BRUNNER & SUDDARTH'S

TEXTBOOK OF Medical-Surgical Nursing

16TH EDITION

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Preface

Since 1964, when Lillian Sholtis Brunner and Doris Smith Suddarth introduced the first edition of the *Textbook of Medical-Surgical Nursing*, the practice of nursing has flexed, changed, evolved, and advanced immensely to meet changing health needs and expectations for health care. With each subsequent edition of this textbook, Lillian and Doris and their successors, Suzanne C. Smeltzer and Brenda Bare (and eventually we, the current authors), updated and revised content to reflect changes and challenges that shaped the practice of nursing, considering complex and interconnected influences and maintaining a focus upon salient social, cultural, economic, and environmental factors. This latest edition focuses on incorporating issues related to diversity, equity, and inclusion as well as social determinants of health as appropriate to the content. In this context, we recognize people have diverse identities and have striven, to the extent possible, to use inclusive language unless it is clinically appropriate to use a less inclusive term, for example, when referring to a person's sex. However, when describing or referencing study populations used in prior research, we use the terminology reported by the sources cited to maintain fidelity to the research.

Organization

Brunner & Suddarth's Textbook of Medical-Surgical Nursing, 16th Edition, is organized into 16 units. These units mirror those found in previous editions with the incorporation of some changes. Content was updated throughout all units, with cross-references to specific chapters included as appropriate. Units 1 through 3 cover foundational principles and core concepts related to medical–surgical nursing practice. Units 4 through 15 discuss adult health conditions that are treated medically or surgically. Unit 16 describes community-based challenges that affect medical–surgical nursing practice.

Units 4 through 15 are structured in the following way to better facilitate comprehension:

- The first chapter in the unit covers assessment and includes a review of normal anatomy and physiology of the body system being discussed.
- Subsequent chapters in the unit cover management of specific disorders.
- Pathophysiology, clinical manifestations, assessment and diagnostic findings, medical management, and nursing management are presented. Nursing Process sections, provided for select conditions, clarify and expand on the nurse's role in caring for patients with these conditions.

There is one additional chapter in this edition than in the 15th edition. The comprehensive and lengthy Chapter 19 in the 15th edition that focused on disorders of the chest and lower respiratory tract is now split into two chapters. The first of these chapters, Chapter 19 in this edition, focuses on acute disorders while the second, Chapter 20, focuses on nonacute disorders of the chest and lower respiratory tract and pulmonary vascular disorders.

Cited references and resources are updated to include the most relevant and recent sources for each chapter. Many of these are web-based and may be in a state of flux. References note retrieval dates; however, resources only note the websites, some of which may remain active, while others may migrate or be unavailable. Rest assured that at the time of writing each chapter, the resource websites were active and vetted as valuable resources for students to review.

Special Features

When caring for patients, nurses assume many different roles, including practitioner, educator, advocate, and researcher. Many of the features in this textbook have been developed to help nurses fulfill these varied responsibilities. Key updates to practice-oriented features in the 16th edition focus on highlighting the Clinical Judgment Measurement Model (CJMM). The CJMM is introduced in Chapter 1 and has been integrated into all other chapters with the inclusion of relevant Clinical Judgment Alerts and *new* Clinical Judgment Exercises at the end of each chapter. Clinical Judgment Alerts indicate which step(s) in the CJMM (i.e., recognize cues, analyze cues, prioritize hypotheses, generate solutions, take actions, and evaluate outcomes) are addressed in the context of the highlighted content. The Clinical Judgment Exercises provide situation-based questions that ask students to apply their knowledge to patient care and support clinical decision making.

Nursing Research Profile charts, Genetics in Nursing Practice charts, Ethical Dilemma charts, and Patient Education charts offer updated information.

The Nursing Process continues to serve as the framework for discussion of nursing management of patients with various disorders, as it has for numerous editions. Whenever salient nursing diagnoses are identified, the diagnoses used are those devised and validated by the International Council of Nursing in the *International Classification for Nursing Practice (ICNP) Catalogue*. Because of the global foci of these nursing diagnoses, they are spelled in the British English manner. American spellings of other terms are used in every other context within the textbook.

The 16th edition has two *Considerations* sections. *Gerontologic Considerations* sections highlight information that pertains specifically to the care of the older adult patient. *Veterans Considerations* sections include information applicable to the special care needs of military veterans.

The textbook also provides pedagogical features developed to help readers engage and learn critical content. Unfolding Patient Stories (case study vignettes) based on vSim for Nursing patients continue as a feature.

Read the User's Guide that follows the Preface for a full explanation and visual representation of all special features.

A Comprehensive Package for Teaching and Learning

To further facilitate teaching and learning, a carefully designed ancillary package has been developed to assist faculty and students.

Instructor Resources

Tools to assist you with teaching your course are available upon adoption of this text at thepoint.lww.com/Brunner16e.

- An **e-Book** gives you access to the book's full text and images online.
- A thoroughly revised and augmented **Test Bank** contains more than 2,900 NCLEX-style questions mapped to chapter learning outcomes.
- An extensive collection of materials is provided for each book chapter:
  - **PowerPoint Presentations** provide an easy way to integrate the textbook with your students' classroom experience; multiple-choice and true/false questions are included to promote class participation.
  - **Guided Lecture Notes** are organized by outcome and provide corresponding PowerPoint slide numbers to simplify preparation for lecture.
  - Plans of Nursing Care, provided for select disorders, illustrate how the nursing process is applied to meet the patient's health care and nursing needs.
  - **Journal Articles** offer access to current articles relevant to each chapter and available in Wolters Kluwer journals to familiarize students with nursing literature.
- Sample **Syllabi** are provided for one- and two-semester courses.
- An **Image Bank** lets you use the photographs and illustrations from this textbook in your course materials.

Study Guide

A comprehensive study aid for reviewing key concepts, ***Study Guide for Brunner & Suddarth's Textbook of Medical-Surgical Nursing*, 16th Edition**, has been thoroughly revised and presents a variety of exercises, including case studies and practice NCLEX-style questions, to reinforce textbook content and enhance learning.

vSim for Nursing

Available for separate purchase, vSim for Nursing, jointly developed by Laerdal Medical and Wolters Kluwer, offers innovative scenario-based learning modules consisting of web-based virtual simulations, course learning materials, and curriculum tools designed to develop critical thinking skills and promote clinical confidence and competence. vSim for Nursing | Medical-Surgical includes 10 virtual simulations based on the National League for Nursing Volume I Complex patient scenarios. Students can progress through suggested readings, pre- and postsimulation assessments, documentation assignments, and guided reflection questions, and will receive an individualized feedback log immediately upon completion of the simulation. Throughout the student learning experience, the product offers remediation back to trusted Lippincott resources, including *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*, as well as Lippincott Nursing Advisor and Lippincott Nursing Procedures—two online, evidence-based, clinical information solutions used in health care facilities throughout the United States. This innovative product provides a comprehensive patient-focused solution for learning and integrating simulation into the classroom.

Contact your Wolters Kluwer sales representative or visit thepoint.lww.com/vsim for options to enhance your medical–surgical nursing course with vSim for Nursing.

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Available for separate purchase, Lippincott DocuCare combines web-based academic electronic health record (EHR) simulation software with clinical case scenarios, allowing students to learn how to use an EHR in a safe, true-to-life setting, while enabling instructors to measure their progress. Lippincott DocuCare's nonlinear solution works well in the classroom, simulation lab, and clinical practice.

Contact your Wolters Kluwer sales representative or visit thepoint.lww.com/DocuCare for options to enhance your medical–surgical nursing course with DocuCare.

Lippincott® CoursePoint+

Lippincott® CoursePoint+ is an integrated, digital curriculum solution for nursing education that provides a completely interactive experience geared to help students understand, retain, and apply their course knowledge and be prepared for practice. The time-tested, easy-to-use, and trusted solution includes engaging learning tools, evidence-based practice, case studies, and in-depth reporting to meet students where they are in their learning, combined with the most trusted nursing education content on the market to help prepare students for practice. This easy-to-use digital learning solution of *Lippincott® CoursePoint+*, combined with unmatched support, gives instructors and students everything they need for course and curriculum success!

Lippincott® CoursePoint+ includes the following:

- Leading content provides a variety of learning tools to engage students of all learning styles.
- A personalized learning approach gives students the content and tools they need at the moment they need it, giving them data for more focused remediation and helping to boost their confidence and competence.
- Powerful tools, including varying levels of case studies, interactive learning activities, and adaptive learning powered by PrepU, help students learn the critical thinking and clinical judgment skills to help them become practice-ready nurses.
- Preparation for Practice tools improve student competence, confidence, and success in transitioning to practice.
  - vSim® for Nursing: Codeveloped by Laerdal Medical and Wolters Kluwer, vSim® for Nursing simulates real nursing scenarios and allows students to interact with virtual patients in a safe, online environment.

- Lippincott® Advisor for Education: With over 8,500 entries covering the latest evidence-based content and drug information, Lippincott® Advisor for Education provides students with the most up-to-date information possible, while giving them valuable experience with the same point-of-care content they will encounter in practice.
- Unparalleled reporting provides in-depth dashboards with several data points to track student progress and help identify strengths and opportunities for improvement.
- Unmatched support includes training coaches, product trainers, and nursing education consultants to help educators and students implement CoursePoint with ease.

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User's Guide

Brunner & Suddarth's Textbook of Medical-Surgical Nursing, 16th Edition, has been revised and updated to reflect the complex nature of nursing practice today. This textbook includes many features to help you gain and apply the knowledge that you need to pass NCLEX and successfully meet the challenges and opportunities of clinical practice. In addition, features have been developed specifically to help you manage the varied roles that nurses assume in practice.

Features to Develop the Nurse as Practitioner

One of the central roles of the nurse is to provide holistic care to patients and their families, both independently and through collaboration with other health care professionals. Special features throughout chapters are designed to assist readers with clinical practice.

- **Nursing Process sections** are organized according to the nursing process framework—the basis for all nursing practice—and help clarify the nurse's responsibilities in caring for patients with select disorders.

NURSING PROCESS

The Patient With Impaired Physical Mobility

Problems commonly associated with immobility include weakened muscles, joint contracture, and deformity. Each joint of the body has a normal range of motion; if the range is limited, the functions of the joint and the muscles that move the joint are impaired, and painful deformities may develop. The nurse must identify patients at risk for such complications. The nurse needs to assess, plan, and intervene to prevent complications of immobility.

Another frequent challenge in rehabilitation nursing is an altered ambulatory/mobility pattern. Patients may be either temporarily or permanently unable to walk independently and unaided. The nurse assesses the mobility of the patient and designs care that promotes independent mobility within the prescribed therapeutic limits. If a patient cannot exercise and move their joints through a full range of motion, contractures may develop. A contracture is a shortening of the muscle and tendon that leads to deformity and limits joint mobility. When the contracted joint is moved, the patient experiences pain; in addition, more energy is required to move when joints are contracted.

Disability brings change to the patient and family unit as well as adjustments in lifestyle, mobility, and interactions as members of a community. Whether temporary or permanent, patients grieve the loss of health and need to process the loss as part of the adaptation process and plan of care (Taylor et al., 2023).

Assessment

Impaired mobility is related to pain, paralysis, loss of muscle strength, systemic disease, an immobilizing device (e.g., cast, brace), or prescribed limits to promote healing. Assessment of mobility includes positioning, ability to move, muscle strength and tone, joint function, and the prescribed mobility limits. Nurses must collaborate with physical therapists or other team members to assess mobility.

- **Assessment charts** focus on data that should be collected as part of the assessment step of the nursing process.

Chart 2-6 Assessment

Assessing the Home Environment

The following items may be part of an assessment of the home environment.

Exterior environment assessment may include:

- ❑ Walks, driveway, curbs, steps, hand rails, and porch
- ❑ Threshold/entry into home
- ❑ Residence numbers visible from street
- ❑ Lighting at all walks, entry
- ❑ Litter
- ❑ Noise
- ❑ Overgrown shrubs
- ❑ Doorbell or notification system

Interior environment assessment may include:

- ❑ Adequate width of halls and doors, considering knobs and hinges/doorswing
- ❑ Level of floor surfaces and thresholds
- ❑ Stairs and hand rails
- ❑ Clutter and trash management
- ❑ Sleeping arrangements and equipment

- **Risk Factors charts** outline factors that can impair health and should be considered in the context of evolving research and social determinants of health and systemic racism.

Chart 11-5 Risk Factors

Distributive Shock

Septic Shock

- Chronic illness
- Emergent and multiple surgeries
- Extremes of age (<1 and >65 years)
- Immunosuppression
- Invasive procedures
- Malnourishment

Neurogenic Shock

- Depressant action of medications
- Lack of glucose (e.g., insulin reaction)
- Spinal anesthesia
- Spinal cord injury

- **Genetics in Nursing Practice charts** summarize and highlight nursing assessments and management issues related to the role of genetics in select disorders.

Chart 8-1 Genetics in Nursing Practice
Genetics Concepts and the Older Adult

Genetic conditions in the older adult may occur from a specific gene mutation or arise as a result of a genetic predisposition combined with other factors (multifactorial). The following are examples of select adult-onset genetic conditions:

- Alzheimer disease and other types of dementia
- Many types of cancer (see Chapter 12, Chart 12-1)
- Hemochromatosis
- Huntington disease
- Polycystic kidney disease

The following are some examples of diseases with multifactorial components, which may include a genetic predisposition, in the older adult:

- Diabetes
- Emphysema
- Heart disease

Nursing Assessments

Refer to Chapter 4, Chart 4-2: Genetics in Nursing Practice: Genetic Aspects of Health Assessment

Family History Assessment Specific to the Older Adult

- Collect and assess family history on both maternal and paternal sides of the family for three generations.
- Determine whether genetic testing has occurred with other family members.
- Assess for person and family perceptions and beliefs around topics related to genetics.

Patient Assessment Specific to the Older Adult and Genetic Illness

- Assess older adult patient's knowledge and understanding of genetics, genetic testing, and gene-based therapies.
- Assess the patient's understanding of genetic information and decipher health literacy needs.
- Perform cultural, social, and spiritual assessment.
- Assess patient's communication capacities so that communication strategies about genetics are tailored to their needs and abilities.
- Identify patient's support system.

Management Issues Specific to Genetics and the Older Adult

- Refer for further genetic counseling and evaluation as warranted so that family members can discuss inheritance, risk to other family members, and availability of genetic testing and gene-based interventions.
- Offer appropriate genetic information and resources that take into consideration older patient's literacy needs.
- Evaluate older patient's understanding before, during, and after the introduction of genetic information and services.
- Take the time to clearly explain the concepts of genetic testing to older patients and provide written information that reinforces the topic of discussion.
- Participate in the management and coordination of care of older patients with genetic conditions and people predisposed to develop or pass on a genetic condition.

Genetics Resources

See Chapter 6, Chart 6-5: Components of Genetic Counseling for additional resources.

- **Pharmacology charts and tables** display important considerations related to administering medications and monitoring drug therapy.

Chart 24-3 Pharmacology
Self-Administration of Nitroglycerin

Most patients with angina pectoris self-administer nitroglycerin on an as-needed basis. A key nursing role in such cases is educating patients about the medication and how to take it. Sublingual nitroglycerin comes in tablet and spray forms.

- Instruct the patient to moisten the tablet with saliva, and place it under the tongue, allowing it to completely dissolve before swallowing.
- Explain that nitroglycerin is volatile and inactivated by heat, moisture, air, light, and time and therefore should be stored securely in its original container (e.g., capped dark glass bottle in a cool place); tablets should never be removed and carried in an unapproved container (e.g., clear plastic pillbox) or in a pocket close to the body.
- Advise the patient to keep the medication with them at all times and to renew the nitroglycerin supply every 3–6 months.
- Inform the patient that the medication should be taken prior to any activity that may produce pain. Because nitroglycerin increases tolerance for exercise and stress when taken prophylactically (i.e., before angina-producing activity, such as exercise, stair-climbing, or sexual activity), it is best taken before pain develops.

- **New! Clinical Judgment Alerts** identify key steps in the Clinical Judgment Measurement Model (CJMM) that the nurse takes to support clinical decision making and evidence-based practice and to avoid making errors.

Clinical Judgment Alert

Take Actions

Inadvertent removal of an ET tube can cause laryngeal swelling, hypoxemia, bradycardia, hypotension, and even death. Nurses should implement measures in accordance with evidence-based practice to minimize the risk of premature or inadvertent removal.

- **Veterans Considerations sections** highlight information applicable to the special care needs of veterans of the military. Veterans—who may include people from all age groups, sexes, races, and socioeconomic strata—may have unique health risks, based upon dates of service and assignment locale.

Veterans Considerations

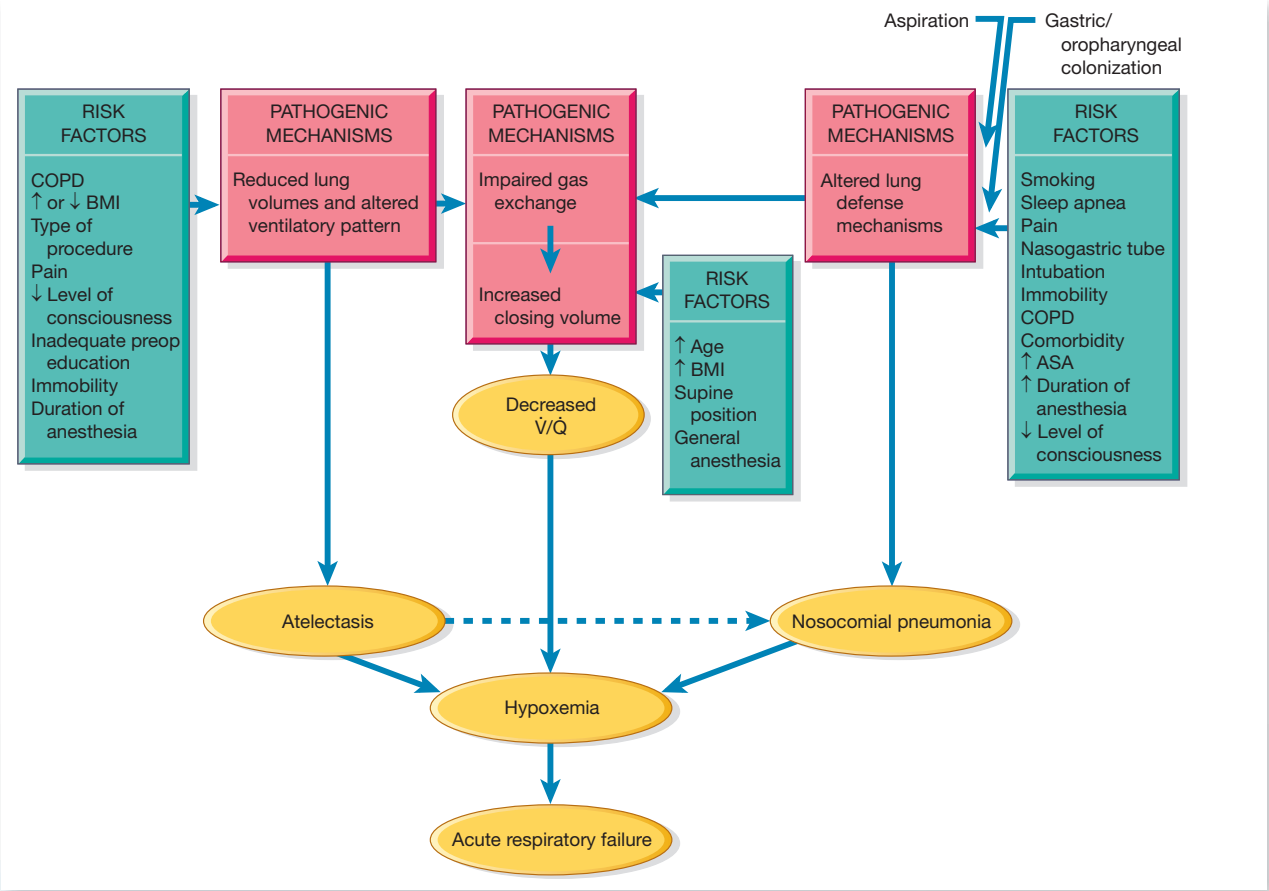
When conducting a health assessment, a key part of the nurse's role is to ask all adult patients if they have served in the U.S. military, and if so, their branch of service, length of service, and assigned duty stations (i.e., geographic locations and type of assignments). Patients who are veterans should specifically be asked about their experiences with violence and war, regardless of their ages, genders, lengths of service, and assignments. Asking about violent experiences works best when treated as a normal and natural part of the nursing assessment. The nurse's approach to gathering this information should be similar to that used when asking patients about sleep or activity difficulties or dietary or sexual concerns.

- **Gerontologic Considerations sections** highlight information that pertains specifically to the care of the older adult patient. In the United States, older adults comprise the fastest-growing segment of the population.

Gerontologic Considerations

Effective health care for older adults requires assessment of sexual health (Weber & Kelley, 2022). Older adults may be stereotyped by misconceptions that they are not able to have sexual intercourse or are not interested in sex (Eliopoulos, 2022); however, sexual activity continues in later life. Literature supports that people remain sexually active for a long time into older age, and that sexuality and intimacy are positively associated with satisfaction with life (Buczak-Stec et al., 2021). However, the frequency of intercourse may diminish and age-related changes can impact the sexual function of older adults (Eliopoulos, 2022). For many people, sexuality encompasses more than the physical act of sexual intercourse. This includes sharing and the intimate exchanges of words and touches between partners (Eliopoulos, 2022).

- **Physiology/Pathophysiology figures** include illustrations and algorithms describing normal physiologic and pathophysiologic processes.



Features to Develop the Nurse as Educator

Health education is a primary responsibility of the nursing profession. Nursing care is directed toward promoting, maintaining, and restoring health; preventing illness; and helping patients and families adapt to the residual effects of illness. Patient education and health promotion are central to all of these nursing activities.

- **Patient Education charts** help the nurse prepare the patient and family for procedures, assist them in understanding the patient's condition, and explain to them how to provide self-care.

Chart 41-3 Patient Education
The Patient With Peptic Ulcer Disease

At the completion of education, the patient or caregiver will be able to:

- State the impact of peptic ulcer disease on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- Explain the importance and necessity of adherence to prescribed medication regimen.
- Demonstrate methods of keeping track of the medication regimen and storage of the prescribed medications and use reminders such as pillboxes or phone or smartwatch alarms.
- State the names, doses, side effects, frequency, and schedule for all medications.
- Identify foods and other substances to avoid (e.g., food and drinks with extreme temperatures, coffee and other caffeinated beverages, alcohol, foods that were not tolerated in the past, nicotine).
- Identify side effects and complications that should be reported to primary provider:
  - Hemorrhage—cool skin, confusion, increased heart rate, labored breathing, blood in stool (either bright red or tarry black)
  - Penetration and perforation—severe abdominal pain, rigid and tender abdomen, vomiting, elevated temperature, increased heart rate
  - Gastric outlet obstruction—nausea and vomiting, distended abdomen, abdominal pain
- State how to reach primary provider with questions or complications.
- State time and date of follow-up appointments and testing.
- Identify the need for health promotion (e.g., cessation of use of tobacco products, stress management), disease prevention, and screening activities.

ADLs, activities of daily living; IADLs, instrumental activities of daily living.

- **Health Promotion charts** review important points that the nurse should discuss with the patient to prevent common health problems from developing.

Chart 8-3 Health Promotion
Nursing Strategies for Promoting Cognitive Function

Nurses can support the processes by which older adults learn by using the following strategies:

- Supply mnemonics to enhance recall of related data.
- Encourage ongoing learning.
- Link new information with familiar information.
- Use visual, auditory, and other sensory cues.
- Encourage learners to wear prescription glasses and hearing aids.
- Provide glare-free lighting.
- Provide a quiet, nondistracting environment.
- Set short-term goals with input from the learner.
- Prioritize the most important information and focus on the priority information.
- Keep education periods short.
- Pace learning tasks according to the endurance of the learner.
- Encourage verbal participation by learners.
- Reinforce successful learning in a positive manner.

Features to Develop the Nurse as Patient Advocate

Nurses advocate for patients by protecting their rights (including the right to health care) and assisting patients and their families in making informed decisions about health care.

- **All new! Ethical Dilemma charts** provide a clinical scenario, discussion points, and questions to help analyze fundamental ethical principles related to the dilemma.

Chart 16-6 Ethical Dilemma

Can Social Media Posts Hamper or Enhance Nurse–Patient Relationships?

