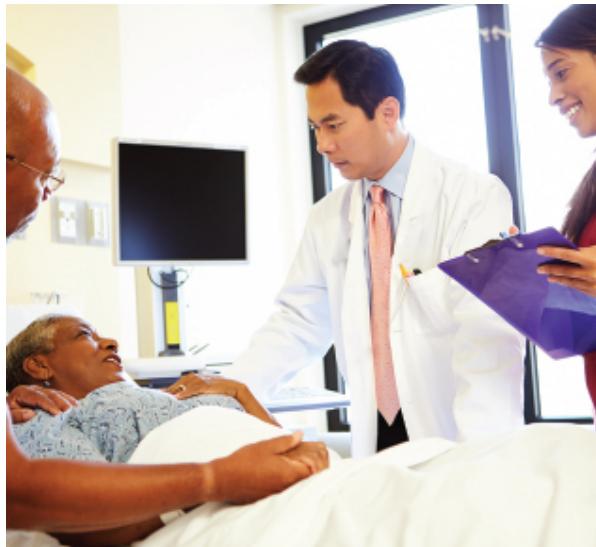


UNIT 2 Concepts and Principles of Patient Management

Case Study

PROMOTING TEAMWORK AND COLLABORATION IN PALLIATIVE CARE



A 56-year-old woman diagnosed with advanced adenocarcinoma of the lung arrives at her outpatient palliative care appointment with delirium, lethargy, and confusion. Her blood work is drawn, and she is transferred to the inpatient oncology unit where you work for further evaluation. The admission profile reports that she is prescribed gabapentin 300 mg three times daily and fentanyl 50 mcg transdermal patch to be replaced every 72 hours. Upon assessment you find the patient confused and unable to rate her pain. The family is concerned about the confusion and worried that she is unable to tolerate the medications ordered for pain. Because this patient has many underlying components to her care, you request a case conference with the family, palliative care team, pharmacist, and the oncologist to determine the best plan of care at this time.

QSEN Competency Focus: Teamwork and Collaboration

The complexities inherent in today's health care system challenge nurses to demonstrate integration of specific interdisciplinary core competencies. These competencies are aimed at ensuring the delivery of safe, quality patient care (Institute of Medicine, 2003). The Quality and Safety Education for Nurses project (Cronenwett, Sherwood, Barnsteiner, et al., 2007; QSEN, 2020) provides a framework for the knowledge, skills, and attitudes (KSAs) required for nurses to demonstrate competency in these key areas, which include ***patient-centered care, interdisciplinary teamwork and collaboration, evidence-based practice, quality improvement, safety, and informatics.***

Teamwork and Collaboration Definition: Function effectively within nursing and interprofessional teams, fostering open communication, mutual respect, and shared decision-making to achieve quality patient care.

SELECT PRE- LICENSURE KSAs	APPLICATION AND REFLECTION
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Knowledge

Recognize contributions of other individuals and groups in helping patient/family achieve health goals	Describe the nursing role in participating in a case conference. How do the various health care team members work with the patient and family and as a team to develop the best plan to manage care for this patient's pain while managing and minimizing the deleterious effects of delirium?
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Skills

Function competently within own scope of practice as a member of the health care team	Even though the patient is unable to rate her pain, what other methods of assessment can you use and rely on to assess pain in this patient? What interventions for pain can a nurse implement as part of a nursing care plan?
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Attitudes

Contribute to resolution of conflict and disagreement	After the case conference, the palliative care team recommends that the pain medication be administered to the patient as prescribed. The family is in conflict with the decision because the patient is confused and is not rating her pain. How can you best resolve the conflict? Are there other members of the team that may be instrumental in communicating with the family?
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Cronenwett, L., Sherwood, G., Barnsteiner, J., et al. (2007). Quality and safety education for nurses. *Nursing Outlook*, 55(3), 122–131; Institute of Medicine. (2003). *Health professions education: A bridge to quality*. Washington, DC: National Academies Press; QSEN Institute. (2020). *QSEN competencies: Definitions and pre-licensure KSAs; Teamwork and collaboration*. Retrieved on 8/15/2020 at: qsen.org/competencies/pre-licensure-ksas/#teamwork_collaboration

9 Pain Management

LEARNING OUTCOMES

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

1. Describe the fundamental concepts of pain including the types of pain, the four processes of nociception, and neuropathic pain.
2. Explain and demonstrate methods to perform a pain assessment.
3. List the first-line agents from the three groups of analgesic agents.
4. Identify the unique effects of select analgesic agents on older adults.
5. Describe practical nonpharmacologic methods that can be used in the clinical setting in patients with pain.
6. Use the nursing process as a framework for care of the patient with pain.

NURSING CONCEPTS

Addiction

Comfort

GLOSSARY

acute pain: pain that results from tissue damage that generally abates as healing occurs; serves as a warning signal that something is wrong or needs attention

adjuvant analgesic agent: a substance or medication added to an analgesic medication regimen to improve analgesia (*synonym:* co-analgesic agent)

agonist: a medication that binds to an opioid receptor mimicking the way endogenous substances provide analgesia

agonist–antagonist: a type of opioid (e.g., nalbuphine and butorphanol) that binds to the kappa opioid receptor site acting as an agonist (capable of producing analgesia) and simultaneously to the mu opioid receptor site acting as an antagonist (reversing mu agonist effects)

allodynia: pain due to a stimulus that does not normally provoke pain, such as touch; typically experienced in the skin around areas affected by nerve injury and commonly seen with many neuropathic pain syndromes

antagonist: a medication that competes with agonists for opioid receptor binding sites; can displace agonists, thereby inhibiting their action

breakthrough pain: a transitory increase in pain that occurs in the context of otherwise controlled persistent pain

ceiling effect: an analgesic dose above which further dose increments produce no change in effect

central sensitization: a key central mechanism of neuropathic pain; the abnormal hyperexcitability of central neurons in the spinal cord, which results from complex changes induced by the incoming afferent barrages of nociceptors and results in an increased nociceptive neuron response

