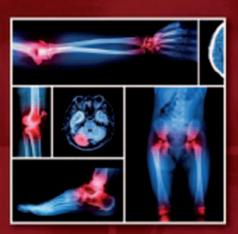
# ACUTE CARE HANDBOOK for PHYSICAL THERAPISTS

Fifth Edition

JAIME C. PAZ
MICHELE P. WEST
KATHRYN PANASCI
KRISTIN C. GREENWOOD







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Fifth Edition

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K.C.G



# **Preface**

In the early 1990s a handful of clinicians from Massachusetts General Hospital in Boston, Massachusetts, sought to develop a resource they believed was needed in the acute care setting. With good fortune, a connection was made with Butterworth-Heinemann, and the first edition of the Acute Care Handbook for Physical Therapists was published. Twenty-two years later, with Elsevier, the fifth edition of the Acute Care Handbook for Physical Therapists has now published. Many ideas with lots of energy were directed toward this project. Initially, the goal was to provide clinicians with a handy, pocket-sized reference for patient care in the hospital setting. It was created primarily for physical therapy students and clinicians unfamiliar with entry-level acute care practice. This handbook was never intended to be a formal comprehensive textbook; however, over the past four editions it has evolved to become one of the resources available to students and physical therapists seeking information for this content area.

Throughout the past 5 years, peer-reviewed literature and clinical resources related to acute care physical therapy practice have grown tremendously. Several efforts have been successfully undertaken by collective stakeholders to maximize patient outcomes and patient safety within the acute care setting, resulting in position papers and references to help enhance practice in real time. Many of the updates in this edition incorporate these advances pertinent to the evolution of the care of patients in the acute care environment as well as developments in medical science. Although it is impossible to keep up-to-date in the moment of practice, we have provided categories and links, both here and throughout the text, along with brief explanations of their relevance, to extend the usefulness of this handbook into practice. Some examples are provided:

- Resources available to members on the American Physical Therapy Association website (apta.org):
  - Physical Therapy Outcomes Registry: http://www.ptoutcomes.com/home.aspx
  - PTNow: https://www.ptnow.org/Default.aspx
    - Clinical Practice Guidelines, including:
      - Role of Physical Therapists in the Management of Individuals at Risk for or Diagnosed with Venous Thromboembolism: Evidence-Based Clinical Practice Guideline https://www.ptnow.org/clinical-practice-guideline-detail/role-of-physical-therapists-in-management-of-indiv
      - Early Rehabilitation for Patients in the Intensive Care Unit (ICU) https://www.ptnow.org/clinicalsummaries-detail/early-rehabilitation-patients-inintensive-care-unit
    - Clinical Summaries:
      - Stroke https://www.ptnow.org/clinical-summariesdetail/stroke-2#ViewComplete

- Documents created by the Academy of Acute Care Physical Therapy and available for free on their website (https://www.acutept.org/):
  - Laboratory values interpretation resources: 2017 Update and Point-of-Care document: https://www.acutept.org/ store/ViewProduct.aspx?id=10758036
  - Resources specific to entry-level education:
    - Core Competencies for Entry-Level Practice in Acute Care Physical Therapy https://www.acutept.org/store/ ViewProduct.aspx?id=10758435
    - Core Competencies for Entry-Level Physical Therapy Assistants in the Acute Care Setting https://www .acutept.org/store/ViewProduct.aspx?id=10758519
- Interprofessional Practice Competencies:
  - Interprofessional Education Collaborative: Core Competencies for Interprofessional Collaborative Practice: 2016
     Update: https://www.ipecollaborative.org/resources.html.

With this fifth edition, editors Jaime Paz and Michele West have recruited two talented associate editors, Kathryn Panasci and Kristin Curry Greenwood, to help make the peer review process more robust as well as to provide a transition for future editions. For both Jaime and Michele, this will signal our last edition as editors of the *Acute Care Handbook*. Since the first publication in 1997, we have been thankful to be able to contribute to this area of clinical practice. Along with our new associate editors, we acknowledge the work of past contributors, some who have stayed on for many or all editions, and we welcome the new contributors that have joined the team. Many of the new and returning contributors are either faculty or clinicians in highly specialized acute care centers. The overall combination of our contributors allows material to remain current with literature while providing clinically relevant information.

This edition of the *Acute Care Handbook for Physical Thera- pists* has been revised and updated to serve its original purpose. All chapters include updated literature, available at the time of revision, to assist with implementing evidence-based practice in this setting. Each chapter centers on either a major body system or health condition and includes:

- A review of body structure and function
- An overview of the medical-surgical evaluation of a patient admitted to the hospital, including pertinent diagnostic procedures and laboratory tests
- A review of health conditions that emphasizes information pertinent to physical therapy management
- Guidelines and considerations for physical therapy examination and intervention

Clinical Tips appear throughout each chapter. These helpful hints are intended to maximize safety, quality, and efficiency of care. As an essential member of the health care team, the

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physical therapist is often expected to understand hospital protocol, safety, medical-surgical "lingo," and the many aspects of patient care from the emergency room setting to the ICU to the general ward. These Clinical Tips are suggestions from the editors and contributors that, from clinical experience, have proved to be beneficial in acclimating therapists to the acute care setting. With advances in literature pertaining to acute care, more of these tips have become validated and thus are referenced accordingly in this new edition.

It is important to remember that all information presented in this book is intended to serve as a guide to stimulate independent critical thinking within the scope of practice of physical therapy and the spectrum of medical-surgical techniques and trends. To implement evidence-based practice, this reference should serve as one of the many resources involved with clinical decision making. As health care continues to evolve, clinicians will need to adapt information to the best of their ability. Developing and maintaining a rapport with the medical-surgical team is essential to facilitate high-quality and safe patient care. We believe the new edition of the *Acute Care Handbook for Physical Therapists* can enhance the content knowledge in the acute care environment by providing valuable information to help interpret medical record reviews, prepare for physical therapy examination and intervention, assist in sound clinical decision making, and support patient and interprofessional interaction.

J.C.P. M.P.W. K.P. K.C.G

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# INTRODUCTION

# CHAPTER 1

# The Acute Care Setting

Kathryn Panascia

# **CHAPTER OUTLINE**

Introduction

Safe Caregiver and Patient

Environment

Fall Risk

**Use of Restraints** 

**Medication Reconciliation** 

Latex Allergy

**Effects of Prolonged Bed Rest** 

# **Intensive Care Unit Setting**

Common Patient and Family Responses to the Intensive Care Unit

Critical Illness Myopathy, Polyneuropathy, and

Polyneuromyopathy Sleep Pattern Disturbance

Substance Use and Withdrawal Severity of Injury and Illness

# **End-of-Life Considerations**

Resuscitation Status
Withholding and Withdrawing
Medical Therapies
Palliative and Hospice Care
Advance Directives
Coma, Vegetative State, and Brain
Death

# **CHAPTER OBJECTIVES**

The objectives of this chapter are the following:

- 1. Review basic safety guidelines and principles for the physical therapist and patient in the hospital setting
- 2. Identify the multisystem effects of prolonged bed rest that can occur with hospitalization and the relevant physical therapy considerations
- 3. Describe the unique characteristics of, and patient response(s) to, the intensive care unit
- 4. Review alcohol use disorders and alcohol withdrawal syndrome
- 5. Identify assessment scales used for defining severity of injury or illness
- 6. Discuss end-of-life considerations and palliative care concepts

# Introduction

The physical therapist must have an appreciation for the distinct aspects of inpatient acute care. This chapter provides a brief overview of the acute care environment, including safety and the use of physical restraints; the effects of prolonged bed rest; end-of-life considerations; and some of the unique circumstances, conditions, and patient responses encountered in the hospital setting.

The acute care or hospital setting is a unique environment with protocols and standards of practice and safety that may not be applicable to other areas of health care delivery, such as an outpatient clinic or school system. Hospitals are designed to accommodate a wide variety of routine, urgent, or emergent patient care needs. The clinical expertise of the staff and the medical-surgical equipment used in the acute care setting (see Chapter 18) reflect these needs. The nature of the hospital setting is to provide 24-hour care; thus the patient, family, and caregivers are faced with the physical, psychological, and emotional sequelae of illness and hospitalization. This can include the response(s) to a change in daily routine, lack of privacy and independence, or a potential lifestyle change, medical crisis, critical illness, or long-term illness.

# Safe Caregiver and Patient Environment

Patient safety is a top priority. Physical therapists should strive to keep the patient safe at all times, comply with hospital initiatives that maximize patient safety, and understand patient safety goals established by their accrediting agency. Basic guidelines for providing a safe caregiver and patient environment include the following:

Reduce the risk of health care—associated infections by always following Standard Precautions, including compliance with hand hygiene guidelines.<sup>1</sup> Refer to Table 13.3 for a summary of precautions to prevent infection, including standard, airborne, droplet, and contact precautions.

<sup>&</sup>lt;sup>a</sup>The authors acknowledge Michele P. West for prior contributions to this chapter.

- Know the facility's policy regarding accidental exposure to chemicals, wastes, or sharps, as well as emergency procedures for evacuation, fire, internal situation, and natural disaster. Know how to contact the employee health service and hospital security.
- Confirm that you are with the correct patient before initiating physical therapy intervention, according to the facility's policy. Most acute care hospitals require two patient identifiers (by patient report or on an identification bracelet), such as name and hospital identification (ID) number, telephone number, or another patient-specific number. A patient's room number or physical location may not be used as an identifier.<sup>1</sup> Notify the nurse if a patient is missing an ID bracelet.
- Elevate the height of the bed as needed to ensure your use of proper body mechanics when performing a bed-level intervention (e.g., stretching, therapeutic exercise, bed mobility training, or wound care).
- Leave the bed or chair (e.g., stretcher chair, recliner chair) in
  the lowest position with wheels locked when the patient will
  be seated at the edge of the surface, exiting or entering the
  surface, and after physical therapy intervention is complete.
  Leave the top bed rails up for all patients.
- Use only equipment (e.g., assistive devices, recliner chairs, wheelchairs) that is in good working condition. If equipment is unsafe, then label it as such and contact the appropriate personnel to repair or discard it.
- Keep the patient's room as neat and clutter free as possible to
  minimize the risk of trips and falls. Pick up objects that have
  fallen on the floor. Secure electrical cords (e.g., for the bed or
  intravenous pumps) out of the way. Keep small equipment
  used for physical therapy intervention (e.g., cuff weights,
  Theraband) in a drawer or closet. Do not block the doorway
  or pathway to and from the patient's bed.
- Store assistive devices at the perimeter of the room when not in use. However, when patients are allowed to ambulate independently with an assistive device, the device should be in safe proximity to the patient.
- Provide enough light for the patient to move about the room or clearly see reading materials.
- Reorient a patient who is confused or disoriented. Often, patients who are confused are assigned rooms closer to the nursing station.
- Always leave the patient with the call bell or other communication devices within close reach. These include eyeglasses and hearing aids. Be sure to inquire if the patient will be using the room telephone, personal cellular phone, or both, and be sure it is within reach.
- Provide recommendations to nursing staff for the use of bathroom equipment (e.g., tub bench, bedside commode, or raised toilet seat) if the patient has functional limitations that may pose a safety risk.
- Dispose of soiled linens, dressings, sharps, and garbage according to the policies of the facility.

# Fall Risk

A fall is defined as "an event which results in a person coming to rest inadvertently on the ground or floor or other lower level."<sup>2</sup> A fall by this definition applies to the conscious or unconscious patient. For hospitalized patients, a fall is one of the most common adverse events and accounts for increased hospital personnel needs, length of stay, cost, and morbidity and mortality, especially among older adults.<sup>3</sup> Fall prevention during hospitalization includes a fall risk assessment performed on admission by nursing staff. Further prevention of falls involves a multitude of strategies and safety initiatives, including personal alarms, proper footwear, medication review, frequent toileting, adequate room lighting, and routine mobilization. The standardized fall risk assessment performed on admission varies by facility; however, common risk factors considered include prior falls, advanced age, medications, visual acuity, muscle strength, functional abilities (e.g., gait, balance, and mobility), and any medical conditions associated with falls (e.g., neurologic, orthopedic, or cognitive impairments; postural hypotension).<sup>4</sup> On the basis of the fall risk score and the subsequent designation of increased fall risk, a patient is identified as such (depending on hospital policy) by a specialized wristband and/or colored hospital gown, on a sign at the doorway to the room, and in the medical record.

# **Use of Restraints**

The use of a restraint may be indicated for the patient who is at risk of self-harm or harm to others, including health care providers, facility staff, and other patients. A restraint is defined as "any manual method, physical or mechanical device, material or equipment that immobilizes or reduces the ability of a patient to move his or her arms, legs, body or head freely; or a drug or medication when it is used as a restriction to manage the patient's behavior or restrict the patient's freedom of movement and is not a standard treatment or dosage for the patient's condition." It should also be noted that "a restraint does not include devices such as orthopedically prescribed devices, surgical dressings or bandages, protective helmets, or other methods that involve the physical holding of a patient for the purpose of conducing routine physical examinations or tests, or to protect the patient from falling out of bed, or to permit the patient to participate in activities without the risk of physical harm."5

The most common types of physical restraints in the acute care setting are wrist or ankle restraints, mitt restraints, vest restraints, or an enclosure bed. Side rails on a bed are considered a restraint when they are elevated with the intent of preventing the patient from exiting the bed, such as when all four rails are raised. The use of restraints requires an order from a licensed independent practitioner (LIP) that must be updated approximately every 24 hours. At that time, an LIP must assess the patient in person before documenting a new order to continue restraint use. A patient on restraints must be monitored frequently, either continuously or at predetermined time increments, depending on the type of restraint used and in accordance with facility policy and procedure and state law.

Although restraints are used with the intent to prevent injury, morbidity and mortality risks are associated with physical restraint use. Most notably, the presence of the restraint and the resultant limitation of patient mobility can increase agitation. New-onset pressure ulcers or alterations in skin integrity, urinary incontinence, constipation, and physical deconditioning can also occur. Musculoskeletal or nerve injury from prolonged positioning or from pushing or pulling on the restraint or strangulation/asphyxiation from the restraint as a result of entrapment can occur if the patient is not monitored closely. Many clinical guidelines and hospital care plans and policies reflect the trend of minimizing restraint use and deferring to alternatives, including regular assessment for personal needs such as scheduled toileting, food and fluids, sleep, and walking; bed and chair alarms to alert staff when a patient has moved from a bed or chair unassisted; diversions such as reading material, activity kits, or movement activities; recruitment of help from family or other patient care companions; relaxation techniques; alternative methods of camouflaging or securing medical devices, lines, or wires; and adequate pain management.<sup>6,7</sup> Use of restraints should be a last-resort option after all alternatives have been explored.

General guidelines most applicable to the physical therapist for the use of restraints include the following:

- Use a slipknot rather than a square knot to secure a restraint
  if the restraint does not have a quick-release connector. This
  ensures that the restraint can be untied rapidly in an emergency.
- Do not secure the restraint to a movable object (e.g., the bed rail), to an object that the patient is not lying or sitting on, or within reach of the patient so that he or she could easily remove it. If using restraints in the hospital bed, they should be secured to a part of the bed that will move with the patient if the head or feet settings are adjusted for positioning.
- Ensure the restraint is secure but not too tight. Place two
  fingers between the restraint and the patient to be sure circulation and skin integrity are not impaired.
- Always replace the restraint after a physical therapy session.
- Be sure the patient does not trip on the ties or "tails" of the restraint during functional mobility training.
- Consult with the health care team to determine whether a
  patient needs to have continued restraint use, especially if
  you feel the patient's behavior and safety have improved.
- Remember that the side effects of a chemical restraint may
  make a patient drowsy or alter his or her mental status; thus
  participation in a physical therapy session may be limited.

# **Medication Reconciliation**

Medication reconciliation is the process of comparing a list of the medication(s) a patient is taking with that ordered on admission, on transfer between areas of the hospital, and on discharge for the purpose of ensuring an up-to-date medication list.<sup>8</sup> Medication reconciliation is an important safety initiative in hospitals to prevent medication errors, such as inadvertent omission or duplication of a medication, incorrect dosing, and drug interactions. This also ensures that all health care providers can access the same complete medication list.<sup>8</sup>

# Latex Allergy

Latex allergy is a hypersensitivity to the proteins in natural rubber latex. If the reaction is immediate, then it is immunoglobulin E (IgE) mediated, with systemic symptoms resulting from histamine release. If the reaction is delayed, typically 48 to 96 hours after exposure, it is T-cell mediated, with symptoms localized at the area of contact and related to the processing chemicals used in the production of natural rubber latex. Signs and symptoms of an allergic reaction to latex range from mild to severe and may include hives, edema, contact dermatitis, rhinitis, headache, eye or throat irritation, abdominal cramping, respiratory difficulty (e.g., wheezing or shortness of breath), chest tightness, and possible anaphylaxis. S-11

Natural rubber latex is used in a multitude of products and equipment found in the acute care setting. This commonly includes gloves, stethoscopes, blood pressure cuffs, airway and intravenous tubing, adhesive tape, dressings, electrode pads, catheters, tubes, therapy/resistance bands, and hand grips on assistive devices. Many hospitals have minimized or eliminated latex products, replacing them with nonlatex or low-protein, powder-free latex products for the benefit of both the patient and health care provider. Pele Effective January 1, 2018, the U.S. Food and Drug Administration officially banned powdered surgeon's gloves, powdered patient examination gloves, and absorbable powder for lubricating a surgeon's glove because of the "unreasonable and substantial risk of illness or injury... including health care worker and patient sensitization to natural rubber latex." 13

Less than 1% of the general population has a sensitization to latex; health care workers have a greater incidence, between 8% and 17%. <sup>9,10</sup> Persons with spina bifida, congenital or urogenital defects, multiple childhood surgeries or medical treatments, occupational exposures to latex, or certain food allergies are at increased risk for latex allergy. <sup>9,10</sup> An association exists between latex sensitivity and food allergies, in which a person can have a cross-reactive allergy to a food (often a fruit) that is linked allergenically to natural rubber latex. This cross-reactivity is known as latex—food syndrome or latex—fruit allergy, and the foods most strongly associated with allergic reactions include banana, kiwi, avocado, and chestnuts. Apples, carrots, celery, papaya, potato, tomato, and melons have a moderate association with latex allergies. <sup>9-11,14</sup> Although not all people with latex sensitivity will also be allergic to these foods, awareness of the possibility is important.

If a patient has an allergy or hypersensitivity to latex, then it is documented in the medical record and at the patient's bedside. Hospitals will provide a special latex-free kit or cart, which consists of latex-free products for use during patient care. Health care providers may be at risk for developing a latex allergy because of their increased exposure to latex in the work setting. The latex allergy protein reaches the skin during exposure to the latex product. This risk is increased with powdered glove use because the skin is exposed to increased amounts of the latex protein through the powder. Aerosolized latex protein powder can also become an irritant to the airways and eyes, resulting in the respiratory and ocular symptoms noted previously. <sup>12</sup> If you suspect a latex hypersensitivity or allergy, seek assistance from the employee health office or a primary care physician.