Case Scenario

You work as a staff nurse in a hospital-based postanesthesia care unit (PACU) for the past 5 years. You enjoy your working environment, where you have cultivated solid working relationships. You follow several of your colleagues on their various social media sites. M.W. is a colleague you have worked closely with for many years and have occasionally socialized with outside of work. M.W. recently posted a video on their social media site of themselves in working scrubs dancing to music and singing voice-over to "Shake It Off." In the video, M.W. moans that most patients recovering from surgery that they have seen " … cannot shake it off and get out of my recovery room fast enough! They whine, they cry, they puke!" M.W. has not identified any patients; nonetheless, you feel discomfort at having seen this video.

Discussion

Had any patient identification been disclosed by M.W. in the video that would have been a clear violation of the Health Insurance Portability and Accountability Act (HIPAA) Privacy Rule (U.S. Department of Health and Human Services [HHS], 2003), which protects patient confidentiality (see Chapter 1 for further discussion). Although M.W. has not singled out any specific patients, they disparage patients under their care in the post. The American Nurses Association Code of Ethics (2015) espouses nursing practice that demonstrates compassion and respect for patients. A video such as one posted by M.W. clearly lacks compassion and is disrespectful of patients. Moreover, patients recovering from the effects of anesthesia are considered vulnerable, making M.W.'s posting even more difficult to reconcile with ethical nursing practice.

Analysis

- Identify the ethical principles that are in conflict in this case (see Chapter 1, Chart 1-5). Is M.W.'s post potentially harmful to patients? Justify your position on whether or not you consider the post nonmaleficent, given that no individual patient was identified.
- Is it ever justifiable (or perhaps even beneficent) for nurses to make generic posts about their nursing practice on social media sites, so long as the posts are HIPAA-compliant? Give some examples of how this might be achieved.
- Describe what you might do at this juncture. Would you confront M.W. about the post? Would your interaction with M.W. depend upon whether or not your worksite (i.e., the hospital and the PACU) was identified in the post? Do you

Features to Develop the Nurse as Researcher

Nurses identify potential research problems and questions to increase nursing knowledge and improve patient care. The use and evaluation of research findings in nursing practice are essential to further the science of nursing.

- **All new! Nursing Research Profiles** identify the implications and applications of nursing research findings for evidence-based nursing practice.

Chart 3-3 Nursing Research Profile

Usability of a Smartphone Medication Reminder

Sherwin, L., Deroche, C., Yevu-Johnson, J., et al. (2021). Usability evaluation of a smartphone medication reminder application in patients treated with short-term antibiotic. *CIN: Computers, Informatics, Nursing*, *39*(10), 547–553.

Purpose

The purpose of this study was to examine the usability and feasibility of the "MediSafe" medication reminder smartphone application (app) in adults with irritable bowel syndrome undergoing short-term antibiotic therapy. Each participant utilized the app with backup from a (human) "Medfriend" from their social support network. The study was completed to determine if use of the "MediSafe" app increased adherence with the three-times-per-day antibiotic therapy.

Design

In this mixed methods study, each of the 20 participants accessed the MediSafe application on their smartphone and identified a (human) "Medfriend" from their social support network. All patient participants used the MediSafe application daily for 14 days. Demographic data were collected, and app usage evaluation data were collected via a questionnaire, individual usage logs, and semi-structured interviews.

Findings

Patient participants rated the usefulness dimension favorably, whereas Medfriend participants rated the app's usefulness slightly less favorably. Ease of use, ease of learning, and satisfaction scales were rated highest by both patient participants and Medfriends, whereas usefulness was rated lowest by both groups, with Medfriends' usefulness rating significantly lower than that of patient participants. In the semi-structured interview qualitative portion of the study, the participants identified that having a Medfriend was beneficial. Two Medfriends identified that they believed by sending reminder texts they were instrumental in facilitating adherence.

Nursing Implications

Nurses should be aware that the MediSafe app is one of many health and medication reminder smartphone apps that are readily available free of charge. The MediSafe app is easy to use and accepted by both patients and their social support network. Additional research in larger studies is necessary to investigate the clinical effectiveness smartphone apps and other health tools have on medication adherence, social support engagement, and quality of life, especially in diverse patient populations.

Features to Facilitate Learning

In addition to practice-oriented features, special features have been developed to help readers learn key information.

- **Unfolding Patient Stories,** written by the National League for Nursing, are an engaging way to begin meaningful conversations in the classroom. These vignettes, which appear throughout the text near related content, feature patients from Wolters Kluwer's vSim for Nursing | Medical-Surgical (codeveloped by Laerdal Medical) and DocuCare products; however, each Unfolding Patient Story in the book stands alone, not requiring purchase of these products.

Unfolding Patient Stories: Skyler Hansen • Part 1

Skyler Hansen is an 18-year-old male recently diagnosed with type 1 diabetes. He lives with his parents and two younger siblings and is active in high school sports. He now requires insulin injections, glucose monitoring, and a diabetic diet. Examine how the psychological, physical, and teaching needs for managing a new diagnosis can be a great source of stress to the patient and family. How can the nurse generate solutions for this patient to cope with and adapt to diabetes management? (Skyler Hansen's story continues in Chapter 47.)

Care for Skyler and other patients in a realistic virtual environment: ***vSim*** *for Nursing* (thepoint.lww.com/vSimMedicalSurgical). Practice documenting these patients' care in DocuCare (thepoint.lww.com/DocuCareEHR).

- **All new! Clinical Judgment Exercises** challenge you to apply textbook knowledge to clinical scenarios while using tenets of the CJMM.

CLINICAL JUDGMENT EXERCISES

1 A college student comes to the student health center where you work. They report trouble sleeping, not being able to concentrate on courses, and generally feeling very stressed. They used to exercise regularly but do not feel they have time now to do so, have gained 10 lb (4.5 kg) this semester, and are using cannabis daily to cope with stress. After analyzing these cues, what problem do you suspect this student is experiencing? What three priority nursing actions should you take to ensure the patient's safety? You also refer them to a campus mindfulness program. What measures will you use to evaluate if this program has helped the student to meet expected outcomes and goals?

2 A 75-year-old attends a health promotion fair in the local hospital clinic where you work. They tell the nurse coordinator, "I am only here to have my cholesterol checked as my doctor said that it is a little higher than it was at my last visit. I don't need to participate in anything else, as I'm doing just fine." One of the volunteers convinces them to have their blood pressure checked and it is 150/90 mm Hg. Which cues are concerning to you? What priority actions should you take?

- **References** cited are listed at the end of each chapter and include updated, current sources at the time of revision.
- **Resources** lists at the end of each chapter include sources of additional information, websites, agencies, and patient education materials all current at the time of revision.

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**Asterisk indicates nursing research.*

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Resources

American Association of Colleges of Nursing, www.aacnnursing.org
American Nurses Association, www.nursingworld.org
American Nurses Association Center for Ethics and Human Rights, www.nursingworld.org/ethics
Campaign for Action: Future of Nursing, campaignforaction.org
Centers for Medicare & Medicaid Services, www.cms.hhs.gov
Healthy People 2030, www.healthypeople.gov
Institute for Healthcare Improvement, www.ihi.org
International Council of Nurses, www.icn.ch
Interprofessional Education Collaborative, www.ipecollaborative.org
NANDA International, www.nanda.org
National Academies of Sciences, Engineering, and Medicine, www.nationalacademies.org
National Center for Ethics in Health Care, www.ethics.va.gov
National League for Nursing, www.nln.org
QSEN Institute: Quality and Safety Education for Nurses, www.qsen.org
The Hastings Center, www.thehastingscenter.org
The Joint Commission, www.jointcommission.org
The TIGER Initiative, www.himss.org/professional-development/tiger-initiative
World Health Organization, www.who.int

Contents

Management of Patients With Arrhythmias and Conduction Disorders

23

LEARNING OUTCOMES

On completion of this chapter, the learner will be able to:

1. Correlate the components of the normal electrocardiogram with physiologic events of the heart.
2. Define the electrocardiogram as a waveform that represents the cardiac electrical event in relation to the lead placement.
3. Analyze elements of an electrocardiographic rhythm strip.
4. Identify the electrocardiographic criteria, causes, and management of arrhythmias, and use the nursing process as a framework for care of the patient with an arrhythmia, including conduction disturbances.
5. Compare the different types of pacemakers, their uses, possible complications, and nursing management.
6. Describe the key points of using a defibrillator; identify the purpose of an implantable cardioverter defibrillator, the types available, possible complications, and nursing management.
7. Describe the nursing management of patients with implantable cardiac devices.

It is essential for the heart to have a regular rate and rhythm to perform efficiently as a pump to circulate oxygenated blood and other life-sustaining nutrients to all of the body's tissues and organs. With an irregular or erratic rhythm, the heart is considered to be arrhythmic (also called *dysrhythmic*). This is a potentially dangerous condition.

Nurses may encounter patients with arrhythmias in all health care settings. Some arrhythmias are acute and others chronic; some require emergent interventions, while others may not. Because patients with arrhythmias are frequently encountered in many different types of settings, nurses must identify and provide appropriate first-line treatment of arrhythmias.

Arrhythmias are disorders of the formation or conduction (or both) of the electrical impulse within the heart. These disorders can cause disturbances of the heart rate, the heart rhythm, or both. Arrhythmias may initially be evidenced by the hemodynamic effect they cause (e.g., a change in conduction may change the pumping action of the heart and cause decreased blood pressure) and are diagnosed by analyzing the electrocardiographic (ECG) waveform. Arrhythmias are named according to the site of origin of the electrical impulse and the mechanism of formation or conduction involved. For example, an impulse that originates in the sinoatrial (SA) node and at a slow rate is called *sinus bradycardia*. Arrhythmia treatment is based on the frequency and severity of symptoms produced.

Normal Electrical Conduction

The electrical impulse that stimulates and paces the cardiac muscle normally originates in the SA node, also called the *sinus node*, an area located near the superior vena cava in the right atrium. In adults, the electrical impulse usually occurs at a rate of 60 to 100 times a minute. The electrical impulse quickly travels from the SA node through the atria to the atrioventricular (AV) node (Fig. 23-1); this process is known as **conduction**. The electrical stimulation of the muscle cells of the atria causes them to contract. The structure of the AV node slows the electrical impulse, giving the atria time to contract and fill the ventricles with blood. This part of atrial contraction is referred to as the *atrial kick* and accounts for approximately one third of the total volume ejected during ventricular contraction (Fuster et al., 2023). The electrical impulse then travels very quickly through the bundle of His to the right and left bundle branches and the Purkinje fibers, located in the ventricular muscle.

The electrical stimulation is called **depolarization**, and the mechanical contraction is called *systole*. Electrical relaxation is called **repolarization**, and mechanical relaxation is called *diastole*. The process from sinus node electrical impulse generation through ventricular repolarization completes the electromechanical circuit, and the cycle begins again. (See Chapter 22 for a more complete explanation of cardiac function.)

Influences on Heart Rate and Contractility

The heart rate is influenced by the autonomic nervous system, which consists of sympathetic and parasympathetic fibers. Sympathetic nerve fibers (also referred to as *adrenergic fibers*) are attached to the heart and arteries as well as several other areas in the body. Stimulation of the sympathetic system results in positive **chronotropy** (increased heart rate), positive **dromotropy** (increased AV conduction), and positive

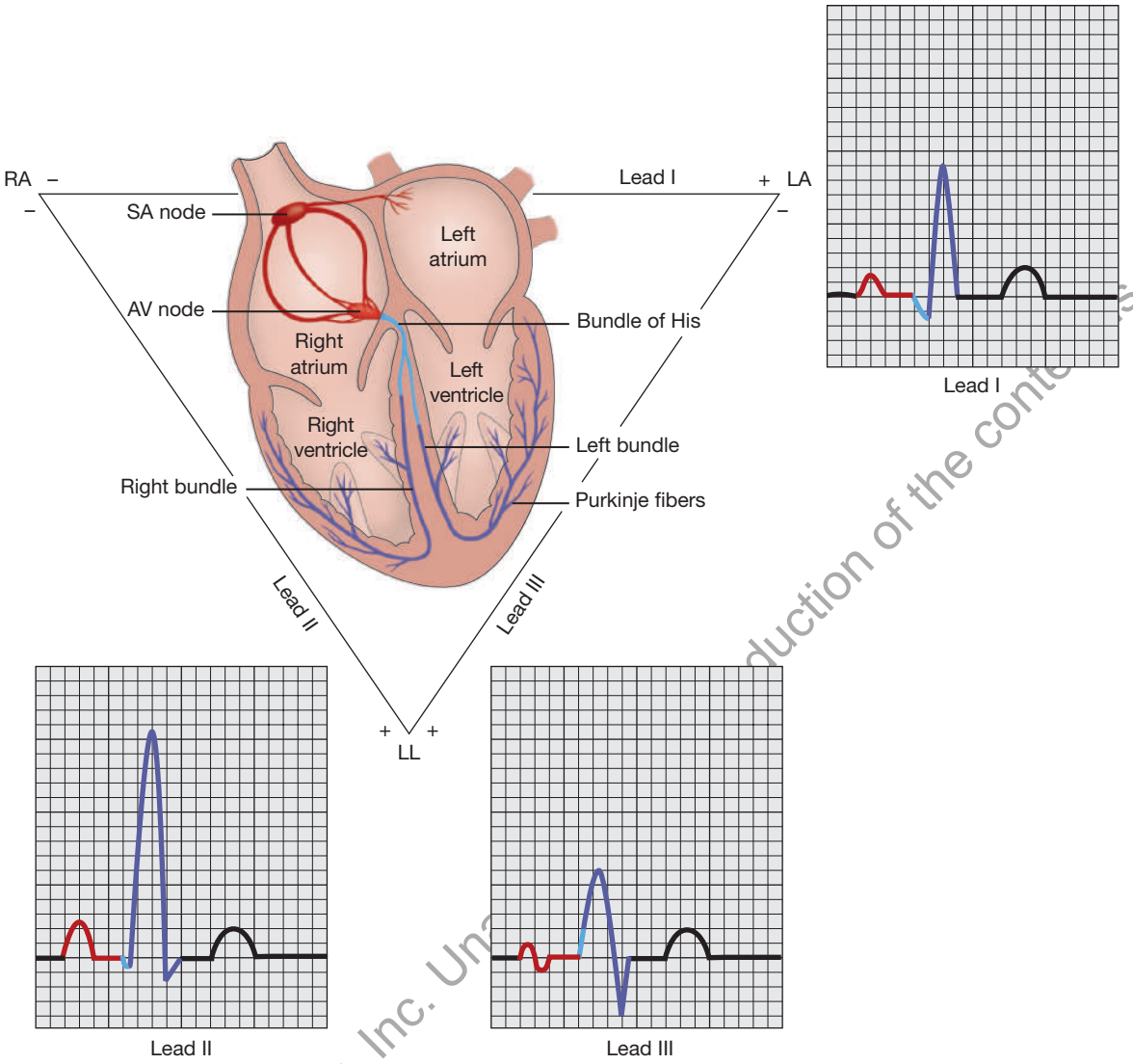

Figure 23-1 • Relationship of electrocardiographic (ECG) complex, lead system, and electrical impulse. The heart conducts electrical activity, which the ECG measures and shows. The configurations of electrical activity displayed on the ECG vary depending on the lead (or view) of the ECG and on the rhythm of the heart. Therefore, the configuration of a normal rhythm tracing from lead I will differ from the configuration of a normal rhythm tracing from lead II, lead II will differ from lead III, and so on. The same is true for abnormal rhythms and cardiac disorders. To make an accurate assessment of the heart's electrical activity or to identify where, when, and what abnormalities occur, the ECG needs to be evaluated from every lead, not just from lead II. Here, the different areas of electrical activity are identified by color. AV, atrioventricular; LA, left arm; LL, left leg; RA, right arm; SA, sinoatrial.

inotropy (increased force of myocardial contraction). Sympathetic stimulation also constricts peripheral blood vessels, therefore, increasing blood pressure. Parasympathetic nerve fibers are also attached to the heart and arteries. Parasympathetic stimulation reduces the heart rate (negative chronotropy), AV conduction (negative dromotropy), and the force of atrial myocardial contraction. The decreased sympathetic stimulation results in dilation of arteries, thereby lowering blood pressure.

Manipulation of the autonomic nervous system may increase or decrease the incidence of arrhythmias. Increased sympathetic stimulation (e.g., caused by exercise, anxiety, fever, or administration of catecholamines such as dopamine, aminophylline, or dobutamine) may increase the incidence of arrhythmias. Decreased sympathetic stimulation (e.g., with rest, anxiety reduction methods such as therapeutic communication or meditation, or administration of beta-adrenergic blocking agents) may decrease the incidence of arrhythmias.

The Electrocardiogram

The electrical impulse that travels through the heart can be viewed by means of ECG, the end product of which is an **electrocardiogram (ECG)**. Each phase of the cardiac cycle is reflected by specific waveforms on the screen of a cardiac monitor or on a strip of ECG graph paper.

Obtaining an Electrocardiogram

An ECG is obtained by placing electrodes on the body at specific areas. Biomonitoring electrodes come in various shapes and sizes, but all have two components: (1) an

adhesive substance that attaches to the skin to secure the electrode in place and (2) a substance that reduces the skin's electrical impedance to enhance conductivity. Washing the area with soap and water prior to placement is recommended to enhance adhesion of electrodes. If the amount of chest hair prevents the electrode from having good contact with the skin, the hair may need to be clipped (Jepsen et al., 2018; see Chapter 22, Chart 22-5, "Applying Electrodes"). Poor electrode adhesion will cause significant **artifact** (distorted, irrelevant, and extraneous ECG waveforms), which may distort capturing an accurate ECG waveform.

The number and placement site of the electrodes depend on the type of ECG being obtained. Most continuous monitors use two to five electrodes, usually placed on the limbs and the chest. These electrodes create an imaginary line, called a *lead*, which serves as a reference point from which the electrical activity is viewed. A lead is like an eye of a camera—It has a narrow peripheral field of vision, looking only at the electrical activity directly in front of it. Therefore, the ECG waveforms that appear on the ECG paper and cardiac monitor represent the electrical impulse in relation to the lead (see Fig. 23-1). A change in the waveform can be caused by a change in the electrical impulse (where it originates or how it is conducted) or by a change in the lead. Electrodes are attached to cable wires, which are connected to one of the following:

- An ECG machine placed at the patient's side for an immediate recording (standard 12-lead ECG)
- A cardiac monitor at the patient's bedside for continuous reading; this kind of monitoring, usually called *hard-wire monitoring*, is used in intensive care units.
- A small box that the patient carries and that continuously transmits the ECG information by radiowaves to a central monitor located elsewhere (called *telemetry*)
- A small, lightweight recorder-like machine (called *continuous ECG monitoring*, which might include a *Holter monitor* or a *patch monitor*) that the patient wears for a prescribed period of time and that continuously records the ECG, which is later viewed and analyzed with a scanner
- A very small device inserted under the skin or worn externally on a wrist band (called *intermittent monitoring* using a *looped recorder*) can perform ECG monitoring on demand whenever a patient is symptomatic (see Chapter 22 for further discussion of ECG monitoring systems).

A patient may undergo an electrophysiology study (EPS), in which electrodes are placed inside the heart in order to obtain an intracardiac ECG. This is used not only to diagnose the arrhythmia but also to determine the most effective treatment plan. However, because an EPS is invasive, it is performed in the hospital and may require that the patient be admitted (see later discussion).

During open heart surgery, temporary pacemaker wires may be lightly sutured to the epicardium and brought through the chest wall. These wires may be used not only for temporary pacing but also, when connected to the V lead cable, to obtain an atrial ECG, which can be helpful in the differential diagnosis of tachyarrhythmias (see Chapter 24 for further discussion).

The placement of electrodes for monitoring varies with the type of technology, the purpose of monitoring, and the protocols used in the health care facility. For a standard 12-lead ECG, 10 electrodes (six on the chest and four on the limbs) are placed on the body (Fig. 23-2). To prevent interference from the electrical activity of skeletal muscle, the limb electrodes are placed on areas that are not bony and that do not have significant movement. The six chest electrodes are applied at very specific areas, and the nurse must locate the specific intercostal space for the correct placement of each chest electrode. Diagnostic errors can occur if electrodes are incorrectly placed. Sometimes, when a patient in the hospital needs to be monitored more closely for ECG changes, the chest electrodes are left in place to ensure the same placement for follow-up 12-lead ECGs.

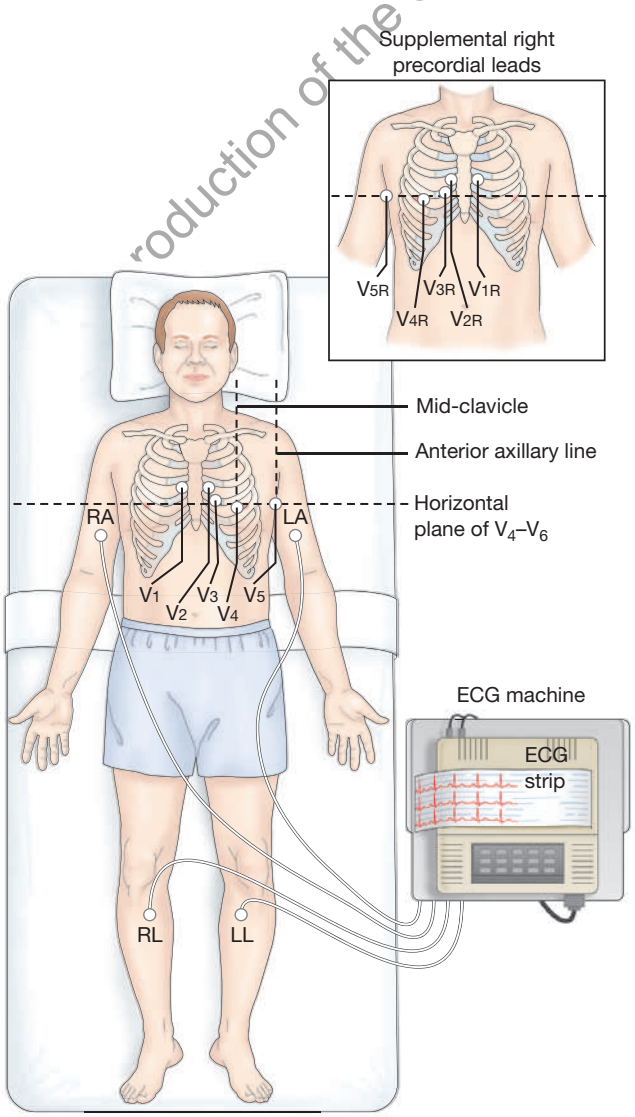

Figure 23-2 • ECG electrode placement. The standard left precordial leads are V_1—fourth intercostal space, right sternal border; V_2—fourth intercostal space, left sternal border; V_3—diagonally between V_2 and V_4; V_4—fifth intercostal space, left midclavicular line; V_5—same level as V_4, anterior axillary line; V_6 (not illustrated)—same level as V_4 and V_5, midaxillary line. The right precordial leads, placed across the right side of the chest, are the mirror opposite of the left leads. LA, left arm; LL, left leg; RA, right arm; RL, right leg.

A standard 12-lead ECG reflects the electrical activity primarily in the left ventricle (LV). Placement of additional electrodes for other leads may be needed to obtain more complete information. For example, in patients with suspected right-sided heart damage, right-sided precordial leads are required to evaluate the right ventricle (RV) (see Fig. 23-2).

Components of the Electrocardiogram

The ECG waveform reflects the function of the heart's conduction system in relation to the specific lead. The ECG offers important information about the electrical activity of the heart and is useful in diagnosing arrhythmias. ECG waveforms are printed on graph paper or viewed electronically on cardiac monitors and are divided by vertical and horizontal lines at standard intervals (Fig. 23-3). Time and rate are measured on the horizontal axis of the graph, and amplitude or voltage is measured on the vertical axis. When an ECG waveform moves toward the top of the graph, it is called a *positive deflection*. When it moves toward the bottom of the graph, it is called a *negative deflection*. When reviewing an ECG, each waveform should be examined and compared with the others.

Waves, Complexes, and Intervals

The ECG is composed of waveforms (including the P wave, the QRS complex, the T wave, and possibly a U wave) and of segments and intervals (including the PR interval, the ST segment, and the QT interval) (see Fig. 23-3).

The **P wave** represents the electrical impulse starting in the SA node and spreading through the atria. Therefore, the P wave represents atrial depolarization. It is normally 2.5 mm or less in height and 0.11 seconds or less in duration.

The **QRS complex** represents ventricular depolarization. Not all QRS complexes have all three waveforms. The Q wave is the first negative deflection after the P wave. The Q wave is normally less than 0.04 seconds in duration and less than 25% of the R-wave amplitude. The R wave is the first positive deflection after the P wave, and the S wave is the first negative deflection after the R wave. The QRS complex is normally less than 0.12 seconds in duration.