chronic or persistent pain: pain that may or may not be time limited but that persists beyond the usual course/time of tissue healing

co-analgesic agent: one of many medications that can either improve the effectiveness of another analgesic agent or independently have analgesic action (*synonym:* adjuvant analgesic agent)

comfort–function goal: the pain rating identified by the individual patient above which the patient experiences interference with function and quality of life (e.g., activities the patient needs or wishes to perform)

efficacy: the extent to which a medication or another treatment “works” and can produce the intended effect—analgesia in this context

half-life: the time it takes for the plasma concentration (amount of medication in the body) to be reduced by 50% (after starting a medication, or increasing its dose; four to five half-lives are required to

approach a steady-state level in the blood, irrespective of the dose, dosing interval, or route of administration; after four to five half-lives, a medication that has been discontinued generally is considered to be mostly eliminated from the body)

hydrophilic: a substance or medication that is readily absorbed in aqueous solution

hyperalgesia: an increasingly intense experience of pain resulting from a noxious stimulus

intraspinal: “within the spine”; refers to the spaces or potential spaces surrounding the spinal cord into which medications can be given

lipophilic: a substance or medication that is readily absorbed in fatty tissues

metabolite: the product of biochemical reactions during medication metabolism

mu agonist: any opioid that binds to the mu opioid receptor subtype and produces analgesic effects (e.g., morphine); used interchangeably with the terms *full agonist*, *pure agonist*, and *morphinelike medication*

multimodal analgesia or multimodal pain management: the intentional, concurrent use of more than one pharmacologic or nonpharmacologic intervention with different methods of action with the goal to achieve better analgesia while using lower doses of medications with fewer adverse effects

neuraxial: of the central nervous system

neuropathic (pathophysiologic) pain: pain caused by injury or dysfunction (lesion or disease) of one or more nerves of the peripheral or central nervous systems with resultant impaired processing of sensory input

neuroplasticity: the ability of the peripheral and central nervous systems to change both structure and function as a result of noxious stimuli

nociceptive (physiologic) pain: pain that is sustained by ongoing activation of the sensory system that conducts the perception of noxious stimuli; implies the existence of damage to somatic or visceral tissues sufficient to activate the nociceptive system

nociceptor: a type of primary afferent neuron that has the ability to respond to a noxious stimulus or to a stimulus that would be noxious if prolonged

nonopioid: refers to analgesic medications that include acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs)

NSAID: an acronym for nonsteroidal anti-inflammatory drug (pronounced “en said”)

opioid: refers to morphine and other natural, semisynthetic, and synthetic medications that relieve pain by binding to multiple types of opioid receptors; term is preferred to “narcotic”

opioid dose-sparing effect: occurs when a nonopioid or co-analgesic medication is prescribed in addition to an opioid, enabling the opioid dose to be lower without diminishing analgesic effects

opioid-induced hyperalgesia: a phenomenon in which exposure to an opioid induces increased sensitivity, or a lowered threshold, to the neural activity conducting pain perception; it is the “flip side” of tolerance

opioid naïve: denotes a person who has not recently taken enough opioid on a regular enough basis to become tolerant to the opioid’s effects

opioid tolerant: denotes a person who has taken opioids long enough at doses high enough to develop tolerance to many of the opioid’s effects, including analgesia and sedation

pain: an unpleasant experience that is either emotional or sensory resulting from actual or possible damage to tissues and is uniquely experienced and described by each person

peripheral sensitization: a key peripheral mechanism of neuropathic pain that occurs when there are changes in the number and location of ion channels; in particular, sodium channels abnormally accumulate in injured nociceptors, producing a lower nerve depolarization threshold, ectopic discharges, and an increase in the response to stimuli

physical dependence: the body’s normal response to administration of an opioid for 2 or more weeks; withdrawal symptoms may occur if an opioid is abruptly stopped or an antagonist is given

placebo: any medication or procedure, including surgery, that produces an effect in a patient because of its implicit or explicit intent and not because of its specific physical or chemical properties

preemptive analgesic agents: pre-injury pain treatments (e.g., preoperative epidural analgesia and preincision local anesthetic infiltration) to prevent the development of peripheral and central sensitization of pain

refractory: nonresponsive or resistant to therapeutic interventions such as analgesic agents

substance use disorder (SUD): problematic use of substances such as opioids, benzodiazepines, or alcohol based on identification of at least two of the diagnostic criteria listed by the American Psychiatric Association. It is characterized by craving the substance; continuing use despite harm; inability to stop using; and experiencing withdrawal

symptoms when abruptly not using the substance; formerly known as addiction

titration: upward or downward adjustment of the amount (dose) of an analgesic agent

tolerance: a normal physiologic process characterized by decreasing effects of a medication at its previous dose, or the need for a higher dose of medication to maintain an effect

withdrawal: result of abrupt cessation or rapid decrease in dose of a substance upon which one is physically dependent. It is not necessarily indicative of substance use disorder

Pain serves as a survival tactic that guides individuals not only to avoid damage in the moment, but also to learn to avoid danger in the future (Martin, Power, Boyle, et al., 2017). Nurses in all settings play a key role in the management of pain as experts in assessment, medication administration, and patient education. They are uniquely positioned to assume this role as members of the health care team most consistently at the patient's bedside. These characteristics have led to nurses' distinction as the primary managers of patients who are experiencing pain (Curtis & Wrona, 2018).

Fundamental Concepts

Understanding the definition, effects, and types of pain lays the foundation for proper pain assessment and management.

Definition of Pain

The American Pain Society (APS, 2016) defines **pain** as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage” (p. 2). This definition describes pain as a complex phenomenon that can impact a person’s psychosocial, emotional, and physical functioning. The clinical definition of pain reinforces that pain is a highly personal and subjective experience: “Pain is whatever the experiencing person says it is, existing whenever he says it does” (McCaffery, 1968, p. 8). The self-report by the patient is the standard; it is considered to be the most reliable indicator of pain and the most essential component of pain assessment (DiMaggio, Clark, Czarenecki, et al., 2018).