# **Effects of Prolonged Bed Rest**

The effects of short-term (days to weeks) or long-term (weeks to months) bed rest can be deleterious and affect every organ system in the body. For the purposes of this discussion, the term bed rest includes immobilization, disuse, and prolonged recumbency. The physical therapist must recognize that a patient in the acute care setting is likely to have an alteration in physiology (e.g., a traumatic or medical-surgical disease or dysfunction) superimposed on bed rest, a second abnormal physiologic state.<sup>15</sup> Many patients on bed rest have been in the intensive care unit (ICU) for extended periods of time with multisystem organ failure or hemodynamic instability, requiring sedation and mechanical ventilation. Other clinical situations classically associated with long-term bed rest include severe burns; multiple trauma injuries; spinal cord injury; acute respiratory distress syndrome (ARDS); or nonhealing, full-thickness wounds (e.g., stage 4 pressure injuries) of the lower extremity or sacrum.

Physiologic consequences of bed rest include, but are not limited to, fluid volume redistribution, altered distribution of body weight and pressure, muscular inactivity, and aerobic deconditioning. <sup>16</sup> The degree of impairment in each system is directly related to the severity of illness or injury and the duration of bed rest. It has been noted that the cardiovascular and pulmonary systems deteriorate more rapidly and recover more slowly compared with the musculoskeletal system. This is seen more dramatically in the older adult population. <sup>16</sup> It is beyond the scope of this book to discuss in detail the physiologic and cellular mechanisms of the sequelae of prolonged bed rest; however, Table 1.1 lists the major systemic changes.

# **Physical Therapy Considerations**

- Monitor vital signs carefully, especially during mobilization at the edge and out of the bed.
- Progressively raise the head of the bed before or during a
  physical therapy session to allow blood pressure to regulate.
  Stretcher chairs (chairs that can position the patient from
  supine to different degrees of reclined or upright sitting)
  are also useful if orthostatic hypotension or activity intolerance prevents standing activity or if the patient may need to
  quickly return to the supine position.
- If hypotension persists after more than a few treatment sessions, consider the use of lower extremity antiembolism stockings, with or without elastic wrapping, when performing initial static sitting activities to minimize pooling of blood in the lower extremities. An abdominal binder may also be helpful to increase return from the abdominal vasculature to the central system.
- Time frames for physical therapy goals will likely be longer for the patient who has been on prolonged bed rest.
- Supplement formal physical therapy sessions with independent or family-assisted therapeutic exercise for a more timely recovery.
- Be aware of the psychosocial aspects of prolonged bed rest.
   Sensory deprivation, boredom, depression, and a sense of loss of control can occur.<sup>17</sup> These feelings may manifest as emotional lability or irritability, and caregivers may incorrectly perceive the patient to be uncooperative.

- As much as the patient wants to be off bed rest, he or she
  will likely be fearful the first time out of bed, especially if
  the patient has insight into his or her muscular weakness
  and impaired aerobic capacity. Additional assistance (e.g.,
  rehabilitation technicians, other licensed rehabilitation clinicians, nurses) may be necessary the first time up to the
  edge or out of the bed to ensure patient safety and medical
  stability.
- After treatment, leave necessities and commonly used items (e.g., call bell, television control, telephone, reading material, beverages, food, tissues) within reach to minimize feelings of confinement.
- Be sure to use bed or chair alarm systems or restraints, if ordered, to minimize the risk of falls.

# **Intensive Care Unit Setting**

As its name suggests, the ICU is a place of intensive medicalsurgical care for patients who require continuous monitoring, usually in conjunction with certain therapies or medical interventions, such as vasoactive medications, sedation, circulatory assist devices, and mechanical ventilation. ICUs are often named according to the specialized care that they provide, for example, coronary care unit (CCU) or surgical ICU (SICU). The patient in the ICU requires a high level of care; thus the nurse-to-patient ratio is 1:1 or 1:2.

# Common Patient and Family Responses to the Intensive Care Unit

Admission to the ICU has an effect on both the patient and family members that extends beyond the question of recovery from injury or illness. Patients in the ICU experience psychological, behavioral, and social challenges as a result of the stress experienced during their time in critical care. 18,19 The stress experienced by the patient and family members is not attributed to poor coping mechanisms or underlying psychological disorders; rather, they become overwhelmed by the number and severity of environmental and psychological stressors experienced in these care units. 19 Patients are faced with environmental stressors such as physical restrictions, sleep deprivation, unfamiliar medical equipment, crowding, excessive lighting, odors, noises, and touch associated with procedures. 18,19 Psychological stressors can include diminished dignity and self-esteem, powerlessness, loss of autonomy, vulnerability, boredom, pain, fear, anxiety, isolation, uncertainty about the future, and spiritual distress. 18,19

The patient's family can also become overwhelmed by the ICU setting. Family members may experience fear, shock, anxiety, stress, helplessness, anger, and denial.<sup>20,21</sup> Like the patient, the family may be overwhelmed by the stimuli and technology of the ICU, in addition to the stress of a loved one's critical or life-threatening illness. Holistic, patient-centered care must include family members and their well-being in addition to the primary concern, which is care of the patient. Health care providers should make every effort to recognize the needs of the family and identify and implement strategies to meet those needs to the best of their ability.<sup>21</sup>

TABLE 1.1 Systemi	TABLE 1.1 Systemic Effects of Prolonged Bed Rest		
Body System	ody System Effects		
Cardiac	Increased heart rate at rest and with submaximal exercise Decreased stroke volume, $VO_{2max}$ , left ventricular volume, and cardiac output Myocardial thinning Orthostatic hypotension		
Hematologic	Decreased total blood volume, red blood cell mass, and plasma volume Increased hematocrit Venous stasis, hypercoagulability, and blood vessel damage (Virchow triad), causing increased risk of venous thromboembolism		
Respiratory	Decreased lung volumes and capacities, especially FRC, FVC, and FEV <sub>1</sub> Decreased mucociliary clearance Increased risk of pneumonia, atelectasis, and pulmonary embolism Decreased arterial oxygen saturation		
Gastrointestinal	Decreased appetite, fluid intake, bowel motility, and gastric bicarbonate secretion Gastroesophageal reflux Difficulty swallowing		
Genitourinary	Increased mineral excretion, kidney stones, difficulty voiding, and urinary retention Increased risk of urinary tract infection		
Endocrine	Altered temperature and sweating responses, circadian rhythm, regulation of hormones, increased cortisol secretion, and glucose intolerance Decreased overall metabolism		
Musculoskeletal	Muscle: increased muscle weakness (especially in antigravity muscles), atrophy, decreased muscle endurance, risk of contracture, weakened myotendinous junction, and altered muscle excitation  Bone: disuse osteoporosis  Joints: degeneration of cartilage, synovial atrophy, and ankylosis		
Neurologic	Sensory and social deprivation Decreased dopamine, noradrenaline, and serotonin levels Depression, restlessness, insomnia Decreased balance, coordination, and visual acuity Increased risk of compression neuropathy Reduced pain threshold		
Integumentary	Increased risk of pressure injury formation		
Immune system	Increased risk of reactivation of latent viruses Reduced immune response and immunity		
Psychological	Impaired self-worth and self-esteem Increased risk of delirium, depression, and posttraumatic stress syndrome		
Body composition	Increased sodium, calcium, potassium, phosphorus, sulfur, and nitrogen loss Increased body fat and decreased lean body mass Fluid shift from the legs to the abdomen/thorax/head, diuresis, natriuresis, dehydration		

FEV<sub>1</sub>, Forced expiratory volume in 1 second; FRC, functional residual capacity; FVC, forced vital capacity; VO<sub>2max</sub>, maximum oxygen update.

Data from Dean E, Butcher S. Mobilization and exercise: physiological basis for assessment, evaluation, and training. In: Frownfelter D, Dean E, ed. Cardiovascular and Pulmonary Physical Therapy Evidence to Practice. 5th ed. St. Louis, Elsevier; 2012; Bartels M, Prince DZ. Acute medical conditions. In: Cifu DX, ed. Braddom's Physical Medicine and Rehabilitation. 5th ed. Philadelphia: Elsevier; 2016; Knight J, Nigam Y, Jones A. Effects of bed rest 1: cardiovascular, respiratory, and haemotological systems. Effects of bed rest 2: Gastrointestinal, endocrine, renal, reproductive, and nervous systems. Effects of bed rest 3: musculoskeletal and immune systems, skin and self-perception (website): http://www.nursingtimes.net. Accessed August 27, 2018.

An acute state of delirium, often termed *ICU delirium, ICU syndrome*, or *ICU psychosis*, is a state of delirium that can occur during the stay in the ICU. Delirium is a "disturbance in consciousness with inattention accompanied by a change in cognition or perceptual disturbance that develops over a short period of time (hours to days) and fluctuates over time." <sup>22</sup> The exact pathophysiology of ICU delirium is unknown, but research has demonstrated that ICU patients may have 10 or more identified risk factors and are at a much higher risk for the development of delirium compared with other populations. <sup>22</sup> A strong association has been found between delirium and variables such as mechanical ventilation, emergency surgery, polytrauma,

organ failure, hypertension, metabolic acidosis, coma, history of dementia, and advanced age.<sup>23</sup> Other possible risk factors include hypoxemia, use of certain benzodiazepine and narcotic medications, infection, immobilization, and pain.<sup>22</sup> ICU delirium is associated with increased time on mechanical ventilation, long-term cognitive impairments, extended length of ICU and hospital stay, increased cost, and higher rates of mortality.<sup>22</sup>

ICU delirium may present as hyperactive (characterized by agitation and restlessness), hypoactive (characterized by withdrawal and flat affect or by decreased responsiveness), or mixed (a fluctuation between the two).<sup>24</sup> Most patients will experience delirium of either mixed or hypoactive nature.

Purely hyperactive delirium is reported in less than 5% of ICU patients. <sup>24</sup> It is important to recognize the distinction between ICU delirium and dementia. ICU delirium has a sudden onset of new cognitive and behavior changes attributed to the current medical situation. Dementia has a more gradual onset and would not be triggered by an ICU admission. <sup>24</sup>

Current practice guidelines recommend that adult ICU patients be regularly assessed for ICU delirium. Accepted assessment tools are the Confusion Assessment Method for the Intensive Care Unit (CAM-ICU) and the Intensive Care Delirium Screening Checklist (ICDSC).<sup>22</sup> These tools are most appropriately used in tandem with an additional validated assessment of consciousness, one example being the Richmond Agitation-Sedation Scale (RASS).<sup>24,25</sup> Both the CAM-ICU and the ICDSC have been validated for use with verbal patients as well as those on mechanical ventilation.<sup>25</sup>

Management strategies for delirium include both pharmacologic and nonpharmacologic interventions. Pharmacologic strategies focus on a thorough review of current medications to identify and eliminate or decrease those that may be causing or exacerbating the delirium (e.g., sedatives, benzodiazepines, analgesics, and/or anticholinergic medications).<sup>22</sup> Current literature is limited with regard to the use of specific medications for the treatment of ICU delirium.<sup>22</sup> Nonpharmacologic interventions include minimizing all risk factors; repeated reorientation and cognitive stimulation throughout the day; maximization of the normal sleep pattern; hydration; pain management; early mobility; removal of lines, wires, and restraints, when appropriate; use of eyeglasses and hearing aids; and minimizing noxious noises or environmental stimuli.<sup>22</sup>

The transfer of a patient from the ICU to a general floor unit can be a stressful and anxiety-provoking event for the patient and family. Referred to as *relocation stress syndrome*, the patient and family may voice concerns about leaving staff members whom they have come to recognize and know by name; they may have to learn to trust new staff, or fear that the level of care may be inferior to that in the ICU.<sup>20</sup> To minimize this anxiety, the physical therapist may continue to treat the patient (if staffing allows), gradually transition the care to another therapist, or assure the patient and family that the general goals of physical therapy will remain unchanged.

# Critical Illness Myopathy, Polyneuropathy, and Polyneuromyopathy

Together, critical illness myopathy (CIM), critical illness polyneuropathy (CIP), and critical illness polyneuromyopathy (CIPNM, a combination of both CIM and CIP) are the leading causes of ICU-related neuromuscular weakness, often resulting in difficulty or failure to successfully wean from mechanical ventilation. <sup>26</sup> These processes involve sensorimotor axons and skeletal muscles, resulting in significant limb and respiratory muscle weakness. Diagnosis is ideally confirmed through electrodiagnostic studies (e.g., nerve conduction and electromyography [EMG]); however, the feasibility of these studies can be limited in the ICU setting. Therefore muscle biopsy and examination of the phrenic nerve and diaphragm can also be helpful diagnostic tools. <sup>26,27</sup> Differential diagnoses may include

Guillain-Barré syndrome, amyotrophic lateral sclerosis, sarcoidosis, myasthenia gravis, rhabdomyolysis, medication toxicity, metabolic neuropathies, ongoing neuromuscular blockage, and muscular dystrophy. 26,27 Diagnosis is often first considered when the patient demonstrates failure to wean from mechanical ventilation. Because of the complex medical state of the patient (e.g., sedation, drug-induced paralysis, and possible encephalopathy), it is often difficult to determine the actual time of onset.<sup>26</sup> CIM and CIPNM are more often reported, with occurrence rates between 25% and 83%. The likelihood of pure CIP is still being debated among experts.<sup>26</sup> The patient's underlying medical condition is a large factor in determining the development and severity of CIM, CIP, and CIPNM. Acute respiratory distress, respiratory failure, mechanical ventilation, trauma, use of vasopressors, intravenous steroid use, sepsis, systemic inflammatory response syndrome, multiple organ failure, inadequate blood glucose control, renal replacement therapy, and prolonged immobility have all been identified as possible variables that increase the risk of developing CIM, CIP, or CIPNM.<sup>26,27</sup>

CIM presents as flaccid, symmetric weakness that is more proximal than distal and includes respiratory muscles. Atrophy will be noted as the illness progresses. Sensation is preserved, but reflexes are generally decreased or absent. 26-28 Electrodiagnostic testing demonstrates reduced amplitude of motor action potentials and an inability to excite muscle tissue despite direct stimulation. Sensory findings are near or at normal. Muscle biopsy will reveal evidence of denervation and reinnervation with fiber-type grouping and atrophy. 26

CIP presents as flaccid, bilateral, distal extremity weakness, sensory loss, and paresthesia. Although deep tendon reflexes usually remain intact initially, they will likely decrease over time. <sup>26-28</sup> The lower extremities are generally affected more compared with the upper extremities, and findings may not be symmetric in nature. <sup>26</sup> Nerve conduction studies demonstrate decreased amplitude of both sensory and motor action potentials without conduction velocity impairment. <sup>26,28</sup> Nerve biopsy reveals widespread motor and sensory nerve degeneration. <sup>26</sup>

CIPNM is a condition with overlap of both CIM and CIP such that electrodiagnostic and clinical features of both will be noted.  $^{26}$ 

Initial symptoms of CIM, CIP, and CIPNM are generally noted within 2 weeks of admission to the ICU but have been reported as soon as 72 hours. <sup>26</sup> Medical management includes supportive and symptomatic care, treatment of the causative factor, and physical therapy. No proven cure exists; however, an intensive insulin regimen is the only medical intervention with notable clinical benefit. <sup>26</sup>

# Sleep Pattern Disturbance

The biological need for sleep is well understood. Ill and injured individuals require increased amounts of sleep over the healthy population to facilitate recovery. However, adequate sleep, in both quantity and quality, can be difficult to obtain in the acute care setting. <sup>29-32</sup> Sleep disturbance is defined as "insufficient duration or stages of sleep that results in discomfort and interferes with quality of life." <sup>30</sup> Inadequate or disrupted sleep can result in impaired health and well-being (e.g., immune, metabolic,

cardiorespiratory, and neurologic system dysfunction); delayed healing or recovery; decreased energy levels; alterations in cognition, mental status, and mood; and increased stress, anxiety, and pain.<sup>29,31,32</sup> The defining characteristics of sleep disturbance are difficulty falling or remaining asleep with or without fatigue on awakening, drowsiness during the day, decreased overall functioning, inability to concentrate, and mood alterations.<sup>33</sup>

In the acute care setting, sleep disturbance may be related to both environmental and nonenvironmental causes. Environmental concerns include frequent awakenings associated with a medical procedure or the need for nursing intervention (e.g., monitoring of vital signs); elevated noise levels, light exposure (e.g., disruptive light at night and inadequate light during the day), and the challenges of sleeping in an unfamiliar environment.<sup>29,31,32</sup> Nonenvironmental causes of disrupted sleep may include the severity of the current disease or illness, pain, medications, and certain medical interventions (e.g., ventilation, dialysis, urinary catheters, external orthopedic hardware or devices, and other lines and wires).<sup>29,31,32</sup> Sleep disturbance is often more prevalent in the older adult population because of changes in circadian rhythms, coexisting health conditions, and dementia.<sup>34</sup>

Possible interventions for sleep disruption include appropriate and adequate management of the current illness, injury, and pain; review of possible sleep-related pharmacologic side effects; reduction in night time noise, light, and interruptions; increased daytime stimulation with reduction in daytime sleeping; and relaxation techniques such as guided imagery, aromatherapy, or music. <sup>31,32</sup> The physical therapist should be aware of the patient who has altered sleep patterns or difficulty sleeping because lack of sleep can affect a patient's ability to participate during a therapy session. The patient may have trouble concentrating and performing higher-level cognitive tasks. The pain threshold may be decreased, and the patient also may exhibit decreased emotional control. <sup>31</sup>

# Substance Use and Withdrawal

Casual or habitual abuse of alcohol, illicit drugs (e.g., cocaine), or medications (e.g., opioids) is a known contributing factor in acute and chronic illness, traumatic accidents, drowning, burn injury, and suicide.<sup>35</sup> A patient in the acute care setting may present with acute intoxication or drug overdose or with a known (i.e., documented) or unknown substance abuse problem. When a patient with an unknown substance abuse disorder is hospitalized, it becomes a challenge to the hospital staff when substance withdrawal occurs. The physical therapist is not usually involved in the care of the patient admitted with substance intoxication or overdose until medical stability is achieved. However, the physical therapist may become involved when the stabilized patient presents with impaired strength, balance, coordination, or functional mobility. Physical therapy may also be consulted to assist in developing a safe discharge plan.