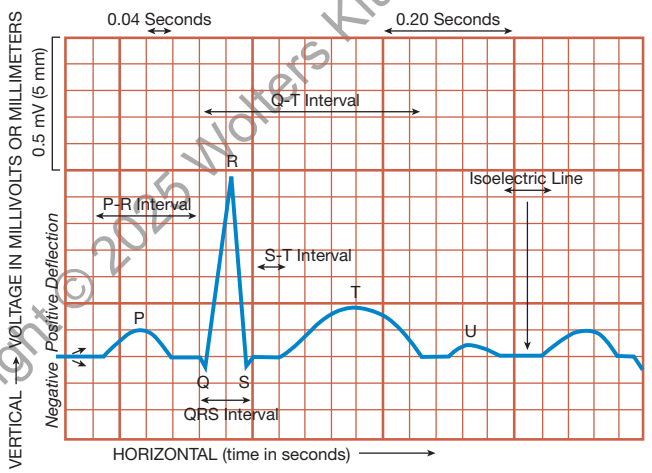

Figure 23-3 • ECG graph and commonly measured components. Each large box represents 0.20 seconds on the horizontal axis and 5 mm or 0.5 mV on the vertical axis. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-16). Wolters Kluwer.

The **T wave** represents ventricular repolarization (when the cells regain a negative charge; also called the *resting state*). It follows the QRS complex and is usually in the same direction (deflection) as the QRS complex. Atrial repolarization also occurs but is not visible on the ECG because it occurs at the same time as ventricular depolarization (i.e., the QRS).

The **U wave** is thought to represent repolarization of the Purkinje fibers; although this wave is rare, it sometimes appears in patients with hypokalemia (low potassium levels), hypertension, or heart disease. If present, the U wave follows the T wave and is usually smaller than the P wave. If larger in amplitude, it may be mistaken for an extra P wave.

The **PR interval** is measured from the beginning of the P wave to the beginning of the QRS complex and represents the time needed for sinus node stimulation, atrial depolarization, and conduction through the AV node before ventricular depolarization. In adults, the PR interval normally ranges from 0.12 to 0.20 seconds in duration.

The **ST segment**, which represents early ventricular repolarization, lasts from the end of the QRS complex to the beginning of the T wave. The beginning of the ST segment is usually identified by a change in the thickness or angle of the terminal portion of the QRS complex. The end of the ST segment may be more difficult to identify because it merges into the T wave. The ST segment is normally isoelectric (see later discussion of TP interval). It is analyzed to identify whether it is above or below the isoelectric line, which may be, among other signs and symptoms, a sign of cardiac ischemia (see Chapter 24).

The **QT interval**, which represents the total time for ventricular depolarization and repolarization, is measured from the beginning of the QRS complex to the end of the T wave. The QT interval varies with heart rate, gender, and age; therefore, the measured interval may be corrected (QT_c) for these variables through specific calculations. The QT_c may be automatically calculated by the ECG technology, or a nurse may manually calculate or use a resource that contains a chart of these calculations. The QT interval is usually 0.32 to 0.40 seconds in duration if the heart rate is 65 to 95 bpm.

The **TP interval** is measured from the end of the T wave to the beginning of the next P wave—an isoelectric period (see Fig. 22-3). When no electrical activity is detected, the line on the graph remains flat; this is called the *isoelectric line*. The ST segment is compared with the TP interval to detect ST-segment changes. The PR segment is sometimes used to determine the isoelectric line. However, because the PR segment sometimes is altered due to ischemic conditions, the TP interval is the preferred reference for the isoelectric line.

The **PP interval** is measured from the beginning of one P wave to the beginning of the next P wave. The PP interval is used to determine atrial rate and rhythm.

The **RR interval** is measured from one QRS complex to the next QRS complex. The RR interval is used to determine ventricular rate and rhythm (see later discussion).

Analyzing the Electrocardiogram Rhythm Strip

The ECG rhythm strip is analyzed in a systematic manner to determine the patient's cardiac rate and rhythm and to detect arrhythmias and conduction disorders, as well as evidence of myocardial ischemia, injury, and infarction.

Determining Heart Rate From the Electrocardiogram

Heart rate can be obtained from the ECG rhythm strip by several methods. A 1-minute rhythm strip contains 300 large boxes and 1,500 small boxes. Therefore, an easy and accurate method of determining heart rate with a regular rhythm is to count the number of small boxes within an RR interval and divide 1,500 by that number. If, for example, there are 10 small boxes between two R waves, the heart rate is 1,500/10 or 150 bpm; if there are 25 small boxes, the heart rate is 1,500/25 or 60 bpm (Fig. 23-4).

An alternative but less accurate method for estimating heart rate, which is usually used when the rhythm is irregular, is to count the number of RR intervals in 6 seconds and multiply that number by 10. The top of the ECG paper is usually marked at 3-second intervals, which is 15 large boxes horizontally. The RR intervals are counted, rather than QRS complexes, because a computed heart rate based on the latter might be inaccurately high. The same methods may be used for determining atrial rate, using the PP interval instead of the RR interval.

Determining Heart Rhythm From the Electrocardiogram

The rhythm is often identified at the same time the rate is determined. Chart 23-1 provides an example of a method to analyze the patient's rhythm. The RR interval is used to determine ventricular rhythm and the PP interval to determine atrial rhythm. If the intervals are the same or if the difference between the intervals is less than 0.8 seconds throughout the strip, the rhythm is called *regular*. If the intervals are different, the rhythm is called *irregular*.

Once the rhythm has been analyzed, the findings are compared with and matched to the ECG criteria for arrhythmias to determine a diagnosis. It is important for the nurse to identify the arrhythmia and assess the patient to determine the physiologic effect of the arrhythmia and identify possible causes. Treatment of an arrhythmia is based on clinical evaluation of the patient with identification of the arrhythmia's etiology and physiologic effect, not on its presence on ECG alone.

Most cardiac monitoring has functionality that includes the ability to continuously monitor the rhythm and alert health care personnel with an auditory and visual alarm when a clinically significant change in the rhythm occurs. However, a high rate of triggered, clinically insignificant alarms may lead to alarm fatigue, which has been linked to nurses ignoring, disabling, or silencing alarms (Sendelbach & Jepsen, 2018)—putting patients at increased risk of adverse events.

Chart 23-1 Interpreting Arrhythmias: Systematic Analysis of the Electrocardiogram

When examining an electrocardiogram (ECG) rhythm strip to learn more about a patient's arrhythmia:

1. Determine the ventricular rate.
2. Determine the ventricular rhythm.
3. Determine the QRS duration.
4. Determine whether the QRS duration is consistent throughout the strip. If not, identify other duration.
5. Identify the QRS shape; if not consistent, then identify other shapes.
6. Identify P waves; is there a P in front of every QRS?
7. Identify the P-wave shape; identify whether it is consistent or not.
8. Determine the atrial rate.
9. Determine the atrial rhythm.
10. Determine each PR interval.
11. Determine if the PR intervals are consistent, irregular but with a pattern to the irregularity or just irregular.
12. Determine how many P waves for each QRS (P:QRS ratio).

In many cases, the nurse may use a checklist and document the findings next to the appropriate ECG criterion.

Clinical Judgment Alert

Recognize Cues, Analyze Cues, & Generate Solutions

It is vital that the nurse assesses the cause(s) of a cardiac monitor's alarm and then adjusts the alarm default settings and individualizes the alarm parameter limits and levels. The assessment should also include an evaluation and discussion with the primary provider to validate that the patient needs to remain on continuous cardiac monitoring.

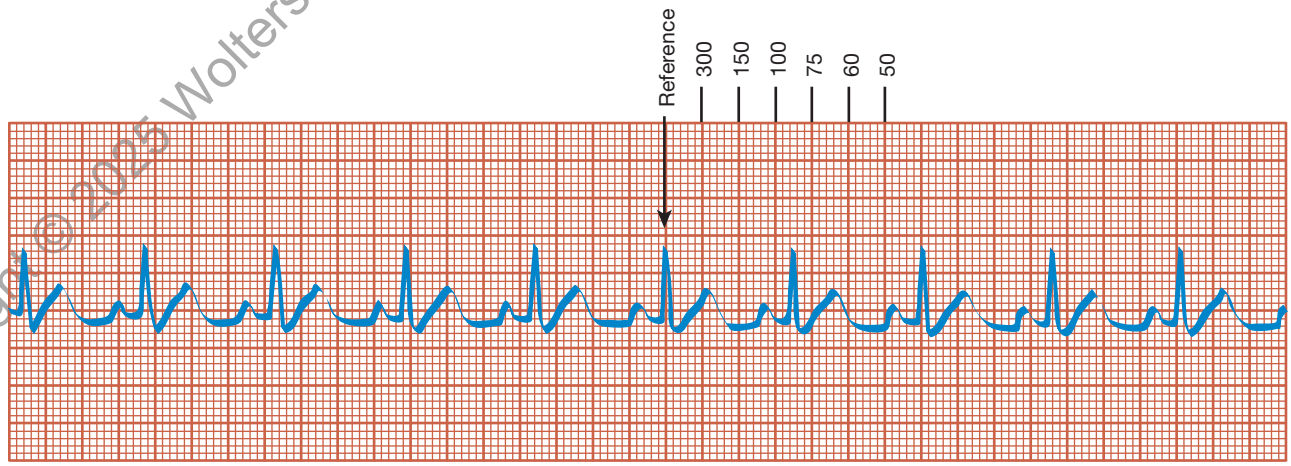

Figure 23-4 • Method for estimating heart rate. The number of small boxes in the RR interval is 17.5; divide this by 1,500, and the heart rate is approximately 86 bpm. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-18–[bottom portion]). Wolters Kluwer.

Normal Sinus Rhythm

Electrical conduction that begins in the SA node generates a **sinus rhythm**. Normal sinus rhythm occurs when the electrical impulse starts at a regular rate and rhythm in the SA node and travels through the normal conduction pathway. Normal sinus rhythm has the following characteristics (Fig. 23-5):

Ventricular and atrial rate: 60 to 100 bpm in the adult
Ventricular and atrial rhythm: Regular
QRS shape and duration: Usually normal but may be regularly abnormal
P wave: Normal and consistent shape; always in front of the QRS
PR interval: Consistent interval between 0.12 and 0.20 seconds
P:QRS ratio: 1:1

Types of Arrhythmias

Arrhythmias include sinus, atrial, junctional, and ventricular arrhythmias and their various subcategories, as well as conduction abnormalities.

Sinus Node Arrhythmias

Sinus node arrhythmias originate in the SA node; these include sinus bradycardia, sinus tachycardia, and sinus arrhythmia.

Sinus Bradycardia

Sinus bradycardia occurs when the SA node creates an impulse at a slower-than-normal rate. Causes include lower metabolic needs (e.g., sleep, athletic training, and hypothyroidism), vagal stimulation (e.g., from vomiting, suctioning, and severe pain), medications (e.g., calcium channel blockers [e.g., nifedipine, amiodarone] and beta-blockers [e.g., metoprolol]), idiopathic sinus node dysfunction, increased intracranial pressure, and coronary artery disease, especially myocardial infarction (MI) of the inferior wall. Unstable and symptomatic bradycardia is frequently due to hypoxemia but may also include acute altered mental status (e.g., delirium) and acute decompensated heart failure (Fuster et al., 2023). Sinus bradycardia has the following characteristics (Fig. 23-6):

Ventricular and atrial rate: Less than 60 bpm in the adult
Ventricular and atrial rhythm: Regular
QRS shape and duration: Usually normal but may be regularly abnormal
P wave: Normal and consistent shape; always in front of the QRS

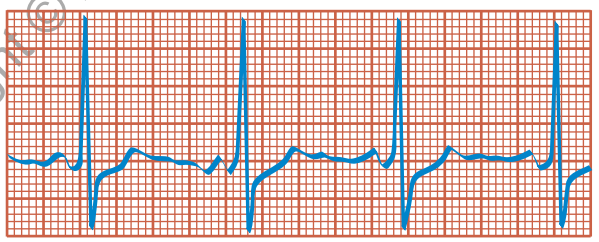

Figure 23-5 • Normal sinus rhythm. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-19A). Wolters Kluwer.

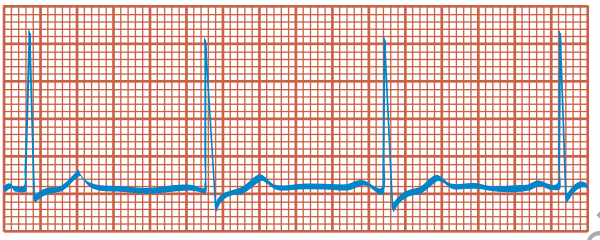

Figure 23-6 • Sinus bradycardia. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (11th ed., Fig. 14-19C). Wolters Kluwer.

PR interval: Consistent interval between 0.12 and 0.20 seconds
P:QRS ratio: 1:1

All characteristics of sinus bradycardia are the same as those of normal sinus rhythm, except for the rate. The patient is assessed to determine the hemodynamic effect and the possible cause of the arrhythmia. If the decrease in heart rate results from stimulation of the vagus nerve, such as with bearing down during defecation or vomiting, attempts are made to prevent further vagal stimulation. If the bradycardia is caused by a medication, such as a beta-blocker, the medication may be withheld. If the slow heart rate causes significant hemodynamic changes resulting in shortness of breath, acute alteration of mental status, angina, hypotension, ST-segment changes, or premature ventricular complexes (PVCs), treatment is directed toward increasing the heart rate. Slow heart rate may be due to sinus node dysfunction. Sinus node dysfunction is more common in older patients but may occur at any age. The causes of sinus node dysfunction are intrinsic (e.g., degenerative idiopathic fibrosis and cardiac remodeling) or extrinsic (e.g., medications and metabolic abnormalities) to the SA node. Many extrinsic causes are reversible. ECG findings often will include sinus bradycardia, sinus pauses, or *tachybrady syndrome*; an alternating bradycardia and tachycardia (Hawks et al., 2021).

Medical Management

The medical management depends on the cause and symptoms, and resolving the causative factors may be the only treatment needed. If the bradycardia produces signs and symptoms of clinical instability (e.g., acute alteration in mental status, chest discomfort, or hypotension), 0.5 mg of atropine may be given rapidly as an intravenous (IV) bolus and repeated every 3 to 5 minutes until a maximum dosage of 3 mg is given. Rarely, if the bradycardia is unresponsive to atropine, emergency transcutaneous pacing can be instituted, or medications, such as dopamine, isoproterenol, or epinephrine, are given (Kusumoto et al., 2019; see later discussion).

Sinus Tachycardia

Sinus tachycardia occurs when the sinus node creates an impulse at a faster-than-normal rate. Causes may include:

Physiologic or psychological stress (e.g., acute blood loss, anemia, shock, hypervolemia, hypovolemia, heart failure, pain, hypermetabolic states, fever, exercise, and anxiety)

Medications that stimulate the sympathetic response (e.g., catecholamines, aminophylline, and atropine), stimulants (e.g., caffeine and nicotine), and illicit drugs (e.g., amphetamines, cocaine, and ecstasy)

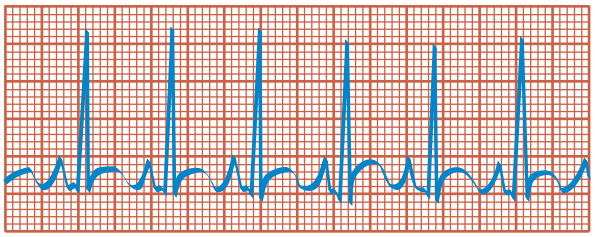

Figure 23-7 • Sinus tachycardia. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-19B). Wolters Kluwer.

Enhanced **automaticity** (the innate ability of the cardiac cells to initiate an electrical impulse) of the SA node, a condition called *inappropriate sinus tachycardia*. Autonomic dysfunction, which results in a type of sinus tachycardia referred to as *postural orthostatic tachycardia syndrome* (POTS). POTS is characterized by tachycardia without hypotension and by presyncopal symptoms such as palpitations, lightheadedness, weakness, and blurred vision, which occur with sudden posture changes.

Sinus tachycardia has the following characteristics (Fig. 23-7):

Ventricular and atrial rate: Greater than 100 bpm in the adult but usually less than 120 bpm
Ventricular and atrial rhythm: Regular
QRS shape and duration: Usually normal but may be regularly abnormal
P wave: Normal and consistent shape; always in front of the QRS but may be buried in the preceding T wave
PR interval: Consistent interval between 0.12 and 0.20 seconds
P:QRS ratio: 1:1

All aspects of sinus tachycardia are the same as those of normal sinus rhythm, except for the rate. As the heart rate increases, the diastolic filling time decreases, possibly resulting in reduced cardiac output and subsequent symptoms of syncope and low blood pressure. If the rapid rate persists and the heart cannot compensate for the decreased ventricular filling, the patient may develop acute pulmonary edema.

Medical Management

Medical management of sinus tachycardia is determined by the severity of symptoms and directed at identifying and abolishing its cause. Vagal maneuvers, such as carotid sinus massage, gagging, bearing down against a closed glottis (as if having a bowel movement), forceful and sustained coughing, and applying a cold stimulus to the face (such as applying an ice-cold wet towel to the face), or administration of adenosine should be considered to interrupt the tachycardia. If the tachycardia is persistent and causing hemodynamic instability (e.g., acute alteration in mental status, chest discomfort, and hypotension), synchronized **cardioversion** (i.e., electrical current given in synchrony with the patient's own QRS complex to stop an arrhythmia) is the treatment of choice, if vagal maneuvers and adenosine are unsuccessful or not feasible (see later discussion). Beta-blockers (Class II antiarrhythmic) and calcium channel blockers (Class IV antiarrhythmic) (Table 23-1) may also be considered in treating hemodynamically stable sinus tachycardia. Catheter ablation

TABLE 23-1 Summary of Antiarrhythmic Medications[a]

Class[a]	Action	Drug Names	Side Effects	Nursing Interventions
IA	Moderate depression of depolarization; prolongs repolarization Treats and prevents atrial and ventricular arrhythmias	Quinidine, procainamide, disopyramide	Decreased cardiac contractility Prolonged QRS, QT Proarrhythmic Hypotension with IV administration Diarrhea with quinidine, constipation with disopyramide Cinchonism with quinidine Lupuslike syndrome with procainamide Anticholinergic effects: dry mouth, urinary hesitancy with disopyramide	Observe for HF. Monitor BP with IV administration. Monitor QRS duration for increase of >50% from baseline. Monitor for prolonged QT. Monitor *N*-acetyl procainamide (NAPA) laboratory values during procainamide therapy. If given for atrial fibrillation, ensure that the patient has been pretreated with a medication to control AV conduction.
IB	Minimal depression of depolarization; shortened repolarization Treats ventricular arrhythmias	Lidocaine, mexiletine	CNS changes (e.g., confusion and lethargy) Bradycardia GI distress Tremors	Monitor for CNS changes and tremors. Discuss with the primary provider decreasing lidocaine dose in older adult patients and patients with cardiac/liver dysfunction.
IC	Marked depression of depolarization; little effect on repolarization Treats atrial and ventricular arrhythmias	Flecainide, propafenone	Proarrhythmic HF Dizziness, visual disturbances, dyspnea	Decrease dose with renal dysfunction and strict vegetarian diets. Avoid use in patients with structural heart disease (e.g., coronary artery disease and HF).

(*continued*)

TABLE 23-1 Summary of Antiarrhythmic Medications[a] (*continued*)

Class[a]	Action	Drug Names	Side Effects	Nursing Interventions
II	Decreases automaticity and conduction Treats atrial and ventricular arrhythmias	Acebutolol[c], atenolol, bisoprolol/HCTZ, esmolol[c], labetalol, metoprolol, nadolol, propranolol[c], sotalol (also has class III actions)[c], timolol	Bradycardia, AV block Decreased contractility Bronchospasm Nausea Hypotension with and without symptoms Masks hypoglycemia and thyrotoxicosis CNS disturbances (e.g., confusion, dizziness, fatigue, and depression)	Monitor heart rate, PR interval, signs and symptoms of HF, especially in those also taking calcium channel blockers. Monitor blood glucose levels in patients with type 2 diabetes. Caution the patient about abrupt withdrawal to avoid tachycardia, hypertension, and myocardial ischemia.
III	Prolongs repolarization Amiodarone treats and prevents ventricular and atrial arrhythmias, especially in patients with ventricular dysfunction. Dofetilide and ibutilide treat and prevent atrial arrhythmias.	Amiodarone, dofetilide, dronedarone, ibutilide	Pulmonary toxicity (amiodarone) Corneal microdeposits (amiodarone) Photosensitivity (amiodarone) Bradycardia Hypotension, especially with IV administration Polymorphic ventricular arrhythmias (rare with amiodarone) Nausea and vomiting Potentiates digoxin (amiodarone) See beta-blockers earlier (sotalol)	Make sure that the patient is sent for baseline pulmonary function tests (amiodarone). Closely monitor the patient. Assess for contraindications prior to administration. Monitor QT duration. Continuous ECG monitoring with the initiation of dofetilide and ibutilide. Monitor renal function.
IV	Blocks calcium channel Treats and prevents paroxysmal atrial arrhythmias[b]	Verapamil, diltiazem	Bradycardia, AV blocks Hypotension with IV administration HF, peripheral edema Constipation, dizziness, headache, nausea	Monitor heart rate, PR interval. Monitor BP closely with IV administration. Monitor for signs and symptoms of HF. Do not crush sustained-release medications.

AV, atrioventricular; BP, blood pressure; CNS, central nervous system; ECG, electrocardiogram; GI, gastrointestinal; HCTZ, hydrochlorothiazide; HF, heart failure; IV, intravenous.
[a]Based on Vaughan-Williams classification.
[b]There are other calcium channel blockers, but they are not approved or used for arrhythmias.
[c]Beta-blocker with labeled use for arrhythmias.
From Al-Khatib et al. (2018); Fuster et al. (2023); Lippincott Solutions (2024).

(see later discussion) of the SA node may be used in cases of persistent inappropriate sinus tachycardia unresponsive to other treatments. Treatment for POTS often involves a combination of approaches, with treatment targeted at the underlying problem. For example, patients with hypovolemia may be advised to increase their fluid and sodium intake, or use salt tablets if necessary.

Sinus Arrhythmia

Sinus arrhythmia occurs when the sinus node creates an impulse at an irregular rhythm; the rate usually increases with inspiration and decreases with expiration. Nonrespiratory causes include heart disease and valvular disease, but these are rare. Sinus arrhythmia has the following characteristics (Fig. 23-8):

- *Ventricular and atrial rate:* 60 to 100 bpm in the adult
- *Ventricular and atrial rhythm:* Irregular
- *QRS shape and duration:* Usually normal but may be regularly abnormal
- *P wave:* Normal and consistent shape; always in front of the QRS
- *PR interval:* Consistent interval between 0.12 and 0.20 seconds
- *P:QRS ratio:* 1:1

Medical Management

Sinus arrhythmia does not cause any significant hemodynamic effect and, therefore, is not typically treated.

Atrial Arrhythmias

Atrial arrhythmias originate from foci within the atria and not the SA node. These include aberrancies such as premature atrial complexes (PACs) as well as atrial fibrillation and atrial flutter.

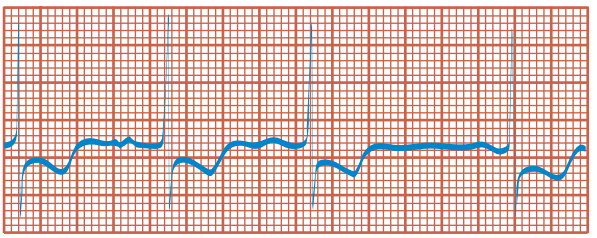

Figure 23-8 • Sinus arrhythmia. Note irregular RR and PP intervals. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-19D). Wolters Kluwer.

Premature Atrial Complex

A PAC is a single ECG complex that occurs when an electrical impulse starts in the atrium before the next normal impulse of the sinus node. The PAC may be caused by caffeine, alcohol, nicotine, stretched atrial myocardium (e.g., as in hypervolemia), anxiety, hypokalemia, hypermetabolic states (e.g., with pregnancy), or atrial ischemia, injury, or infarction. PACs are often seen with sinus tachycardia. PACs have the following characteristics (see Fig. 23-9):

Ventricular and atrial rate: Depends on the underlying rhythm (e.g., sinus tachycardia)

Ventricular and atrial rhythm: Irregular due to early P waves, creating a PP interval that is shorter than the others. This is sometimes followed by a longer-than-normal PP interval but one that is less than twice the normal PP interval. This type of interval is called a *noncompensatory pause*.

QRS shape and duration: The QRS that follows the early P wave is usually normal, but it may be abnormal. It may even be absent.

P wave: An early and different P wave may be seen or may be hidden in the T wave; other P waves in the strip are consistent.