Effects of Pain

Pain affects individuals of every age, gender, race, and socioeconomic class (APS, 2016). It is the primary reason people seek health care and one of the

most common conditions that nurses treat (U.S. Department of Health & Human Services [HHS], 2019). Unrelieved pain has the potential to affect every system in the body and cause numerous harmful effects, some of which may last a lifetime ([Table 9-1](#)). Despite many advances in the understanding of the underlying mechanisms of pain and the availability of improved analgesic agents and technology, as well as nonpharmacologic pain management methods, all types of pain continue to be undertreated (Jungquist, Vallerand, Sicoutris, et al., 2017).

Types and Categories of Pain

Pain can be categorized in many ways, and clear distinctions are not always possible. Pain often is described from the perspective of duration, as being acute or chronic (persistent) (APS, 2016). **Acute pain** involves tissue damage as a result of surgery, trauma, burn, or venipuncture, and is expected to have a relatively short duration and resolve with normal healing. **Chronic or persistent pain** is subcategorized as being of cancer or noncancer origin and can persist throughout the course of a person's life. Examples of noncancer chronic pain include peripheral neuropathy from diabetes, back or neck pain after injury, and osteoarthritis pain from joint degeneration. Chronic pain may be intermittent, occurring with flares, or it may be continuous. Some conditions can produce both acute and chronic pain. For example, some patients with cancer have continuous chronic pain and also experience more intense acute exacerbations of pain periodically, which is called **breakthrough pain** (BTP). Patients may also endure acute pain from repetitive painful procedures during cancer treatment (APS, 2016).

Pain is also classified by its inferred pathology as being either nociceptive pain or neuropathic pain ([Table 9-2](#)). **Nociceptive (physiologic) pain** refers to the normal functioning of physiologic systems that leads to the perception of noxious stimuli (tissue injury) as being painful (International Association for the Study of Pain [IASP], 2017). This is the reason why nociception is described as "normal" pain transmission. **Neuropathic (pathophysiologic) pain** is pathologic and results from abnormal processing of sensory input by the nervous system as a result of damage to the peripheral or central nervous system (CNS) or both (IASP, 2017).

TABLE 9-1 Harmful Effects of Unrelieved Pain

Domains Affected	Specific Responses to Pain
Endocrine	↑ Adrenocorticotropic hormone (ACTH), ↑ cortisol, ↑ antidiuretic hormone (ADH), ↑ epinephrine, ↑ norepinephrine, ↑ growth hormone (GH), ↑ catecholamines, ↑ renin, ↑ angiotensin II, ↑ aldosterone, ↑ glucagon, ↑ interleukin-1; ↓ insulin, ↓ testosterone
Metabolic	Gluconeogenesis, hepatic glycogenolysis, hyperglycemia, glucose intolerance, insulin resistance, muscle protein catabolism, ↑ lipolysis
Cardiovascular	↑ Heart rate, ↑ cardiac workload, ↑ peripheral vascular resistance, ↑ systemic vascular resistance, hypertension, ↑ coronary vascular resistance, ↑ myocardial oxygen consumption, hypercoagulation, deep vein thrombosis
Respiratory	↓ Flows and volumes, atelectasis, shunting, hypoxemia, ↓ cough, sputum retention, infection
Genitourinary	↓ Urinary output, urinary retention, fluid overload, hypokalemia
Gastrointestinal	↓ Gastric and bowel motility
Musculoskeletal	Muscle spasm, impaired muscle function, fatigue, immobility
Cognitive	Reduction in cognitive function, mental confusion
Immune	Depression of immune response
Developmental	↑ Behavioral and physiologic responses to pain, altered temperaments, higher somatization; possible altered development of the pain system, ↑ vulnerability to stress disorders, addictive behavior, and anxiety states
Future pain	Debilitating chronic pain syndromes: postmastectomy pain, post thoracotomy pain, phantom pain, postherpetic neuralgia
Quality of life	Sleeplessness, anxiety, fear, hopelessness, ↑ thoughts of suicide

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Patients may have a combination of nociceptive and neuropathic pain. For example, a patient may have nociceptive pain as a result of tumor growth, and also report radiating sharp and shooting neuropathic pain if the tumor is pressing against a nerve plexus. Sickle cell disease pain is usually a combination of nociceptive pain from the various hematologic changes of sickled cells as well as neuropathic pain from nerve ischemia (Belvis, Henderson, & Benzon, 2018).

Nociceptive Pain

Nociception includes four specific processes: transduction, transmission, perception, and modulation (Ellison, 2017). Figure 9-1 illustrates these processes and following is an overview of each.

TABLE 9-2 Classification of Pain by Inferred Pathology

	Nociceptive Pain	Neuropathic Pain	Mixed Pain
Physiologic Processes	Normal processing of stimuli that damages tissues or has the potential to do so if prolonged; can be somatic or visceral	Abnormal processing of sensory input by the peripheral or central nervous system or both	Components of both nociceptive and neuropathic pain; poorly defined
Categories and Examples	<p>Somatic Pain: Arises from bone joint, muscle, skin, or connective tissue. It is usually described as aching or throbbing in quality and is well localized</p> <p><i>Examples:</i> Surgical, trauma; wound and burn pain; cancer pain (tumor growth) and pain associated with bony metastases; labor pain (cervical changes and uterine contractions); osteoarthritis and rheumatoid arthritis pain; osteoporosis pain; pain of Ehlers–Danlos syndrome; ankylosing spondylitis</p> <p>Visceral Pain: Arises from visceral organs, such as the GI tract and pancreas. This may be subdivided:</p> <ul style="list-style-type: none"> • Tumor involvement of the organ capsule that causes aching and fairly well-localized pain • Obstruction of hollow viscus, which causes intermittent cramping and poorly localized pain <p><i>Examples:</i> Organ-involved cancer pain; ulcerative colitis; irritable bowel syndrome; Crohn's disease; pancreatitis</p>	<p>Centrally Generated Pain</p> <p><i>Deafferentation pain:</i> Injury to either the peripheral or central nervous system; burning pain below the level of a spinal cord lesion reflects injury to the central nervous system</p> <p><i>Examples:</i> Phantom pain as a result of peripheral nerve damage; poststroke pain; pain following spinal cord injury</p> <p><i>Sympathetically maintained pain:</i> Associated with dysregulation of the autonomic nervous system</p> <p><i>Example:</i> Complex regional pain syndrome</p> <p>Peripherally Generated Pain</p> <p><i>Painful polyneuropathies:</i> Pain is felt along the distribution of many peripheral nerves.</p> <p><i>Examples:</i> Diabetic neuropathy; posttherapeutic neuralgia; alcohol–nutritional</p>	<p>No identified categories</p> <p><i>Examples:</i> Fibromyalgia; some types of neck, shoulder, and back pain; some headaches; pain associated with HIV; some myofascial pain; pain associated with Lyme disease</p>