This text will focus on alcohol withdrawal because of its relatively high incidence. The *Diagnostic and Statistical Man*nal of Mental Disorders, fifth edition (DSM-V) has integrated two previously identified disorders, alcohol abuse and alcohol dependence, into a single diagnosis, alcohol use disorder (AUD). AUD is further divided into mild, moderate, and severe categories based on the number of identified symptoms.<sup>36</sup>

An estimated 15 million persons, age 18 years or older, in the United States have an AUD. <sup>36</sup> Data suggest that one in four patients admitted to a hospital has an AUD. <sup>19</sup> Alcohol withdrawal syndrome (AWS) is the sudden reduction or cessation of alcohol intake after heavy and prolonged alcohol consumption, with the development of two or more physical manifestations within hours to days. <sup>37</sup> Noted signs and symptoms must result in "significant distress or impairment in social, occupational, or other important areas of functioning" and may not be attributed to any other condition or cause. <sup>37</sup> Clinical manifestations of AWS are classified as mild, moderate, or severe. <sup>19,38</sup>

- Mild signs/symptoms of AWS are observed within 3 to 6 hours after alcohol use is discontinued and typically last 1 to 2 days. Symptoms include tachycardia, palpitations, tremor, diaphoresis, headache, nausea and vomiting, anxiety, and insomnia.
- Moderate signs/symptoms of AWS include persistence or worsening of the previously listed conditions and a new onset of generalized tonic-clonic seizures (within 12–48 hours, but may occur as soon as 1 hour after the last drink) and visual hallucinations (within 12–24 hours of alcohol cessation). The patient is aware, and often very distressed, that he or she is hallucinating.<sup>37-39</sup>
- Severe AWS and delirium tremens (DT) symptoms begin 48 to 96 hours after the last drink and last up to 7 days. Clinical manifestations include uncontrolled shaking, tachycardia, hypertension, hallucinations, disorientation, agitation, diaphoresis, and fever.<sup>37-39</sup> It should be noted that DT-related hallucinations are more global in nature, with alterations in vital signs. The timing of onset and the nature of the hallucinations are the factors that distinguish alcoholic hallucinations (described above) from DT-related halluciations.<sup>38</sup>

Medical interventions to prevent or minimize AWS include hydration; electrolyte, vitamin, and glucose replacement (e.g., intravenous infusion of thiamine, folic acid, magnesium, and a multivitamin in saline—often referred to as a "banana bag"); adequate nutrition; and the use of benzodiazepines. <sup>38</sup> Ideally, an objective scale is used to grade AWS symptoms and dose medication or other interventions accordingly. The Prediction of Alcohol Withdrawal Severity Score (PAWSS) assessment tool may be used to screen for risk of development of withdrawal symptoms. <sup>37</sup> The Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar) is a commonly used tool for grading withdrawal severity and guiding medical treatment. <sup>19,38</sup>

# Severity of Injury and Illness

Determination of severity of injury and illness often focuses on identifying the extent of actual or anticipated tissue damage and the associated physiologic response of the body.<sup>40</sup> Other dimensions to consider are the cognitive, psychological, and functional effects of the injury or illness on the patient.<sup>41</sup> This information may assist in predicting patient morbidity, disability, mortality, hospital readmission, and use of health care resources.<sup>40,42</sup>

A multitude of host-related factors contribute to injury severity, including preexisting medical, nutritional, and psychological conditions; age; sex; prior level of function; environmental factors; and personal characteristics. 40,41

Initial determination of injury and illness severity generally occurs by first responders or emergency room and ICU physicians. Table 1.2 outlines some commonly used injury and illness severity scoring systems. The acute care physical therapist will encounter results of these assessments within the patients' medical record and should have a basic understanding of their implication in predicted outcomes.

# **End-of-Life Considerations**

End-of-life care may include complex moral, ethical, or legal dilemmas, or a combination of these, with regard to a patient's vital physiologic functions, medical-surgical prognosis, quality of life, and personal values and beliefs. 43 End-of-life considerations faced by patients, family, and caregivers include the following:

- Resuscitation status
- Withholding and withdrawing medical therapies
- Palliative and hospice care
- Advance directives
- · Coma, vegetative state, and brain death

# **Resuscitation Status**

It is imperative that health care providers are aware of the patient's wishes regarding resuscitation should an emergency situation arise. Therefore every patient has a "code" status, which refers to the decision of whether or not life-saving interventions should be initiated after cardiopulmonary arrest. The designation full code means all appropriate efforts will be made to revive a patient after cardiopulmonary arrest. Do not resuscitate (DNR) is the predetermined decision to decline cardiopulmonary resuscitation (CPR), including defibrillation and pharmacologic cardioversion in the case of cardiopulmonary arrest. The code status do not intubate (DNI) is the predetermined decision to decline intubation for the purpose of subsequent mechanical ventilation in the event of respiratory arrest. Either full code or DNR and/or DNI status is officially documented in the medical record by the attending physician. If a patient is DNR or DNI, he or she will wear a wristband with that designation. The physical therapist must be aware of each patient's resuscitation or code status. DNR/DNI orders do not directly affect the physical therapy plan of care.

# Withholding and Withdrawing Medical Therapies

Withholding care is the decision to defer initiation of a treatment because it is not beneficial for or desired by the patient. Withdrawing care refers to the discontinuation of a treatment that is no longer beneficial or consistent with the goals of the patient and/or family. In both situations, a specific medical intervention is removed from the patient's plan of care (e.g., mechanical ventilation, hemodialysis, chemotherapy, or artificial nutrition and hydration). By forgoing life-sustaining medical treatment, the underlying disease process is allowed to take its course and

natural death occurs. <sup>44</sup> In these situations, an order for "comfort measures only" (CMO) is written by the physician. The patient on CMO status continues to receive care to maintain overall comfort (e.g., medications for pain control or sedation or to otherwise eliminate distress). Any intervention that causes discomfort is discontinued. <sup>44</sup> The patient on CMO status does not receive physical therapy.

# **Palliative and Hospice Care**

Palliative and hospice care provide meaningful but distinctly different services to patients with a serious illness (e.g., chronic obstructive pulmonary disease [COPD], Parkinson's disease, cancer, Alzheimer's disease). The goal of palliative care is the management or relief of distressing symptoms of illness to "achieve the best quality of life for patients and their families, consistent with their values." 46,47 Palliative care is appropriate for any patient, regardless of age, stage of illness, or prognosis. This team-based care model is applied alongside any other medical treatments or interventions for the underlying disease condition and focuses on physical, spiritual, and emotional well-being; symptom management; family involvement and support; and access to any therapies that may provide comfort or piece of mind. 46-48

Hospice care is a form of palliative care that is specific to any patient with a life-limiting illness in his or her last 6 months of life. He Patients are no longer seeking curative intervention; the focus is on comfort and relief of symptoms, maximizing the quality of the remaining life, and providing support to the patient and family. Hike palliative care, hospice care has a team-based approach and can occur in any setting (e.g., hospital, long-term care facility, or the patients home). He

The role of physical therapy in patients receiving palliative care or hospice services will depend on the needs and desires of the patient and family. In either care situation, physical therapy may be consulted to provide recommendations and interventions for positioning, pain management, energy conservation, mobility strategies, edema management, family education, equipment training, and home modifications.<sup>50</sup> Because the focus of hospice is end-of-life care, intervention will be centered on comfort and maintenance of desired physical abilities to enhance the quality of remaining life, while preparing the patient and family for the anticipated decline as the disease progresses. Physical therapy intervention in the palliative care population focuses on current symptom management and optimizing physical function to improve the quality of life.<sup>50</sup> Physical therapists are uniquely equipped to meet the needs of this population because of the ability to provide a continuum of care, to provide services when a patient has a change in medical status, and to use a knowledge base encompassing movement dysfunction, ergonomics, and pain management.<sup>51</sup> The role of physical therapy in hospital-based palliative care may be consultative or ongoing.

### **Advance Directives**

An advance directive allows individuals to document their wishes regarding medical decisions and end-of-life care should they be unable to speak for themselves.<sup>52</sup> This is a legal document

TABLE 1.2 Critical Illness and Injury Severity Scoring Systems		
Scoring System	Name	Description
APACHE II	Acute Physiologic Assessment and Chronic Health Evaluation	Measurement of disease severity through assessment of 12 routine physiologic variables (temperature; MAP; HR; RR; oxygenation; arterial pH; serum sodium, potassium, and creatinine; Hct; WBC; and GCS), age, and underlying health status. Scores range from 0 to 71, with higher scores indicating increased severity of disease and risk of mortality. A third and fourth version have been developed but are more cumbersome and less widely utilized.
GCS	Glasgow Coma Scale	Rapid assessment of level of consciousness and severity of brain injury via eye opening and verbal and motor responses. Scores range from 3 to 15. Lower scores indicate increasingly compromised neurologic function, with a score of less than 8 typically suggestive of a comatose state. Developed for determination of neurologic prognosis in patients with head trauma but useful in monitoring severity of coma or impaired levels of consciousness in other populations. Refer to Chapter 6 for more information.
MPM III	Mortality Prediction Model	Predictor of hospital mortality and length of stay through the assessment of three physiologic variables (GCS, HR, SBP), presence of concurrent diagnoses, age, code status at admission, and admission and medical management related variables. MPM II may still be more widely used.
SAPS 3	3rd Simplified Acute Physiology Score	Assessment of illness severity and prediction of mortality during hospitalization through 20 variables including preadmission characteristics (age, health status and comorbidities, and medical management before current ICU admission), current admission information (reason for admission, surgical status, and presence of infection), and nine physiologic variables (HR, SBP, temperature, GCS, bilirubin, creatinine, leukocytes, pH, platelets, and oxygenation). Scores range from 5 to 124, with higher scores indicating increased likelihood of hospital mortality. It should be noted that SAPS II is still in use in some facilities.
SOFA	Sequential Organ Failure Assessment	Measurement of organ failure severity through the assessment of six organ systems (respiratory: PaO <sub>2</sub> /FiO <sub>2</sub> , coagulation: platelets, liver: bilirubin, cardiovascular: hypotension, CNS: GCS, and renal: creatinine or urine output). Prediction of mortality is not the focus, but can be estimated by SOFA score. Scores range from 0 to 24, with higher scores indicating increased degree of organ failure and associated risk of adverse outcomes and mortality.

BUN, Blood urea nitrogen; CNS, central nervous system; FiO<sub>2</sub>, fraction of inspired oxygen; Hct, hematocrit; HR, heart rate; MAP, mean arterial pressure; PaO<sub>2</sub>, partial pressure of arterial oxygen; RR, respiratory rate; SBP, systolic blood pressure; WBC, white blood cells.

Data from Merck Manual Professional Version: Critical care scoring systems (website): https://merckmanuals.com. Accessed August 30, 2018; Rapsang AG, Shyam DC: Scoring systems in the intensive care unit: a compendium, Indian J Crit Care Med. 18(4),220-228, 2014; Salluh JIF, Soares M: ICU severity of illness scores: APACHE, SAPS, and MPM. Curr Opin Crit Care. 20:557-565, 2014. DOI:10.1097/MCC.0000000000000135; Moreno RP: SAPS 3—from evaluation of the patient to evaluation of the intensive care unit. Part 2: development of a prognostic model for hospital mortality at ICU admission, Intensive Care Med. 10,1345-1355, 2005; Jentzer JC, Bennett C, Wiley BM, et al. Predictive value of the sequential organ failure assessment score for mortality in a contemporary cardiac intensive care unit population. J Am Heart Assoc. 18(7),e008169, 2018.

that remains in effect until the individual properly changes or amends it. State laws regarding advance directives vary; therefore the advance directive documents in one state may not be valid in another. <sup>52</sup> Advance directives typically include the designation of a medical power of attorney (POA) (also referred to as a *durable power of attorney* or *health care proxy*) and documentation of a living will. The medical POA is the individual who will make medical decisions on behalf of the patient should he or she be unable to do so. <sup>52,53</sup> A living will is a written statement that expresses the individual's desires regarding treatment decisions, such as resuscitation, mechanical ventilation, dialysis, and artificial nutrition and hydration. <sup>52,53</sup>

It is imperative that the patient or family present advance directive documents on admission to the hospital setting so that the patient's wishes may be honored in the event that he or she is no longer able to communicate them. The medical POA and living will go into effect only after a physician has determined that the patient is no longer capable of making his or her own medical decisions.<sup>52</sup>

# Coma, Vegetative State, and Brain Death

A diagnosis of coma, vegetative state, or brain death can be devastating. These conditions involve unconsciousness and absence of self-awareness but are distinct in terms of neurologic function and recovery. Coma is a state of uninterrupted unconsciousness without arousal or awareness, characterized by lack of eye opening and sleep/wake cycles. While brainstem reflex responses remain intact, no meaningful interaction with the environment occurs. 54,55 Coma is typically a symptom of another condition, such as neurologic disease (e.g., stroke), a mass (e.g., brain tumor), trauma (e.g., traumatic brain injury), or a metabolic derangement (e.g., encephalopathy); drug or alcohol overdose; poisoning; infection; or it may be psychogenic in nature.<sup>54</sup> A vegetative state (VS) is a transient state of wakefulness characterized by cyclic sleep patterns, spontaneous eye opening, involuntary movement, and normal body temperature, yet there is a lack of purposeful awareness or responsiveness to stimuli, cognitive function, and speech. VS is considered persistent if it lasts longer than 1 month after an acute trauma; it is considered permanent 3 months after a nontraumatic brain injury or 12 months after a traumatic brain injury.<sup>55</sup> The clinical criteria for brain death includes absence of brainstem reflexes, cerebral motor responses, and spontaneous respirations in the setting of a known irreversible cause.<sup>56,57</sup> Brain death can be confirmed with cerebral angiography, electroencephalography, transcranial Doppler sonography, radionuclide imaging, or computed tomography angiography.<sup>57</sup> Refer to Chapter 6 for more information on these neurologic diagnostic tests.

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# CHAPTER 2

# The Medical Record

Kathryn Panascia

# **CHAPTER OUTLINE**

Introduction
Confidentiality
Physical Therapist
Documentation
Components of the Medical
Record
Orders
Admission Note Format
Progress Notes
Reports

# **CHAPTER OBJECTIVES**

The objectives for this chapter are the following:

- 1. Briefly describe the medical record, including related confidentiality and security regulations
- 2. Review documentation standards for the physical therapist
- 3. Describe the different components of the medical record, including a detailed outline of the admission history and physical

# Introduction

The medical record is a legal document that contains information regarding a patient's health status, treatments received, provider plans, and an outline of delivery of care. Specifically, the medical record contains information about past and present history of illness and medical conditions, including current diagnosis; current medication list; known allergies and sensitivities and patient reaction; physical examination findings; diagnostic test and laboratory results; treatment plans and interventions; record of preventative services and screening; and follow-up plans. Both primary care and ancillary service providers, such as physical therapists, will be contributing information to the medical record. This document serves as the primary means of communication among providers and a complete and accurate medical record aids in the continuity of quality care. The medical record may also be used for educational purposes, performing quality improvement studies, conducting research, and resolving legal issues such as competency or disability.

In the past, medical records were paper-based and proved to have significant challenges and consequences. The National Academy of Medicine highlighted that a paper-based health care system resulted in "redundant tests; increased costs; uncoordinated and fragmented care; medical decisions made with incomplete data, leading to adverse events; and potential clinical innovations left undiscovered, hidden in patient files." As a response, the *Health Information Technology for Economic and Clinical Health (HITECH) Act* went into effect in February 2009 as a means to expand and protect meaningful use of health information technology via electronic health records (EHRs). Federally funded incentive programs were designed to encourage Medicare and Medicaid providers, both individual and organizational, to adopt the use of EHRs with ongoing demonstration of risk assessments to ensure compliance with the *Health Insurance Portability and Accountability Act (HIPAA)*. All eligible providers were required to use EHRs by 2015, with increasing annual penalties for those not in compliance. Data shows that nearly all U.S. hospitals and 80% of private practice providers have adopted the use of EHRs. This has allowed for increased sharing of health-related information among systems and a positive effect on quality, safety, and efficiency of health care services.

# Confidentiality

The HIPAA *Privacy Rule* e stablished standards for defining and protecting all "individually identifiable health information," which is considered "protected health information (PHI)" and should be kept confidential.<sup>6,7</sup> All health care providers and covered entities (e.g., health care plans; health care clearinghouses; and any business associates involved in data analysis, utilization review, billing, and claims processing) must safeguard the availability and integrity of PHI in oral, written, media, or electronic forms.<sup>6</sup> PHI includes any information that pertains to the past, present, or future physical or mental health conditions of an individual, including provision of care, payment

<sup>&</sup>lt;sup>a</sup>The authors acknowledge Michele P. West for prior contributions to this chapter.

TABLE 2.1 Prohibitive Abbreviations	TABLE 2.	1 Prohibitive	Abbreviations
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Do Not Use	Potential Problem	Use Instead
U, u (unit)	Mistaken for "0" (zero), the number "4" (four) or "cc"	Write "unit"
IU (International Unit)	Mistaken for IV (intravenous) or the number 10 (ten)	Write "International Unit"
Q.D, QD, q.d., qd (daily) QOD., QOD, q.o.d., qod (every other day) TIW or tiw	Mistaken for each other Period after Q mistaken for "I" and the "O" mistaken for the "I" Mistaken for 3 times a day or twice in 1 week	Write "daily" Write "every other day" Write "3 times weekly"
Trailing zero (X.0 mg) <sup>b</sup> Lack of trailing zero (.X mg)	Decimal point is missed	Write X mg Write 0.X mg
MS MSO <sub>4</sub> and MgSO <sub>4</sub>	Can mean morphine sulfate or magnesium sulfate Confused for one another	Write "morphine sulfate" Write "magnesium sulfate"
> and < (greater than and less than)	Mistaken as opposite of intended; mistakenly use the incorrect symbol	Write "greater than" or "less than"

<sup>&</sup>lt;sup>a</sup>Applies to all orders and all medication-related documentation that is handwritten (including free-text computer entry) or on preprinted forms.

<sup>b</sup>Exception: A "trailing zero" may be used only where required to demonstrate the level of precision of the value being reported, such as for laboratory results, imaging studies that report size of lesions, or catheter/tube sizes. It may not be used in medication orders or other medication-related documentation.

Data from The Joint Commission Official "Do Not Use" List. www.jointcommission.org. Last accessed August 24, 2018.; Institute for Safe Medication Practices: Unsafe

of care, and demographics.<sup>6</sup> The *Privacy Rule* does allow for, but not require, disclosure of PHI without patient authorization for the purposes of research and public health. It is recommended that the covered entity rely on best judgment and professional ethics when making the decision to disclose such information.<sup>6</sup>

medical abbreviations (website): http://www.consumermedsafety.org. Accessed November 10, 2018.

The HIPAA *Security Rule* addresses the confidentiality of electronic PHI (e-PHI).<sup>7</sup> The *Security Rule* states that a covered entity must ensure the integrity and availability of e-PHI that it creates, receives, maintains, or transmits.<sup>7</sup> The goal of the *Security Rule* is to protect e-PHI as covered entities adopt new and efficient technologies.<sup>7</sup>

All health care providers, individual and institutional, who exchange health-related information electronically and for the purposes of financial or administrative health care activities (e.g., referral, authorization, coordination of benefits, claims, and payment) must comply with HIPAA.<sup>8,9</sup> This would include the physical therapist, who must also adhere to the American Physical Therapy Association's *Guide for Professional Conduct* and *Code of Ethics for the Physical Therapist*, <sup>10,11</sup> all policies and procedures of the facility, and state law with regard to sharing medical record information with the patient, family, caregivers, visitors, or third parties.

# **CLINICAL TIP**

To ensure confidentiality of PHI in the acute care setting, the physical therapist should log off the computer when not in use, cover any paperwork kept on clipboards when moving within the facility, and use discretion when discussing patient information in shared rooms, hallways, and/or elevators.

# **Physical Therapist Documentation**

The physical therapist should comply with documentation standards, including, but not limited to, the policies/procedures of the organization, the state, and the American Physical Therapy Association's *Guidelines: Physical Therapy Documentation of Patient/Client Management.* 12

In general, documentation must be<sup>1,12</sup>:

- Completed for every visit/encounter, at the time of care or soon after
- Dated and timed with authenticated signature and credentials
- Legible and in blue or black ink (when applicable)
- Clearly labeled with the appropriate patient identification (e.g., name and identification number)
- Complete, accurate, and objective
- Cosigned for a physical therapist assistant (if required by state practice act) or student physical therapist
- Free of ambiguous acronyms or abbreviations to minimize misinterpretation and prevent errors that could result in patient safety issues (Table 2.1)<sup>13</sup>

Errors on handwritten documentation should be corrected with a single line through the error, followed by the author's initials and the date. Electronic documentation systems vary but will allow the author to indicate a change without deleting the original record.