PR interval: The early P wave has a shorter-than-normal PR interval but still between 0.12 and 0.20 seconds.

P:QRS ratio: Usually 1:1

PACs are common in normal hearts. A pulse deficit (a difference between the apical and radial pulse rate) may exist.

Medical Management

If PACs are infrequent; no treatment is necessary. If there are more than 6 per minute, this may herald a worsening disease state or the onset of more serious arrhythmias, such as atrial fibrillation. Medical management is directed toward treating the underlying cause (e.g., reduction of caffeine intake and correction of hypokalemia).

Atrial Fibrillation

Atrial fibrillation is a very common arrhythmia, with an estimated 12.1 million Americans predicted to be living with atrial fibrillation by 2030 (Centers for Disease Control and Prevention [CDC], 2023). Atrial fibrillation can result from diverse pathophysiologic etiologies and risks (Chart 23-2).

Atrial fibrillation results from abnormal impulse formation that occurs when structural or electrophysiologic abnormalities alter the atrial tissue causing a rapid, disorganized, and uncoordinated twitching of the atrial musculature (January et al., 2014, 2019). The autonomic nervous system has a significant role in atrial fibrillation with both intrinsic and extrinsic regulatory factors (Kusayama et al., 2021). Separate from the extrinsic nervous system, which includes the brain and spinal cord, the cardiac autonomic nervous system (CANS) consists of a highly interconnected network of autonomic ganglia and nerve cell bodies embedded within the epicardium, largely within the atrial myocardium and pulmonary veins. Hyperactive autonomic ganglia in the CANS are thought to play a critical role in atrial fibrillation, resulting in impulses that are initiated from the pulmonary veins and conducted through the AV node. The ventricular rate of response depends on the conduction of atrial impulses through the AV node, presence of accessory electrical conduction pathways, and therapeutic effect of medications.

Lack of consistency in describing patterns or types of atrial fibrillation has led to the use of numerous labels, such as **paroxysmal** (i.e., sudden onset with spontaneous termination), persistent, and permanent. The recommended classification system is noted in Chart 23-3.

Atrial fibrillation has the following characteristics (Fig. 23-10):

Ventricular and atrial rate: Atrial rate is 300 to 600 bpm; ventricular rate is usually 120 to 200 bpm in untreated atrial fibrillation.

Ventricular and atrial rhythm: Highly irregular

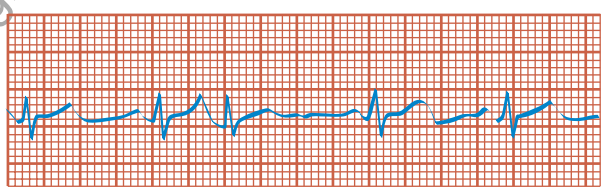

Figure 23-9 • Premature atrial complex (PAC). Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-22A). Wolters Kluwer.

Chart 23-2 Risk Factors
Atrial Fibrillation

- Alcohol use disorder
- Cardiothoracic surgery
- Diabetes
- European ancestry
- Exercise
- Family history
- Female sex
- Heart failure
- Hyperthyroidism
- Hypertension
- Increased pulse pressure
- Increasing age
- Myocardial infarction
- Obstructive sleep apnea
- Overweight and obesity
- Smoking
- Valvular heart disease

Adapted from CDC (2023); January et al. (2014, 2019).

Chart 23-3 Atrial Fibrillation Classification System

Type	Description
Paroxysmal	Sudden onset with termination that occurs spontaneously or after an intervention; lasts <7 d but may recur
Persistent	Continuous, lasting >7 d
Long-standing persistent	Continuous, lasting >12 mo
Permanent	Persistent, but the decision has been made not to restore or maintain sinus rhythm
Nonvalvular	Absence of moderate-to-severe mitral stenosis or mechanical heart valve

Adapted from January et al. (2014, 2019).

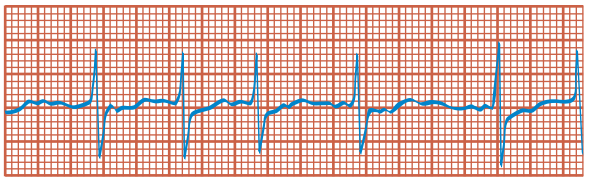

Figure 23-10 • Atrial fibrillation. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-22D). Wolters Kluwer.

QRS shape and duration: Usually normal but may be abnormal

P wave: No discernible P waves; irregular undulating waves that vary in amplitude and shape are seen and referred to as *fibrillatory* or *f waves*.

PR interval: Cannot be measured

P:QRS ratio: Many:1

Patients with atrial fibrillation are at increased risk of heart failure, myocardial ischemia, and embolic events such as stroke (January et al., 2014, 2019). A rapid and irregular ventricular response reduces the time for ventricular filling, resulting in a smaller stroke volume. Because atrial fibrillation causes a loss in AV synchrony (the atria and ventricles contract at different times), the atrial kick (the last part of diastole and ventricular filling, which accounts for 25% to 30% of the cardiac output) is also lost. Some patients with atrial fibrillation are without symptoms while others experience palpitations and clinical manifestations of heart failure (e.g., shortness of breath, hypotension, dyspnea on exertion, and fatigue; see Chapter 26). In addition, a high ventricular rate of response (greater than 80 bpm) during atrial fibrillation can eventually lead to mitral valve dysfunction, mitral regurgitation, intraventricular conduction delays, and dilated ventricular cardiomyopathy.

Patients with atrial fibrillation may exhibit a pulse deficit—a numeric difference between apical and radial pulse rates. The shorter time in diastole reduces the time available for coronary artery perfusion, thereby increasing the risk of myocardial ischemia with the onset of anginal symptoms (see Chapter 24). Decreasing the ventricular rate may avoid and correct these effects.

The erratic nature of atrial contraction, alterations in ventricular ejection, and atrial myocardial dysfunction promote the formation of thrombi, especially within the left atrium, increasing the risk of an embolic event. For patients with nonvalvular atrial fibrillation, up to 90% of the embolus origin is within the left atrial appendage (LAA) (Yin et al., 2022). A therapeutic approach to addressing the role of the LAA in atrial fibrillation, left atrial appendage occlusion (LAAO), is discussed later in this chapter.

Assessment and Diagnostic Findings

The clinical evaluation of atrial fibrillation should include a history and physical examination that identifies the onset and nature of signs and symptoms, including their frequency, duration, any precipitating factors, along with any response to medications, and any risk factors (see Chart 23-2). A 12-lead ECG is performed to verify the atrial fibrillation rhythm, as well as to identify LV hypertrophy, bundle branch block, prior myocardial ischemia, or other arrhythmias. The RR, QRS, and QT intervals are analyzed to verify the effectiveness of any prescribed antiarrhythmic medications (January et al., 2014, 2019). A transesophageal echocardiogram (TEE) can identify the presence of valvular heart disease, provide information about LV and RV size and function, RV pressures to identify pulmonary hypertension, LV hypertrophy, and presence of left atrial thrombi (January et al., 2014, 2019). Blood tests to screen for diseases that are known risks for atrial fibrillation (see Chart 23-2), including thyroid, renal, and hepatic function, are assessed in the patient with a new onset of atrial fibrillation, as well as when the ventricular rate is difficult to control (January et al., 2014, 2019). Additional tests may include a chest x-ray (to evaluate pulmonary vasculature in a patient suspected of having pulmonary hypertension), exercise stress test (to exclude myocardial ischemia or reproduce exercise-induced atrial fibrillation), Holter or event monitoring (see Chapter 22), and an EPS (January et al., 2014, 2019; see later discussion).

Medical Management

Treatment of atrial fibrillation depends on the cause, pattern, and duration of the arrhythmia, the ventricular response rate and the presence of structural or valvular heart disease, and other cardiac conditions such as coronary artery disease or heart failure. Strategies for both rhythm control and rate control are dependent on shared clinical decision making between the patient and primary provider. In some cases, atrial fibrillation spontaneously converts to sinus rhythm within 24 to 48 hours and without treatment. However, in instances where atrial fibrillation is concomitant with significant other morbid conditions (e.g., severe heart failure), the atrial fibrillation may be classified as "permanent," meaning that the patient and primary provider have made a joint decision to stop further attempts to restore or maintain sinus rhythm. Therefore, the management of atrial fibrillation may be different among various patients and may change over time.

Medical management revolves around preventing embolic events such as stroke with anticoagulant medications, controlling the ventricular rate of response with antiarrhythmic agents, and treating the arrhythmia as indicated so that it is converted to a sinus rhythm.

Pharmacologic Therapy

Antithrombotic Medications. Antithrombotic drugs may include anticoagulants and antiplatelet drugs. Oral antithrombotic therapy is indicated for most patients with nonvalvular atrial fibrillation (e.g., absence of mechanical heart valve) because it reduces the risk of stroke (January et al., 2014, 2019). Atrial fibrillation guidelines recommend the use of a scoring system to assist in the assessment of stroke risk. Antithrombotic therapy is then selected based on risk factors outlined in the mnemonic CHA_2DS_2-VASc (Chart 23-4) with each risk factor assigned points tallied for a total score that indicates an overall risk of stroke (January et al., 2014, 2019).

According to pharmacologic treatment guidelines (January et al., 2014, 2019):

- Patients with nonvalvular atrial fibrillation with a CHA_2DS_2-VASc score of 0 may choose the option of no antithrombotic therapy.
- Patients with nonvalvular atrial fibrillation with a CHA_2DS_2-VASc score of 1 may choose no

Chart 23-4 Stroke Risk Assessment for the Patient With Atrial Fibrillation: The CHA_2DS_2-VASc Scoring System

	Risk Factor	Points
C	Congestive heart failure (left ventricular systolic dysfunction)	1
H	Hypertension (BP >130/80 mm Hg)	1
A_2	Age ≥75 y	2
D	Diabetes	1
S_2	Prior stroke/TIA/thromboembolism	2
V	Vascular disease (i.e., prior MI, PAD, or aortic plaque)	1
A	Age 65–74 y	1
Sc	Sex category (female)	1

BP, blood pressure; MI, myocardial infarction; PAD, peripheral artery disease; TIA, transient ischemic attack.
Adapted from January et al. (2014, 2019).

antithrombotic therapy, treatment with an oral anticoagulant, or aspirin.

- Patients with nonvalvular atrial fibrillation with a CHA_2DS_2-VASc score of 2 or higher for males and 3 or higher for females may choose warfarin, a direct thrombin inhibitor (e.g., dabigatran), or a Factor Xa inhibitor (e.g., rivaroxaban, apixaban, and edoxaban).

Patients with atrial fibrillation with valvular heart disease or bioprosthetic heart valves may be prescribed warfarin, a direct-acting oral anticoagulant (DOAC), or a Factor Xa inhibitor (Samoš et al., 2022). For patients with mechanical heart valves, warfarin is recommended (January et al., 2014, 2019). If immediate or short-term anticoagulation is necessary, the patient may be placed on IV or low–molecular-weight heparin until warfarin therapy can be started and the international normalized ratio (INR) level reaches a therapeutic range consistent with antithrombosis, usually defined as an INR between 2.0 and 3.0 (see Chapter 27 for further discussion).

Medication selection for all patients with atrial fibrillation depends upon stroke and bleeding risks as well as specific patient needs and preferences (January et al., 2014, 2019; Samoš et al., 2022). For instance, treatment with warfarin will require weekly INR testing during initiation of therapy, as well as ongoing monitoring (see Chapter 27 for further discussion). Home monitoring of therapy is an option for some patients. DOAC and Factor Xa inhibitors require baseline assessment of hemoglobin and hematocrit, as well as liver and renal function, along with INR. Advantages of these medications include fewer drug–drug interactions, dietary limitations, and the elimination of frequent INR testing.

Medications That Control the Heart Rate. Maintaining a resting ventricular heart rate of less than 80 bpm is recommended in order to manage symptoms of atrial fibrillation (January et al., 2014, 2019). To decrease the ventricular rate in patients with paroxysmal, persistent, or permanent atrial fibrillation, a beta-blocker (Class II antiarrhythmic, see Table 23-1) or non–dihydropyridine calcium channel blocker (Class IV antiarrhythmic, see Table 23-1) is generally recommended (January et al., 2014, 2019).

Medications That Convert the Heart Rhythm or Prevent Atrial Fibrillation. For patients with atrial fibrillation lasting 48 hours or longer, anticoagulation is recommended prior to attempts to restore sinus rhythm pharmacologically or with electrical cardioversion. In the absence of therapeutic anticoagulation, TEE may be performed to identify left atrial thrombus formation prior to cardioversion (January et al., 2014, 2019). If no thrombus is identified, cardioversion can proceed.

Medications given to achieve pharmacologic cardioversion to sinus rhythm may include flecainide, dofetilide, propafenone, amiodarone, and IV ibutilide administered within 7 days of the onset of atrial fibrillation. Despite a degree of risk, dofetilide is a preferred medication because it is highly effective at converting atrial fibrillation to sinus rhythm, has fewer drug-to-drug interactions, and is better tolerated by patients than other medications (January et al., 2014, 2019).

Preoperative administration of beta-blockers (see Table 23-1) has resulted in a significant reduction in atrial fibrillation after cardiac surgery (Boons et al., 2021). Cholesterol-lowering drugs such as the HMG-CoA (3-hydroxy-3-methylglutaryl coenzyme A) reductase inhibitors (also called statins; see Chapter 24, Table 24-1) may also be prescribed to prevent new-onset atrial fibrillation following cardiac surgery (Nomani et al., 2021). If symptomatic, a catheter ablation may be indicated for rhythm control of paroxysmal atrial fibrillation that is refractory to at least one Class I or Class III antiarrhythmic medication (see Table 23-1) (January et al., 2014, 2019; see later discussion).

Electrical Cardioversion for Atrial Fibrillation

Electrical cardioversion is indicated for patients with atrial fibrillation who are hemodynamically unstable (e.g., acute alteration in mental status, chest discomfort, and hypotension) and do not respond to medications (January et al., 2014, 2019). Medications given to achieve pharmacologic cardioversion may be used before electrical cardioversion for enhanced outcomes.

Clinical Judgment Alert

Recognize Cues, Analyze Cues, & Generate Solutions

The nurse recognizes the cue of atrial fibrillation and analyzes that the patient is at high risk for thrombus formation. When electrical cardioversion is indicated for atrial fibrillation, the nurse anticipates that a transesophageal echocardiogram may be performed to evaluate for possible atrial thrombi before this procedure is conducted.

Because atrial function may be impaired for several weeks after cardioversion, antithrombotic therapy (e.g., warfarin) is indicated for at least 4 weeks after the procedure (January et al., 2014, 2019). Repeated attempts at electrical cardioversion may be made, following administration of an antiarrhythmic medication (see later discussion on "Electrical Cardioversion" section).

Cardiac Rhythm Therapies

Atrial fibrillation that does not respond to medications or electrical cardioversion may be treated by cardiac rhythm therapies, including catheter ablation, maze or mini-maze procedure, or convergent procedure.

Catheter Ablation Therapy

Catheter **ablation** destroys specific cells that are the cause of a tachyarrhythmia. Catheter ablation is performed most often today for atrial fibrillation, although it may also be useful in treating atrioventricular nodal reentry tachycardia (AVNRT) and recurrent ventricular tachycardia (VT) (see later discussion of these arrhythmias). Ablation is a procedure similar to a cardiac catheterization (see Chapter 22) involving a special catheter that is advanced at or near the origin of the arrhythmia. During these treatments, electromagnetic thermal techniques (heating or freezing) are used to destroy targeted tissue, and laser ablation is used to scar the tissue and eliminate the arrhythmia (Zhang et al., 2022). The tissue damage is more specific to the arrhythmic tissue, with less trauma to the surrounding cardiac tissue.

An EPS may be performed to induce the arrhythmia prior to the catheter ablation. During the ablation procedure, defibrillation pads, an automatic blood pressure cuff, and a pulse oximeter are used. The patient is usually given moderate sedation (see Chapter 15) and IV heparin to reduce the risk of periprocedural thromboembolism. Immediately after, the patient is monitored for another 30 to 60 minutes and then retested to ensure that the arrhythmia does not recur when attempting to induce. Successful ablation is achieved when the arrhythmia cannot be induced. Complications are rare, but more serious complications are often related to the catheterization process itself, including vascular injury, tricuspid valve damage, pulmonary embolism, hemorrhage, pericardial tamponade, MI, and stroke (Homoud et al., 2022).

Nursing Management. Postprocedural care for the patient post ablation is similar to the nursing management of a patient post cardiac catheterization (see Chapter 22). Postprocedural nursing interventions include frequent monitoring for sedation recovery, for arrhythmias, and for signs and symptoms of a stroke and vascular access site complications. Because of the prolonged time required for the procedure as well as the time needed in bed to obtain hemostasis at the vascular access site, it is not unusual for the patient to have back discomfort. In addition to administering any pain medications, the nurse may help to alleviate this pain by placing rolled towels under the patient's knees and waist.

Maze and Mini-Maze Procedures

The maze procedure is an open heart surgical procedure for refractory atrial fibrillation, which is typically only used for patients undergoing a concurrent cardiac surgery procedure. Small transmural incisions are made throughout the atria. The resulting formation of scar tissue prevents reentry conduction of the aberrant electrical impulse. Because the procedure requires significant time and cardiopulmonary bypass, its use is reserved only for those patients undergoing cardiac surgery for another reason (e.g., coronary artery bypass; January et al., 2014, 2019). Some patients may need a permanent pacemaker after this surgery because of subsequent injury to the SA node.

A modification of the maze procedure, minimally invasive maze surgery, or mini-maze, may be performed by making small incisions between the ribs, through which video-guided instruments are inserted. This surgery eliminates the need for opening the sternum, heart–lung bypass, and the use of cardioplegia (see Chapter 24 for further discussion of cardiac surgery). This results in a shorter recovery time and decreased risk of complications (January et al., 2014, 2019).

Convergent Procedure

The convergent procedure utilizes a hybrid approach to ablation, requiring the skills of both a cardiothoracic surgeon and an electrophysiologist, a cardiologist with specialized training. This procedure is associated with lower rates of arrhythmia recurrence than catheter ablation, and improvements have been made to minimize complications, making the procedure less complex and safer (Wats et al., 2020). The surgeon performs ablation of the epicardial wall in the area around the pulmonary veins, and the electrophysiologist performs ablation around the endocardial area of the pulmonary veins. The coordination between the surgeon, electrophysiologist, and the staff is essential to success. Epicardial mapping of the posterior wall scar utilizing 3D mapping systems allows for surgeons to create additional epicardial lesions. This process includes pulmonary vein isolation and posterior wall isolation followed by catheter ablation. The patient may need to recover in the hospital post procedure; however, most patients have this procedure done in a same-day surgery setting.

Left Atrial Appendage Occlusion

LAAO is an alternative to anticoagulant medications for stroke prevention in patients with nonvalvular atrial fibrillation (Kleinecke et al., 2021). As noted previously, the LAA is the area where the majority of stroke-causing blood clots form in patients with nonvalvular atrial fibrillation. However, concerns about the risk of long-term anticoagulant use and the risk of bleeding can complicate effective management (Ojo et al., 2020).

Candidates for LAAO include those patients with increased risk of stroke based on CHA_2DS_2-VASc scores of 1 or higher (see Chart 23-4) and those patients seeking a nonpharmacologic alternative to treatment for nonvalvular atrial fibrillation (Kleinecke et al., 2021). Commonly used is the WATCHMAN, a device typically inserted while the patient is under anesthesia. Similar to a percutaneous coronary intervention (PCI) procedure (see Chapter 24), a small incision is made, usually in the radial area, and in some cases in the femoral area for high-risk patients, and a catheter is then inserted that guides the device into position (Watson et al., 2021). The use of radial access for PCI in high-risk patients remains low despite an increase in adoption of radial access for PCI. The parachute-shaped device is threaded through to the opening of the LAA, sealing it off and preventing it from releasing clots (Fig. 23-11).

Patients, typically, fully recover as outpatients and resume activities of daily living within 24 hours after placement of a WATCHMAN device; however, some patients may need to stay in the hospital overnight. The nursing management of patients who received this device is similar to that of patients post cardiac catheterization (see Chapter 22). Patients are prescribed aspirin and warfarin post procedure; approximately

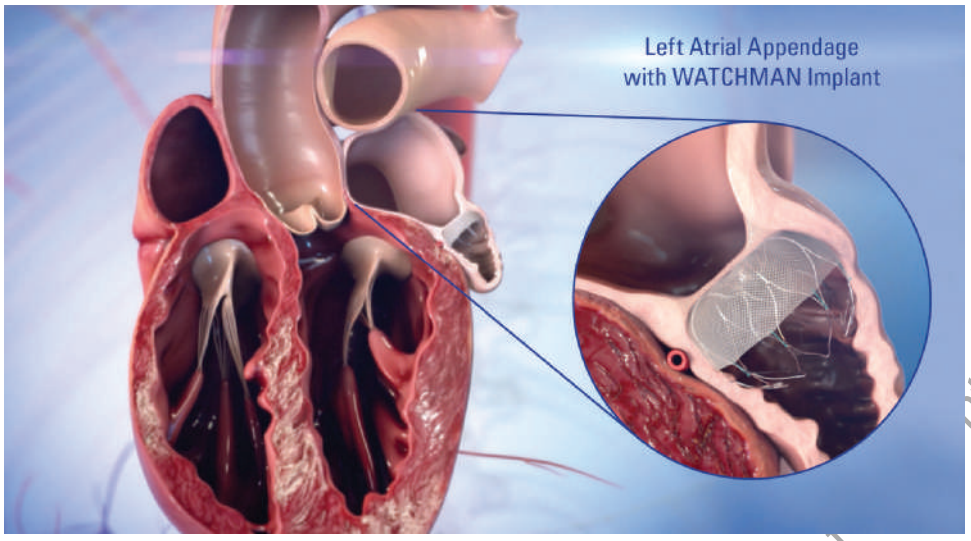

Figure 23-11 • The WATCHMAN device in place over the left atrial appendage. Copyright © 2021 Boston Scientific Corporation or its affiliates. All rights reserved.

6 weeks post procedure, they should return to the cardiology clinic for a TEE to confirm that the device has effectively occluded the LAA. If LAAO has occurred, then the patient may stop taking warfarin and is prescribed clopidogrel, an antiplatelet medication. After 6 months, the patient may stop taking clopidogrel but must continue taking daily aspirin indefinitely (Magdi et al., 2021).

Wolff-Parkinson-White Syndrome

In the patient with atrial fibrillation, if the QRS is wide and the ventricular rhythm is very fast and irregular, an *accessory pathway* should be suspected. An accessory pathway is typically congenital tissue between the atria, bundle of His, AV node, Purkinje fibers, or ventricular myocardium. This anomaly is known as Wolff-Parkinson-White (WPW) syndrome. Electrical cardioversion is the treatment of choice for atrial fibrillation in the presence of WPW syndrome that causes hemodynamic instability. Medications that block AV conduction (e.g., digoxin, diltiazem, and verapamil) should be avoided in WPW because they can increase the ventricular rate. If the patient is hemodynamically stable, procainamide, propafenone, flecainide, or amiodarone are recommended to restore sinus rhythm (January et al., 2014, 2019). Catheter ablation is performed for long-term management (see previous discussion).

Atrial Flutter

Atrial flutter occurs because of a conduction defect in the atrium and causes a rapid, regular atrial impulse at a rate between 250 and 400 bpm. Because the atrial rate is faster than the AV node can conduct, not all atrial impulses are conducted into the ventricle, causing a therapeutic block at the AV node. This is an important feature of this arrhythmia. If all atrial impulses were conducted to the ventricle, the ventricular rate would also be 250 to 400 bpm, which would result in ventricular fibrillation, a life-threatening arrhythmia. Atrial flutter risk factors mirror those for atrial fibrillation (Fuster et al., 2023; see Chart 23-2).

Atrial flutter has the following characteristics (Fig. 23-12):

Ventricular and atrial rate: Atrial rate ranges between 250 and 400 bpm; ventricular rate usually ranges between 75 and 150 bpm.

Ventricular and atrial rhythm: The atrial rhythm is regular; the ventricular rhythm is usually regular but may be irregular because of a change in the AV conduction.

QRS shape and duration: Usually normal but may be abnormal or absent

P wave: Saw-toothed shape; these waves are referred to as *F waves*.

PR interval: Multiple F waves may make it difficult to determine the PR interval.

