructed, which slows blood flow. The result is an increase in pressure in the pulmonary arteries, making it more difficult for the right ventricle to eject blood to the pulmonary arteries.

Pulmonary stenosis: Narrowing or constriction of the pulmonic valve that creates a pressure gradient.

Pulse oximetry: A noninvasive method of monitoring the percentage of hemoglobin that is saturated with oxygen.

Pulse pressure variation (PPV): An alternative, less invasive method of evaluating cardiac output. It may be used as a means for determining the patient's ability to respond to fluid. PPV is the difference between the maximum and minimum values of the arterial pulse pressure during one mechanical breath divided by the mean of the two values. In the evaluation of the Frank-Starling curve, an increase in preload causes a decrease in PPV; decreasing preload causes an increase in PPV and contractility.

Pulsus alternans: An exaggeration of the normal variation in the pulse during the inspiratory phase of respiration, in which the pulse becomes weaker as the person inhales and stronger as the person exhales. It is an indicator of the presence of severe ventricular systolic failure and decreased myocardial contractility.

Right-to-left shunting: The flow of blood from the right to left side of the heart, usually through an opening between the two atria or ventricles. Great vessels in the chest may be affected as well. Right-to-left shunting can be attributed to a patent foramen ovale, especially with conditions that increase right atrial pressure (e.g., tricuspid stenosis). An example is a patient with an atrial septal defect. The affected patient may have periods of cyanosis.

Right ventricular assist device (RVAD): A type of mechanical support for the right ventricle. Blood is drained through a pump from the right ventricle to the pulmonary artery.

Robot-assisted coronary artery bypass (RACAB): A cutting-edge surgical technique. Unaccommodating places is what robot-assisted surgery is about; the human surgeon is not optimized for tiny spaces. In RACAB, the surgical robot consists of a collection of wristed tools called manipulators, which receive digital instructions from an interfaced computer. The surgeon, who is seated at a computer console with a three-dimensional display, acts as the "driver" of the computer. The surgeon initiates the digital instructions by controlling the hand grips. By using the hand grips, the surgeon's hand movements at the console are then duplicated in the robot, with software filtering out physiologic hand tremors.

Saphenous vein: A vein in the patient's leg that runs near the leg's surface. It is used as a graft for coronary artery bypass procedures. There are actually two saphenous veins in the leg—the great (large) and small veins. When harvested for bypass procedures, long incisions are usually made. Almost directly upon harvest, the surface of the saphenous vein becomes vulnerable to platelet aggregation

because of the loss of the vascular endothelium. For this reason, patients require antithrombotic therapy to prevent graft closure.

Square wave test: Also referred to as a fast flush or dynamic response test; a test that is performed to assure that the waveforms that appear on the monitoring screen accurately reflect pressures. It is accomplished by pulling and releasing the "pigtail" or squeezing the button of the flush device so that the flow through the tubing increases (from 3 mL/hr obtained with a pressure bag inflated to 300 mm Hg). The sudden rise in pressure in the system generates a square wave on the monitor oscilloscope.

Stabilizer: A device used in minimally invasive cardiac surgery that provides a direct view of the operating field, dampens the movement of the epicardium, and permits the surgeon to maintain a nontraumatic grip on the beating heart. The device helps the surgeon isolate the diseased vessel and stabilizes the localized region of epicardium for anastomosis.

Steroid pulse: Administration of large doses of steroids over a short period of time to treat heart transplant rejection.

Stroke volume: The amount of blood ejected by the left ventricle with each contraction. Stroke volume is affected by the amount of blood in the ventricle and by the force of contraction of the ventricle. It can also be affected if the aortic valve restricts flow out of the left ventricle.

Stroke volume variation (SVV): An alternative, less invasive method of evaluating cardiac output. It may be used as a means for determining the patient's ability to respond to fluid. SVV occurs due to changes in intrathoracic pressure during spontaneous breathing. It produces data on changes in preload that occur with mechanical ventilation. SVV is the difference between the maximum and minimum stroke volume during one mechanical breath relative to the mean stroke volume.

Subendocardial resection (SER): A procedure that involves surgical removal of scar tissue, portions of an aneurysm, or other sites of abnormal electrograms. It is used to treat ventricular tachycardia.

Superficial sternal wound infection: An infection involving only the skin and subcutaneous fat. It is characterized by drainage from the wound and local inflammation while the underlying sternum remains stable.

Surgical anterior ventricular endocardial restoration (SAVER): A procedure that is a modification of the original Dor procedure. It is associated with a significant reduction of left ventricular volume and a significant increase in ejection fraction as well as significant reductions in hospitalizations for heart failure.