These standards apply to documentation of the physical therapy examination, evaluation, diagnosis, prognosis, and plan of care (including interventions). <sup>12</sup> Routine physical therapy documentation includes the initial examination/evaluation, visit/encounter notes, reexamination, and discharge or discontinuation summary. <sup>12</sup> A documentation entry is required for every physical therapist encounter and should include the following, as applicable <sup>14</sup>:

- Phone calls or conversations with other health care providers, referral sources, or payment entities
- Handouts provided to the patient, including exercise programs or educational materials
- Follow-up information provided to the patient
- The use of interpreter services
- Therapist response and assessment of an adverse event or situation (completed outside the medical record via a facility approved incident report form)

- Be sure to document when a patient/family member declines or refuses therapy intervention or requests a specific time of day for therapy, including a rationale for such.
- Documentation of deferring or "holding" therapy should include a rationale and the source of the deferral, whether it is from the physician, nursing, physical therapist, or other provider.
- Documentation of patient unavailability (e.g., off the floor at a test or procedure) is also suggested.

# Components of the Medical Record

The organization of the medical record will vary from institution to institution; however, it is typically composed of the following basic sections:

# **Orders**

The "orders" section is a log of all instructions within the plan of care for the patient, including, but not limited to, medications, diagnostic or therapeutic tests and procedures, vital sign parameters, activity level or restrictions, mobility precautions, diet, request for consultation services, and resuscitation status. Orders may be written by a physician, a physician assistant, or a nurse practitioner. A verbal order, given either via telephone or face to face, may be taken by a nurse or other licensed and authorized health care provider, including a physical therapist, according to departmental, facility, and state policies. 15 In the interests of patient safety and error prevention, the process of taking a verbal order, especially for medications, has been minimized in most facilities.<sup>15</sup> In the event that a verbal order is communicated, it must be read back to the ordering practitioner and cosigned by that individual at a subsequent time.<sup>15</sup>

# **Physical Therapy Considerations**

- The orders section of the patient's medical record should be reviewed before the initial evaluation and any subsequent physical therapy intervention(s) for the following (as applicable): the order for physical therapy services, patient activity level, weight-bearing status, need for any bracing or orthotic devices, vital sign parameters, and positioning restrictions. On subsequent physical therapy sessions, the review of the orders section offers a "snapshot" of change(s) in the patient. Look for new or discontinued medications, changes in PO (by mouth) status, and new laboratory or diagnostic testing orders or results.
- If an order appears incomplete or ambiguous, the order should be clarified before beginning physical therapy.

# **Admission Note Format**

The following outline summarizes the basic format of the initial admission note (often referred to as the "H&P," that is, history and physical) written by a physician, a physician's assistant, or a nurse practitioner in the medical record. 16 The italicized items indicate the standard information the physical therapist should review before beginning an initial evaluation.

- I. History (subjective information)
  - A. Data that identify the patient, including the source and degree of reliability of the information

- B. History of present illness (HPI), including the chief complaint/concern (CC) and a chronological list of the events associated with, and leading up to, arrival at the hospital
- C. Past medical and surgical history (PMH, PSH), risk factors for disease, and if there are any known allergies and sensitivities, including reactions, to medication, food, or other substances
- D. Family health history, including age and health or age and cause of death for immediate family members as well as a relevant familial medical history
- E. Personal and social history, including occupation, lifestyle, leisure activities, substance use (e.g., tobacco, alcohol, illicit drugs), functional mobility status, the need for home or outpatient services, support system availability, and architectural barriers at home
- F. Current medications, including over-the-counter and nontraditional therapies, as well as overall level of compliance
- II. Physical examination (objective information). Findings are described in detail according to the following:
  - A. General information, including vital signs and physical appearance
  - B. Skin
  - C. Head, eyes, ears, nose, throat (HEENT); oropharynx; and thyroid
  - D. Neck, including lymph nodes and carotids
  - E. Heart
  - F. Lungs
  - G. Abdomen
  - H. Genitalia/pelvic/rectal examination
  - Extremities, including range of motion (ROM), strength, and pulses
  - Neurologic system (mental status, cranial nerves, motor strength, sensation, reflexes, cerebellar function/observed ambulation)
- III. Laboratory and test results (e.g., radiographic studies, electrocardiography [ECG], etc.)
- IV. Assessment. The assessment is a statement of the condition and prognosis of the patient in regard to the chief complaint and/or medical-surgical status. If the etiology of the problem(s) is unclear, then differential diagnoses are listed.
- V. Plan. The plan of care may include further observation, tests, laboratory analysis, consultation with additional specialty services or providers, pharmacologic therapies, other interventions, and discharge planning.

# **Physical Therapy Considerations**

- It is always advisable to note if a patient has a preferred name that may differ from the legal name documented in the medical record. This may be reported to the physical therapist by nursing staff, documented in the patient's room, or learned via direct conversation with the patient or their family.
- A patient's gender identity may differ from that assigned at birth or noted within the medical record. It is important to inquire about the patient's preferred name and pronouns (e.g., he/she, him/her, they/them). 17

# **Progress Notes**

A progress note is a shortened version of the initial admission note with an emphasis on any new findings, an updated assessment, and plan. The progress note section is typically multidisciplinary, with documentation from all caregivers in chronological order. The nursing staff documents its own admission assessment, problem list, and care plan(s). Medication reconciliation sheets, flow sheets, clinical pathways, consult service notes from other physicians and health care professionals, and operative and procedural notes are also included in this section.

# **Reports**

A variety of reports are filed chronologically in individual sections in the medical record (e.g., radiologic or laboratory reports). Each report includes an interpretation or normal reference ranges, or both, for various diagnostic or laboratory test results. Other types of reports include those from pulmonary function tests, electroencephalography (EEG), and stress testing.

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# **BODY SYSTEMS**

# CHAPTER 3

# Cardiac System

Konrad J. Dias Sean M. Collins

# **CHAPTER OUTLINE**

### Introduction

**Body Structure and Function** 

Cardiac Cycle

Cardiac Output

Coronary Perfusion

Systemic Circulation

### **Cardiac Evaluation**

**Patient History** 

**Physical Examination** 

Diagnostic and Laboratory Measures

### **Health Conditions**

Hypertension

**Acute Coronary Syndrome** 

Rhythm and Conduction

Disturbance

Valvular Heart Disease

Myocardial and Pericardial Hear

Disease

**Heart Failure** 

# Management

Revascularization and Reperfu-

sion of the Myocardium

Ablation Procedure Cardioversion

Life Vest

Valve Replacement

Percutaneous Aortic Valvotomy

and Transcatheter Aortic Valve

Implantation

Cardiac Transplantation

Cardiac Medications

Physical Therapy Intervention

# **Patient Education**

APPENDIX 3A: Description of Electrocardiography (ECG) Characteristics and Associated

APPENDIX 3B: Common Rhythm
Disturbances

# **CHAPTER OBJECTIVES**

The objectives of this chapter are the following:

- 1. Provide a brief overview of the structure and function of the cardiovascular system
- 2. Give an overview of cardiac evaluation, including physical examination and diagnostic testing
- 3. Describe cardiac diseases and disorders, including clinical findings and medical and surgical management
- 4. Establish a framework on which to base physical therapy evaluation and intervention in patients with cardiovascular disease

# Introduction

Physical therapists in acute care facilities commonly encounter patients with cardiac system dysfunction as either a primary morbidity or a comorbidity. The 2017 updated report from the American Heart Association on heart disease and stroke statistics reported that cardiovascular disease (CVD) accounts for approximately 800,000 deaths in the United States. <sup>1</sup> This accounts for one out of every three deaths in the United States. Globally CVD accounts for 31% of all deaths, with an estimated cost of \$1044 billion by 2030. <sup>1,2</sup>

Smoking, lack of physical activity, and obesity continue to be significant risk factors for CVD. Physical therapists play a critical role in educating patients on the negative effects of these risk factors and educate them on ways to modify lifestyle and reduce the risk for cardio-vascular events. Smoking, including secondhand smoke, accounts for an estimated 6.3 million deaths worldwide. Additionally, one in three adults in the United States do not meet current recommendations for physical activity, with an estimated 37.7% of adults categorized as obese. 1,2

In the acute care setting, the role of the physical therapist in managing patients with CVD is rooted in examination, evaluation, intervention, and discharge planning for the purpose of improving functional capacity and maximizing participation. The physical therapist must be prepared to safely accommodate for the effects of dynamic (pathologic, physiologic, medical, and surgical interventions) changes into his or her evaluation and plan of care.

The normal cardiovascular system provides the necessary pumping force to circulate blood through the coronary, pulmonary, cerebral, and systemic circulation. To perform work, such as during functional tasks, energy demands of the body increase, thus increasing the oxygen demands of the heart. A variety of pathologic states can create impairments in the cardiac system's structure and function, ultimately leading to functional limitations. To fully address these functional limitations, the physical therapist must understand normal and abnormal cardiovascular structure, function, clinical tests, and medical and surgical management.

# **Body Structure and Function**

The heart and the roots of the great vessels (Fig. 3.1) occupy the pericardium, which is located in the mediastinum. The sternum, the costal cartilages, and the medial ends of the third to fifth ribs on the left side of the thorax create the anterior border of the mediastinum. It is bordered inferiorly by the diaphragm, posteriorly by the vertebral column and ribs, and laterally by the pleural cavity (which contains the lungs). Specific cardiac structures and vessels and their respective functions are outlined in Tables 3.1 and 3.2.

*Note:* The mediastinum and the heart can be displaced from their normal positions with changes in the lungs secondary to various disorders. For example, a tension pneumothorax shifts the mediastinum away from the side of dysfunction (see Chapter 4 for a further description of pneumothorax).

The cardiovascular system must adjust the amount of nutrient- and oxygen-rich blood pumped out of the heart (cardiac output [CO]) to meet the spectrum of daily energy (metabolic) demands of the body.

The heart's ability to pump blood depends on the following characteristics<sup>3</sup>:

• Automaticity: The ability to initiate its own electrical impulse

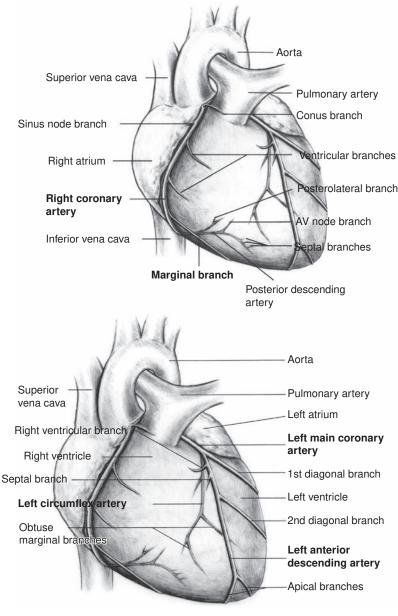


FIG. 3.1

Anatomy of the right coronary artery and left coronary artery, including left main, left anterior descending, and left circumflex coronary arteries. (From Becker RC. Chest Pain: The Most Common Complaints Series. Boston: Butterworth-Heinemann; 2000.)

- Excitability: The ability to respond to electrical stimulus
- Conductivity: The ability to transmit electrical impulse from cell to cell within the heart
- Contractility: The ability to stretch as a single unit and then passively recoil while actively contracting
- Rhythmicity: The ability to repeat the cycle in synchrony with regularity

# Cardiac Cycle

Blood flow throughout the cardiac cycle depends on circulatory and cardiac pressure gradients. The right side of the heart is a low-pressure system with little vascular resistance in the pulmonary arteries, whereas the left side of the heart is a high-pressure system with high vascular resistance from the systemic circulation. The cardiac cycle is the period from the beginning of one contraction, starting with sinoatrial (SA) node depolarization, to the beginning of the next contraction. *Systole* is the period of contraction, and *diastole* is the period of relaxation. Systole and diastole can also be categorized into atrial and ventricular components:

- Atrial diastole is the period of atrial filling. The flow of blood is directed by the higher pressure in the venous circulatory system.
- Atrial systole is the period of atrial emptying and contraction. Initial emptying of approximately 70% of blood occurs as a result of the initial pressure gradient between the atria

TABLE 3.1 Primary Structures of the Heart		
Structure	Description	Function
Pericardium	Double-walled sac of elastic connective tissue, a fibrous outer layer, and a serous inner layer	Protects against infection and trauma
Epicardium	Outermost layer of cardiac wall, covers surface of heart and great vessels	Protects against infection and trauma
Myocardium	Central layer of thick muscular tissue	Provides major pumping force of the ventricles
Endocardium	Thin layer of endothelium and connective tissue	Lines the inner surface of the heart, valves, chordae tendineae, and papillary muscles
Right atrium	Heart chamber	Receives blood from the venous system and is a primer pump for the right ventricle
Tricuspid valve	Atrioventricular valve between right atrium and ventricle	Prevents back flow of blood from the right ventricle to the atrium during ventricular systole
Right ventricle	Heart chamber	Pumps blood to the pulmonary circulation
Pulmonic valve	Semilunar valve between right ventricle and pulmonary artery	Prevents back flow of blood from the pulmonary artery to the right ventricle during diastole
Left atrium	Heart chamber	Acts as a reservoir for blood and a primer pump for the left ventricle
Mitral valve	Atrioventricular valve between left atrium and ventricle	Prevents back flow of blood from the left ventricle to the atrium during ventricular systole
Left ventricle	Heart chamber	Pumps blood to the systemic circulation
Aortic valve	Semilunar valve between left ventricle and aorta	Prevents back flow of blood from the aorta to the left ventricle during ventricular diastole
Chordae tendineae	Tendinous attachment of atrioventricular valve cusps to papillary muscles	Prevents valves from everting into the atria during ventricular systole
Papillary muscle	Muscle that connects chordae tendineae to floor of ventricle wall	Constricts and pulls on chordae tendineae to prevent eversion of valve cusps during ventricular systole

TABLE 3.2 Great Vessels of the Heart and Their Fu	Inction
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Structure	Description	Function
Aorta	Primary artery from the left ventricle that ascends and then descends after exiting the heart	Ascending aorta delivers blood to neck, head, and arms Descending aorta delivers blood to visceral and lower body tissues
Superior vena cava	Primary vein that drains into the right atrium	Drains venous blood from head, neck, and upper body
Inferior vena cava	Primary vein that drains into the right atrium	Drains venous blood from viscera and lower body
Pulmonary artery	Primary artery from the right ventricle	Carries blood to lungs

and the ventricles. Atrial contraction then follows, squeezing out the remaining 30%.<sup>3</sup> This is commonly referred to as the *atrial kick*.

- Ventricular diastole is the period of ventricular filling. It initially occurs with ease; then, as the ventricle is filled, atrial contraction is necessary to squeeze the remaining blood volume into the ventricle. The amount of stretch placed on the ventricular walls during diastole, referred to as *left ventricular end diastolic pressure (LVEDP)*, influences the force of contraction during systole. (Refer to the Factors Affecting Cardiac Output section for a description of preload.)
- Ventricular systole is the period of ventricular contraction. The initial contraction is isovolumic (i.e., it does not eject blood), which generates the pressure necessary to serve as the catalyst for rapid ejection of ventricular blood. The left ventricular ejection fraction (EF) represents the percent of end diastolic volume ejected during systole and is normally approximately 60%.<sup>3</sup>

# Cardiac Output

CO is the quantity of blood pumped by the heart in 1 minute. Regional demands for tissue perfusion (based on local metabolic needs) compete for systemic circulation, and total CO adjusts to meet these demands. Adjustment to CO occurs with changes in heart rate (HR—chronotropic) or stroke volume (SV—inotropic). Normal resting CO is approximately 4 to 8 liters per minute (L/min), with a resting HR of 70 beats per minute (bpm); resting SV is approximately 71 mL/beat. The maximum value

of CO represents the functional capacity of the circulatory system to meet the demands of physical activity.

CO 
$$(L/min) = HR (bpm) \times SV (L)$$

CO also can be described relative to body mass as the cardiac index (CI), the amount of blood pumped per minute per square meter of body mass. Normal CI is between 2.5 and 4.2 L/min/m². This wide normal range makes it possible for CO to decline by almost 40% and still remain within the normal limits. Although several factors interrupt a direct correlation between CI and functional aerobic capacity, CI below 2.5 L/min/m² represents a marked disturbance in cardiovascular performance and is always clinically relevant.<sup>5</sup>

#### **Factors Affecting Cardiac Output**

**Preload.** Preload is the amount of tension on the ventricular wall before it contracts. It is related to venous return and affects SV by increasing left ventricular end diastolic volume in addition to pressure and therefore contraction.<sup>3</sup> This relationship is explained by the Frank-Starling mechanism and is demonstrated in Fig. 3.2.

**Frank-Starling Mechanism.** The Frank-Starling mechanism defines the normal relationship between the length and tension of the myocardium. The greater the stretch on the myocardium before systole (preload), the stronger is the ventricular contraction. The length—tension relationship in skeletal muscle is based on the response of individual muscle fibers; however, relationships between cardiac muscle length and tension consist

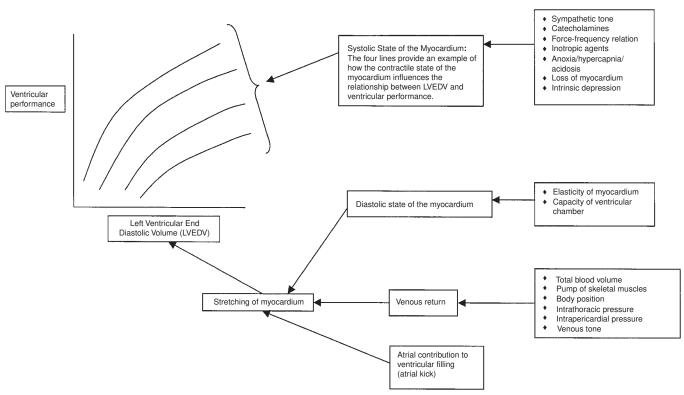


FIG. 3.2
Factors affecting left ventricular function. (Modified from Braunwal E, Ross J, Sonnenblick E et al. *Mechanisms of Contraction of the Normal and Failing Heart.* 2nd ed. Boston: Little, Brown; 1976.)

of the whole heart. Therefore length is considered in terms of volume; tension is considered in terms of pressure. A greater volume of blood returning to the heart during diastole equates to greater pressures generated initially by the heart's contractile elements. Ultimately facilitated by elastic recoil, a greater volume of blood is ejected during systole. The effectiveness of this mechanism can be reduced in pathologic situations.<sup>4</sup>

**Afterload.** Afterload is the force against which a muscle must contract to initiate shortening.<sup>6</sup> Within the ventricular wall, this is equal to the tension developed across its wall during systole. The most prominent force contributing to afterload in the heart is blood pressure (BP), specifically vascular compliance and resistance. BP affects aortic valve opening and is the most obvious load encountered by the ejecting ventricle. An example of afterload is the amount of pressure in the aorta at the time of ventricular systole.<sup>3</sup>

#### **Cardiac Conduction System**

A schematic of the cardiac conduction system and a normal electrocardiography (ECG) result are presented in Fig. 3.3. Normal

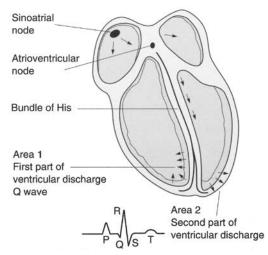


FIG. 3.3
Schematic representation of the sequence of excitation in the heart. (From Walsh M, Crumbie A, Reveley S. Nurse Practitioners: Clinical Skills and Professional Issues. Boston: Butterworth-Heinemann; 1999.)

conduction begins in the SA node and travels throughout the atrial myocardium (atrial depolarization) via intranodal pathways to the atrioventricular (AV) node, where it is delayed momentarily. It then travels to the bundle of His, to the bundle branches, to the Purkinje fibers, and finally to the myocardium, resulting in ventricular contraction. Disturbances in conduction can decrease CO (refer to the Health Conditions section for a discussion of rhythm and conduction disturbances).