P:QRS ratio: 2:1, 3:1, or 4:1

Medical Management

Atrial flutter can cause serious signs and symptoms, such as chest pain, shortness of breath, and low blood pressure. Medical management involves the use of vagal maneuvers or a trial administration of adenosine, which causes sympathetic block and slowing of conduction through the AV node. This may terminate the tachycardia or facilitate visualization of flutter waves for diagnostic purposes. Adenosine is given IV by rapid administration and immediately followed by a 20-mL saline flush and elevation of the arm with the IV line to promote rapid circulation of the medication. Atrial flutter is treated with antithrombotic therapy, rate control, and rhythm control

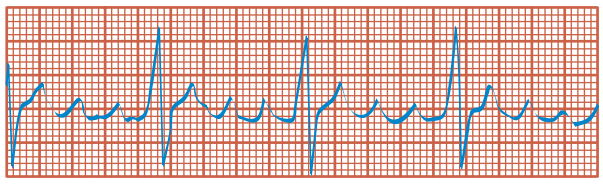

Figure 23-12 • Atrial flutter. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-22C). Wolters Kluwer.

in the same manner as atrial fibrillation (January et al., 2014, 2019). Electrical cardioversion is often successful in converting atrial flutter to sinus rhythm (see later discussion).

Junctional Arrhythmias

Junctional arrhythmias originate within AV nodal tissue and may include premature junctional complexes, junctional rhythms, nonparoxysmal junctional tachycardia, and AV nodal reentry tachycardia.

Premature Junctional Complex

A premature junctional complex is an impulse that starts in the AV nodal area before the next normal sinus impulse reaches the AV node. Premature junctional complexes are less common than PACs. Causes include digitalis toxicity, heart failure, and coronary artery disease. The ECG criteria for premature junctional complex are the same as for PACs, except for the P wave and the PR interval. The P wave may be absent, may follow the QRS, or may occur before the QRS but with a PR interval of less than 0.12 seconds. This arrhythmia rarely produces significant symptoms. Treatment for frequent premature junctional complexes is the same as for frequent PACs.

Junctional Rhythm

Junctional rhythm occurs when the AV node becomes the pacemaker of the heart. When the sinus node slows (e.g., from increased vagal tone) or when the impulse cannot be conducted through the AV node (e.g., because of complete heart block), the AV node automatically discharges an impulse. Junctional rhythm not caused by complete heart block has the following characteristics (Fig. 23-13):

- *Ventricular and atrial rate:* Ventricular rate 40 to 60 bpm; atrial rate also 40 to 60 bpm if P waves are discernible.
- *Ventricular and atrial rhythm:* Regular
- *QRS shape and duration:* Usually normal but may be abnormal
- *P wave:* May be absent, after the QRS complex or before the QRS; may be inverted, especially in lead II
- *PR interval:* If the P wave is in front of the QRS, the PR interval is less than 0.12 seconds.
- *P:QRS ratio:* 1:1 or 0:1

Medical Management

Junctional rhythm may produce signs and symptoms of reduced cardiac output. If this occurs, the treatment is the same as for sinus bradycardia, and emergency pacing may be needed (see later discussion under "Pacemaker Therapy").

Nonparoxysmal Junctional Tachycardia

Junctional tachycardia is caused by enhanced automaticity in the junctional area, resulting in a rhythm similar to junctional rhythm, except at a higher rate of 70 to 120 bpm. Although this rhythm generally does not have any detrimental hemodynamic effect, it may indicate a serious underlying condition, such as digitalis toxicity, myocardial ischemia, hypokalemia, or chronic obstructive pulmonary disease.

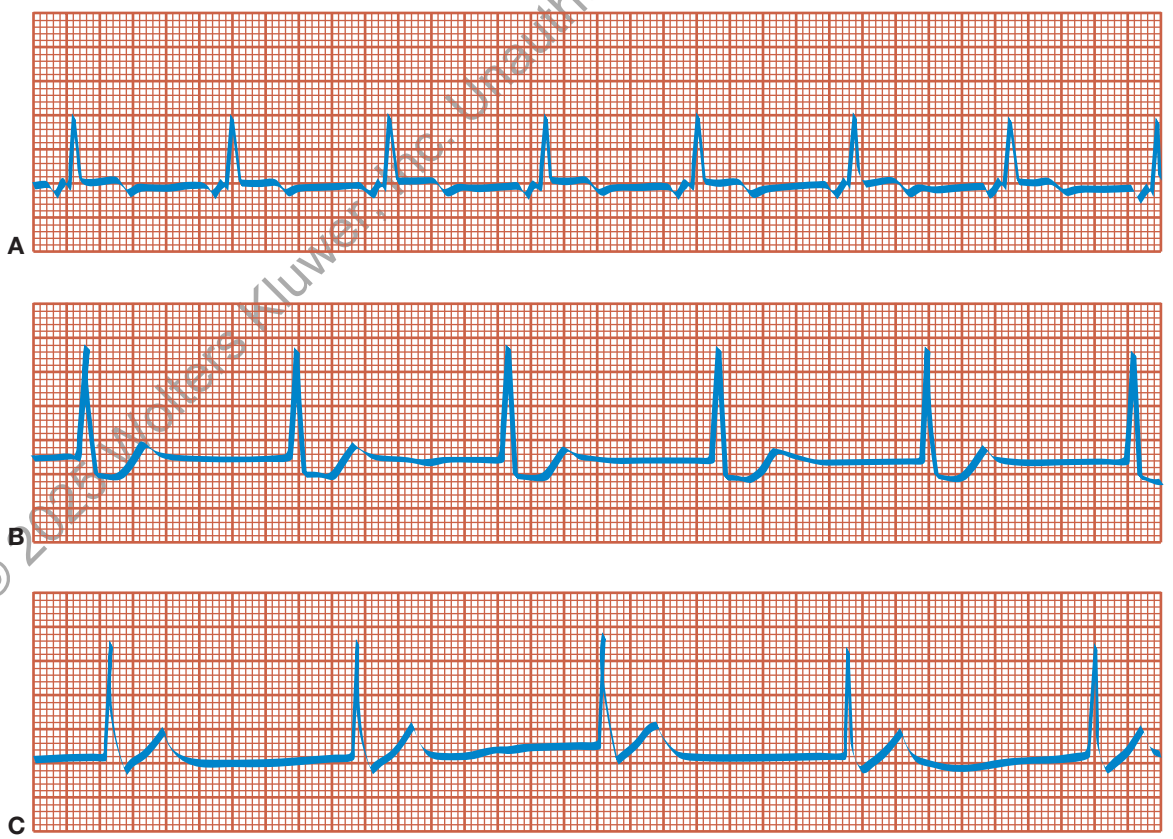

Figure 23-13 • Junctional rhythm. **A.** Note that the inverted P wave appears before the normal QRS complex. **B.** Note that the inverted P wave is buried inside the QRS complex. **C.** Note that the inverted P wave follows the QRS complex. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-23A-C). Wolters Kluwer.

Atrioventricular Nodal Reentry Tachycardia

AVNRT is a common arrhythmia that occurs when an impulse is conducted to an area in the AV node that causes the impulse to be repeatedly rerouted back into the same area at a very fast rate. Each time the impulse is conducted through this area, it is also conducted down into the ventricles, causing a fast ventricular rate. AVNRT that has an abrupt onset and an abrupt cessation with a QRS of normal duration has frequently been called *paroxysmal atrial tachycardia (PAT)* and also *paroxysmal supraventricular tachycardia (PSVT)*. AVNRT also occurs when the duration of the QRS complex is 0.12 seconds or greater and a block in the bundle branch is known to be present. This arrhythmia may last for seconds or several hours. Factors associated with the development of AVNRT include caffeine, nicotine, hypoxemia, and stress. Underlying pathologies include coronary artery disease and cardiomyopathy; however, it occurs more often in females and not in association with underlying structural heart disease. AVNRT has the following characteristics (Fig. 23-14):

- *Ventricular and atrial rate:* Atrial rate is usually 150 to 250 bpm; ventricular rate is usually 120 to 200 bpm.
- *Ventricular and atrial rhythm:* Regular; sudden onset and termination of the tachycardia
- *QRS shape and duration:* Usually normal but may be abnormal
- *P wave:* Usually very difficult to discern
- *PR interval:* If the P wave is in front of the QRS, the PR interval is less than 0.12 seconds.
- *P:QRS ratio:* 1:1, 2:1

Clinical symptoms vary with the rate and duration of the tachycardia and the patient's underlying condition. The tachycardia usually is of short duration, resulting only in palpitations. A fast rate may also reduce cardiac output, resulting in significant signs and symptoms such as restlessness, chest pain, shortness of breath, pallor, hypotension, and loss of consciousness.

Medical Management

Because AVNRT is generally a benign non–life-threatening arrhythmia, the goal of medical management is to alleviate symptoms and improve quality of life. Patients with minimum symptoms with an AVNRT that terminates spontaneously or with minimal treatment may choose just to be monitored and self-treat. Patients who become significantly symptomatic and require emergency department visits to terminate the rhythm may want to initiate therapy immediately.

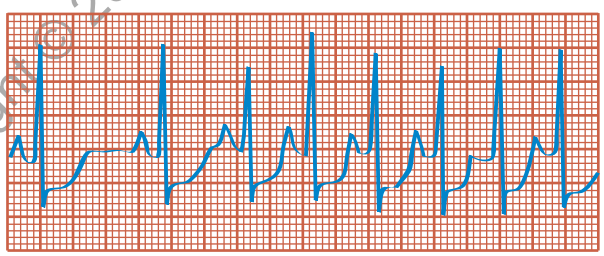

Figure 23-14 • Atrioventricular nodal reentry tachycardia (AVNRT), also called paroxysmal atrial tachycardia (PAT), and paroxysmal supraventricular tachycardia (PSVT). Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 17-22B). Wolters Kluwer.

The aim of therapy is to break the reentry of the impulse. Catheter ablation is the initial treatment of choice. It attempts to immediately eliminate the area that permits the rerouting of the impulse causing the tachycardia (Hafeez & Armstrong, 2023; see previous discussion of "Atrial Fibrillation" and "Catheter Ablation Therapy" sections). Vagal maneuvers may be used to interrupt AVNRT. These techniques increase parasympathetic stimulation, causing slower conduction through the AV node and blocking the reentry of the rerouted impulse. Some patients use some of these methods to terminate the episode on their own.

Pharmacologic Therapy

If the vagal maneuvers are ineffective, the patient may then receive a bolus of adenosine to correct the rhythm; this is usually effective in terminating AVNRT. Because the effect of adenosine is so short, AVNRT may recur; the first dose may be followed by two additional doses. If the vagal maneuvers and adenosine are ineffective, IV non–dihydropyridine calcium channel blockers (e.g., verapamil), IV beta-blockers, or IV digoxin may be considered (Hafeez & Armstrong, 2023). If the patient is unstable or does not respond to the medications, electrical cardioversion is the treatment of choice (see later discussion).

If P waves cannot be identified, the rhythm may be called *supraventricular tachycardia* (SVT), or PSVT if it has an abrupt onset, until the underlying rhythm and resulting diagnosis are determined. SVT and PSVT indicate only that the rhythm is not VT. SVT could be atrial fibrillation, atrial flutter, or AVNRT, among others. Vagal maneuvers and adenosine may be used to convert the rhythm or at least slow conduction in the AV node to allow visualization of the P waves.

Ventricular Arrhythmias

Ventricular arrhythmias originate from foci within the ventricles; these may include PVCs, VT, ventricular fibrillation, idioventricular rhythms, and ventricular asystole.

Premature Ventricular Complex

A PVC is an impulse that starts in a ventricle and is conducted through the ventricles before the next normal sinus impulse. PVCs can occur in healthy people, especially with the intake of caffeine, nicotine, or alcohol. PVCs may be caused by cardiac ischemia or infarction, increased workload on the heart (e.g., heart failure and tachycardia), digitalis toxicity, hypoxia, acidosis, or electrolyte imbalances, especially hypokalemia. In a rhythm referred to as *bigeminy*, every other complex is a PVC. In trigeminy, every third complex is a PVC, and in quadrigeminy, every fourth complex is a PVC. PVCs have the following characteristics (Fig. 23-15):

- *Ventricular and atrial rate:* Depends on the underlying rhythm
- *Ventricular and atrial rhythm:* Irregular due to early QRS, creating one RR interval that is shorter than the others. The PP interval may be regular, indicating that the PVC did not depolarize the sinus node.
- *QRS shape and duration:* Duration is 0.12 seconds or longer; shape is bizarre and abnormal. When these bizarrely shaped, widened QRS complexes resemble each other, they are called unifocal. When they have at least two different morphologic appearances, they are called multifocal.

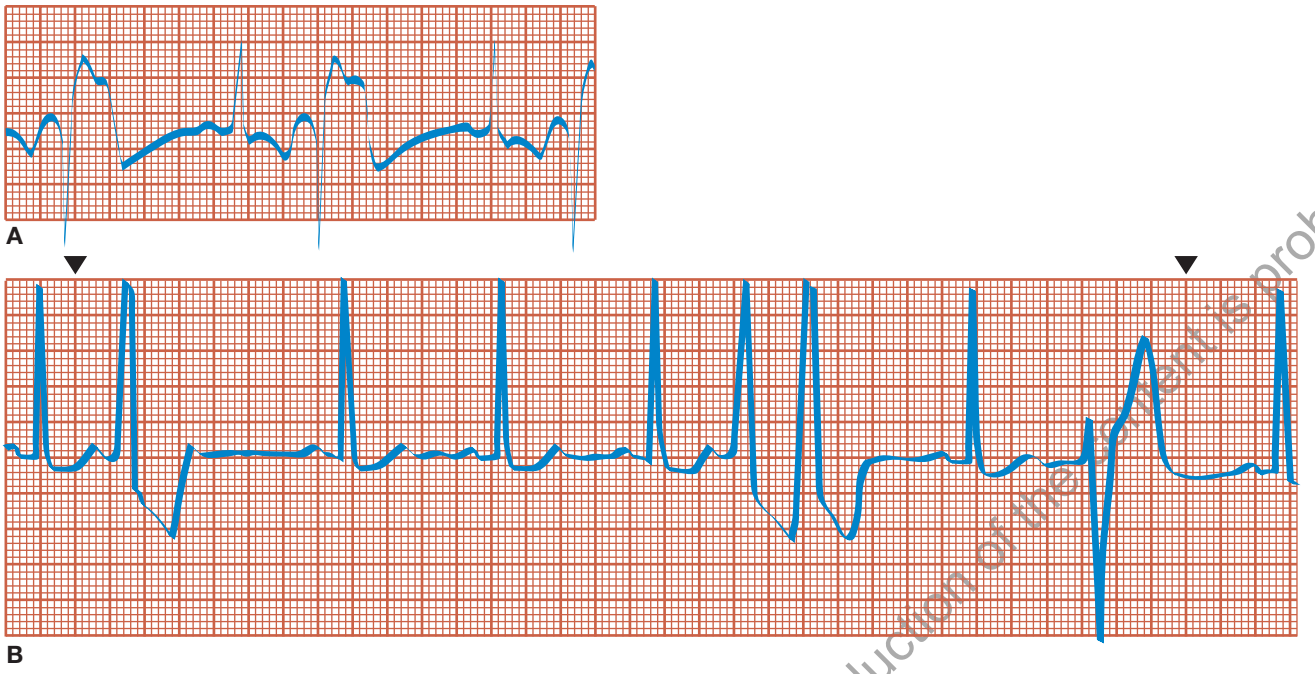

Figure 23-15 • Premature ventricular complexes (PVCs). **A.** Ventricular bigeminy that is unifocal; note that every other beat is a PVC, with the same morphologic appearance. **B.** Multifocal PVCs; note that there are at least two different appearing PVCs. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-25B and C). Wolters Kluwer.

P wave: Visibility of the P wave depends on the timing of the PVC; may be absent (hidden in the QRS or T wave) or in front of the QRS. If the P wave follows the QRS, the shape of the P wave may be different.

PR interval: If the P wave is in front of the QRS, the PR interval is less than 0.12 seconds.

P:QRS ratio: 0:1; 1:1

The patient may feel nothing or may say that the heart "skipped a beat." The effect of a PVC depends on its timing in the cardiac cycle and how much blood was in the ventricles when they contracted.

Medical Management

PVCs are a common occurrence and may increase in frequency with age (Al-khatib et al., 2018). Initial treatment is aimed at correcting the cause. PVCs that are frequent and persistent may be treated with amiodarone or beta-blockers, but long-term pharmacotherapy for PVCs is not usually indicated. PVCs are not considered a warning for ensuing VT. However, studies have shown an association of PVCs with adverse outcomes; therefore, patients may need to be evaluated for underlying causes (e.g., ischemic heart disease and LV dysfunction) (Al-khatib et al., 2018).

Ventricular Tachycardia

VT is defined as three or more PVCs in a row, occurring at a rate exceeding 100 bpm. The causes are similar to those of PVC. Patients with larger MIs and lower ejection fractions are at higher risk of lethal VT. VT is an emergency because the patient is nearly always unresponsive and pulseless. VT has the following characteristics (Fig. 23-16):

Ventricular and atrial rate: Ventricular rate is 100 to 200 bpm; atrial rate depends on the underlying rhythm.

Unfolding Patient Stories: Kenneth Bronson • Part 2

Recall from Chapter 19 Kenneth Bronson, who came to the emergency department with difficulty breathing after a week of flulike symptoms, productive cough, and high fever. He was diagnosed with a right lower lobe pneumonia. Sinus tachycardia with occasional unifocal premature ventricular contractions (PVCs) is seen on the cardiac monitor. What cues do you notice as potential causes for the tachycardia and PVCs when considering his age, symptoms experienced over the last week, and the clinical manifestations associated with his diagnosis?

Care for Kenneth and other patients in a realistic virtual environment: ***vSim*** *for Nursing* (**thepoint.lww.com/vSimMedicalSurgical**). Practice documenting these patients' care in **thepoint.lww.com/DocuCareEHR**.

Ventricular and atrial rhythm: Usually regular; atrial rhythm may also be regular.

QRS shape and duration: Duration is 0.12 seconds or more; bizarre, abnormal shape

P wave: Very difficult to detect, so the atrial rate and rhythm may be indeterminable.

PR interval: Very irregular, if P waves are seen.

P:QRS ratio: Difficult to determine, but if P waves are apparent, there are usually more QRS complexes than P waves.

The patient's tolerance or lack of tolerance for this rapid rhythm depends on the ventricular rate and severity of ventricular dysfunction.

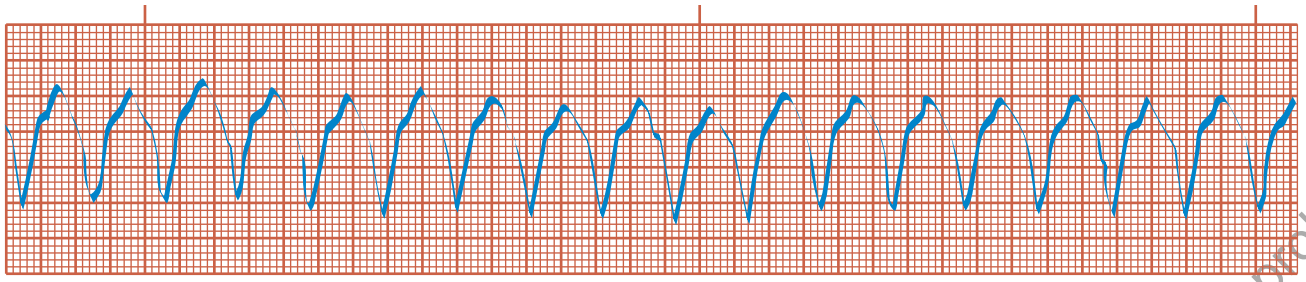

Figure 23-16 • Ventricular tachycardia. Reprinted with permission from Huff, J. (2006). *ECG workout* (5th ed., p. 199). Lippincott, Williams & Wilkins.

Medical Management

Several factors determine the initial treatment, including: identifying the rhythm as monomorphic (having a consistent QRS shape and rate) or polymorphic (having varying QRS shapes and rhythms), determining the existence of a prolonged QT interval before the initiation of VT, any comorbidities, and ascertaining the patient's heart function. If the patient is stable, continuing the assessment, especially obtaining a 12-lead ECG, may be the only action necessary.

The patient may need antiarrhythmic medications, antitachycardia pacing, or direct cardioversion or defibrillation. Procainamide, amiodarone, sotalol, and lidocaine are all antiarrhythmic medications that may be considered based on the type of VT (e.g., monomorphic or polymorphic), clinical presentation, and patient comorbidities (e.g., impaired cardiac function and acute MI).

Cardioversion is the treatment of choice for monophasic VT in a patient who is symptomatic. **Defibrillation**, which uses an electrical current given to stop the arrhythmia that is not set to synchronize with the patient's QRS complex, is the treatment of choice for pulseless VT. Any type of VT in a patient who is unconscious and without a pulse is treated in the same manner as ventricular fibrillation: Immediate defibrillation is the action of choice (see later discussion on "Cardioversion and Defibrillation" section).

For long-term management, patients with an ejection fraction less than 35% should be considered for an implantable cardioverter defibrillator (ICD) (see later discussion). Those with an ejection fraction greater than 35% may be managed with antiarrhythmic medication.

Torsades de pointes is a polymorphic VT preceded by a prolonged QT interval, which could be congenital or acquired. Common causes include central nervous system disease; certain medications (e.g., ciprofloxacin, erythromycin, haloperidol, lithium, and methadone); or low levels of potassium, calcium, or magnesium. Congenital QT prolongation is another cause. Because this rhythm is likely to cause the patient to deteriorate and become pulseless, immediate treatment is required by identifying and treating the underlying cause and includes correction of any electrolyte imbalance, such as administration of IV magnesium, and with IV isoproterenol pacing if associated with bradycardia (Frampton et al., 2023; Wyckoff et al., 2022).

Ventricular Fibrillation

The most common arrhythmia in patients with cardiac arrest is ventricular fibrillation, which is a rapid, disorganized ventricular rhythm that causes ineffective quivering of the ventricles. No atrial activity is seen on the ECG. The most common cause of ventricular fibrillation is acute coronary artery disease and resulting acute MI (Frampton et al., 2023; Wyckoff et al., 2022). Other causes include untreated or unsuccessfully treated VT, cardiomyopathy, valvular heart disease, Brugada syndrome, several proarrhythmic medications, acid–base and electrolyte abnormalities, and electrical shock. Ventricular fibrillation has the following characteristics (Fig. 23-17):

Ventricular rate: Greater than 300 bpm

Ventricular rhythm: Extremely irregular, without a specific pattern

QRS shape and duration: Irregular, undulating waves with changing amplitudes. There are no recognizable QRS complexes.

Medical Management

Ventricular fibrillation is characterized by the absence of an audible heartbeat, a palpable pulse, and respirations. Because there is no coordinated cardiac activity, cardiac arrest and death are imminent if the arrhythmia is not corrected. Immediate electrical defibrillation and cardiopulmonary resuscitation (CPR) are essential for survival. Antiarrhythmic drugs such as amiodarone may be indicated when defibrillation attempts are unsuccessful or in the setting of recurrent ventricular fibrillation (Frampton et al., 2023; see Chapter 26 for further discussion on interventions during cardiac arrest).

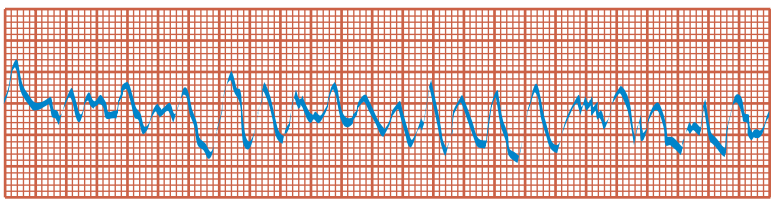

Figure 23-17 • Ventricular fibrillation. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-27C). Wolters Kluwer.

Idioventricular Rhythm

Idioventricular rhythm, also called *ventricular escape rhythm*, occurs when the impulse starts in the conduction system below the AV node. When the sinus node fails to create an impulse (e.g., from increased vagal tone) or when the impulse is created but cannot be conducted through the AV node (e.g., due to complete AV block), the Purkinje fibers automatically discharge an impulse. When idioventricular rhythm is not caused by AV block, it has the following characteristics (Fig. 23-18):

Ventricular rate: Between 20 and 40 bpm; if the rate exceeds 40 bpm, the rhythm is known as accelerated idioventricular rhythm.
Ventricular rhythm: Regular
QRS shape and duration: Bizarre, abnormal shape; duration is 0.12 seconds or more.

Medical Management

Idioventricular rhythm commonly causes the patient to lose consciousness and experience other signs and symptoms of reduced cardiac output. In such cases, the treatment is the same as for asystole and pulseless electrical activity (PEA) (see Chapter 26) if the patient is in cardiac arrest or for bradycardia if the patient is not in cardiac arrest. Interventions should follow current advanced cardiovascular life support (ACLS) guidelines once identifying the underlying cause; administering IV epinephrine, atropine, and vasopressor medications; and initiating emergency transcutaneous pacing as needed. In some cases, idioventricular rhythm may cause no symptoms of reduced cardiac output.