			neuropathy; some types of neck, shoulder, and back pain; pain of Guillain–Barré syndrome
		<i>Painful mononeuropathies:</i> Usually associated with a known peripheral nerve injury; pain is felt at least partly along the distribution of the damaged nerve	
Pharmacologic Treatment	Most responsive to nonopioids, opioids, and local anesthetics	Co-analgesic agents, such as antidepressants, anticonvulsants, and local anesthetics, but there is wide variability in terms of efficacy and adverse-effect profiles	Co-analgesic agents, such as antidepressants, anticonvulsants, and local anesthetics, but there is wide variability in terms of efficacy and adverse-effect profiles

GI, gastrointestinal; HIV, human immune deficiency virus.

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Transduction

Transduction refers to the processes by which noxious stimuli, such as a surgical incision or burn, activate primary afferent neurons called **nociceptors**, located throughout the body in the skin, subcutaneous tissue, and visceral (organ), and somatic (musculoskeletal) structures (Montgomery, Mallick-Searle, Peltier, et al., 2018). These neurons have the ability to respond selectively to noxious stimuli generated as a result of tissue damage from

mechanical (e.g., incision, tumor growth), thermal (e.g., burn, frostbite), chemical (e.g., toxins, chemotherapy), and infectious sources. Noxious stimuli cause the release of a number of excitatory compounds (e.g., serotonin, bradykinin, histamine, substance P, and prostaglandins), which move pain along the pain pathway (Ringkamp, Dougherty, & Raja, 2018) (see Fig. 9-1A). In addition, sodium, calcium, and potassium ion channels are stimulated to open, resulting in electrical impulses that are transmitted through the large, rapid conducting A-delta and smaller, peripheral C-fiber nociceptors (Ellison, 2017).

Prostaglandins are lipid compounds that initiate inflammatory responses that increase tissue swelling and pain at the site of injury (Baral, Udit, & Chiu, 2019). They form when the enzyme phospholipase breaks down phospholipids into arachidonic acid. In turn, the enzyme cyclo-oxygenase (COX) acts on arachidonic acid to produce prostaglandins (Fig. 9-2). COX-1 and COX-2 are isoenzymes of COX and play an important role in producing the effects of the **nonopioid** analgesic agents, which include the nonsteroidal anti-inflammatory drugs (**NSAIDs**) and acetaminophen. NSAIDs produce pain relief by mediating inflammation at the site of trauma, primarily by blocking the formation of prostaglandins (Leppert, Malec-Milewska, Zajaczkowska, et al., 2018). The nonselective NSAIDs, such as ibuprofen, naproxen, diclofenac, and ketorolac, inhibit both COX-1 and COX-2, and the COX-2 selective NSAIDs, such as celecoxib, inhibit only COX-2. As Figure 9-2 illustrates, both types of NSAIDs produce anti-inflammation and pain relief through the inhibition of COX-2. Acetaminophen is known to be a COX inhibitor that has minimal peripheral effect, is not anti-inflammatory, and can both relieve pain and reduce fever by preventing the formation of prostaglandins in the CNS (Slattery & Klegeris, 2018).