**Neural Input.** The SA node has its own inherent rate. However, neural input can influence HR, heart rate variability (HRV), and contractility through the autonomic nervous system.<sup>3,9</sup>

Parasympathetic system (vagal) neural input generally decelerates cardiac function, thus decreasing HR and contractility. Parasympathetic input travels through the vagus nerves. The right vagus nerve stimulates primarily the SA node and affects rate, whereas the left vagus nerve stimulates primarily the AV node and affects AV conduction.<sup>3,9</sup>

Sympathetic system neural input is through the thoracolumbar sympathetic system and increases HR and augment ventricular contractility, thus accelerating cardiac function.<sup>3</sup>

**Endocrine Input.** In response to physical activity or stress, a release in catecholamines increases HR, contractility, and peripheral vascular resistance for a net effect of increased cardiac function (Table 3.3).<sup>3</sup>

**Local Input.** Tissue pH, concentration of carbon dioxide (CO<sub>2</sub>), concentration of oxygen (O<sub>2</sub>), and metabolic products (e.g., lactic acid) can affect vascular tone.<sup>3</sup> During exercise, increased levels of CO<sub>2</sub>, decreased levels of O<sub>2</sub>, decreased pH, and increased levels of lactic acid at the tissue level dilate local blood vessels and therefore increase CO distribution to that area.

#### Cardiac Reflexes

Cardiac reflexes influence HR and contractility and can be divided into four general categories: baroreflex (pressure), Bainbridge reflex (stretch), chemoreflex (chemical reflex), ergoreflex (ergoreceptors).

Baroreflexes are activated through a group of mechanoreceptors located in the heart, great vessels, and intrathoracic and

TABLE 3.3 Cardiac Effects of Hormones			
Hormone	Primary Site	Stimulus	Cardiac Effect
Norepinephrine	Adrenal medulla	Stress/exercise	Vasoconstriction
Epinephrine	Adrenal medulla	Stress/exercise	Coronary artery vasodilation
Angiotensin	Kidney	Decreased arterial pressure	Vasoconstriction, increased blood volume
Vasopressin	Posterior pituitary	Decreased arterial pressure	Potent vasoconstrictor
Bradykinin	Formed by polypeptides in blood when activated	Tissue damage/inflammation	Vasodilation, increased capillary permeability
Histamine	Throughout tissues of body	Tissue damage	Vasodilation, increased capillary permeability
Atrial natriuretic peptides	Atria of heart	Increased atrial stretch	Decreased blood volume
Aldosterone	Adrenal cortex	Angiotensin II (stimulated) by hypovolemia or decreased renal perfusion	Increased blood volume, kidneys excrete mor potassium

cervical blood vessels. These mechanoreceptors are most plentiful in the walls of the internal carotid arteries.<sup>3</sup> Mechanoreceptors are sensory receptors that are sensitive to mechanical changes, such as pressure and stretch. Activation of the mechanoreceptors by high pressures results in an inhibition of the vasomotor center of the medulla that increases vagal stimulation. This chain of events is known as the *baroreflex* and results in vasodilation, decreased HR, and decreased contractility.

Mechanoreceptors located in the right atrial myocardium respond to stretch. An increased volume in the right atrium results in an increase in pressure on the atrial wall. This reflex, known as the *Bainbridge reflex*, stimulates the vasomotor center of the medulla, which, in turn, increases sympathetic input and increases HR and contractility.<sup>3</sup> Respiratory sinus arrhythmia, an increased HR during inspiration and decreased HR during expiration, may be facilitated by changes in venous return and SV caused by changes in thoracic pressure induced by the respiratory cycle. At the beginning of inspiration when thoracic pressure is decreased, venous return is greater; therefore a greater stretch is exerted on the atrial wall.<sup>10</sup>

Chemoreceptors located on the carotid and aortic bodies have a primary effect on increasing rate and depth of ventilation in response to  $CO_2$  levels, but they also have a cardiac effect. Changes in  $CO_2$  during the respiratory cycle also may result in sinus arrhythmia.<sup>3</sup>

Ergoreceptors and the ergoreflex regulates hemodynamics through activation of mechanosensitive afferents to inhibit the sustained vagal effects on the heart caused by an increase heart rate during physical loading.<sup>11</sup>

#### **Coronary Perfusion**

For a review of the major coronary arteries, refer to Fig. 3.1. Blood is pumped to the large superficial coronary arteries during

ventricular systole. At this time, myocardial contraction limits the flow of blood to the myocardium; therefore myocardial tissue is perfused during diastole.

# **Systemic Circulation**

For a review of the distribution of systemic circulation, refer to Fig. 3.4. Systemic circulation is affected by total peripheral resistance (TPR), which is the resistance to blood flow by the force created by the aorta and arterial system. Two factors that contribute to resistance are (1) vasomotor tone, in which vessels dilate and constrict; and (2) blood viscosity, in which greater pressure is required to propel thicker blood. TPR, also called *systemic vascular resistance*, and CO influence BP.<sup>3</sup> This relationship is illustrated in the following equation:

$$BP = CO \times TPR$$

#### Cardiac Evaluation

Cardiac evaluation uses patient history, physical examination (which consists of observation, palpation, BP measurement, and heart sound auscultation), laboratory tests, and diagnostic procedures to summarize the patient's situation by connecting features of body structure, function, activity, and participation to develop a plan of care.

## **Patient History**

In addition to the general chart review presented in Chapter 2, the following pertinent information about patients with cardiac dysfunction should be obtained before physical examination<sup>4,12–14</sup>:

• Presence of chest pain (see Chapter 17 for an expanded description of characteristics and etiology of chest pain)

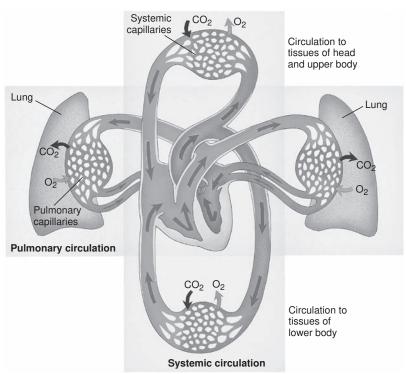


FIG. 3.4
Schematic of systemic circulation. (From Thibodeau GA. Structure and Function of the Body. 13th ed. St. Louis: Mosby; 2007.)

- Location and radiation
- Character and quality (crushing, burning, numbing, hot)
- Angina equivalents (what the patient feels as angina [e.g., jaw pain, shortness of breath, dizziness, lightheadedness, diaphoresis, burping, nausea, or any combination of these])
- Aggravating factors: Activity, recreational drugs
- Alleviating factors: Pain stops when activity ceases, relieved by sitting forward or resting
- Precipitating factors
- Medical treatment sought and its outcome
- Presence of palpitations
- Presence of cardiac risk factors (Box 3.1)
- Family history of cardiac disease
- History of dizziness or syncope
- Previous myocardial infarction (MI), cardiac studies, or procedures
- Medications ordered: Nitroglyercin, beta-blockers, calcium channel blockers, antihypertensive medications, antiplatelet therapy

# **CLINICAL TIP**

When discussing angina with a patient, use the patient's terminology. If the patient describes the angina as "crushing" pain, ask the patient if he or she experiences the crushing feeling during treatment as opposed to asking the patient if he or she has chest pain. The common medical record abbreviation for chest pain is CP.

# **Physical Examination**

#### Observation

Key components of the observation portion of the physical examination include the following<sup>4,8</sup>:

- Facial color, skin color and tone, or the presence of diaphoresis
- Obvious signs of edema, discoloration in the extremities
- Respiratory rate
- Signs of trauma (e.g., paddle burns or ecchymosis from cardiopulmonary resuscitation)
- Presence of jugular venous distention (JVD), which results from the backup of fluid into the venous system from rightsided congestive heart failure (CHF) (Fig. 3.5)
- Make sure the patient is in a semirecumbent position (45 degrees).
- Have the patient turn his or her head away from the side being evaluated.
- Observe pulsations in the internal jugular neck region. Pulsations are normally seen 3 to 5 cm above the sternum. Pulsations higher than this or absent pulsations indicate JVD.

#### **Palpation**

Palpation is the second component of the physical examination and is used to evaluate and identify the following:

- Pulses for circulation quality, HR, and rhythm (Table 3.4, Fig. 3.6)
- Extremities for pitting edema bilaterally (Table 3.5)

Major Independent Risk Factors	Predisposing Risk Factors	Conditional Risk Factors	
Smoking	Physical inactivity	Elevated triglycerides	
Hypertension	Obesity	Small LDL particles	
Elevated serum cholesterol, total (and LDL)	Body mass index >30 kg/m <sup>2</sup>	Elevated homocysteine	
Decreased HDL cholesterol	Abdominal obesity (waist-hip ratio)	Elevated lipoprotein (a)	
Diabetes mellitus	Men >40 inches	Elevated inflammatory markers	
Advancing age	Women >35 inches	C-reactive protein	
	Family history of premature heart disease	Fibrinogen	
	Psychosocial factors		
	Job strain		
	Ethnic characteristics		

 $LDL, \ Low-density \ lipoprotein; HDL, \ high-density \ lipoprotein.$ 

Data from Grundy SM, Pasternak R, Greenland P, et al. Assessment of cardiovascular risk by use of multiple-risk-factor assessment equations: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. *Circulation*. 1999;100:1481-1492; Belkic KL, Landsbergis PA, Schnall PL, Baker D. Is job strain a major source of cardiovascular disease risk? *Scand J Work Environ Health*. 2004;30(2):85-128.

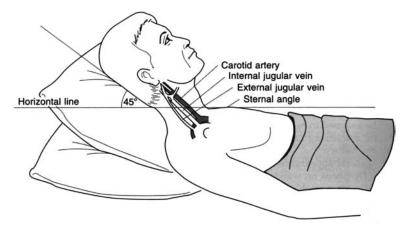


FIG. 3.5

**TABLE 3.4 Pulse Amplitude Classification and Pulse Abnormalities** 

#### **Pulse Amplitude Classification** Scale Degree Description 0 Absent pulse No pulse—no circulation 1+ Diminished pulse Reduced stroke volume and ejection fraction, increased vascular 2+ Normal pulse Normal resting conditions, no pathologies 3+ Moderately increased Slightly increased stroke volume and ejection fraction 4+ Markedly increased (bounding)<sup>a</sup> Increased stroke volume and ejection fraction, can be diminished with vasoconstriction

#### **Pulse Abnormalities**

Abnormality	Palpation	Description
Pulsus alternans	Regular rhythm with strong pulse waves alternating with weak pulse waves	Indicates left ventricular failure when present at normal heart rates
Bigeminal pulses	Every other pulse is weak and early	Result of premature ventricular contractions (bigeminy)
Pulsus paradoxus	Reduction in strength of the pulse with an abnormal decline in blood pressure during inspiration	May be caused by chronic obstructive lung disease, pericarditis, pulmonary emboli, restrictive cardiomyopathy, and cardiogenic shock

<sup>&</sup>lt;sup>a</sup>Corrigan's pulse is a bounding pulse visible in the carotid artery that occurs with aortic regurgitation.

Data from Woods SL, Sivarajian-Froelicher ES, Underhill-Motzer S, eds. Cardiac Nursing. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2000.

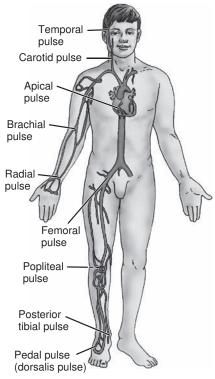


FIG. 3.6
Arterial pulses. (From Pierson FM. *Principles and Techniques of Patient Care.* 4th ed. St. Louis: Saunders; 2008.)

When palpating HR, counting the pulse rate for 15 seconds and multiplying by 4 is sufficient with normal rates and rhythms. If rates are faster than 100 bpm or slower than 60 bpm, palpate the pulse for 60 seconds. If the rhythm is irregularly

TABLE 3.5 Pitting Edema Sca
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Scale	Degree	Description
1 + Trace	Slight	Barely perceptible depression
2+ Mild	0–0.6 cm	Easily identified depression (EID) (skin rebounds in <15 sec)
3+ Moderate	0.6–1.3 cm	EID (rebound 15-30 sec)
4+ Severe	1.3–2.5 cm	EID (rebound >30 sec)

Data from Woods SL, Sivarajian Froelicher ES, Underhill-Motzer S, eds. *Cardiac Nursing*. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2000; Hillegass E. *Essentials of Cardiopulmonary Physical Therapy*. 4th ed. St. Louis: Elsevier; 2017.

irregular (e.g., during atrial fibrillation) or regularly irregular (e.g., premature ventricular contractions [PVCs]), perform auscultation of heart sounds to identify the apical HR for a full minute. In these cases, palpation of pulse cannot substitute for ECG analysis to monitor the patient's rhythm, but it may alert the therapist to the onset of these abnormalities.

#### **CLINICAL TIP**

Use caution in palpating pulses because manual pressure on the carotid sinus may cause baroreflex drops in heart rate (HR), blood pressure (BP), or both.

#### **Blood Pressure**

BP measurement with a sphygmomanometer (cuff) and auscultation is an indirect, noninvasive measurement of the force exerted against the arterial walls during ventricular systole (systolic blood pressure [SBP]) and during ventricular

<b>TABLE 3.6</b>	Normal	Rlood	Pressure	Randes
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Age Ranges	Systolic	Diastolic
Age 8 years	85–114 mmHg	52–85 mmHg
Age 12 years	95–135 mmHg	58-88 mmHg
Adult	<120 mmHg	<80 mmHg
Elevated	120–129 mmHg	<80 mmHg
Hypertension		
Stage 1	130–139 mmHg	80-89 mmHg
Stage 2	≥140 mmHg	≥90 mmHg
Normal exercise	Increases 5–12 mmHg per MET increase in workload	±g mmHg

MET, Metabolic equivalent.

Data from Wright JD, Hughes JP, Ostchega Y, Yoon SS, Nwankwo T. Mean systolic and diastolic blood pressure in adults aged 18 and over in the United States, 2001-2008. *Natl Health Stat Rep.* 2011(35):1-22, 24; Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol.* 2018;138:e426-e483; American College of Sports Medicine, Armstrong LE, et al. ACSM's Guidelines for Exercise Testing and Prescription. Philadelphia: Lippincott Williams & Wilkins; 2005.

diastole (diastolic blood pressure [DBP]). BP is affected by total peripheral resistance (blood volume and elasticity of arterial walls) and CO. Table 3.6 lists normal BP ranges. Occasionally, BP measurements can be performed only on certain limbs secondary to the presence of such conditions as a percutaneously inserted central catheter, arteriovenous fistula for hemodialysis, blood clots, scarring from brachial artery cutdowns, or lymphedema (e.g., status post mastectomy). BP of the upper extremity should be measured in the following manner:

- Check for posted signs, if any, at the bedside that indicate which arm should be used in taking BP. BP variations of 5 to 10 mmHg between the right and left upper extremity are considered normal. Patients with arterial compression or obstruction may have differences of more than 10 to 15 mmHg.<sup>14</sup>
- 2. Use a properly fitting cuff. The inflatable bladder should have a width of approximately 40% and length of approximately 80% of the upper arm circumference.<sup>15</sup>
- 3. Position the cuff 2.5 cm above the antecubital crease.
- 4. Rest the relaxed arm at the level of the heart.
- 5. To determine how high to inflate the cuff, palpate the radial pulse, inflate until no longer palpable, and note the cuff inflation pressure. Deflate the cuff.
- 6. Place the bell of the stethoscope gently over the brachial artery.
- 7. Reinflate the cuff to 30 to 40 mmHg greater than the value in step 5. Then slowly deflate the cuff. Cuff deflation should occur at approximately 2 to 3 mmHg per second.<sup>15</sup>
- 8. Listen for the onset of tapping sounds, which represents blood flow returning to the brachial artery. This is the SBP.
- 9. As the pressure approaches diastolic pressure, the sounds

will become muffled and in 5 to 10 mmHg will be completely absent. These sounds are referred to as *Korotkoff* sounds (Table 3.7).<sup>14,15</sup>



In situations when it is difficult to auscultate or discern a distinct diastolic pressure, the patient's blood pressure may be noted as systolic BP/P (i.e., "BP is 90 over palp") or systolic BP over 2 diastolic pressures (e.g., 140/85/62) denoting the onset of muffling sounds and the disappearance of sounds.<sup>13</sup>

#### **Physical Therapy Considerations**

- Recording preexertion, paraexertion, and postexertion BP is
  important for identification of BP responses to activity. During recovery from exercise, blood vessels dilate to allow for
  greater blood flow to muscles. In cardiac-compromised or
  very deconditioned individuals, total CO may be unable to
  support this increased flow to the muscles and may lead to
  decreased output to vital areas, such as the brain.
- If you are unable to obtain BP on the arm, the thigh is an appropriate alternative, with auscultation at the popliteal artery.
- Falsely high readings occur if the cuff is too small or applied loosely or if the brachial artery is lower than the heart level.
- Evaluation of BP and HR in different postures can be used to monitor orthostatic hypotension with repeat measurements on the same arm 1 to 5 minutes after position changes. The symbols that represent patient position are shown in Fig. 3.7.
- The same extremity should be used when serial BP recordings will be compared for an evaluation of hemodynamic response.
- A BP record is kept on the patient's vital sign flow sheet.
   This is a good place to check for BP trends throughout the day and, depending on your hospital's policy, to document BP changes during the therapy session.
- An auscultatory gap is the disappearance of sounds between phase 1 and phase 2 and is common in patients with high BP, venous distention, and severe aortic stenosis. Its presence can create falsely low SBPs if the cuff is not inflated enough (prevented by palpating for the disappearance of the pulse before measurement), or falsely high DBPs if the therapist stops measurement during the gap (prevented by listening for the phase 3 to phase 5 transitions).<sup>13</sup>

#### Auscultation

Evaluation of heart sounds can yield information about the patient's condition and tolerance to medical treatment and physical therapy through the evaluation of valvular function, rate, rhythm, valvular compliance, and ventricular compliance.<sup>4</sup> To listen to heart sounds, a stethoscope with a bell and a diaphragm is necessary. For a review of normal and abnormal heart sounds, refer to Table 3.8. The examination should follow a systematic pattern using the bell (for low-pitched sounds) and diaphragm (for high-pitched sounds) and should cover all

TABLE 3.7	Korotkoff	Sounds
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Phase	Sound	Indicates
1	First sound heard, faint tapping sound with increasing intensity or sharp "thud" heard	Systolic pressure (blood starts to flow through compressed artery)
2	Start blowing or swishing sound	Blood flow continues to be heard; sounds are beginning to change because of the changing compression on the artery
3	Sounds increase in intensity with a distinct tapping	Blood flow is increasing as artery compression is decreasing
4	Sounds become muffled	Diastolic pressure in children <13 years of age and in adults who are exercising, pregnant, or hyperthyroid (see phase 5)
5	Disappearance	Diastolic pressure in adults—occurs 5–10 mmHg below phase 4 in normal adults. In states of increased rate of blood flow, it may be >10 mmHg below phase 4. In these cases, the phase 4 sound should be used as diastolic pressure in adults

Data from Woods SL, Sivarajian-Froelicher ES, Underhill-Motzer S, eds. Cardiac Nursing. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2000; Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking. Philadelphia: Lippincott Williams & Wilkins; 2003; Ball JW, Dains JE, Flynn JA, Solomon BS, Stewart RW. Seidel's Guide to Physical Examination: An Interprofessional Approach. 9th ed. St. Louis: Elsevier; 2019.