Ventricular Asystole

Commonly called *flatline*, ventricular asystole is characterized by absent QRS complexes confirmed in two different leads. Occasionally, P waves may be apparent for a short duration of time. There is no heartbeat, no palpable pulse, and no respiration. Without immediate treatment, ventricular asystole is fatal.

Medical Management

Ventricular asystole is treated the same as PEA, focusing on high-quality CPR with minimal interruptions and identifying underlying and contributing factors. The key to successful treatment is a rapid assessment to identify possible causes, which are usually identified with a helpful mnemonic known as the Hs and Ts. The Hs include *H*ypovolemia, *H*ypoxia, *H*ydrogen ion (acidosis), *H*ypo/*H*yperkalemia, and *H*ypothermia. The Ts include *T*ension pneumothorax, *T*amponade (cardiac), *T*oxins, and *T*hrombosis (both pulmonary and coronary). If identified, these must be immediately treated (Jordan et al., 2023). After the initiation of CPR, intubation and establishment of IV access are the next recommended actions, with no or minimal interruptions in chest compressions (see Chapter 26).

Conduction Abnormalities

When assessing the rhythm strip, the underlying rhythm is first identified. Then, the PR interval is assessed for the possibility of an AV block. AV blocks occur when the conduction of the impulse through the AV node or bundle of His area is decreased or stopped. These blocks can be caused by medications (e.g., digitalis, calcium channel blockers, and beta-blockers), Lyme disease, myocardial ischemia and infarction, hypothyroidism, or activities that cause an increase in vagal tone (Kusumoto et al., 2019). If the AV block is caused by increased vagal tone (e.g., long-term athletic training, sleep, coughing, suctioning, pressure above the eyes or on large vessels, and anal stimulation), it is commonly accompanied by sinus bradycardia. AV block may be temporary and resolve on its own, especially after correcting the underlying cause, but in some cases, it may be permanent and require permanent pacing (Frampton et al., 2023).

The clinical signs and symptoms of a heart block vary with the resulting ventricular rate and the severity of any underlying disease processes. Whereas first-degree AV block rarely causes any hemodynamic effect, the other blocks may result in decreased heart rate, causing a decrease in perfusion to vital organs. The treatment is based on the hemodynamic effect of the rhythm, and providers must always keep in mind the need to treat the patient, not the rhythm. The treatment is based on the hemodynamic effect of the rhythm.

First-Degree Atrioventricular Block

First-degree AV block occurs when all the atrial impulses are conducted through the AV node into the ventricles at a rate slower than normal. This conduction disorder has the following characteristics (Fig. 23-19):

Ventricular and atrial rate: Depends on the underlying rhythm
Ventricular and atrial rhythm: Depends on the underlying rhythm
QRS shape and duration: Usually normal but may be abnormal
P wave: In front of the QRS complex; shows sinus rhythm, regular shape

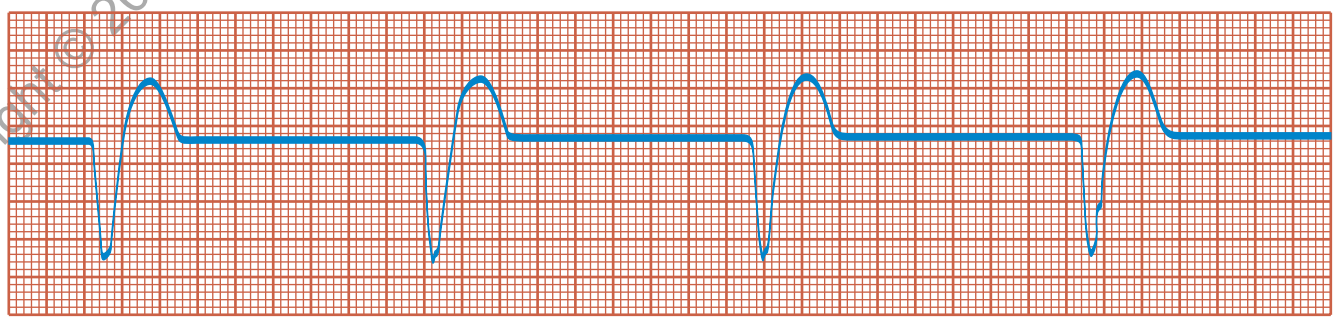

Figure 23-18 • Idioventricular rhythm.

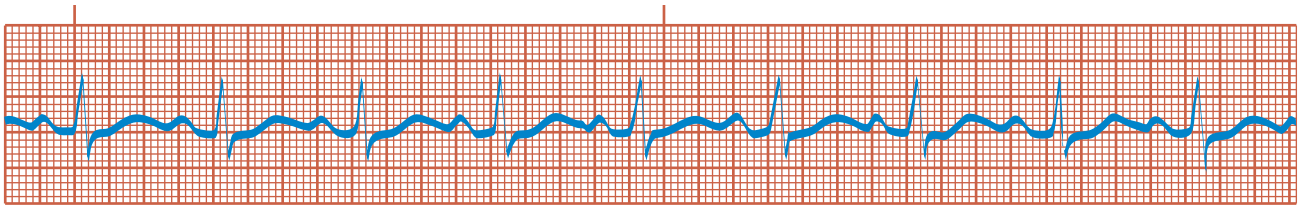

Figure 23-19 • First-degree atrioventricular block. Note that the PR interval is constant but greater than 0.20 seconds. Reprinted with permission from Huff, J. (2006). *ECG workout* (5th ed., p. 150). Lippincott, Williams & Wilkins.

PR interval: Greater than 0.20 seconds; PR interval measurement is constant.
P:QRS ratio: 1:1

Second-Degree Atrioventricular Block, Type I (Wenckebach)

Second-degree AV block, type I, occurs when there is a repeating pattern in which all but one of a series of atrial impulses are conducted through the AV node into the ventricles (e.g., every four of five atrial impulses are conducted). Each atrial impulse takes a longer time for conduction than the one before, until one impulse is fully blocked. Because the AV node is not depolarized by the blocked atrial impulse, the AV node has time to fully repolarize so that the next atrial impulse can be conducted within the shortest amount of time. Second-degree AV block, type I, has the following characteristics (Fig. 23-20):

Ventricular and atrial rate: Depends on the underlying rhythm, but the ventricular rate is lower than the atrial rate.
Ventricular and atrial rhythm: The PP interval is regular if the patient has an underlying normal sinus rhythm; the RR interval characteristically reflects a pattern of change. Starting from the RR that is the longest, the RR interval gradually shortens until there is another long RR interval.
QRS shape and duration: Usually normal but may be abnormal
P wave: In front of the QRS complex; shape depends on underlying rhythm.
PR interval: The PR interval becomes longer with each succeeding ECG complex until there is a P wave not followed by a QRS. The changes in the PR interval are repeated between each "dropped" QRS, creating a pattern in the irregular PR interval measurements.
P:QRS ratio: 3:2, 4:3, 5:4, and so forth

Second-Degree Atrioventricular Block, Type II

Second-degree AV block, type II, occurs when only some of the atrial impulses are conducted through the AV node into the ventricles. Second-degree AV block, type II, has the following characteristics (Fig. 23-21):

Ventricular and atrial rate: Depends on the underlying rhythm, but the ventricular rate is lower than the atrial rate
Ventricular and atrial rhythm: The PP interval is regular if the patient has an underlying normal sinus rhythm. The RR interval is usually regular but may be irregular, depending on the P:QRS ratio.
QRS shape and duration: Usually abnormal but may be normal
P wave: In front of the QRS complex; shape depends on underlying rhythm.
PR interval: The PR interval is constant for those P waves just before QRS complexes.
P:QRS ratio: 2:1, 3:1, 4:1, 5:1, and so forth

Third-Degree Atrioventricular Block

Third-degree AV block occurs when no atrial impulse is conducted through the AV node into the ventricles. In third-degree AV block, two impulses stimulate the heart: One stimulates the ventricles, represented by the QRS complex, and one stimulates the atria, represented by the P wave. P waves may be seen, but the atrial electrical activity is not conducted down into the ventricles to initiate the QRS complex, the ventricular electrical activity. Having two impulses

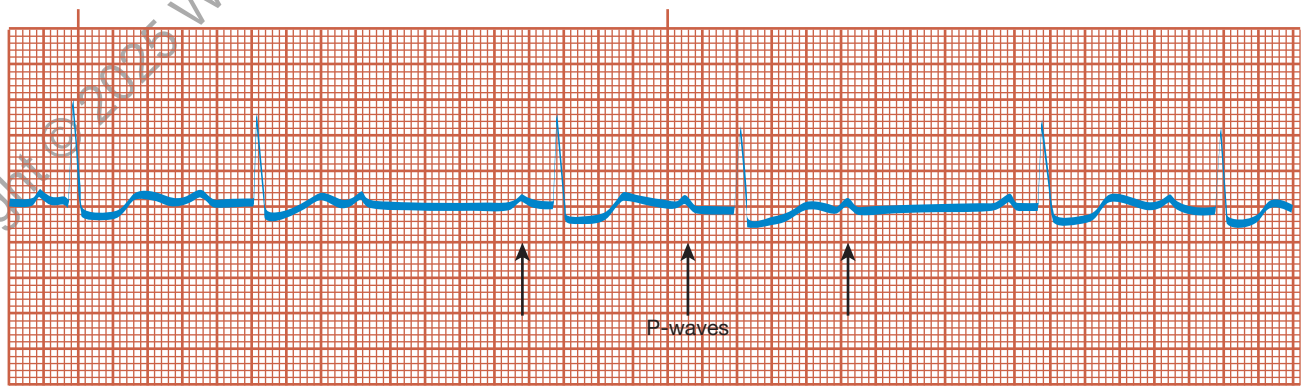

Figure 23-20 • Second-degree atrioventricular block, type I. Note progressively longer PR durations until there is a nonconducted P wave. Reprinted with permission from Huff, J. (2017). *ECG workout: Exercises in arrhythmia interpretation* (7th ed., Fig. 8-20). Wolters Kluwer.

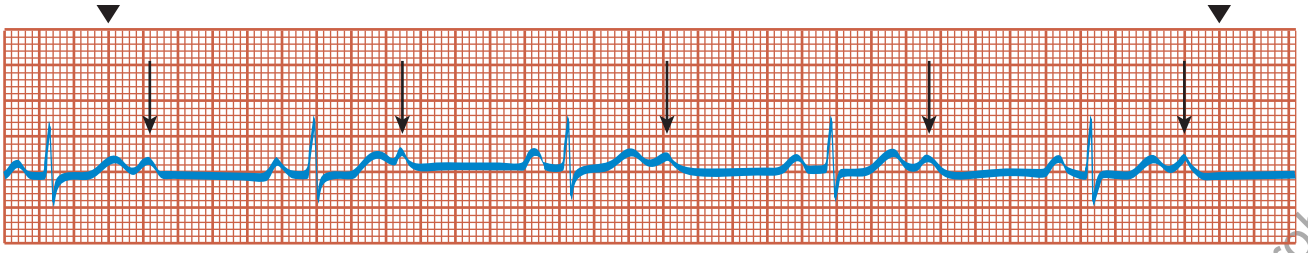

Figure 23-21 • Second-degree atrioventricular block, type II. Note constant PR interval and the presence of more P waves than QRS complexes. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-29C). Wolters Kluwer.

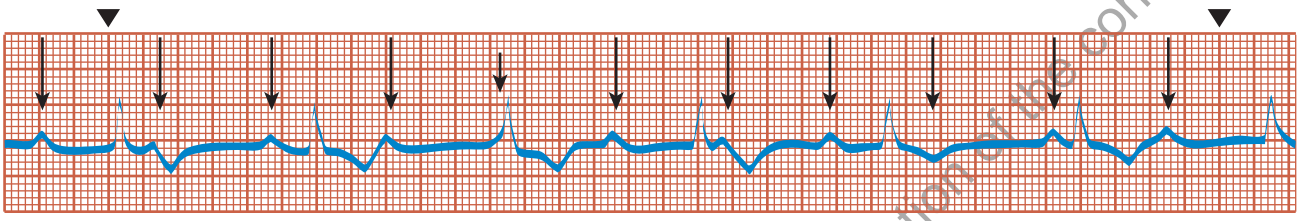

Figure 23-22 • Third-degree atrioventricular block; devoid relationship between P waves and QRS complexes. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 14-29D). Wolters Kluwer.

stimulate the heart results in a condition referred to as AV *dissociation*, which may also occur during VT. Complete block (third-degree AV block) has the following characteristics (Fig. 23-22):

Ventricular and atrial rate: Depends on the escape rhythm (junctional or idioventricular) and underlying atrial rhythm, but the ventricular rate is lower than the atrial rate.

Ventricular and atrial rhythm: The PP interval is regular and the RR interval is regular, but the PP interval is not equal to the RR interval.

QRS shape and duration: Depends on the escape rhythm; with junctional rhythm, QRS shape and duration are usually normal; with idioventricular rhythm, QRS shape and duration are usually abnormal.

P wave: Depends on underlying rhythm

PR interval: Very irregular

P:QRS ratio: More P waves than QRS complexes

Medical Management of Conduction Abnormalities

Based on the cause of the AV block and the stability of the patient, treatment is directed toward increasing the heart rate to maintain a normal cardiac output. If the patient is stable without symptoms, no treatment may be indicated or it may simply consist of decreasing or eliminating the cause (e.g., withholding the medication or treatment). If the cause of the block is directly related to medication necessary for treating other conditions, permanent pacemaker implantation may be indicated. In some patients, temporary pacing may be necessary. Temporary pacing is most often used to treat patients with severe symptomatic bradycardias or tachycardias with the goal to restore compromised hemodynamic status until causes are resolved or a permanent pacemaker is placed (He et al., 2023).

NURSING PROCESS

The Patient With an Arrhythmia

Assessment

The priority assessment for a patient with an arrhythmia should begin with identifying the possible cause. Other major areas of assessment include contributing factors and the arrhythmia's effect on the heart's ability to maintain adequate perfusion. When cardiac output is reduced, the amount of oxygen reaching the tissues and vital organs is diminished. This diminished oxygenation produces the signs and symptoms associated with arrhythmias, which may cause significant distress and disrupt activities of daily living.

A health history is obtained to identify any previous occurrences of arrhythmias, decreased cardiac output, such as syncope, lightheadedness, dizziness, fatigue, chest discomfort, and palpitations. Possible causes of the arrhythmia need to be identified. All prescribed and over-the-counter medications, including herbs and nutritional supplements, are reviewed. If a patient is taking an antiarrhythmic medication, an assessment for treatment adherence, side effects, adverse reactions, and potential contraindications is necessary. For example, some medications (e.g., digoxin) can cause arrhythmias. Laboratory results are reviewed to assess levels of medications as well as factors that could contribute to the arrhythmia (e.g., anemia). A thorough psychosocial assessment is performed to identify the possible effects of the arrhythmia, the patient's perception and understanding of the arrhythmia and its treatment, and whether anxiety is a significant contributing factor.

The nurse conducts a physical assessment to confirm the data obtained from the history and to observe for signs of diminished cardiac output during the arrhythmic event, especially changes in the level of consciousness. The nurse assesses the patient's skin, which may be pale and cool. Signs of fluid retention, such as neck vein distention and crackles and wheezes auscultated in the lungs, may be detected. The rate and rhythm of apical and peripheral pulses are also assessed, and any

pulse deficit is noted. The nurse auscultates for extra heart sounds (especially S_3 and S_4) and for heart murmurs, measures blood pressure, and determines pulse pressures. A declining pulse pressure indicates reduced cardiac output. One assessment may not disclose significant changes in cardiac output; therefore, the nurse compares multiple assessment findings over time, especially those that occur with and without the arrhythmia.

Diagnosis

Nursing Diagnoses

Based on the assessment data, major nursing diagnoses may include:

- Impaired cardiac output associated with inadequate ventricular filling or altered heart rate
- Anxiety associated with fear of the unknown outcome of altered health state
- Lack of knowledge of disease and treatment regime

Collaborative Problems/Potential Complications

Potential complications may include:

- Cardiac arrest (see Chapter 26)
- Heart failure (see Chapter 26)
- Thromboembolic event, especially with atrial fibrillation (see Chapter 27)

Planning and Goals

The major goals for the patient may include eliminating or decreasing the occurrence of the arrhythmia (by addressing contributory factors) to maintain cardiac output; verbalizing reduction in anxiety; verbalizing an understanding about the arrhythmia, tests used to diagnose the problem, and its treatment; and developing or maintaining self-management skills.

Nursing Interventions

Monitoring and Managing the Arrhythmia to Maintain Cardiac Output

The nurse evaluates the patient's blood pressure, pulse rate and rhythm, rate and depth of respirations, and breath sounds on an ongoing basis to determine the arrhythmia's hemodynamic effect. The nurse also asks the patient about possible symptoms of the arrhythmia (e.g., episodes of lightheadedness, dizziness, or fainting) as part of the ongoing assessment. If a patient with an arrhythmia is hospitalized, the nurse may obtain a 12-lead ECG, continuously monitor the patient, and analyze rhythm strips to track the arrhythmia.

Control of the occurrence or the effect of the arrhythmia, or both, is often achieved with antiarrhythmic medications. The nurse assesses and observes for the benefits and adverse effects of each medication. The nurse, in collaboration with the primary provider, also manages medication administration carefully so that a constant serum level of the medication is maintained. The nurse may also conduct a 6-minute walk test as prescribed, which is used to identify the patient's ventricular rate in response to exercise. The patient is asked to walk for 6 minutes, covering as much distance as possible while the nurse monitors the patient for any symptoms (Matos Casano & Anjum, 2023). At the end, the nurse records the distance covered and the pre- and postexercise heart rate as well as the patient's response. The nurse assesses for factors that contribute to the arrhythmia (e.g., oxygen deficits, acid–base and electrolyte imbalances, caffeine, or nonadherence to the medication regimen). The nurse also monitors for ECG changes (e.g., widening of the QRS, prolongation of the QT interval, and increased heart rate) that increase the risk of an arrhythmic event.

Reducing Anxiety

When the patient experiences episodes of arrhythmia, the nurse stays with the patient and provides assurance of safety and security while maintaining a calm and reassuring attitude to reduce anxiety and build a trusting relationship with the patient. The nurse seeks the patient's view of the events and discusses the emotional response to the arrhythmia. The nurse emphasizes successes with the patient to promote a sense of self-management of the arrhythmia. For example, if a patient is experiencing episodes of arrhythmia and a medication is given that begins to reduce the incidence of the arrhythmia, the nurse communicates that information to the patient and explores the patient's response to this information. In addition, the nurse can help the patient develop a system to identify possible causative, influencing, and alleviating factors to maximize the patient's control of their health and to make the episode less threatening.

Promoting Home, Community-Based, and Transitional Care

Educating Patients About Self-Care

When educating patients about arrhythmias, the nurse first assesses the patient's understanding, clarifies misinformation, and then shares needed information in terms that are understandable and in a manner that is not frightening or threatening. The nurse clearly explains the etiology of the arrhythmia and treatment options to the patient and family. If necessary, the nurse explains the importance of maintaining therapeutic serum levels of antiarrhythmic medications so that the patient understands why medications should be taken regularly as prescribed and the importance of regular blood testing as prescribed. If the medication has the potential to alter the heart rate, the patient is taught how to take their pulse before each dose and to notify the primary provider if the pulse is outside the identified parameters. In addition, the relationship between an arrhythmia and cardiac output is explained so that the patient recognizes the symptoms of the arrhythmia and the rationale for the treatment regimen. If the patient is prescribed an anticoagulant medication, patient education points about taking anticoagulant medications are reviewed and summarized (see Chapter 27, Chart 27-8). The patient and family are educated about measures to take to decrease the risk of recurrence of the arrhythmia. If the patient has a potentially lethal arrhythmia, the nurse establishes with the patient and family a plan of action to take in case of an emergency and, if appropriate, encourages a family member to obtain CPR training. The patient and family are also educated about the potential risks of the arrhythmia and the signs and symptoms specific to the arrhythmia. For example, the patient with atrial fibrillation should be educated about the possibility of an embolic event.

Continuing and Transitional Care

A referral for home, community-based, or transitional care is often necessary for the patient with an arrhythmia with hemodynamic instability, significant symptoms of decreased cardiac output, or other manifestations that may impact activities of daily living. Home, community-based, or transitional care is warranted if the patient has significant comorbidities, socioeconomic issues, or limited self-management skills that could increase the risk of nonadherence to the therapeutic regimen and recurrence of arrhythmia. A referral is indicated if the patient had an electronic device implanted recently.

Evaluation

Expected patient outcomes may include:

- Maintaining cardiac output
- Experiencing reduced anxiety
- Expressing understanding of the arrhythmia and its treatment

Adjunct Modalities and Management

Arrhythmia treatments depend on whether the disorder is acute or chronic, the cause of the arrhythmia, and its actual or potential hemodynamic effects.

Acute arrhythmias are treated with medications or with emergency defibrillation, cardioversion, or pacing. Many antiarrhythmic medications are used to treat atrial and ventricular tachyarrhythmias (see Table 23-1). The choice of medication depends on the specific arrhythmia and its duration, the presence of structural heart disease (e.g., heart failure), and the patient's response to previous treatment. The nurse is responsible for monitoring and documenting the patient's responses to the medication and for ensuring that the patient has the knowledge and ability to manage the medication regimen.

If medications alone are ineffective in eliminating or decreasing the arrhythmia, certain adjunct mechanical therapies are available. The most common therapies are elective cardioversion and defibrillation for acute tachyarrhythmia, and implantable electronic devices for bradycardias (pacemakers) and chronic tachyarrhythmias (ICDs). Surgical treatments, although less common, are also available. The nurse is responsible for assessing the patient's understanding of and response to mechanical therapy, as well as the patient's self-management abilities. The nurse explains that the purpose of the device is to help the patient lead a life that is as active and productive as possible while minimizing the impact of arrhythmias.

Cardioversion and Defibrillation

Cardioversion and defibrillation treat tachyarrhythmias by delivering an electrical current that depolarizes a critical mass of myocardial cells. When the cells repolarize, the SA node is usually able to recapture its normal functional role as the heart's pacemaker.

The same type of device, called a *defibrillator*, is used for both cardioversion and defibrillation. The electrical voltage required to defibrillate the heart is usually greater than that required for cardioversion and may cause more myocardial damage. Biphasic defibrillators deliver an electrical charge in both directions, either between hands-free defibrillator pads or from hands-on defibrillator paddles. Because the delivery of the electrical charge varies among devices, the manufacturer's recommended dose should be followed for the first and subsequent defibrillations. In some patients who may not respond to initial defibrillation attempts, *double sequential*, or delivery of shocks by two defibrillators simultaneously, is considered (Merchant et al., 2020; Wyckoff et al., 2022).

The electrical current is delivered externally through the skin with the use of paddles or with conductor pads. The paddles or pads may be placed on the front of the chest (standard placement) (Fig. 23-23), or one pad may be placed on the front of the chest and the other pad placed under the patient's back just left of the spine (anteroposterior placement) (Fig. 23-24).

Defibrillator multifunction conductor pads contain a conductive medium and are connected to the defibrillator to allow for hands-off defibrillation. This method reduces the risk of touching the patient during the procedure and increases electrical safety. Automated external defibrillators (AEDs), which are now found in many public areas, use this type of delivery for the electrical current.

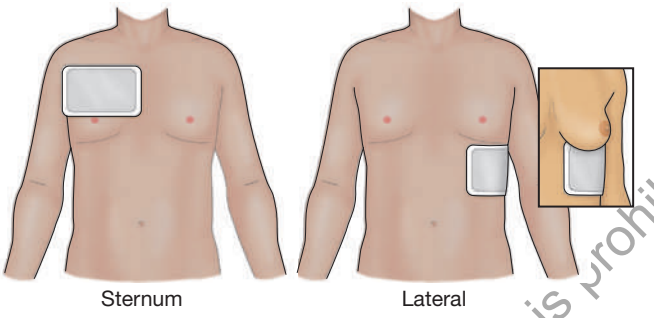

Figure 23-23 • Standard pad placement for defibrillation.

Clinical Judgment Alert

Generate Solutions & Take Actions

When using paddles, the appropriate conductant should be applied between the paddles and the patient's skin. Any other type of conductant, such as ultrasound gel, should not be substituted due to lack of electrical conductivity.