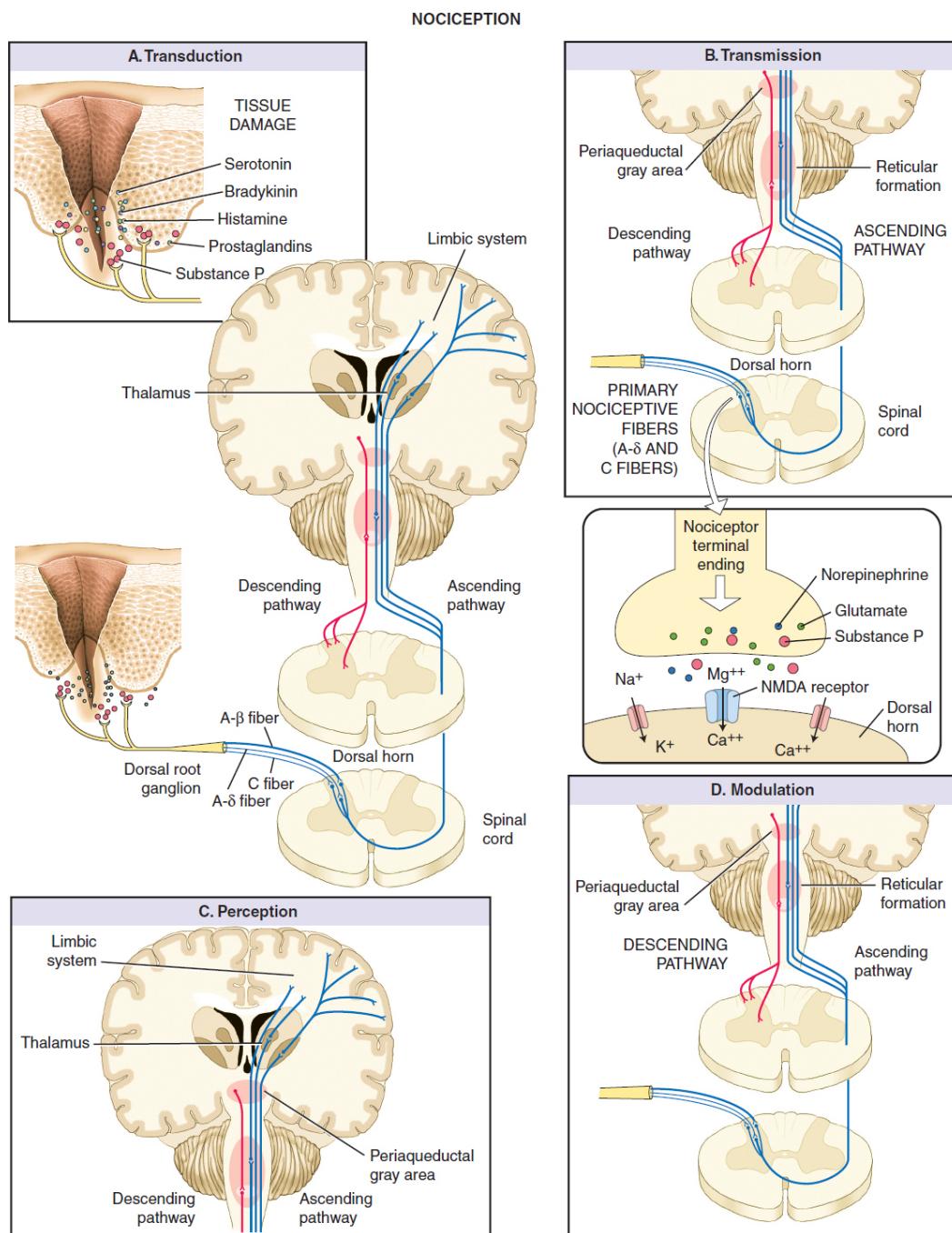


Figure 9-1 • Nociception. **A.** Transduction. **B.** Transmission. **C.** Perception. **D.** Modulation. Redrawn from Pasero, C., & McCaffery, M. (2011). *Pain assessment and pharmacologic management* (p. 5). St. Louis, MO: Mosby-Elsevier. Copyright 2011, Pasero, C., & McCaffery, M. Used with permission.

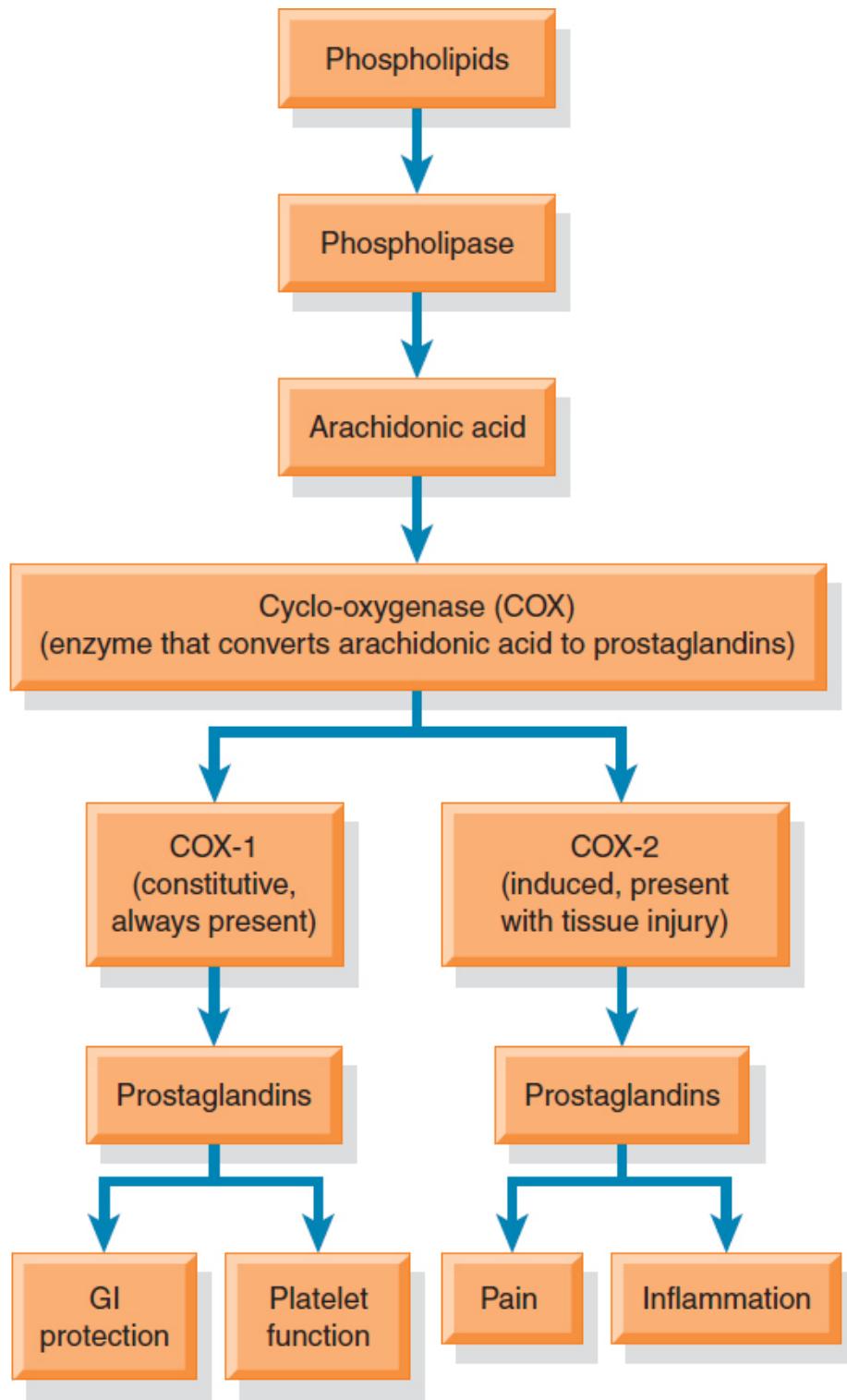


Figure 9-2 • Enzyme pathway: COX-1 and COX-2. Redrawn from Pasero, C., & McCaffery, M. (2011). *Pain assessment and pharmacologic management* (p. 6). St. Louis, MO: Mosby-Elsevier. Copyright 2004, Pasero, C., & McCaffery, M. Used with permission.