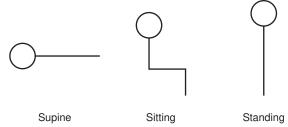


FIG. 3.7 Orthostatic blood pressure symbols.

auscultatory areas, as illustrated in Fig. 3.8. Abnormal sounds should be noted with a description of the conditions in which they were heard (e.g., after exercise or during exercise).

#### **Physical Therapy Considerations**

- Always ensure proper function of a stethoscope by tapping the diaphragm before use with a patient.
- Avoid rubbing any part of the stethoscope on extraneous objects because this can add noise and detract from the examination.
- Avoid auscultation of heart sounds over clothing, which can muffle the intensity of normal and abnormal sounds.
- If the patient has an irregular cardiac rhythm, determine HR through auscultation (apical HR). To save time, listen for the HR during a routine auscultatory examination with the stethoscope's bell or diaphragm in any of the auscultation locations (see Fig. 3.8).
- Heart sounds can be heard online at the Auscultation Assistant, available at: http://www.med.ucla.edu/wilkes/intro.html.

# **Diagnostic and Laboratory Measures**

The diagnostic and laboratory measures discussed in this section provide information used to determine medical diagnoses, guide interventions, and assist with determining prognoses. The clinical relevance of each test varies according to the pathology. This section is organized across

a spectrum—from the least invasive to the most invasive measures. When appropriate, detailed information for test results most pertinent to the physical therapist are provided. For clinical decision making, physical therapists usually need information that helps identify indications for intervention, relative or absolute contraindications for intervention, possible complications during activity progression, and indicators of performance. Box 3.2 provides the clinician with important information to obtain from the medical record before patient encounter. In addition, the clinician should review the radiographic report, echocardiographic report and other specialized tests to obtain valuable knowledge of the patient's cardiovascular status.

#### Oximetry

Oximetry (SaO<sub>2</sub>) is used to indirectly evaluate the oxygenation of a patient and can be used to titrate supplemental oxygen. Refer to Chapter 4 for a further description of oximetry.

#### Electrocardiography

ECG provides a graphic analysis of the heart's electrical activity. ECG commonly is used to detect arrhythmias, heart blocks, and myocardial perfusion. It also can detect atrial or ventricular enlargement. ECG used for continuous monitoring of patients in the hospital typically involves a 3- to 5-lead system. A lead represents a particular portion, or "view," of the heart. The patient's rhythm usually is displayed in his or her room, in the hall, and at the nurses' station. Diagnostic ECG involves a 12-lead analysis, the description of which is beyond the scope of this book. For a review of basic ECG rate and rhythm analysis, refer to Table 3.9 and Fig. 3.3.

**Holter and Event Monitoring.** *Holter monitoring* refers to 24- or 48-hour ECG analysis conducted to detect cardiac arrhythmias and corresponding symptoms during a patient's daily activity. <sup>14</sup> Holter monitoring is different from telemetric monitoring because the ECG signal is recorded and analyzed later.

**CHAPTER 3** 

TABLE 3.8 Normal and Abnormal Heart Sounds			
Sound	Location	Description	
S1 (normal)	All areas	First heart sound; signifies closure of atrioventricular valves and corresponds to onset of ventricular systole	
S2 (normal)	All areas	Second heart sound; signifies closure of semilunar valves and corresponds with onset of ventricular diastole	
S3 (abnormal)	Best appreciated at apex	Immediately after S2; occurs early in diastole and represents filling of the ventricle. In young, healthy individuals, it is considered normal and called a <i>physiologic third sound</i> . In the presence of heart disease, it results from decreased ventricular compliance (a classic sign of congestive heart failure)	
S4 (abnormal)	Best appreciated at apex	Immediately preceding S1; occurs late in ventricular diastole; associated with increased resistance to ventricular filling; common in patients with hypertensive heart disease, coronary heart disease, pulmonary disease, or myocardial infarction, or after coronary artery bypass grafts	
Murmur (abnormal)	Over respective valves	Indicates regurgitation of blood through valves; can also be classified as systolic or diastolic murmurs. Common pathologies resulting in murmurs include mitral regurgitation and aortic stenosis	
Pericardial friction rub (abnormal)	Third or fourth intercostal space, anterior axillary line	Sign of pericardial inflammation (pericarditis), associated with each beat of the heart; sounds like a creak or leather being rubbed together	

Data from Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking. Philadelphia: Lippincott Williams & Wilkins; 2003.

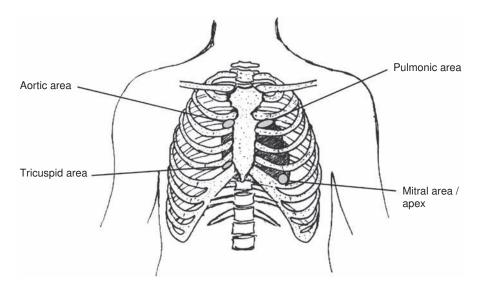


FIG. 3.8 Areas for heart sound auscultation. (Courtesy Barbara Cocanour, PhD, Department of Physical Therapy, University of Massachusetts, Lowell, MA.)

Indications for Holter monitoring include the evaluation of syncope, dizziness, shortness of breath with no other obvious cause, palpitations, antiarrhythmia therapy, pacemaker functioning, activity-induced silent ischemia, and risk of cardiac complications with the use of HRV.

Event monitoring refers to extended ECG monitoring, with recordings saved only when an event is either automatically identified (device algorithms) or self-identified by the patient.

Heart Rate Variability. HRV has been discussed in the literature to possibly reflect cardiac autonomic nervous system regulation. A common overall measure of HRV is the standard deviation of all RR intervals on ECG during a 24-hour period (SDNN).9 Evidence regarding the potential clinical utility of HRV for cardiology is growing; however, HRV continues to be used primarily in research. 16 In healthy populations, low HRV is a risk factor for all causes of cardiac mortality<sup>17–19</sup> and for new onset of hypertension.<sup>20</sup> Low HRV is also a risk for mortality in patients who have had an MI<sup>21-23</sup> or have coronary artery disease<sup>24</sup> or CHF.<sup>25</sup> Evidence suggests that altered HRV during exercise may provide valuable information for risk assessment.<sup>26</sup> This is likely related to the increasingly accepted relationship between delayed heart rate recovery (HRR) after exercise and cardiac risk. Delayed HRR (<46 bpm) 3 minutes after an exercise test is a predictor of long-term (≈15 years) mortality.<sup>27</sup> HRR is discussed further in the section on Physical Therapy Intervention.

Telemetric Electrocardiography Monitoring. Telemetric ECG monitoring provides real-time ECG visualization via radiofrequency transmission of the ECG signal to a monitor. Benefits of telemetry include monitoring with no hardwire

BOX 3.2 Medical Chart Review			
Medical Test	Rationale for Use of the Test	Interpretation of the Test	
B-type natriuretic peptide (BNP)	Assesses biomarkers of heart failure, including natriuretic peptides that include BNP and N-terminal pro-BNP (NT-proBNP). <sup>6</sup>	Normal levels of BNP are less than 100 pg/mL. Values above 500 are generally considered to be positive for heart failure. BNP has been found to be a statistically significant ( $P < 0.05$ ) prognostic indicator of heart failure, and studies have discovered moderate to strong correlations between BNP and peak oxygen uptake ( $VO_{2max}$ )	
Blood urea nitrogen (BUN), Plasma creatinine levels, and urine output	Assesses renal function, which may be reduced in patients with HF.	Normal BUN: 6–30 mg/dL Normal creatinine: 0.5–2.0 mg/dL Values above normal ranges indicate renal failure Oliguria <400 mL of urine in 24 hours Anuria: <100 mL of urine in 24 hours	
Hemoglobin and hemato- crit levels	Assess the oxygen carrying capacity within the system.	Normal hemoglobin 12–14 gm/ $100$ mL of blood in adult males and $14$ – $16$ g/ $100$ mL of blood in adult women Normal hematocrit: $45\%$ men; $40\%$ women	
Creatine kinase—myocardial band CK-MB index	Creatine kinase MB subunit (CK-MB), an isoenzyme, is released into blood and elevates with an acute MI. <sup>8</sup>	Normal level: 0%–3% Minor cardiac dysfunction: 5% Major cardiac dysfunction: 10% Peak Levels: 14–36 hours	
Troponin I	Troponin I is the most frequently used marker to assess the presence of an acute MI. <sup>8</sup> Troponin I is an isotype found exclusively in the myocardium and is therefore 100% cardiac specific.	Normal level: 0–0.2 mcg/mL Minor cardiac dysfunction: 5 mcg/mL Major cardiac dysfunction: 10 mcg/mL Peak levels: 24–36 hours	
C-reactive protein (CRP)	CRP is a test that measures the amount of a protein in the blood that signals acute inflammation. To determine a person's risk for heart disease, a more sensitive CRP test called a high-sensitivity C-reactive protein (hs-CRP) assay is available.	hs-CRP lower than 1.0 mg/L indicates a low risk of developing cardiovascular disease hs-CRP between 1 and 3 mg/L indicates an average risk of developing cardiovascular disease hs-CRP higher than 3.0 mg/L indicates a high risk of developing cardiovascular disease	
Electrolyte assessment	Appropriate levels of potassium, calcium, and magnesium allow for normal electrical conduction through the heart.	Hypokalaemia, low potassium (usually <3.5 mEq/L), produces arrhythmias with flattened T waves and depressed ST segments, as well as bilateral lower extremity muscle cramping. An inverted U-wave may also be noticed on the electrocardiogram. Hypocalcaemia (low blood serum calcium levels) and hypomagnesaemia (low magnesium in blood) have the potential of increasing ventricular ectopy within the heart	

 $Data\ from:\ Academy\ of\ Acute\ Care\ Physical\ The rapy.\ \textit{Laboratory}\ \textit{Values}\ \textit{Interpretation}\ \textit{Resource}.\ 2017\ (Reference\ \#:\ C)$ 

TABLE 3.9	Electrocardiograph	Interpretation
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Waya/Codmont	Duration (seconds)	Amulituda (mm)	Indicatos
Wave/Segment	Duration (seconds)	Amplitude (mm)	Indicates
P wave	< 0.10	1–3	Atrial depolarization
PR interval	0.12–0.20	Isoelectric line	Elapsed time between atrial depolarization and ventricular depolarization
QRS complex	0.06–0.10	25–30 (maximum)	Ventricular depolarization and atrial repolarization
ST segment	0.12	−½ to +1	Elapsed time between end of ventricular depolarization and beginning of repolarization
QT interval (QTc)	0.42–0.47	Varies	Elapsed time between beginning of ventricular repolarization and end of repolarization (QTc is corrected for heart rate)
T wave	0.16	5–10	Ventricular repolarization

connection between the patient and the visual display unit and real-time graphic display of the ECG signal using the standard ECG monitor attachment.

# **CLINICAL TIP**

Some hospitals use an activity log with Holter monitoring. If so, document physical therapy intervention on the log. If no log is available, record the time of day and physical therapy intervention in the medical record.

#### **Complete Blood Cell Count**

Relevant values from the complete blood cell count are hematocrit, hemoglobin, and white blood cell counts (see Box 3.2). Hematocrit refers to the number of red blood cells per 100 mL of blood and therefore fluctuates with changes in the total red blood cell count (hemoglobin) and with blood volume (i.e., reduced plasma volume results in relatively more red blood cells in 100 mL of blood). Elevated levels of hematocrit (which may be related to dehydration) indicate increased viscosity of blood that can potentially impede blood flow to tissues. 14 Hemoglobin is essential for the adequate oxygen-carrying capacity of the blood. A decrease in hemoglobin and hematocrit levels (10% below normal is called anemia) may decrease activity tolerance or make patients more susceptible to ischemia secondary to decreased oxygen-carrying capacity. 13,28 Slight decreases in hematocrit resulting from adaptations to exercise (with no change in hemoglobin) are related to increases in blood volume. The concomitant exercise-related decreases in blood viscosity may be beneficial to post-MI patients.<sup>29</sup>

Elevated white blood cell counts can indicate that the body is fighting infection, or they can occur with inflammation caused by cell death, such as in MI. Erythrocyte sedimentation rate (ESR), another hematologic test, is a nonspecific index of inflammation and commonly is elevated for 2 to 3 weeks after MI.<sup>28</sup> Refer to Chapter 7 for more information about these values.

#### **Coagulation Profiles**

Coagulation profiles provide information about the clotting time of blood. Patients who undergo treatment with thrombolytic therapy after the initial stages of MI or who are receiving anticoagulant therapy because of various cardiac arrhythmias require coagulation profiles to monitor anticoagulation in an attempt to prevent complications, such as bleeding. The physician determines the patient's therapeutic range of anticoagulation using prothrombin time (PT), partial thromboplastin time (PTT), and international normalized ratio (INR).<sup>28</sup> Refer to Chapter 7 for details regarding these values and their significance to treatment.

The normal levels for PT-INR is 0.8 to 1.2.30 For patients on heparin, the activated PTT is more appropriate to evaluate. Normal PTT range is 21 to 35 seconds. Typically therapeutic ranges for effectiveness of the anticoagulant is 2 to 2.5 times normal range (60-109 seconds) with variability based on the reagent utilized.<sup>30</sup> Patients with low INR and activated partial

thromboplastin time (aPTT) are at higher risk of thrombosis, especially if they have arrhythmias (e.g., atrial fibrillation) or valvular conditions (e.g., mitral regurgitation) that produce stasis of the blood. Patients values greater than the therapeutic range are at increased risk for bleeding and therefore should be carefully evaluated for balance and fall risk before mobilization.<sup>28</sup>

#### **Blood Lipids**

Elevated total cholesterol levels in blood are a significant risk factor for atherosclerosis and therefore ischemic heart disease.<sup>31</sup> Measuring blood cholesterol level is necessary to determine the risk for development of atherosclerosis and to assist in patient education, dietary modification, and medical management. Normal values can be adjusted for age; however, levels of total cholesterol greater than 240 mg/dL are generally considered high, and levels of less than 200 mg/dL are considered normal.

A blood lipid analysis categorizes cholesterol into high-density lipoproteins (HDLs) and low-density lipoproteins (LDLs) and provides an analysis of triglycerides. HDLs are formed by the liver and are considered beneficial because they are readily transportable and do not adhere to the intimal walls of the vascular system. People with higher amounts of HDLs are at lower risk for coronary artery disease. 28,31 HDL levels of less than 33 mg/dL carry an elevated risk of heart disease. A more important risk for heart disease is an elevated ratio of total cholesterol to HDL. Normal ratios of total cholesterol to HDL range from 3 to 5.14

LDLs are formed by a diet excessive in fat and are related to a higher incidence of coronary artery disease. LDLs are not as readily transportable as HDLs because LDLs adhere to intimal walls in the vascular system.<sup>28</sup> Normal LDL levels are below 100 mg/dL.<sup>14</sup>

Triglycerides are fat cells that are free floating in blood. When not in use, they are stored in adipose tissue. A person's triglyceride levels increase after he or she eats foods high in fat and decrease with exercise. High levels of triglycerides are associated with a risk of coronary heart disease.<sup>28</sup>

#### **CLINICAL TIP**

Cholesterol levels may be elevated falsely after an acute myocardial infarction (MI); therefore preinfarction levels (if known) are used to guide risk factor modification. Values will not return to normal until at least 6 weeks after MI.

#### C-Reactive Protein

C-reactive protein (CRP) is a test that measures the amount of a protein in the blood that signals acute inflammation. To determine a person's risk for heart disease, a more sensitive CRP test, called high-sensitivity C-reactive protein (hs-CRP) assay, is available. A growing number of studies have determined that high levels of hs-CRP consistently predict recurrent coronary events in patients with unstable angina (USA) and acute MI. In addition, elevated hs-CRP levels are associated with lower survival rates in these patients with cardiovascular disease (see Box 3.2).32-34

#### **Biochemical Markers**

After an initial myocardial insult, the presence of tissue necrosis can be determined by increased levels of biochemical markers. Levels of biochemical markers, such as serum enzymes (creatine kinase [CK]) and proteins (troponin I and T), also can be used to determine the extent of myocardial death and the effectiveness of reperfusion therapy. In patients presenting with specific anginal symptoms and diagnostic ECG, these biochemical markers assist with confirmation of the diagnosis of an MI (refer to Box 3.2, Medical Chart Review). Enzymes play a more essential role in the medical assessment of many patients with nonspecific or vague symptoms and inconclusive ECG changes. Such analysis also includes evaluation of isoenzyme levels. Isoenzymes are different chemical forms of the same enzyme that are tissue specific and allow differentiation of damaged tissue (e.g., skeletal muscle versus cardiac muscle).

CK (formally called creatine phosphokinase) is released after cell injury or cell death. CK has three isoenzymes. The CK-MB isoenzyme is related to cardiac muscle cell injury or death. The most widely used value is the CK-MB relative index calculated as 100% (CK-MB/total CK).<sup>35</sup> Temporal measurements of the CK-MB relative index help physicians diagnose MI, estimate the size of infarction, and evaluate the occurrence of reperfusion and possible infarct extension. An early CK-MB peak with rapid clearance is a good indication of reperfusion. 14 Values may increase from skeletal muscle trauma, cardiopulmonary resuscitation (CPR), defibrillation, and open-heart surgery. Coronary artery bypass surgery tends to elevate CK-MB levels secondary to the cross-clamp time in the procedure. Early postoperative peaks and rapid clearance seem to indicate reversible damage, whereas later peaks and longer clearance times with peak values exceeding 50 U/L may indicate an MI.14 Treatment with thrombolytic therapy, such as streptokinase or a tissue plasminogen activator (tPa), has been shown to falsely elevate the values and may create a second peak of CK-MB, which strongly suggests successful reperfusion. 14,35

Troponins are essential contractile proteins found in skeletal and cardiac muscles. Troponin I is an isotype found exclusively in the myocardium and is therefore 100% cardiac specific. Troponin T, another isotype, is sensitive to cardiac damage, but its levels also rise with muscle and renal failure.<sup>35</sup> These markers have emerged as sensitive and cardiac-specific clinical indicators for the diagnosis of MI and for risk stratification.