Whether using pads or paddles, the nurse must observe two safety measures. First, good contact must be maintained between the pads or paddles and the patient's skin (with a conductive medium between them) to prevent electrical current from leaking through the air (arcing) when the defibrillator is discharged. Second, there is no physical contact with the patient or with anything that is touching the patient when the defibrillator is discharged, to minimize the chance that electrical current is conducted to anyone other than the patient. Chart 23-5 provides a review of nursing responsibility when a patient is cardioverted or defibrillated.

Electrical Cardioversion

Electrical cardioversion involves the delivery of a carefully "timed" electrical current to terminate a tachyarrhythmia. In cardioversion, the defibrillator is set to synchronize with the ECG on a cardiac monitor so that the electrical impulse discharges during ventricular depolarization (QRS complex). The synchronization prevents the discharge from occurring during the vulnerable period of repolarization (T wave),

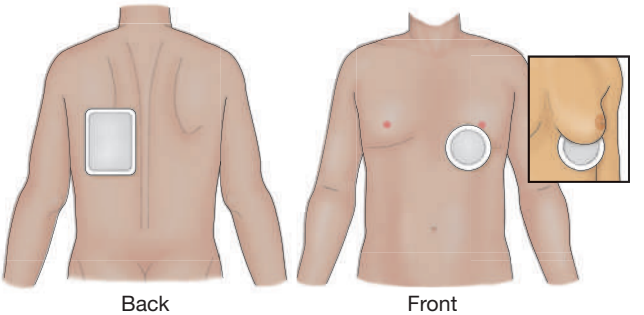

Figure 23-24 • Anteroposterior pad placement for defibrillation.

Chart 23-5 Assisting With External Defibrillation or Cardioversion

When assisting with external defibrillation or cardioversion, the nurse should remember these key points:

- Multifunction conductor pads or paddles are used, with a conducting medium between the paddles and the skin in the proper locations. The conducting medium is available as a sheet, gel, or paste. Gels or pastes with poor electrical conductivity should not be used.
- Paddles or pads should be placed so that they do not touch the patient's clothing or bed linen and are not near medication patches or in the direct flow of oxygen.
- Patients with large breasts should have the left pad or paddle placed underneath or lateral to the left breast.
- During cardioversion, the monitor leads must be attached to the patient in order to set the defibrillator to the synchronized mode ("in sync"). If defibrillating, the defibrillator must *not* be in the synchronized mode (most machines default to the "not-sync" mode).
- When using paddles, 20 to 25 lb of pressure must be used in order to ensure good skin contact.
- When using a manual discharge device, it must not be charged until it is ready to shock; then thumbs and fingers must be kept off the discharge buttons until paddles or pads are on the chest and ready to deliver the electrical charge.
- When it is time to defibrillate, whoever is delivering the charge should announce, "charging to (number of joules)" prior to discharging.
- "Clear!" must be called three times before discharging: As "Clear" is called the first time, the discharger must visually check that they are not touching the patient, bed, or equipment; as "Clear" is called the second time, the discharger must visually check that no one else is touching the bed, the patient, or equipment, including the endotracheal tube or adjuncts; and as "Clear" is called the third time, the discharger must perform a final visual check to ensure that everyone is clear of the patient and anything touching the patient.
- The delivered energy and resulting rhythm are recorded.
- Cardiopulmonary resuscitation (CPR) is immediately resumed after the defibrillation charge is delivered, if appropriate, starting with chest compressions.
- If CPR is necessary, after five cycles (about 2 minutes) of CPR, the cardiac rhythm is checked again and another shock is delivered, if warranted. A vasoactive or antiarrhythmic medication is given as soon as possible after the rhythm check to facilitate a positive response to defibrillation.
- After the event is complete, the skin under the pads or paddles is inspected for burns; if any are detected, the primary provider or a wound care nurse is consulted about appropriate treatment.
- The defibrillator is plugged back into an outlet, and supplies are restocked as needed.

Adapted from Little & Klein (2020); Wyckoff et al. (2022).

which could result in VT or ventricular fibrillation. The ECG monitor connected to the external defibrillator usually displays a mark or line that indicates sensing of a QRS complex. When the synchronization function is turned on, no electrical current is delivered if the defibrillator does not discern a QRS complex. Therefore, it is important to ensure that the patient is connected to the monitor and to select a lead (not "paddles") that has the most appropriate sensing of the QRS. Because there may be a short delay until recognition of the QRS, the discharge buttons of an external manual defibrillator must be held down until the shock has been delivered. In most monitors, the synchronization mode must be reactivated if the initial cardioversion was ineffective and another cardioversion is needed.

If the cardioversion is elective and the arrhythmia has lasted longer than 48 hours, anticoagulation for a few weeks before cardioversion is recommended (January et al., 2014, 2019). Antiarrhythmic medications are commonly withheld for 48 hours before cardioversion to ensure the resumption of sinus rhythm with normal conduction. The patient is instructed not to eat or drink for at least 4 hours before the procedure. Gel-covered conductor pads are positioned anteroposteriorly (front and back) for cardioversion. Before cardioversion, the patient receives moderate sedation IV as well as an analgesic medication or anesthesia. Respiration is then supported with supplemental oxygen as needed, and emergency intubation equipment is available in case of an emergency. The success of electrical cardioversion greatly depends on the amount of electrical energy (joules) administered, the waveform, and placement of the electrode pads. The amount of voltage used varies from 50 to 360 J, depending on the defibrillator's technology, the type and duration of the arrhythmia, and the size and hemodynamic status of the patient (Little & Klein, 2020; Wyckoff et al., 2022). Indications of a successful response are conversion to sinus rhythm, adequate peripheral pulses, adequate blood pressure, and patient recovery. Because of the sedation, airway patency must be maintained and the patient's state of consciousness assessed. Vital signs and oxygen saturation are monitored and recorded until the patient is stable and recovered from sedation and analgesic medications or anesthesia. ECG monitoring is required during and after cardioversion (Kwon et al., 2023).

Defibrillation

Defibrillation is used in emergency situations as the treatment of choice for ventricular fibrillation and pulseless VT, the most common cause of abrupt loss of cardiac function and sudden cardiac death. The sooner defibrillation is used, the better the survival rate (Hedge & Gnugnoli, 2022). Defibrillation is not used for patients who are conscious or have a pulse. Several studies have demonstrated that early defibrillation performed by lay people in a community setting can increase the survival rate (Hedge & Gnugnoli, 2022). The availability and the use of AEDs in public places can shorten the interval from collapse to rhythm recognition and defibrillation, which can significantly improve survival out of the hospital (Hedge & Gnugnoli, 2022).

Epinephrine is given after initial unsuccessful defibrillation to make it easier to convert the arrhythmia to a normal rhythm with the next defibrillation. This medication also increases cerebral and coronary artery blood flow. Antiarrhythmic medications such as amiodarone, lidocaine, or magnesium may be given if ventricular arrhythmia persists (see Table 23-1). This treatment with continuous CPR, medication administration, and defibrillation continues until a stable rhythm resumes or until it is determined that the patient cannot be revived.

Electrophysiology Studies

An EPS is an invasive procedure used to evaluate and treat various chronic arrhythmias that have caused cardiac arrest or significant symptoms. It is also indicated for patients with symptoms that suggest an arrhythmia that has gone undetected and undiagnosed by other methods. Because an EPS is invasive, it is performed in the hospital and may require that the patient be admitted. An EPS is used to:

- Identify the impulse formation and propagation through the cardiac electrical conduction system.
- Assess the function or dysfunction of the SA and AV nodal areas.
- Identify the location (called *mapping*) and mechanism of the arrhythmogenic foci (the exact site where the arrhythmia originates).
- Assess the effectiveness of antiarrhythmic medications and devices for the patient with an arrhythmia.
- Treat certain arrhythmias through the destruction of the causative cells (ablation).

An EPS procedure is a type of cardiac catheterization that is performed in a specially equipped cardiac catheterization laboratory by an electrophysiologist, assisted by other EPS laboratory personnel. The patient is conscious but lightly sedated. Usually, one or more catheters are inserted into the groin, neck, or antecubital fossa, or radially. The electrodes are positioned within the heart at specific locations depending on the type of study being conducted. These electrodes allow the electrical signal to be recorded from within the heart (intracardiogram).

The electrodes also allow the electrophysiologist to introduce a pacing stimulus to the intracardiac area at a precisely timed interval and rate, thereby stimulating the area. An area of the heart may be paced at a rate much faster than the normal rate of automaticity, the rate at which impulses are spontaneously formed. This allows the pacemaker to become an artificial focus of automaticity and to assume control of the sinus node. Then, the pacemaker is stopped suddenly, and the time it takes for the sinus node to resume control is assessed. A prolonged time indicates dysfunction of the sinus node.

One of the main purposes of programmed stimulation is to assess the ability of the area surrounding the electrode to cause a reentry arrhythmia. Premature impulses are delivered to an area in an attempt to cause the tachyarrhythmia. Because the precise location of the suspected area and the specific timing of the pacing needed are unknown, the electrophysiologist may use different techniques to cause the arrhythmia during the study. If the arrhythmia is reproduced by programmed stimulation, it is called *inducible*. Once an arrhythmia is induced, a treatment plan is determined and implemented. If, on the follow-up EPS, the tachyarrhythmia is not induced, then it is determined that the treatment is effective. Different medications are given and combined with cardiac implantable electronic devices to determine the most effective treatment to suppress the arrhythmia.

Patient care, patient education, and associated complications of an EPS are similar to those associated with cardiac catheterization (see Chapter 22). The study is usually about 2 hours in length; however, if the electrophysiologist conducts not only a diagnostic procedure but also treatment, the study can take up to 6 hours.

Patients scheduled for an EPS may be anxious about the procedure and its outcome. A detailed discussion involving the patient, the family, and the electrophysiologist usually occurs to ensure that the patient can give informed consent and to reduce the patient's anxiety about the procedure. Before the procedure, the nurse educates the patient about the EPS and its usual duration, the environment where the procedure is performed, and what to expect. Although an EPS is not painful, it may cause discomfort and fatigue. It may also cause feelings that were experienced when the arrhythmia occurred in the past. In addition, patients are educated about what will be expected of them after the procedure.

The patient should also know that the arrhythmia may occur during the procedure. It often stops on its own; if it does not, treatment is given to restore the patient's normal rhythm. The arrhythmia may have to be terminated using cardioversion or defibrillation, but this is performed under more controlled circumstances than if performed in an emergency. Postprocedural care is similar to that for cardiac catheterization, including restriction of activity to promote hemostasis at the insertion site (see Chapter 22).

Pacemaker Therapy

A pacemaker is an electronic device that provides supplemental electrical stimuli to the heart muscle. Pacemakers are used when a patient has a permanent or temporary slower-than-normal impulse formation, or a symptomatic AV or ventricular conduction disturbance. They are also used to control some tachyarrhythmias that do not respond to medication. Biventricular (both ventricles) pacing, also called **cardiac resynchronization therapy**, may be used to treat advanced heart failure. Pacemaker technology also may be used in conjunction with an ICD.

Pacemakers are permanent or temporary. Temporary pacemakers support patients until they improve or receive a permanent pacemaker (e.g., after acute MI or during open heart surgery) and are used only in hospital settings.

Pacemaker Design and Types

Pacemakers consist of two components: an electronic pulse generator and pacemaker electrodes, which are located on leads or wires. The generator contains the circuitry and batteries that determine the rate (measured in beats per minute) and the strength or output, measured in milliamperes (mA) of the electrical stimulus delivered to the heart. The generator also has circuitry that can detect the intracardiac electrical activity to cause an appropriate response; this component of pacing is called *sensitivity* and is measured in millivolts (mV). Sensitivity is set at the level that the intracardiac electrical activity must exceed to be sensed by the device. Leads, which carry the impulse created by the generator to the heart, can be threaded by fluoroscopy through a major vein into the heart, usually the right atrium and ventricle (endocardial leads), or they can be lightly sutured onto the outside of the heart and brought through the chest wall during open heart surgery (epicardial wires). These epicardial pacemakers are temporary and are removed by a gentle tug a few days after surgery. Because of this, safely securing these leads is essential prior to removal. The endocardial leads are temporarily placed with catheters through a vein by fluoroscopy. The leads may

also be part of a specialized pulmonary artery catheter (see Chapter 22). However, obtaining a pulmonary artery wedge pressure may cause the leads to move out of the pacing position. The endocardial and epicardial wires are connected to a temporary generator, which is about the size of a cellular phone. The energy source for a temporary generator is a common household battery. Monitoring for pacemaker malfunctioning and battery failure is a nursing responsibility.

The endocardial leads also may be placed permanently, passed into the heart through the subclavian, axillary, or cephalic vein, and connected to a permanent generator. These types of pacemakers are also called transvenous pacemakers. Most current leads have a fixation mechanism (e.g., a screw) at the end of the lead that allows precise positioning and avoidance of dislodgement. The permanent generator, which often weighs less than 1 oz (28.35 g) and is smaller than the size of a cellular phone, is usually implanted in a subcutaneous pocket created in the pectoral region, below the clavicle in males or behind the breast in females (Fig. 23-25). This 1-hour procedure is performed in a cardiac catheterization laboratory using a local anesthetic and moderate sedation. Close monitoring of the respiratory status is needed until the patient is fully awake.

Leadless pacemakers, a newer type of permanent pacemaker, are 90% smaller than transvenous pacemakers. They feature a self-contained, single-unit pulse generator and electrode that is inserted transvenously directly into the RV (Kuang et al., 2021).

Permanent pacemaker generators are insulated to protect against body moisture and warmth and have filters that protect them from electrical interference from most household devices, motors, and appliances. Lithium cells are commonly used; they last approximately 5 to 15 years, depending on the type of pacemaker, how it is programmed, and how often it is used. Most pacemakers have an elective replacement indicator (ERI), which is a signal that indicates when the battery is approaching depletion and must be replaced. The pacemaker continues to function for several months after the appearance of ERI to ensure that there is adequate time for a battery replacement. Because the battery is permanently sealed in the pacemaker, the entire generator must be replaced. To replace a failing generator, the old generator is removed, and a new generator is reconnected to the existing leads and reimplanted in the already existing subcutaneous pocket. When leadless pacemaker batteries signal that they must be replaced, a new system is simply implanted and the old battery is then disabled (Vouliotis et al., 2023). This is often an outpatient procedure.

If a patient suddenly develops bradycardia, is symptomatic but has a pulse, and is unresponsive to atropine, emergency pacing may be started with transcutaneous pacing, which most defibrillators are now equipped to perform. Some AEDs are able to do both defibrillation and transcutaneous pacing. Large pacing ECG electrodes are placed on the patient's chest and back. The electrodes are connected to the defibrillator, which acts as the temporary pacemaker generator (Fig. 23-26). Transcutaneous pacing impulses must travel through the patient's skin and tissue before reaching the heart, which can cause significant discomfort and is intended for use only in emergencies for short periods of time. This type of pacing necessitates hospitalization. If the patient is alert, sedation and analgesia are administered. After transcutaneous pacing, the skin under the electrode is inspected for erythema and burns. Transcutaneous pacing is not indicated for pulseless bradycardia (Wyckoff et al., 2022).

Pacemaker Generator Functions

Because of the sophistication and wide use of pacemakers, a universal code has been adopted to provide a means of safe communication about their function. The coding is referred to as the *NASPE-BPEG code* because it is sanctioned by the North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group. The complete code consists of five letters; the fourth and fifth letters are used only with permanent pacemakers (Bernstein et al., 2002; Mulpuru et al., 2017; Chart 23-6).

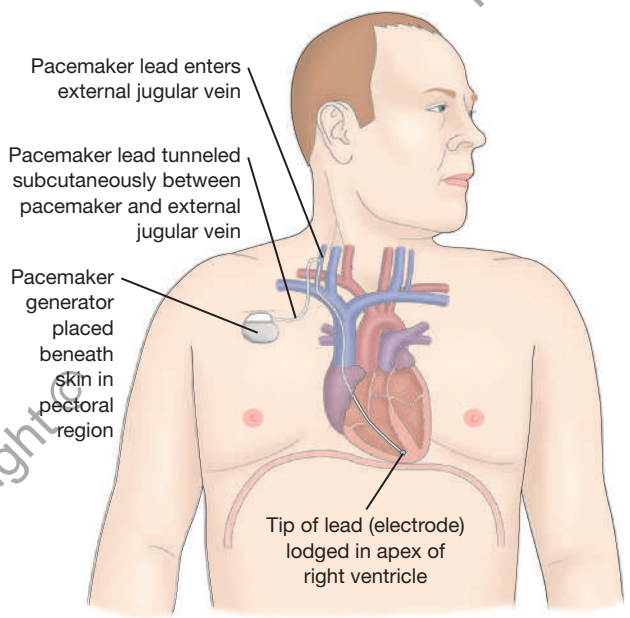

Figure 23-25 • Implanted transvenous pacing lead (with electrode) and pacemaker generator.

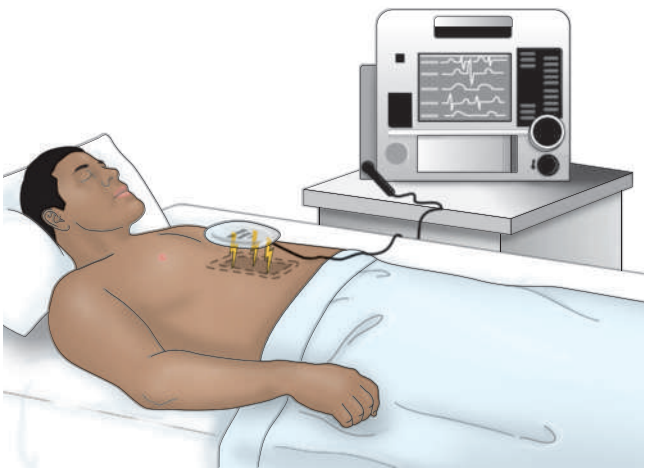

Figure 23-26 • Transcutaneous pacemaker with electrode pads connected to the anterior and posterior chest walls.

Chart 23-6 North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group Code (NASPE-BPEG Code) for Pacemaker Generator Function

- The first letter of the code identifies the chamber or chambers being paced (i.e., the chamber containing a pacing electrode). The letter characters for this code are A (atrium), V (ventricle), or D (dual, meaning both A and V).
- The second letter identifies the chamber or chambers being sensed by the pacemaker generator. Information from the electrode within the chamber is sent to the generator for interpretation and action by the generator. The letter characters are A (atrium), V (ventricle), D (dual), and O (indicating that the sensing function is turned off).
- The third letter of the code describes the type of response that will be made by the pacemaker to what is sensed. The letter characters used to describe this response are I (inhibited), T (triggered), D (dual—inhibited and triggered), and O (none). Inhibited response means that the response of the pacemaker is controlled by the activity of the patient's heart—that is, when the patient's heart beats, the pacemaker does not function, but when the heart does not beat, the pacemaker does function. In contrast, a triggered response means that the pacemaker responds (paces the heart) when it senses intrinsic heart activity.
- The fourth letter of the code is related to a permanent generator's ability to vary the heart rate. This ability is available in most current pacemakers. The possible letters are O, indicating no rate responsiveness, or R, indicating that the generator has rate modulation (i.e., the pacemaker has the ability to automatically adjust the pacing rate from moment to moment based on parameters, such as QT interval, physical activity, acid–base changes, body temperature, rate and depth of respirations, or oxygen saturation). A pacemaker with rate-responsive ability is capable of improving cardiac output during times of increased cardiac demand, such as exercise and decreasing the incidence of atrial fibrillation. All contemporary pacemakers have some type of sensor system that enables them to provide rate-adaptive pacing.
- The fifth letter of the code has two different indications: (1) the permanent generator has multisite pacing capability with the letters A (atrium), V (ventricle), D (dual), and O (none); or (2) the pacemaker has an antitachycardia function.
- Commonly, only the first three letters are used for a pacing code. An example of an NASPE-BPEG code is DVI:
  - **D:** Both the atrium and the ventricle have a pacing electrode in place.
  - **V:** The pacemaker is sensing the activity of the ventricle only.
  - **I:** The pacemaker's stimulating effect is inhibited by ventricular activity—In other words, it does not create an impulse when the pacemaker senses that the patient's ventricle is active.

Adapted from Bernstein et al. (2002); Gillis et al. (2012); Mulpuru et al. (2017).

The pacemaker paces the atrium and then the ventricle when no ventricular activity is sensed for a period of time specific to each patient. 3A straight vertical line usually can be seen on the ECG when pacing is initiated. The line that represents pacing is called a *pacemaker spike*. The appropriate ECG complex should immediately follow the pacing spike; therefore, a P wave should follow an atrial pacing spike (Fig. 23-27A) and a QRS complex should follow a ventricular pacing spike (see Fig. 23-27B). Because the impulse starts in a different place than the patient's normal rhythm, the QRS complex or P wave that responds to pacing looks different from the patient's normal ECG complex. *Capture* is a term used to denote that the appropriate complex followed the pacing spike.

The type of pacemaker generator and the settings selected depend on the patient's electrical cardiac needs, underlying cardiac function, and age. Pacemakers are generally set to sense and respond to intrinsic activity, which is called *on-demand pacing*. If the pacemaker is set to pace but not to sense, it is called a *fixed* or *asynchronous pacemaker*; this is written in pacing code as AOO or VOO. The pacemaker paces at a constant rate, independent of the patient's intrinsic rhythm. VOO pacing may indicate battery failure.

VVI (V, paces the ventricle; V, senses ventricular activity; I, paces only if the ventricles do not depolarize) pacing causes loss of AV synchrony and atrial kick, which may cause a decrease in cardiac output and an increase in atrial distention and venous congestion. Pacemaker syndrome, causing symptoms such as chest discomfort, shortness of breath, fatigue, activity intolerance, and orthostatic hypotension, is most common with VVI pacing (Glikson et al., 2021; Mulpuru et al., 2017). Atrial pacing and dual-chamber (right atrial and RV) pacing have been found to reduce the incidence of atrial fibrillation, ventricular dysfunction, and heart failure (Glikson et al., 2021; Mulpuru et al., 2017).

Single-chamber atrial pacing (AAI) or dual-chamber pacing (DDD) is recommended over VVI in patients with sinus node dysfunction, the most common cause of bradycardias requiring a pacemaker, and a functioning AV node (Glikson et al., 2021; Mulpuru et al., 2017). AAI pacing ensures synchrony between atrial and ventricular stimulation (and therefore contraction), as long as the patient has no conduction disturbances in the AV node. Dual-chamber pacemakers are recommended as the treatment for patients with AV conduction disturbances (Glikson et al., 2021; Mulpuru et al., 2017).

Synchronized biventricular pacing, also called *cardiac resynchronization therapy*, is associated with improved mortality rates in patients with heart failure and in patients with left bundle branch block (Agrawal, 2021). Synchronized biventricular pacing features three leads: one for the right atrium, one for the RV, and one for the LV, usually placed in the left lateral wall. This therapy improves cardiac function, resulting in decreased heart failure symptoms and an improved quality of life. Biventricular pacing may be used with an ICD (Agrawal, 2021).

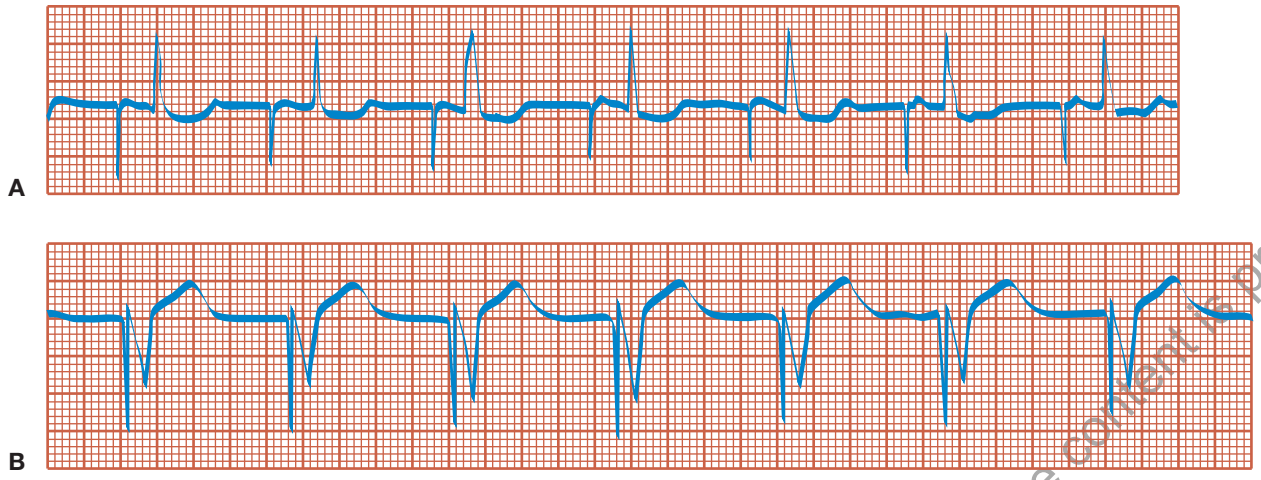

Figure 23-27 • Pacemaker capture. **A.** Atrial pacemaker; note that each vertical pacemaker spike is followed by a P wave. **B.** Ventricular pacemaker; note that each vertical pacemaker spike is followed by a QRS complex. Reprinted with permission from Morton, G. M., & Fontaine, D. K. (2024). *Critical care nursing: A holistic approach* (12th ed., Fig. 15-34A and B). Wolters Kluwer.