Other analgesic agents work at the site of transduction by affecting the flux of ions. For example, sodium channels are closed and inactive at rest but undergo changes in response to nerve membrane depolarization. Transient channel opening leads to an influx of sodium that results in nerve conduction (Nouri, Osuagwu, Boyette-Davis, et al., 2018). Local anesthetics reduce nerve conduction by blocking sodium channels. The calcium channel blocking anticonvulsants that are used to treat neuropathic pain facilitate analgesia by reducing the flux of calcium ions and limiting glutamate, norepinephrine, and substance P release (Peterson, Benson, & Hurley, 2018).

Transmission

Transmission is another process involved in nociception. Effective transduction generates an action potential that is transmitted along the lightly myelinated rapid conducting A-delta fibers and the unmyelinated slower impulse conducting C fibers (Ellison, 2017) (see Fig. 9-1B). The endings of A-delta fibers detect thermal and mechanical injury, allow relatively quick localization of pain, and are responsible for a rapid reflex withdrawal from the painful stimulus. Unmyelinated C fibers respond to mechanical, thermal, and chemical stimuli. They produce poorly localized and often aching or burning pain. A-beta (β) fibers are the largest of the fibers and respond to touch, movement, and vibration but do not normally transmit pain (Ellison, 2017; Vardeh & Naranjo, 2017).

The action potential impulse with the noxious information passes through the dorsal root ganglia, then synapses in the dorsal horn of the spinal cord, and then ascends up to the spinal cord and transmits the information to the brain, where pain is perceived (Ellison, 2017; Ringkamp et al., 2018) (see Fig. 9-1B). Extensive modulation occurs in the dorsal horn via complex neurochemical mechanisms (see Fig. 9-1B inset). The primary A-delta fibers release glutamate and C fibers release substance P and other neuropeptides (Schliessbach & Maurer, 2017). Glutamate is a key neurotransmitter because it binds to the *N*-methyl-*D*-aspartate (NMDA) receptor and promotes pain transmission (Zhou, 2017).

Perception

An additional process involved in nociception is perception, which is the result of the neural activity associated with transmission of noxious stimuli (Ringkamp et al., 2018). It requires activation of higher brain structures for the occurrence of awareness, emotions, and impulses associated with pain (see Fig. 9-1C). Although the physiology of pain perception continues to be studied, it can be targeted by nonpharmacologic therapies, such as distraction, which are based on the belief that innate brain processes can strongly influence pain perception (Chayadi & McConnell, 2019).

Modulation

Modulation is another process involved in nociception. Modulation of the information generated in response to noxious stimuli occurs at every level from the periphery to the cortex and involves many different neurochemicals (Damien, Colloca, Bellei-Rodriguez, et al., 2018) (see [Fig. 9-1D](#)). For example, serotonin and norepinephrine are inhibitory neurotransmitters that are released in the spinal cord and the brain stem by the descending (efferent) fibers of the modulatory system (Nouri et al., 2018). Some antidepressants provide pain relief by blocking the body's reuptake (resorption) of serotonin and norepinephrine, extending their availability to fight pain (Martin et al., 2017). Endogenous opioids are located throughout the peripheral and central nervous systems, and like exogenous opioids, they bind to opioid receptors in the descending system and inhibit pain transmission (Nouri et al., 2018).

Neuropathic Pain

Neuropathic pain is caused by either a lesion or a disease involving the somatosensory nervous system (Bouhassira, 2019). Injuries to peripheral nerves can either be traumatic or nontraumatic, such as diabetic or compression neuropathies (Osborne, Anastakis, & Davis, 2018). Although specific causes may vary based on the underlying pathology, it is theorized that there are changes in the ion channels; imbalance of the stimuli processing between excitatory and inhibitory somatosensory signals; activity of glial cells; or potential differences in modulation of pain that occur with neuropathic pain (Colloca, Ludman, Bouhassira, et al., 2017; Liu, Zhu, Ju, et al., 2019) ([Fig. 9-3](#)). Recent research findings suggest that dysfunction in autophagy (i.e., cellular degradation of unnecessary materials) is involved with neuropathic pain (Liu et al., 2019). Research is ongoing to better define the peripheral and central mechanisms that initiate and maintain neuropathic pain (García, Gutiérrez-Lara, Centurión, et al., 2019; Kwiatkowski & Mika, 2018; Liu et al., 2019; Nishimura, Kawasaki, Suzuki, et al., 2019).

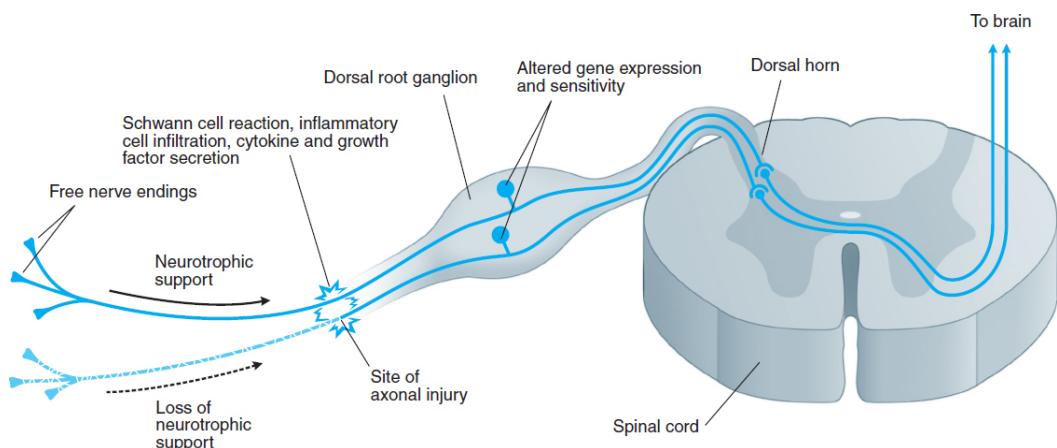


Figure 9-3 • Neuropathic pain. Nociceptive injury or inflammation may result in an altered physiologic response within the nociceptive system. These changes cause release of inflammatory cytokines that may alter gene expression and sensitivity in nociceptive fibers. In turn, these alter nociceptive activity, causing neuropathic pain.
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Peripheral Mechanisms