# **SECTION CAL TIP**

Wait for the final diagnosis of location, size, and type of myocardial infarction (MI) before beginning active physical therapy intervention. This allows for compete information from the medical team for the therapist's clinical decision making and decreases risk of harm. Withhold physical therapy geared toward testing functional capacity or increasing the patient's activity until cardiac enzyme levels have peaked and begin to fall.

#### **Natriuretic Peptides**

Three natriuretic peptides have been identified in humans. These include atrial natriuretic peptide (ANP), B-type natriuretic

peptide (BNP), and C-type natriuretic peptide (CNP).<sup>5</sup> ANP is stored in the right atrium and released in response to increased atrial pressures. BNP is stored in the ventricles and released in response to increased ventricular distending pressures. ANP and BNP cause vasodilatation and natriuresis and counteract the water-retaining effects of the adrenergic and renin angiotensin system. CNP is located primarily in the vasculature. The physiologic role of CNP has not been clarified yet.

Circulating levels of ANP, BNP, and N-terminal (NT)–proBNP (a variation of BNP) are elevated in plasma in patients with heart failure (HF). In normal human hearts, ANP predominates in the atria, with a low-level expression of BNP and CNP. Patients with heart failure demonstrate an unchanged content of ANP in the atria, with a marked increase in the concentrations of BNP and NT-proBNP. The 2017 American Heart Association (AHA)/American College of Cardiology (ACC) guidelines provide a Class I recommendation (Level of Evidence: A) for measurement of BNP or NT-proBNP for establishing prognosis or disease severity in chronic HF.<sup>37</sup> These natriuretic peptide biomarkers in patients assist in the diagnosis and exclusion of HF in those patients presenting with dyspnea.

No level of BNP perfectly distinguishes patients with HF and those without HF. Normal levels include BNP less than 100 pg/mL (see Box 3.2).<sup>5,38</sup> Research has indicated a correlation between circulating BNP concentrations and the severity of CHF, based on the New York Heart Association (NYHA) functional classification system. Values between 100 and 300 pg/mL have been shown to be correlated with NYHA Class I; values greater than 300 pg/mL are associated with Class II heart failure; and values greater than 600 pg/mL and greater than 900 pg/mL correlate with NYHA Class III and IV, respectively.<sup>30</sup>

#### **Arterial Blood Gas Measurements**

Arterial blood gas measurement may be used to evaluate the oxygenation (PaO<sub>2</sub>), ventilation (PaCO<sub>2</sub>), and pH in patients during acute MI and exacerbations of CHF in certain situations (i.e., obvious tachypnea, low SaO<sub>2</sub>). These evaluations can help determine the need for supplemental oxygen therapy and mechanical ventilatory support in these patients. Oxygen is the first drug provided during a suspected MI. Refer to Chapters 4 and 18 for further description of arterial blood gas interpretation and supplemental oxygen, respectively.

#### **Chest Radiography**

Chest radiography ("x-ray") can be ordered for patients to assist in the diagnosis of CHF or cardiomegaly (enlarged heart). Patients in CHF have an increased density in pulmonary vasculature markings, giving the appearance of congestion in the vessels. <sup>4,8</sup> Refer to Chapter 4 for further description of chest radiography.

#### **Echocardiography**

Transthoracic echocardiography (TTE), or "cardiac echo," is a noninvasive procedure that uses ultrasound to evaluate the function of the heart. Evaluation includes the size of the ventricular cavity, the thickness and integrity of the septum, valve Transesophageal echocardiography (TEE) is an approach to echocardiography that provides a better view of the mediastinum in cases of pulmonary disease, chest wall abnormality, and obesity, which make standard echocardiography difficult. <sup>14,39</sup> For this test, the oropharynx is anesthetized, and the patient is given enough sedation to be relaxed but still awake because he or she needs to cooperate by swallowing the catheter. The catheter, a piezoelectric crystal mounted on an endoscope, is passed into the esophagus. Specific indications for TEE include bacterial endocarditis, aortic dissection, regurgitation through or around a prosthetic mitral or tricuspid valve, left atrial thrombus, intracardiac source of an embolus, and interarterial septal defect. Patients usually are required to fast for at least 4 hours before the procedure. <sup>39</sup>

Principal indications for echocardiography are to assist in the diagnosis of pericardial effusion, cardiac tamponade, idiopathic or hypertrophic cardiomyopathy, a variety of valvular diseases, intracardiac masses, ischemic cardiac muscle, left ventricular aneurysm, ventricular thrombi, and a variety of congenital heart diseases. <sup>14</sup>

TTE also can be performed during or immediately after bicycle or treadmill exercise to identify ischemia-induced wall motion abnormalities or during a pharmacologically induced exercise stress test (e.g., dobutamine stress echocardiography [DSE]). This stress echocardiography adds to the information obtained from standard stress tests (ECGs) and may be used as an alternative to nuclear scanning procedures. Transient depression of wall motion during or after stress suggests ischemia. 40

**Contrast Echocardiography.** The ability of echocardiography to diagnose perfusion abnormalities and myocardial chambers is improved by using an intravenously injected contrast agent. The contrast allows for greater visualization of wall motion and wall thickness and calculation of EE.<sup>41</sup>

**Dobutamine Stress Echocardiography.** Dobutamine is a potent alpha-1 ( $\alpha_1$ ) agonist and a beta-receptor agonist with prominent inotropic and less prominent chronotropic effects on the myocardium. Dobutamine (which, unlike dipyridamole (Persantine; see Persantine Thallium Stress Testing in this chapter), increases contractility, HR, and BP in a manner similar to exercise) is injected in high doses into subjects as an alternative to exercise. 40 Dobutamine infusion is increased in a stepwise fashion similar to an exercise protocol. The initial infusion is 0.01 mg/kg and is increased 0.01 mg/kg every 3 minutes until a maximum infusion of 0.04 mg/kg is reached. Typically the echocardiographic image of wall motion is obtained during the final minute(s) of infusion. This image can then be compared with baseline recordings. 40 If needed, atropine occasionally is added to facilitate a greater HR response for the test. 40 Low-dose DSE has the capacity to evaluate the contractile response of the impaired myocardium. Bellardinelli et al.<sup>42</sup> have demonstrated that improvements in functional capacity after exercise can be predicted by low-dose DSE. Patients with a positive contractile response to dobutamine were more likely to increase their VO<sub>2max</sub> after a 10-week exercise program. Having a positive contractile response on the low-dose DSE had a positive predictive value of 84% and a negative predictive value of 59%.  $^{42}$ 

As this study indicates, research has demonstrated the prognostic value of certain medical tests for determining functional prognosis. Therefore physical therapists must be prepared to assess this area of literature critically to assist the medical team in determining the level of rehabilitative care for a patient during recovery.

#### **Exercise Testing**

Exercise testing, or stress testing, is a noninvasive method of assessing cardiovascular responses to increased activity. The use of exercise testing in cardiac patients can serve multiple purposes, which are not mutually exclusive. The most widespread use of exercise testing is as a diagnostic tool for the presence of coronary artery disease. Other uses include determination of prognosis and severity of disease, evaluation of the effectiveness of treatment, early detection of labile hypertension, evaluation of CHF, evaluation of arrhythmias, and evaluation of functional capacity. 40 Exercise testing involves the systematic and progressive increase in intensity of activity (e.g., treadmill walking, bicycling, stair climbing, arm ergometry). These tests are accompanied by simultaneous ECG analysis, BP measurements, and subjective reports, commonly using Borg's Rating of Perceived Exertion (RPE). 43,44 Occasionally, the use of expired gas analysis can provide useful information about pulmonary function and maximal oxygen consumption. 40 Submaximal tests, such as the 12- and 6-minute walk tests, can be performed to assess a patient's function. For further discussion of the 6-minute walk test, refer to Chapter 23.

Submaximal tests differ from maximal tests in that the patient is not pushed to his or her maximum HR; instead the test is terminated at a predetermined end point, usually at 75% of the patient's predicted maximum HR.<sup>45</sup> For a comparison of two widely used exercise test protocols and functional activities, refer to Table 3.10. For a more thorough description of submaximal exercise testing, the reader is referred to the report of Noonan and Dean.<sup>45</sup>

Contraindications to exercise testing include the following 46:

- Recent MI (<48 hours earlier)
- · Acute pericarditis
- Unstable angina
- Ventricular or rapid arrhythmias
- Untreated second- or third-degree heart block
- Decompensated CHF
- Acute illness

Exercise test results can be used for the design of an exercise prescription. On the basis of the results, the patient's actual or extrapolated maximum HR can be used to determine the patient's target HR range and safe activity intensity. RPE with symptoms during the exercise test also can be used to gauge exercise or activity intensity, especially in subjects on betablockers. (Refer to the Physical Therapy Intervention section for a discussion on the use of RPE.)

Any walk test that includes measurement of distance and time can be used to estimate metabolic equivalents of task (METs) and oxygen consumption with the following equations:

			=-	
Oxygen Requirements (mL O <sub>2</sub> /kg/min)	Metabolic Equivalents (METS)	Functional Tasks	Treadmill: Bruce Protocol 3-Minute Stages (mph/elevation)	Bike Ergometer: for 70 kg of Body Weight (kg/min)
52.5	15			
49.5	14			
45.5	13		4.2/16.0	1500
42.0	12			1350
38.5	11			1200
35.0	10	Jogging	3.4/14.0	1050
31.5	9			900
28.0	8			750
24.5	7		2.5/12.0	
21.0	6	Stair climbing		600
17.5	5		1.7/10.0	450
14.0	4	Walking (level surface)		300
10.5	3			150
7.0	2	Bed exercise (arm exercises in supine or sitting)		

Data from American Heart Association, Committee on Exercise Testing and Training of Apparently Healthy Individuals: A Handbook for Physicians. Dallas: AHA; 1972; Brooks GA, Fahey TD, White TP, eds. Exercise Physiology: Human Bioenergetics and Its Applications. 2nd ed. Mountain View, CA: Mayfield Publishing; 1996.

$$\begin{split} \text{VO}_2\,\text{mL/kg/min} &= (\text{mph}) \big(26.83\,\,\text{m/min}\big) \big(0.1\,\,\text{mL/kg/min}\big) \\ &\quad + 3.5\,\,\text{mL/kg/min} \\ \text{METs} &= \text{VO}_2\,\,\text{mL/kg/min} \div 3.5 \\ \text{MPH} &= \big(\text{pace}\,[\text{feet/min}] \times 60\big) \div 5280\,(\text{feet/mile}) \end{split}$$

A direct relationship exists between pace on a level surface and METs (oxygen consumption). A therapist can use walking pace to estimate oxygen consumption and endurance for other functional tasks that fall within a patient's oxygen consumption (aerobic functional capacity). It is important to note METs can only be calculated for individuals without biomechanical insufficiencies that may alter amount of energy needed, especially in older adults.<sup>47</sup>

Fig. 3.9 depicts the relationship between pace (feet/min) and METs for level surface ambulation. Bruce Protocol Stage 1 (because of its incline when at 1.7 miles per hour [mph]) is similar to ambulation at 400 feet/min on a level surface. If a patient cannot sustain a particular pace for at least 10 minutes, it can be concluded that this pace exceeds the patient's anaerobic threshold. If the patient cannot sustain a pace for at least 1 minute, it can be concluded that the pace is close to the patient's maximal MET (oxygen consumption). Therefore continuous aerobic exercise programs should be at a walking pace below anaerobic threshold. For interval aerobic training, work periods at walking paces that can be sustained for 1 to 10 minutes would be appropriate with an equal period of rest. If a patient is required routinely to exceed maximal oxygen consumption during daily tasks, he or she is much more likely to experience signs of fatigue and exhaustion over time, such as during repeated bouts of activity throughout the day.

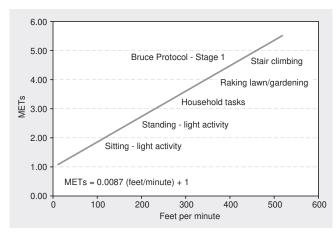


FIG. 3.9
Relationship between walking pace, metabolic equivalents of task (METs), and oxygen consumption on level surfaces. (Data from Fletcher GF, Balady GJ, Amsterdam EA, et al. Exercise standards for testing and training: a statement for healthcare professionals from the American Heart Association. *Circulation*. 2001;104:1694-1740.)

## **CLINICAL TIP**

Synonyms for aerobic exercise tests include exercise tolerance test (ETT) and graded exercise test (GXT).

**Thallium Stress Testing.** Thallium stress testing is a stress test that involves the injection of a radioactive nuclear marker for the detection of myocardial perfusion. The injection is given typically (via an intravenous line) during peak exercise or when

**CHAPTER 3** 

symptoms are reported during the stress test. After the test, the subject is passed under a nuclear scanner to be evaluated for myocardial perfusion by assessment of the distribution of thallium uptake. The subject then returns 3 to 4 hours later to be reevaluated for myocardial reperfusion. This test appears to be more sensitive than stress tests without thallium for identifying patients with coronary artery disease.<sup>14</sup>

Persantine Thallium Stress Testing. Persantine thallium stress testing is the use of dipyridamole (Persantine) to dilate the coronary arteries. Coronary arteries with atherosclerosis do not dilate; therefore dipyridamole shunts blood away from these areas. It is used typically in patients who are very unstable, deconditioned, or unable to ambulate or cycle for exercise-based stress testing. 40 Patients are asked to avoid all food and drugs containing methylxanthines (e.g., coffee, tea, chocolate, cola drinks) for at least 6 hours before the test in addition to phosphodiesterase drugs, such as aminophylline, for 24 hours. While the patient is supine, an infusion of dipyridamole (0.56 mL/kg diluted in saline) is given intravenously over 4 minutes (using a large-vein intracatheter). Four minutes after the infusion is completed, the perfusion marker (thallium) is injected, and the patient is passed under a nuclear scanner to be evaluated for myocardial perfusion by assessment of the distribution of thallium uptake.40

#### Cardiac Catheterization

Cardiac catheterization, classified as either right or left, is an invasive procedure that involves passing a flexible, radiopaque catheter into the heart to visualize chambers, valves, coronary arteries, great vessels, cardiac pressures, and volumes to evaluate cardiac function (estimate EF, CO).

The procedure also is used in the following diagnostic and therapeutic techniques<sup>14</sup>:

- Angiography
- Percutaneous coronary intervention (PCI)
- Electrophysiologic studies (EPSs)
- Cardiac muscle biopsy

Right-sided catheterization involves entry through a sheath that is inserted into a vein (commonly subclavian) for evaluation of right heart pressures; calculation of CO; and angiography of the right atrium, right ventricle, tricuspid valve, pulmonic valve, and pulmonary artery.<sup>14</sup> It also is used for continuous hemodynamic monitoring in patients with present or very recent heart failure to monitor cardiac pressures (see Chapter 18). Indications for right heart catheterization include an intracardiac shunt (blood flow between right and left atria or right and left ventricles), myocardial dysfunction, pericardial constriction, pulmonary vascular disease, valvular heart disease, and status post heart transplantation.

Left-sided catheterization involves entry through a sheath inserted into an artery (commonly femoral) to evaluate the aorta, left atrium, and left ventricle; left ventricular function; mitral and aortic valve function; and angiography of coronary arteries. Indications for left heart catheterization include aortic dissection, atypical angina, cardiomyopathy, congenital heart disease, coronary artery disease, status post MI, valvular heart disease, and status post heart transplant.

TABLE 3.11 Assessment Methods

Method	Region Examined
Aortography	Aorta and aortic valve
Coronary arteriography	Coronary arteries
Pulmonary angiography	Pulmonary circulation
Ventriculography	Right or left ventricle and AV valves

AV, Atrioventricular.

Data from Woods SL, Sivarajian Froelicher ES, Underhill-Motzer S, ed. Cardiac Nursing. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2009.

## **Physical Therapy Considerations**

- After catheterization, the patient is on bed rest for approximately 4 to 6 hours when venous access is performed, or for 6 to 8 hours when arterial access is performed.<sup>14</sup>
- The sheaths typically are removed from the vessel 4 to 6 hours after the procedure, and pressure is applied constantly for 20 minutes after sheath removal.<sup>14</sup>
- The extremity should remain immobile with a sandbag over the access site to provide constant pressure to reduce the risk of vascular complications.<sup>14</sup>
- Some hospitals may use a knee immobilizer to assist with immobilizing the lower extremity.
- Physical therapy intervention should be deferred or limited to bedside treatment within the parameters of these precautions.
- During the precautionary period, physical therapy intervention, such as bronchopulmonary hygiene, or education may be necessary. Bronchopulmonary hygiene is indicated if pulmonary complications or risk of these complications exists. Education is warranted when the patient is anxious and needs to have questions answered regarding his or her functional mobility.
- After the precautionary period, normal mobility can progress to the limit of the patient's cardiopulmonary impairments; however, the catheterization results should be incorporated into the physical therapy treatment plan.

#### **Angiography**

Angiography involves the injection of radiopaque contrast material through a catheter to visualize vessels or chambers. Different techniques are used for different assessments (Table 3.11).

#### **Electrophysiologic Studies**

EPSs are performed to evaluate the electrical conduction system of the heart. 14 An electrode catheter is inserted through the femoral vein into the right ventricle apex. Continuous ECG monitoring is performed internally and externally. The electrode can deliver programmed electrical stimulation to evaluate conduction pathways, formation of arrhythmias, and the automaticity and refractoriness of cardiac muscle cells. EPSs evaluate the effectiveness of antiarrhythmic medication and can provide specific information about each segment of the conduction system.<sup>14</sup> In many hospitals, these studies may be combined with a therapeutic procedure,

- Sinus node disorders
- AV or intraventricular block
- Previous cardiac arrest
- Tachycardia at greater than 200 bpm
- Unexplained syncope

# **SECTION CAL TIP**

Patients undergoing electrophysiologic studies (EPSs) typically remain on bed rest for 4 to 6 hours after the test.