Complications of Pacemaker Use

Complications associated with pacemakers relate to their presence within the body and improper functioning (Chart 23-7). In the initial hours after a temporary or permanent pacemaker is inserted, the most common complication is dislodgment of the pacing electrode. Minimizing patient activity can help prevent this complication. If a temporary electrode is in place, the extremity through which the catheter has been advanced is immobilized. With a permanent pacemaker, the patient is instructed initially to restrict activity on the side of the implantation.

Leadless pacemakers are used to treat bradycardia and conduction disorders, especially in patients with chronic atrial fibrillation, vascular access issues, or who require infrequent pacing. Leadless pacemakers have several advantages over traditional transvenous pacemakers; they do not require a surgical pocket or transvenous leads and are associated with fewer complications (Bicong et al., 2022; Glikson et al., 2021; Vouliotis et al., 2023).

Leadless pacemakers are associated with fewer complications than transvenous pacemakers, including fewer infections, hematomas, lead dislodgement, and lead fracture. However, they provide only single-chamber RV pacing and do not feature concomitant defibrillator capabilities, limiting their usefulness (Bicong et al., 2022; Glikson et al., 2021; Vouliotis et al., 2023).

The ECG is monitored very carefully to detect pacemaker malfunction. Improper pacemaker function, which can arise from failure in one or more components of the pacing system, is outlined in Table 23-2. The following data should be noted on the patient's record: model of pacemaker, type of generator, date and time of insertion, location of pulse generator, stimulation threshold, and pacer settings (e.g., rate, energy output [mA], sensitivity [mV], and duration of the interval between atrial and ventricular impulses [AV delay]). This information is important for identifying normal pacemaker function and diagnosing pacemaker malfunction.

A patient experiencing pacemaker malfunction may develop bradycardia as well as signs and symptoms of decreased cardiac output (e.g., diaphoresis, orthostatic hypotension, and syncope). The degree to which these symptoms become apparent depends on the severity of the malfunction, the patient's level of dependency on the pacemaker, and the

Chart 23-7 Potential Complications From Insertion of a Pacemaker

- Local infection at the entry site of the leads for temporary pacing or at the subcutaneous site for permanent generator placement. Preoperative skin preparation with chlorohexidine, prophylactic antibiotic, and antibiotic irrigation of the subcutaneous pocket prior to generator placement has decreased the rate of infection to a minimal rate.
- Pneumothorax or hemothorax. The risk is reduced if cephalic vein is cut down, contrast venography, or ultrasound guidance is utilized.
- Bleeding and hematoma at the lead entry sites for temporary pacing or at the subcutaneous site for permanent generator placement. This usually can be managed with cold compresses and discontinuation of antiplatelet and antithrombotic medications.
- Ventricular ectopy and tachycardia from irritation of the ventricular wall by the endocardial electrode
- Movement or dislocation of the lead placed transvenously (perforation of the myocardium)
- Phrenic nerve, diaphragmatic (hiccupping may be a sign), or skeletal muscle stimulation if the lead is dislocated or if the delivered energy (mA) is set high. The occurrence of this complication is avoided by testing during device implantation.
- Cardiac perforation resulting in pericardial effusion and, rarely, cardiac tamponade, which may occur at the time of implantation or months later. This condition can be recognized by the change in QRS complex morphology, diaphragmatic stimulation, or hemodynamic instability.
- Twiddler syndrome may occur when the patient manipulates the generator, causing lead dislodgement or fracture of the lead.
- Pacemaker syndrome (hemodynamic instability caused by ventricular pacing and the loss of AV synchrony).

AV, atrioventricular.
Adapted from Diaz et al. (2023); Mulpuru et al. (2017); Wyckoff et al. (2022).

TABLE 23-2 Assessing Pacemaker Malfunction

Problem	Possible Cause	Nursing Considerations
Loss of capture—Complex does *not* follow the pacing spike.	Inadequate stimulus Lead dislodgement Lead wire fracture Catheter malposition Battery depletion Electronic insulation break Medication change Myocardial ischemia	Check the security of all connections; increase milliamperage. Reposition extremity; turn patient to left side. Change battery. Change generator.
Undersensing—Pacing spike occurs at preset interval despite the patient's intrinsic rhythm.	Sensitivity too high Electrical interference (e.g., by a magnet) Faulty generator	Decrease sensitivity. Eliminate interference. Replace generator.
Oversensing—loss of pacing artifact; pacing does not occur at preset interval despite the lack of intrinsic rhythm.	Sensitivity too low Electrical interference Battery depletion Change in medication	Increase sensitivity. Eliminate interference. Change battery.
Loss of pacing—total absence of pacing spikes	Oversensing Battery depletion Loose or disconnected wires Perforation	Change battery. Check the security of all connections. Apply magnet over permanent generator. Obtain 12-lead ECG and portable chest x-ray. Assess for murmur. Contact the primary provider.
Change in pacing QRS shape	Septal perforation	Obtain 12-lead ECG and portable chest x-ray. Assess for murmur. Contact the primary provider.
Rhythmic diaphragmatic or chest wall twitching or hiccupping	Output too high Myocardial wall perforation	Decrease milliamperage. Turn pacer off. Contact the primary provider at once. Monitor closely for decreased cardiac output.

ECG, electrocardiogram.
Adapted from Arcinas & Sheldon (2023); Mulpuru et al. (2017).

patient's underlying condition. Pacemaker malfunction is diagnosed by analyzing the ECG. Manipulating the electrodes, changing the generator's settings, or replacing the pacemaker generator or leads may be necessary.

Inhibition of permanent pacemakers or reversion to asynchronous fixed rate pacing can occur with exposure to strong electromagnetic interference (EMI). Pacemaker technology allows patients to safely use most household electronic appliances and devices, but gas-powered engines should be turned off before working on them. Objects that contain magnets (e.g., the earpiece of a phone, large stereo speakers, and jewelry) should not be near the generator for longer than a few seconds. Patients are advised to place cellular phones at least 6 to 12 in away from the pacemaker generator. Large electromagnetic fields, such as those produced by magnetic resonance imaging (MRI), radio and television transmitter towers and lines, transmission power lines, power tools, and electrical substations may cause EMI. Patients are cautioned to avoid such situations or to simply move farther away from the area if they experience dizziness or a feeling of rapid or irregular heartbeats (palpitations).

In addition, the metal of the pacemaker generator may trigger store and library antitheft devices as well as airport and building security alarms; however, these alarm systems do not interfere with the pacemaker function. Patients should walk through them quickly and avoid standing in or near these devices for prolonged periods of time. The handheld screening devices used in airports may interfere with the pacemaker. Patients should be advised to ask security personnel to perform a hand search instead of using the handheld screening device. Patients also should be educated to wear or carry medical identification to alert personnel to the presence of the pacemaker.

Pacemaker Surveillance

Remote monitoring technology is embedded into new pacemakers to replace the need for frequent in-person follow-up cardiologist visits required with older devices. Remote monitoring systems include transtelephonic monitoring (use of an analog phone with transmission through a landline), inductive monitoring (use of a wand with transmission through a landline or a cellular connection), and remote wandless monitoring (use of wandless transmitter through radiofrequency) (Glikson et al., 2021). With remote monitoring, ECG data are transmitted to the primary provider at the cardiology clinic. In addition, the pacemaker rate and other data concerning pacemaker function (e.g., generator setting, battery status, sensing function, lead integrity, and pacing data, such as number of pacing events) are obtained and evaluated by the cardiologist. This simplifies the

diagnosis of a failing generator, reassures the patient, and improves management when the patient is physically remote from pacemaker testing facilities. A follow-up schedule is dependent upon the patient's needs and pacemaker model.

Implantable Cardioverter Defibrillator

The **implantable cardioverter defibrillator** is an electronic device that detects and terminates life-threatening episodes of tachycardia or fibrillation, especially those that are ventricular in origin. Patients at high risk of VT or ventricular fibrillation and who would benefit from an ICD are those who have survived sudden cardiac death, which usually is caused by ventricular fibrillation, or have experienced spontaneous, symptomatic VT (syncope secondary to VT) not due to a reversible cause (called a *secondary prevention intervention*). Patients with coronary artery disease who are 40 days postacute MI with moderate-to-severe LV dysfunction (ejection fraction less than or equal to 35%) are at risk for sudden cardiac death and therefore an ICD is indicated (called a *primary prevention intervention*). An ICD implantation is also recommended in patients who have been diagnosed with nonischemic dilated cardiomyopathy for at least 9 months and have functional New York Heart Association Class II or III heart failure (see Chapter 26, Table 26-1) (Al-khatib et al., 2018; Ganz, 2023; Wilkoff et al., 2016).

If there is a waiting period for ICD implantation for patients with postacute MI who are at risk for sudden cardiac death, a wearable vestlike automated defibrillator, which works just like an AED, is prescribed by the provider (National Heart, Lung, and Blood Institute [NHLBI], 2023; Fig. 23-28). Prior to the delivery of the shock, the vest may vibrate, alarm, and issue an audible warning to announce that a shock is imminent. The vest weighs about a pound, is worn under the patient's clothing, and is attached to a monitor with a battery that is worn in a holster or on a shoulder strap. The monitor automatically downloads information once a day, usually in the middle of the night. The vest is worn at all times and removed only when showering or bathing. The battery is changed every day. Education is provided to the patient by the device manufacturer. However, the nurse should assess the patient's understanding of the education provided and explore any issues that may prevent the patient from wearing it. The wearable ICD is only successful if worn regularly by the patient.

An ICD has a generator smaller than the size of a cellular phone, similar to a pacemaker, that is implanted in a subcutaneous pocket, usually in the upper chest wall. An ICD also has at least an RV lead that is implanted transvenously and can sense intrinsic electrical activity and deliver an electrical impulse. The implantation procedure, postimplantation care, and lengths of hospital stay are much like those for insertion of a pacemaker (Fig. 23-29).

ICDs are designed to respond to a heart rate that exceeds a predetermined level and a change in the isoelectric line segments. When an arrhythmia occurs, rate sensors require a set duration of time to sense the arrhythmia. The device automatically charges once an arrhythmia is sensed. After the arrhythmia is confirmed, the ICD delivers the programmed charge through the lead to the heart (Libby et al., 2021).

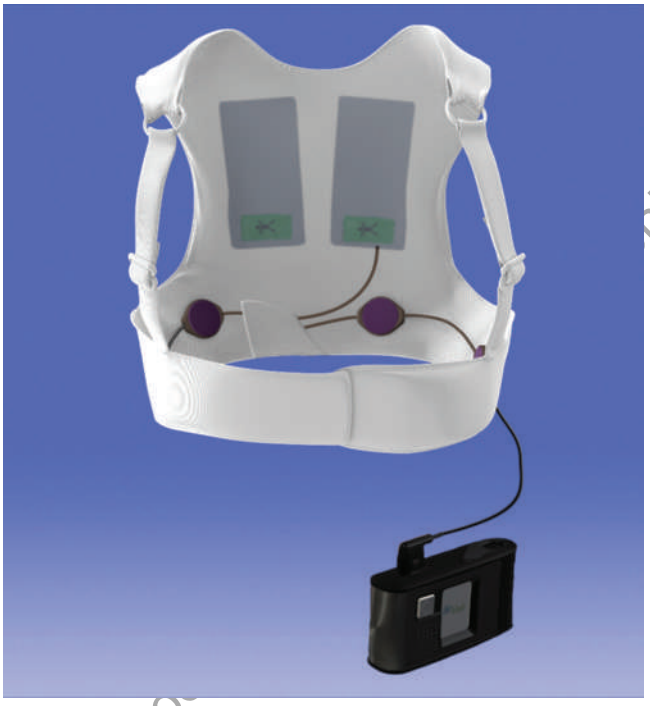

Figure 23-28 • The wearable cardioverter defibrillator vest. Courtesy of ZOLL LifeVest.

The life of the lithium battery varies depending on the ICD and use but may last at least 10 years. ICD surveillance is similar to that of the pacemaker; however, it includes stored endocardial ECGs as well as information about the number and frequency of shocks that have been delivered. Antiarrhythmic medication is commonly given with this

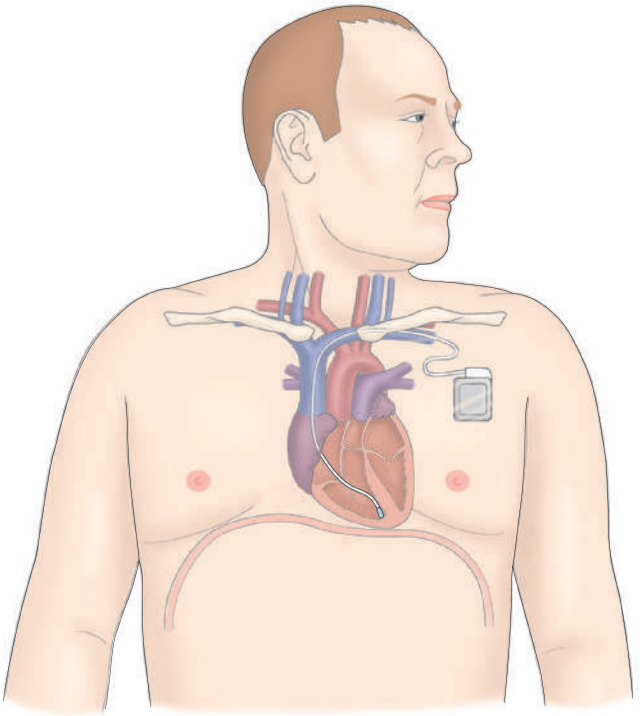

Figure 23-29 • The implantable cardioverter defibrillator consists of a generator and a sensing/pacing/defibrillating electrode.

technology to minimize the occurrence of a tachyarrhythmia and to reduce the frequency of ICD discharge. Some ICDs can respond with antitachycardia pacing, in which the device delivers electrical impulses at a fast rate in an attempt to disrupt the tachycardia, low-energy cardioversion, or defibrillation; others may use all three techniques. Pacing is used to terminate tachycardias caused by a conduction disturbance called *reentry*, which is repetitive restimulation of the heart by the same impulse. An impulse or a series of impulses is delivered to the heart by the device at a fast rate to collide with and stop the heart's reentry conduction impulses, and therefore to stop the tachycardia. Typically, ICDs also have pacemaker capability if the patient develops bradycardia, which sometimes occurs after treatment of the tachycardia.

The subcutaneous ICD is an alternative to the conventional ICD. The main advantages of subcutaneous defibrillators are that the complications associated with vascular access are avoided, infection risks are minimized, and lead failure is extremely rare. Patients without pacing indications of atria or ventricles and who are at risk for sudden cardiac death are the best candidates for this technology (Singh et al., 2020).

Which device is used and how it is programmed depend on the patient's arrhythmia(s). The device may be programmed differently for different arrhythmias (e.g., ventricular fibrillation, VT with a fast ventricular rate, and VT with a slow ventricular rate). As with pacemakers, there is an NASPE-BPEG code for communicating the functions of the ICDs (Bernstein et al., 2002; Wilkoff et al., 2016). The first letter represents the chamber or chambers shocked (O, none; A, atrium; V, ventricle; D, both atrium and ventricle). The second letter represents the chamber that can be antitachycardia paced (O, A, V, D, meaning the same as the first letter). The third letter indicates the method used by the generator to detect a tachycardia (E, electrogram; H, hemodynamics). The last letter represents the chambers that have antibradycardia pacing (O, A, V, D, meaning the same as the first and second letters of the ICD code).

Complications of ICD implantation mirror those associated with pacemaker insertion. The primary potential complication is surgery-related infection, which increases with each battery or lead replacement. A few complications are associated with the technical aspects of the equipment, like those of pacemakers, such as premature battery depletion and dislodged or fractured leads. Inappropriate delivery of ICD therapy, usually due to oversensing or atrial and sinus tachycardias with a rapid ventricular rate response, is the most frequent long-term complication and requires immediate reprogramming of the device.

Nursing Management

After a permanent electronic device is inserted, the patient's heart rate and rhythm are monitored by ECG. The device's settings are documented and compared with the ECG recordings to assess the device's function. Pacemaker malfunction can be detected by examining the pacemaker spike and its relationship to the corresponding ECG complexes. Cardiac output and hemodynamic stability are assessed to identify the patient's response to pacing and the adequacy of pacing. The appearance or increasing frequency of arrhythmia is observed and reported to the primary provider. If the patient has an ICD implanted and develops a shockable rhythm, the ECG should be recorded to note the time between the onset of the arrhythmia and the onset of the device's shock or antitachycardia pacing.

The incision site where the generator was implanted is observed for bleeding, hematoma formation, or infection. A chest x-ray is usually taken after the procedure and prior to discharge to document the position of leads in addition to ensuring that the procedure did not cause a pneumothorax. It is necessary to assess the function of the device throughout its lifetime and especially after changes in the patient's medication regimen. For example, antiarrhythmic agents, beta-blockers, and diuretics may increase the pacing threshold, whereas corticosteroids and alpha-adrenergic agents may decrease the pacing threshold; the opposite effect occurs when the patient is taken off these medications.

In the peri- and postoperative phases, the nurse carefully observes the patient's responses to the device and provides the patient and family with further education as needed. The nurse also assists the patient and family in addressing concerns and in making decisions about self-care and lifestyle changes necessitated by the arrhythmia and resulting device implantation. The patient is also assessed for anxiety, depression, or anger, which may be symptoms of ineffective coping with the implantation. In addition, the level of knowledge and education needs of the patient and family and the history of adherence to the therapeutic regimen should be identified. It is especially important to include the family when providing education and support.

Preventing Infection

The nurse changes the dressing as prescribed or as needed and inspects the insertion site for redness, swelling, soreness, or any unusual drainage. Any change in wound appearance, an increase in the patient's temperature, or an increase in the patient's white blood count should be reported to the primary provider.

Promoting Effective Coping

To promote effective coping strategies, the nurse must recognize both the patient's and family's perceptions of the situation and their resulting emotional state and assist them to explore their reactions and feelings. Because of the unpredictable and possibly painful ICD discharge, patients with ICDs are most vulnerable to feelings of helplessness, leading to depression. The nurse can help the patient identify positive methods to deal with the actual or perceived limitations and manage any lifestyle changes needed. The nurse may help and reassure the patient of activity changes, the emotional responses to the change, and how the patient responds to that emotion. The patient and family should be encouraged to talk about their experiences and emotions with each other and the health care team as well as appropriate support groups. The nurse may also encourage the use of spiritual resources if appropriate for the patient. Instructing the patient about the ICD may help the patient cope with changes that occur as a result of device implantation.

Promoting Home, Community-Based, and Transitional Care

After device insertion, the patient's hospital stay is typically short or may be completed in an outpatient setting, and follow-up in an outpatient clinic, office, or device clinic is common. The patient's anxiety and feelings of vulnerability may interfere with the ability to learn the information provided. The nurse needs to include family and caregivers in the education and provide printed materials. The nurse establishes priorities for learning with the patient and caregiver. Education includes the importance of periodic device monitoring, promoting safety, surgical site care, and avoiding EMI (Chart 23-8). In addition, the educational plan should include information about activities that are safe and those that may be dangerous. The nurse discusses with the patient and family what they have to do when a shock is delivered. Education may include CPR training for the family.

Chart 23-8 Patient Education

Educating the Patient With an Implantable Cardiac Device

At the completion of education, the patient and caregiver will be able to:

- State the impact of device implantation on physiologic functioning, ADLs, IADLs, self-image and roles, relationships (including sexuality), and spirituality.
- State changes in lifestyle (e.g., diet, activity, and mobility/driving restrictions) necessary to maintain health.
- State the name, dose, side effects, frequency, and schedule for all medications.
- For patients with pacemakers, check pulse daily. Report *immediately* any sudden slowing or increasing of the pulse rate. This may indicate pacemaker malfunction.
- Avoid infection at the insertion site of the device.
- Leave the incision uncovered and observe it daily for redness, increased swelling, and heat.
- Take temperature at the same time each day; report any increase.
- Avoid wearing tight, restrictive clothing that may cause friction over the insertion site.
- Initially, avoid soaking in the tub and lotion, creams, or powders in the area of the device.
- Adhere to activity restrictions.
- Restrict movement of arm until the incision heals; do not raise the arm above head for 2 weeks.
- Avoid heavy lifting for a few weeks.
- Discuss safety of activities (e.g., driving) with a primary provider.
- Recognize that although it may take up to 2 to 3 weeks to resume normal activities, physical activity does not usually have to be curtailed, with the exception of contact sports.
- Electromagnetic interference: Understand the importance of the following:
- Avoid large magnetic fields, such as those created by MRI, large motors, arc welding, and electrical substations. Magnetic fields may deactivate the device, negating its effect on an arrhythmia.
- At security gates at airports, government buildings, or other secured areas, show identification card and request a hand (not handheld device) search. Obtain and carry a letter from the primary provider about this requirement.
- Some electrical and small motor devices, as well as products that contain magnets (e.g., cellular phones), may interfere with the functioning of the cardiac device if the electrical device is placed very close to it. Avoid leaning directly over large electrical devices or motors, or ensure that contact is of brief duration; place cellular phone on the opposite side of cardiac device.
- Household appliances (e.g., microwave ovens) should not cause any concern.
- Describe precautions and safety measures to be used.
- Describe what to do if symptoms occur, and notify the primary provider if any discharges seem unusual.
- Maintain a log that records discharges of an ICD. Record events that precipitate the sensation of shock. This provides important data for the primary provider to use in readjusting the medical regimen.
- Encourage family members to attend a cardiopulmonary resuscitation class.
- Call 911 for emergency assistance if feeling of dizziness occurs.
- Wear medical identification (e.g., MedicAlert) that includes primary provider information.
- Avoid frightening family or friends with unexpected shocks from an ICD, which will not harm them. Inform family and friends that in the event they are in contact with the patient when a shock is delivered, they may also feel the shock. It is especially important to warn sexual partners that this may occur.
- Carry medical identification with the primary provider's name, type and model number of the device, manufacturer's name, and the hospital where the device was inserted.
- Identify community resources for peer and caregiver/family support.
- Adhere to appointments for follow-up care that are scheduled to monitor the electronic performance of the cardiac device. This is especially important during the first month after implantation and near the end of the battery life. Remember to take the log of ICD discharges to review with the primary provider.
- Identify the need for health promotion, disease prevention, and screening activities.

ADL, activities of daily living; IADL, instrumental activities of daily living; ICD, implantable cardioverter defibrillator; MRI, magnetic resonance imaging.

CLINICAL JUDGMENT EXERCISES

1. A 68-year-old patient presents to the cardiology clinic where you work with a history of atrial fibrillation (AF). They have a past medical history of hypertension, diabetes, and hyperlipidemia. Their CHA2DS2-VASc score is 4, indicating a moderate-to-high risk of stroke. The patient is currently on warfarin therapy for stroke prevention. The patient has been taking warfarin for the past 2 years, with a well-controlled international normalized ratio (INR) within the therapeutic range (2.0 to 3.0). They attend regular follow-up appointments for INR monitoring, and their medication adherence has been good.

What pathophysiologic cues are important for you to recognize and analyze about AF and the impact on other risks such as stroke?

Describe how you will evaluate the expected outcome of warfarin therapy.

2. A 72-year-old patient is admitted to the cardiac care unit where you work postimplantation of a dual-chamber pacemaker due to symptomatic bradycardia. They have a medical history of hypertension, coronary artery disease, and a previous myocardial infarction. They are taking various cardiac medications, including antiplatelet agents and beta-blockers. The pacemaker implantation procedure was successful, and the patient is recovering well. As a member of the nursing team, you are responsible for providing comprehensive care to ensure optimal healing, monitoring, and patient education regarding pacemaker management.

- You hypothesize that pacemakers are implanted in patients with cardiovascular disease for which common indications?
- When you take action to implement the plan of care, what information will you present to the patient and family that reviews the basic functioning of a dual-chamber pacemaker and how it addresses bradycardia?
- You generate solutions to meet the patient's immediate care priorities post-pacemaker implementation. Which evidence-based actions will be included to achieve optimal patient outcomes?
- Identify important points for patient education regarding pacemaker self-care, activity restrictions, and recognizing signs of potential issues.

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