At any point from the periphery to the CNS, the potential exists for the development of neuropathic pain. Nerve endings in the periphery can become damaged, leading to abnormal reorganization in the nervous system called maladaptive **neuroplasticity**, an underlying mechanism of some neuropathic pain states (Osborne et al., 2018). Changes in ion channels can occur, such as increased sodium channel activity in sensory nerves resulting in heightened excitability, increased transduction, and release of neurotransmitters (Colloca et al., 2017). These and many other processes lead to a phenomenon called **peripheral sensitization**, which is thought to contribute to the maintenance of neuropathic pain and is thought to be reflected in allodynia and hyperalgesia (Osborne et al., 2018). **Allodynia**, or pain from a normally non-noxious stimulus (e.g., touch), is one such type of abnormal sensation and a common feature of neuropathic pain (Chekka & Benzon, 2018; Osborne et al., 2018). In patients with allodynia, the mere weight of clothing or bedsheets on the skin can be excruciatingly painful. **Hyperalgesia** is an increased response of pain sensation from a stimulus which at a usual pain threshold produces a less intense pain response.

Central Mechanisms

Central mechanisms also play a role in the establishment of neuropathic pain. **Central sensitization** is defined as abnormal hyperexcitability of central

neurons in the spinal cord, which results from complex changes induced by incoming afferent barrages of nociceptors, which also can result in allodynia and hyperalgesia (Osborne et al., 2018). Extensive release and binding of excitatory neurotransmitters, such as glutamate, activate the NMDA receptor and cause an increase in intracellular calcium levels into the neuron, resulting in pain (Yan, Li, Zhou, et al., 2017). Similar to what happens in the peripheral nervous system, an increase in the influx of sodium is thought to lower the threshold for nerve activation, increase response to stimuli, and enlarge the receptive field served by the affected neuron (Osborne et al., 2018; Yan et al., 2017).

As in the peripheral nervous system, anatomic changes can occur in the CNS. For example, when the NMDA receptor cells are continuously activated, reorganization in the dorsal horn of the spinal cord can occur (Nouri et al., 2018). Nerve fibers can invade other locations and create abnormal sensations, such as allodynia, in the area of the body served by the injured nerve.

Pain Assessment

The highly subjective nature of pain causes challenges in assessment and management; however, the patient's self-report is the undisputed standard for assessing the existence and intensity of pain (APS, 2016; Herr, Coyne, McCaffery, et al., 2011; McCaffery, Herr, & Pasero, 2011). Self-report is considered the most reliable measure of the existence and intensity of the patient's pain and is recommended by The Joint Commission (Baker, 2017). Accepting and acting on the patient's report of pain are sometimes difficult. Because pain cannot be proven, clinicians may feel vulnerable to inaccurate or untruthful reports of pain. Although clinicians are entitled to their personal opinions, those thoughts cannot interfere with appropriate patient care. [Chart 9-1](#) provides strategies to use when the patient's report of pain is not accepted.

Chart 9-1

Strategies to Use When the Patient's Report of Pain Is Not Accepted

- Acknowledge that everyone is entitled to a personal opinion, but personal opinion does not form the basis for professional practice.
- Clarify that the sensation of pain is subjective and cannot be proved or disproved.
- Quote recommendations from clinical practice guidelines, especially those published by the American Pain Society.
- Ask, "Why is it so difficult to believe that this person hurts?"

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Quality and Safety Nursing Alert

Although accepting and responding to the report of pain may result in administering analgesic agents to an occasional patient who does not have pain, doing so helps to ensure that everyone who does have pain receives appropriate care. Health care professionals do not have the right to deprive any patient of appropriate assessment and treatment simply because they believe a patient is not being truthful. Pain is an extremely personal experience manifested uniquely by each person. It is important to carefully assess and reassess pain when administering analgesic medications.

Performing the Comprehensive Pain Assessment: Patient Interview

A comprehensive pain assessment should be conducted during the admission assessment or initial interview with the patient, with each new report of pain, and whenever indicated by changes in the patient's condition or treatment plan. It serves as the foundation for developing and evaluating the effectiveness of the pain treatment plan. The following are components of a comprehensive pain assessment and tips on how to elicit the information from the patient:

- *Location(s) of pain:* Ask the patient to state or point to the area(s) of pain on the body. Sometimes allowing patients to make marks on a body diagram is helpful in gaining this information.
- *Intensity:* Ask the patient to rate the severity of the pain using a reliable and valid pain assessment tool. [Chart 9-2](#) provides guidance for educating patients and their families on how to use a pain rating scale. Various scales translated in several languages have been

evaluated and made available for use in clinical practice and for educational practice. The most common include the following:

Chart 9-2  **PATIENT EDUCATION**

Educating Patients and Their Families How to Use a Pain Rating Scale^a

Step 1. Show the pain rating scale to the patient and the family and explain its primary purpose.

Example: “This is a pain rating scale that many of our patients use to help us understand their pain and to set goals for pain relief. We will ask you regularly about pain, but any time you have pain you must let us know. We do not always know when you hurt.”

Step 2. Explain the parts of the pain rating scale. If the patient does not like it or understand it, switch to another scale (e.g., vertical presentation, VDS, or faces).

Example: “On this pain rating scale, 0 means no pain and 10 means the worst possible pain. The middle of the scale, around 5, means moderate pain. A 2 or 3 would be mild pain, but 7 or higher means severe pain.”