Surgical revascularization: Also known as coronary artery bypass grafting (CABG). Use of arterial or venous vessels to create a new pathway for blood to reach the coronary arteries, thereby "bypassing" a stenosis.

Sympathomimetics: Agents that activate adrenergic receptors by direct receptor binding, promotion of norepinephrine (NE) release, blockade of NE reuptake, and inhibition of NE inactivation.

Systolic pressure variation (SPV): An alternative, less invasive method of evaluating cardiac output. It may be used as a means for determining the patient's ability to respond to fluid. SPV is the difference between the maximum and minimum systolic blood pressure during one mechanical breath.

Tidal volume: The amount of air inhaled during a normal breath (versus a forced inhalation).

Total artificial heart (TAH): A treatment alternative for patients with biventricular failure who are hospitalized candidates for heart transplant. A TAH replaces the function of

both ventricles and the four heart valves. It is implanted in the patient's chest and attached to the atria. Tubes from the ventricles continue from the patient's chest to a power-generating console. The TAH can deliver cardiac output at a rate as high as 9.5 L/min. It reportedly augments renal and hepatic blood flow and improves survival of heart transplant patients with preoperative biventricular failure.

Totally endoscopic coronary artery bypass (TECAB): A method of performing off-pump coronary artery bypass grafting. It entails using endoscopy, as opposed to the minimally invasive direct coronary artery bypass (MIDCAB) approach, which uses small thoracotomy incisions.

Transmyocardial laser revascularization (TMR): A procedure whereby transmyocardial channels are created from the epicardium into the ventricle via a laser. The channels then allow blood from the ventricle to reach the myocardium directly.

Transplantation: The surgical removal of a diseased heart and replacement with a healthy donor heart.

Transplant vasculopathy: Accelerated coronary artery disease in the transplanted heart.

Tricuspid regurgitation: Incomplete closure of the tricuspid valve leaflets, resulting in a backflow of blood.

Tricuspid stenosis: Narrowing or constriction of the tricuspid valve, which creates a pressure gradient.

Tricuspid valve: The heart valve that is located between the right atrium and ventricle, near the atrioventricular (AV) node, right coronary artery, and coronary sinus. Its function is to maintain forward flow of blood. The tricuspid valve has an annular ring and three leaflets connected via chordae tendinae to papillary muscles that are integrated with the right ventricle

Unassisted aortic end-diastolic pressure: The pressure in the aorta at the end of diastole when counterpulsation via the balloon pump has not assisted that cardiac cycle.

Unassisted systole: The systolic aortic pressure when counterpulsation has not assisted the cycle.

Underdamped waveform: A situation in which a pressure waveform has an overresponse, revealed visually as an exaggerated, narrow, and artificially peaked tracing. In this case, the waveform overestimates the patient's systolic pressure and underestimates the diastolic pressure.

Valvuloplasty: A procedure that entails insertion of a balloon to stretch or enlarge the valve opening.

Venous oxygenation saturation (SvO_2): A method used to determine how much oxygen the tissues are extracting. Venous oxygen saturation reveals the association between oxygen delivery (the amount of oxygen that is carried to the tissues each minute) and oxygen consumption (the amount of oxygen used by the tissues).

Ventricular assist device (VAD): A device used for longer-term mechanical circulatory support. This implanted mechanical pump is used in patients with end-stage heart disease. VADs assist a weakened heart by pumping blood throughout the body.

Ventricular reconstruction: Techniques that are based on the principle that the ventricular wall tension is proportional to the left ventricular radius and pressure and inversely proportional to the wall thickness (Law of Laplace). By changing the size and shape of the ventricle, these techniques seek to reduce wall tension and improve left ventricular function. Specifically, surgical reconstruction techniques remove or isolate dysfunctional myocardium, reduce the diameter of the ventricle, and attempt to restore a more elliptical ventricular shape. Additional goals are to

relieve ischemia by revascularization if possible, and to further reduce ventricular size and volume via mitral valve repair.

Ventricular septal defect (VSD): An opening in the wall (septum) between the right and left ventricles. The result of this opening is the return of oxygenated blood in the left ventricle to the right ventricle, rather than the blood continuing forward into the systemic circulation to deliver oxygen to the cells, organs, muscles, and tissues. A VSD also results in an increase in ventricular workload because greater volumes of blood are being circulated; this effect ultimately leads to heart failure.

Vital capacity: The amount of air that can be exhaled forcibly following a full inspiration.

Zero balance: A process of establishing atmospheric pressure as zero to obtain accurate hemodynamic values.

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