#### **Health Conditions**

When disease and degenerative changes impair the heart's capacity to perform work, a reduction in CO occurs. If cardiac, renal, or central nervous system perfusion is reduced, a vicious cycle resulting in heart failure can ensue. A variety of pathologic processes can impair the heart's capacity to perform work. These pathologic processes can be divided into four major categories: (1) acute coronary syndrome (ACS), (2) rhythm and conduction disturbance, (3) valvular heart disease, and (4) myocardial and pericardial heart disease. CHF occurs when this failure to pump blood results in an increase in the fluid in the lungs, liver, subcutaneous tissues, and serous cavities.<sup>5</sup>

# Hypertension

Hypertension (HTN) is the most prevalent cardiovascular disease in the United States. The National Health and Nutrition Examination Survey (NHAHES) conducted from 2005 to 2008 estimated that approximately 76 million Americans above age 20 years have HTN. <sup>48</sup>

In 2017 the ACC and the AHA published revised guidelines on the stages of HTN and the appropriate threshold values to initiate antihypertensive drug treatment (see Table 3.6).<sup>49</sup> Notably, hypertension is a predominant risk factor for several additional cardiovascular diseases and often goes undetected for decades.<sup>49</sup> An important consideration for all health care professionals, including physical therapists, is the association between undetected hypertension and risk, particular in ambulatory (outpatient) and primary care settings.<sup>49</sup>

#### **Acute Coronary Syndrome**

When myocardial oxygen demand is higher than supply, the myocardium must use anaerobic metabolism to meet energy demands. This system can be maintained for only a short period before tissue ischemia will occur, which typically results in angina (chest pain). If the supply and demand are not balanced by rest, medical management, surgical intervention, or any combination of these, injury of the myocardial tissue will ensue, followed by infarction (cell death). This balance of supply and demand is achieved in individuals with normal coronary circulation; however, it is compromised in individuals with impaired coronary blood flow. The following pathologies can result in myocardial ischemia:

- Coronary arterial spasm is a disorder of transient spasm of coronary vessels that impairs blood flow to the myocardium.
   It can occur with or without the presence of atherosclerotic coronary disease. It results in variant angina (Prinzmetal angina).
- Coronary atherosclerotic disease (CAD) is a multistep process of the deposition of fatty streaks, or plaques, on artery walls (atherosis). The presence of these deposits eventually leads to arterial wall damage and platelet and macrophage aggregation, which then leads to thrombus formation and hardening of the arterial walls (sclerosis). The net effect is a narrowing of coronary walls. It can result in stable angina, unstable angina, or MI. 4,6,14

Clinical syndromes caused by these pathologies are as follows<sup>8,14</sup>:

- Stable (exertional) angina occurs with increased myocardial demand, such as during exercise; is relieved by reducing exercise intensity or terminating exercise; and responds well to nitroglycerin.
- Variant angina (Prinzmetal angina) is a less-common form of angina caused by coronary artery spasm. This form of angina tends to be prolonged, severe, and not readily relieved by nitroglycerin.
- Unstable angina is considered intermediate in severity between stable angina and MI. It usually has a sudden onset, occurs at rest or with activity below the patient's usual ischemic baseline and may be different from the patient's usual anginal pattern. Unstable angina is not induced by activity or increased myocardial demand that cannot be met. It can be induced at rest, when supply is cut down with no change in demand. A common cause of unstable angina is believed to be a rupture of an atherosclerotic plaque.
- MI occurs with prolonged or unmanaged ischemia (Table 3.12). It is important to realize that an evolution occurs from ischemia to infarction. Ischemia is the first phase of tissue response when the myocardium is deprived of oxygen. It is reversible if sufficient oxygen is provided in time. However, if oxygen deprivation continues, myocardial cells will become injured and eventually will die (infarct). The location and extent of cell death are determined by the coronary artery that is compromised and the amount of time that the cells are deprived. A clinical overview is provided in Fig. 3.10.

# **CLINICAL TIP**

ST depression on a patient's ECG of approximately 1 to 2 mm generally is indicative of ischemia; ST elevation generally is indicative of myocardial injury or infarction.

#### **Rhythm and Conduction Disturbance**

Rhythm and conduction disturbances can range from minor alterations with no hemodynamic effects to life-threatening episodes with rapid hemodynamic compromise. A.6.8 Refer to the tables in Appendix 3A for a description of atrial, ventricular, and junctional rhythms and AV blocks. Refer to Appendix 3B for examples of common rhythm disturbances. Physical therapists must be able to identify abnormalities on ECG to determine

<u> </u>		
MI/Wall Affected	Possible Occluded Coronary Artery	Possible Complications
Anterior MI/anterior left ventricle	LCA	Left-sided CHF, pulmonary edema, bundle branch block, AV block, and ventricular aneurysm (which can lead to CHF, dysrhythmias, and embolism)
Inferior MI/inferior left ventricle	RCA	AV blocks (which can result in bradycardia) and papillary muscle dysfunction (which can result in valvular insufficiency and eventually CHF)
Anterolateral MI/anterolateral left ventricle	LAD, circumflex	Brady or tachyarrhythmias, acute ventricular septal defect
Anteroseptal MI/septal region—between left and right ventricles	LAD	Brady or tachyarrhythmias, ventricular aneurysm
Posterior MI/posterior heart	RCA, circumflex	Bradycardia, heart blocks
Right ventricular MI	RCA	Right ventricular failure (can lead to left ventricular failure and therefore cardiogenic shock), heart blocks, hepatomegaly, peripheral edema
Transmural MI (Q-wave MI)	Any artery	Full wall thickness MI, as above
Subendocardial MI (non-Q-wave MI)	Any artery	Partial wall thickness MI, as above, potential to extend to transmural MI

AV, Atrioventricular; CHF, congestive heart failure; LAD, left anterior descending; LCA, left coronary artery; MI, myocardial infarction; RCA, right coronary artery. Data from Woods SL, Sivarajian-Froelicher ES, Underhill-Motzer S, eds. Cardiac Nursing. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2000.

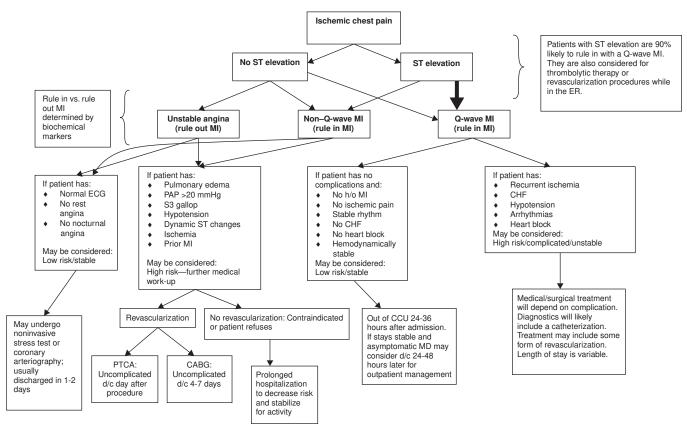


FIG. 3.10

Possible clinical course of patients admitted with chest pain. CABG, Coronary artery bypass graft; CHF, congestive heart failure; CC, coronary care unit; d/c, discharge; ECG, electrocardiogram; b/o, history of; MI, myocardial infarction; PAP, pulmonary arterial pressure; PTCA, percutaneous transluminal coronary angioplasty; ST elevation, electrocardiogram that shows elevation of the ST segment. (Data from American College of Cardiology/American Heart Association. 1999 Update: ACC/AHA guidelines for the management of patients with acute myocardial infarction: executive summary and recommendations. Circulation. 1999;100:1016-1030; American College of Cardiology/American Heart Association: ACC/AHA guidelines for the management of patients with acute myocardial infarction. J Am Coll Cardiol. 1996;28:1328-1428; American College of Cardiology/American Heart Association: ACC/AHA guidelines for the management of patients with unstable angina [USA] and non-ST segment elevation myocardial infarction. J Am Coll Cardiol. 2000;36:971-1048.)

Disease	Symptoms	Signs
Aortic stenosis	Angina, syncope or near syncope, signs of left ventricle failure (dyspnea, orthopnea, cough)	Elevated left ventricular wall pressure, decreased subendocardial blood flow, systolic murmur, ventricular hypertrophy
Chronic aortic regurgitation	Angina, symptoms of left ventricular failure	Dilated aortic root, dilated left ventricle, diastolic murmur, left ventricular hypertrophy
Acute aortic regurgitation	Rapid progression of symptoms of left ventricular failure, pulmonary edema, angina	Sinus tachycardia to compensate for decreased stroke volume, loud S3, diastolic murmur, signs of ventricular failure
Mitral stenosis	Symptoms of pulmonary vascular congestion (dyspnea, orthopnea). If patient develops pulmonary hypertension (which can cause hypoxia, hypotension), he or she may have angina, syncope	Left atrial hypertrophy, pulmonary hypertension, atrial fibrillation, can have embolus formation (especially if in atrial fibrillation), long diastolic murmur
Chronic mitral regurgitation	Symptoms of pulmonary vascular congestion, angina, syncope, fatigue	Left atrial enlargement, atrial fibrillation, elevated left atrial pressure
Acute mitral regurgitation	Rapid progression of symptoms of pulmonary vascular congestion	Sinus tachycardia, presence of S3 or S4, pulmonary edema
Mitral valve prolapse	Most commonly asymptomatic, fatigue, palpitation	Systolic click, may have tachyarrhythmia syncope

Data from Woods SL, Sivarajian-Froelicher ES, Underhill-Motzer S, eds. Cardiac Nursing. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2000; Cheitlin MD, Sokolow M, McIlroy MB. Clinical Cardiology. 6th ed. Norwalk, CT: Appleton & Lange; 1993.

patient tolerance to activity. In particular, physical therapists should understand progressions of common ECG abnormalities so that they can identify, early on, when the patient is not tolerating an intervention. (Refer to the Physical Therapy Intervention section for a discussion on ECG.)

A common form of rhythm disturbance is a PVC, which also can be referred to as a *ventricular premature beat*. These abnormalities originate from depolarization of a cluster of cells in the ventricle (an ectopic foci), which results in ventricular depolarization. From the term *ectopic foci*, PVCs may be referred to as *ventricular ectopy*.

#### Valvular Heart Disease

Valvular heart disease encompasses valvular disorders of one or more of the four valves of the heart (Table 3.13). The following three disorders can occur<sup>4,6</sup>:

- 1. Stenosis, which involves narrowing of the valve
- 2. Regurgitation, the back flow of blood through the valve, which occurs with incomplete valve closure
- 3. Prolapse, which involves enlarged valve cusps (The cusps can become floppy and bulge backward. This condition may progress to regurgitation.)

Over time, these disorders can lead to pumping dysfunction and, ultimately, heart failure.

#### Myocardial and Pericardial Heart Disease

Myocardial heart disease affects the myocardial muscle tissue and also is referred to as *cardiomyopathy* (Table 3.14); pericardial heart diseases affect the pericardium (Table 3.15).

#### **Heart Failure**

The syndrome of HF, evidenced by a decrease of CO, can be caused by a variety of cardiac pathologies. Because CO is not maintained, life cannot be sustained if HF continues without

treatment. HF results in the congestion of the pulmonary circulation and, in certain cases, even the systemic circulation. In light of this, clinically, therapists may see the abbreviation CHF emphasizing the congestion within the heart failure syndrome. The most common pathologic etiology of HF is some type of cardiomyopathy (see Table 3.14).

The following terms are used to classify the types of cardiac impairment in  $HF^{50}$ :

- *Left-sided heart failure*—refers to failure of the left ventricle, resulting in back flow into the lungs.
- Right-sided failure—refers to failure of the right side of the heart, resulting in back flow into the systemic venous system.
- High-output failure—refers to heart failure that is secondary
  to renal system failure to filter off excess fluid. The renal system failure places a higher load on the heart, which cannot
  be maintained.
- Low-output failure—refers to the condition in which the heart is not able to pump the minimal amount of blood to support circulation.
- Systolic dysfunction—refers to a problem with systole or the actual strength of myocardial contraction and is commonly referred to as heart failure with reduced ejection fraction (HFrEF).
- Diastolic dysfunction—refers to a problem during diastole or the ability of the ventricle to allow the filling of blood, and is commonly referred to as Heart Failure with Preserved Ejection Fraction (HFpEF).

Possible signs and symptoms of HF are described in Box 3.3. The AHA/ACC and the NYHA have created two classifications of HF.<sup>1,51</sup> From a structural perspective, HF is staged on the basis of the extent of structural damage to heart tissue. From a functional perspective, HF is classified on the basis of the NYHA functional classification.<sup>52</sup> The two classification systems are combined in Table 3.16.

**CHAPTER 3** 

<b>TABLE 3.14</b>	Myocardial	Diseases—	-Cardiom	yopathies
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Functional Classification			
Cardiomyopathy	Dysfunction	Description	
Dilated	Systolic	Ventricle is dilated, with marked contractile dysfunction of myocardium	
Hypertrophic	Diastolic	Thickened ventricular myocardium, less compliant to filling, and therefore decreased filling during diastole	
Restrictive	Systolic and diastolic	Endocardial scarring of ventricles, decreased compliance during diastole, and decreased contractile force during systole	
Etiologic Classification			
Etiology		Examples	
Inflammatory		Viral infarction, bacterial infarction	

Inflammatory Viral infarction, bacterial infarction Metabolic Selenium deficiency, diabetes mellitus Fibroplastic Carcinoid fibrosis, endomyocardial fibrosis Hypersensitivity Cardiac transplant rejection, methyldopa Genetic Hypertrophic cardiomyopathy, Duchenne's muscular dystrophy Idiopathic Idiopathic hypertrophic cardiomyopathy Infiltrative Sarcoidosis, neoplastic Hematologic Sickle cell anemia Alcohol, bleomycin Toxic Physical agents Heat stroke, hypothermia, radiation Miscellaneous acquired Postpartum cardiomyopathy, obesity

Data from Cahalin L. Cardiac muscle dysfunction. In: Hillegass EA, Sadowsky HS, eds. Essentials of Cardiopulmonary Physical Therapy. 2nd ed. Philadelphia: Saunders; 2001; Hare JM. The dilated, restrictive, and infiltrative cardiomyopathies. In Libby P, Bonow RO, Mann DL, et al. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 8th ed. Philadelphia: Saunders; 2008.

Disease	Symptoms	Signs	
Acute pericarditis	Retrosternal chest pain (worsened by supine and/or deep inspiration), dyspnea, cough, hoarseness, dysphagia, fever, chills, and weakness possible	Pericardial friction rub; diffuse ST segment elevation; decreased QRS voltage in all ECG leads if pericardial effusion also present	
Constrictive pericarditis	Abdominal swelling, peripheral edema, fatigue, dyspnea, dizziness and/or syncope, signs of pulmonary venous congestion, vague nonspecific retrosternal chest pain	Jugular venous distention; QRS voltage diminished on ECG; occasionally atrial fibrillation	
Chronic pericardial effusion (without tamponade)	May have vague fullness in anterior chest, cough, hoarseness, dysphagia	Muffled heart sounds; may have pericardial friction rub; QRS voltage diminished on ECG; chest x-ray with cardiomegaly without pulmonary congestion	
Pericardial tamponade	Symptoms of low cardiac output (dyspnea, fatigue, dizziness, syncope); may have retrosternal chest pain; may have cough, hiccoughs, hoarseness	Jugular venous distention, cardiomegaly, diminished QRS voltage on ECG; becomes tamponade from effusion when right heart catheterization shows equal pressures in right atrium, ventricle, and capillary wedge (signifies left atria pressure), and left heart catheterization shows equal pressure on left side of heart to right side	

ECG, Electrocardiography.

Data from Woods SL, Sivarajian-Froelicher ES, Underhill-Motzer S, eds. Cardiac Nursing. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2000; Cheitlin MD, Sokolow M, McIlroy MB. Clinical Cardiology. 6th ed. Norwalk, CT: Appleton & Lange; 1993.

Activity progression for patients hospitalized with HF is based on the ability of medical treatments (e.g., diuresis, inotropes) to keep the patient out of heart failure. When a patient with HF is medically stabilized, the heart is thought to be "compensated." Conversely, when the patient is unable to maintain adequate circulation, the heart would be "decompensated." Clinical examination findings allow the therapist to evaluate continuously the patient's tolerance to the activity progression. Although MET tables are not commonly used clinically, they do provide a method of progressively increasing a patient's activity level. As greater MET levels are achieved with an appropriate hemodynamic response, the next level of activity can be attempted. See Table 3.10 for MET levels for common activities that patients can perform.

# Management

This section discusses surgical and nonsurgical procedures, pharmacologic interventions, and physical therapy interventions for patients with cardiac health conditions

# Revascularization and Reperfusion of the Myocardium Thrombolytic Therapy

Thrombolytic therapy has been established as an acute management strategy for patients experiencing MI because of the

BOX 3.3 Signs and Symptoms of Heart Failure			
Signs	Symptoms		
Cold, pale, possibly cyanotic extremities Weight gain Peripheral edema Jugular venous distention Tachypnea Crackles (rales) Tubular breath sounds S3 and S4 heart sounds Sinus tachycardia Pulsus Alterans Decreased exercise tolerance and physical work capacity	Dyspnea Tachypnea Paroxysmal nocturnal dyspnea Orthopnea Cough Fatigue		

Data from Hillegass E, Lowers ST, Barker E. Cardiac muscle dysfunction and failure. In: Hillegass EA. *Essentials of Cardiopulmonary Physical Therapy.* 4th ed. St. Louis: Elsevier; 2017.

high prevalence of coronary artery thrombosis during acute MI. Thrombolytic agents, characterized as fibrin-selective and nonselective agents, are administered to appropriate candidates via intravenous access. The most common agents include streptokinase (nonselective), anisoylated plasminogen streptokinase activator complex (nonselective), and t-PA (fibrin-selective). <sup>14</sup> Fibrin-selective agents have a high velocity of clot lysis, whereas the nonselective agents have a slower clot lysis and more prolonged systemic lytic state.

The indication for thrombolytic therapy includes chest pain suggestive of myocardial ischemia and associated with acute ST segment elevation on a 12-lead ECG or a presumed new left ventricular bundle branch block. Hospital protocol regarding the timing of thrombolytic therapy usually varies because clinical trials have led to some controversy. <sup>14</sup> Some studies show benefits only if treatment is conducted within 6 hours of symptoms, whereas others have demonstrated improvement with treatment up to 24 hours after onset of symptoms. <sup>14</sup>

The contraindications to thrombolytic therapy generally include patients who are at risk for excessive bleeding. Because of the variability that can occur among patients, many contraindications are considered relative cautions, and the potential benefits of therapy are weighed against the potential risks. Thrombolytic therapy is used in conjunction with other medical treatments, such as with aspirin, intravenous heparin, intravenous nitroglycerin, lidocaine, atropine, and a beta-blocker. As previously discussed, early peaking of CK-MB is associated with reperfusion. <sup>14</sup>

## **Percutaneous Revascularization Procedures**

Percutaneous revascularization procedures are used to return blood flow through coronary arteries that have become occlusive

<b>TABLE 3.16</b>	AHA/ACC Stages and NYHA Functional Classes of Heart Failure

AHA/ACC Stage	Description	NYHA Class <sup>a</sup>	Description
Stage A	At high risk for developing HF. No identified structural or functional abnormality, no signs or symptoms of HF <sup>b</sup>	NA	
Stage B	Structural heart disease that is strongly associated with the development of HF but no signs and symptoms of HF	Ι	No limitation in physical activity; ordinary physical activity does not cause fatigue, palpitations, or dyspnea
Stage C	Symptomatic HF, associated with underlying structural heart disease	I	No limitation in physical activity; ordinary physical activity does not cause fatigue, palpitations, or dyspnea
		II	Slight limitation of physical activity; comfortable at rest but ordinary activity results in fatigue, palpitations, or dyspnea
		III	Marked limitation of physical activity; comfortable at rest, but less than ordinary activity results in fatigue, palpitations, or dyspnea
		IV	Symptoms at rest; unable to do any physical activity without symptomology
Stage D	Advanced structural disease with marked sympto- mology at rest despite maximal medical therapy	IV	Symptoms at rest; unable to do any physical activity without symptomology

<sup>&</sup>lt;sup>a</sup>Functional capacity refers to subjective symptoms of the patient. This aspect of the classification is identical to the New York Heart Association's Classification. <sup>b</sup>Objective structural assessment was added to the classification system by the American Heart Association in 1994. It refers to measurements such as electrocardiograms, stress tests, echocardiograms, and radiologic images. <sup>46</sup>

Data from Yancy CW, Jessup M, Bozkurt B, et al. 2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America. *J Card Fail*. 2017;23(8):628-651.