Step 3. Discuss pain as a broad concept that is not restricted to a severe and intolerable sensation.

Example: “Pain refers to any kind of discomfort anywhere in your body. Pain also means aching and hurting. Pain can include pulling, tightness, burning, knifelike feelings, and other unpleasant sensations.”

Step 4. Verify that the patient understands the broad concept of pain. Ask the patient to mention two examples of pain they have experienced. If the patient is already in pain that requires treatment, use the present situation as the example.

Example: “I want to be sure that I have explained this clearly, so would you give me two examples of pain you have had recently?” If the patient’s examples include various parts of the body and various pain characteristics, that indicates that they understand pain as a fairly broad concept. An example of what a patient might say is “I have a mild, sort of throbbing headache now, and yesterday my back was aching.”

Step 5. Ask the patient to practice using the pain rating scale with the present pain or select one of the examples mentioned.

Example: “Using the scale, what is your pain right now? What is it at its worst?” OR “Using the pain rating scale and one of your examples of pain, what is that pain usually? What is it at its worst?”

Step 6. Set goals for comfort and function/recovery/quality of life. Ask patients what pain rating would be acceptable or satisfactory, considering the activities required for recovery or for maintaining a satisfactory quality of life.

Example for a surgical patient: “I have explained the importance of coughing and deep breathing to prevent pneumonia and other complications. Now we need to determine the pain rating that will not interfere with this so that you may recover quickly.”

Example for patient with chronic pain or terminal illness: “What do you want to do that pain keeps you from doing? What pain rating would allow you to do this?”

^aWhen a patient is obviously in pain or not focused enough to learn to use a pain rating scale, pain treatment should proceed without pain ratings. Education can be undertaken when pain is reduced to a level that facilitates understanding how to use a pain scale.

VDS, verbal descriptor scale.

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- *Numeric Rating Scale (NRS)*: The NRS is most often presented as a horizontal 0- to 10-point scale, with word anchors of “no pain” at one end of the scale, “moderate pain” in the middle of the scale, and “worst possible pain” at the end of the scale. It may also be put on a vertical axis, which may be helpful for patients who read from right to left.
- *Wong–Baker FACES Pain Rating Scale*: The FACES scale consists of six cartoon faces with word descriptors, ranging from a smiling face on the left for “no pain (or hurt)” to a frowning, tearful face on the right for “worst pain (or hurt).” Patients are asked to choose the face that best reflects their pain. The faces are most commonly numbered using a 0, 2, 4, 6, 8, 10 metric, although 0 to 5 can also be used. Patients are asked to choose the face that best describes their pain. The FACES scale is used in adults and children as young as 3 years (McCaffery et al., 2011). It is important to appreciate that FACES scales are self-report tools; clinicians should not attempt to match a face shown on a scale to the patient’s facial expression to determine pain intensity. Patients may be able to understand the tool better if it is displayed vertically with no pain as the anchor at the bottom.
- *Faces Pain Scale—Revised (FPS-R)*: The FPS-R has six faces to make it consistent with other scales using the 0 to 10 metric. The faces range from a neutral facial expression to one of intense pain and are numbered 0, 2, 4, 6, 8, and 10. As with the Wong–Baker FACES scale, patients are asked to choose the face that best reflects their pain. Faces scales have been shown to be reliable and valid measures in children as young as 3 years of age; however, the ability to optimally quantify pain (identify a number) is not acquired until approximately 8 years of age (Spagrud, Piira, & Von Baeyer, 2003). Ongoing research suggests that the FPS-R is preferred by both patients who are cognitively intact and older adults who are cognitively impaired, and by minority populations (Kang & Demiris, 2018).

- *Verbal descriptor scale (VDS)*: A VDS uses different words or phrases to describe the intensity of pain, such as “no pain, mild pain, moderate pain, severe pain, very severe pain, and worst possible pain.” The patient is asked to select the phrase that best describes pain intensity.
- *Visual Analogue Scale (VAS)*: The VAS is a horizontal (sometimes vertical) 10-cm line with word anchors at the extremes, such as “no pain” on one end and “pain as bad as it could be” or “worst possible pain” on the other end. Patients are asked to make a mark on the line to indicate intensity of pain, and the length of the mark from “no pain” is measured and recorded in centimeters or millimeters. Although often used in research, the VAS is impractical for use in daily clinical practice and rarely used in that setting.
- *Quality*: Ask the patient to describe how the pain feels. Descriptors such as “sharp,” “shooting,” or “burning” may help identify the presence of neuropathic pain.
- *Onset and duration*: Ask the patient when the pain started and whether it is constant or intermittent.
- *Aggravating and relieving factors*: Ask the patient what makes the pain worse and what makes it better.
- *Effect of pain on function and quality of life*: The effect of pain on the ability to perform recovery activities should be regularly evaluated in the patient with acute pain. It is particularly important to ask patients with persistent pain about how pain has affected their lives, what they could do before the pain began that they can no longer do, or what they would like to do but cannot do because of the pain.
- ***Comfort–function goal*** (pain intensity): For patients with acute pain, identify short-term functional goals and reinforce to the patient that good pain control will more likely lead to successful achievement of the goals. For example, surgical patients are told that they will be expected to ambulate or participate in physical therapy postoperatively. Patients with chronic pain can be asked to identify their unique functional or quality-of-life goals, such as being able to work or walk the dog. Success is measured by progress toward meeting those functional goals (Topham & Drew, 2017).
- *Other information*: The patient’s culture, past pain experiences, and pertinent medical history such as comorbidities, laboratory tests, and diagnostic studies are considered when establishing a treatment plan.

Patients who are unable to report their pain are at higher risk for undertreated pain than those who can report (Hargas, 2017; McCaffery et al., 2011). In the adult population, this includes patients who are cognitively impaired, critically ill (intubated, unresponsive), comatose, or imminently dying. Patients who are receiving neuromuscular blocking agents or are sedated from anesthesia and other medications given during surgery are also among this at-risk population.

The Hierarchy of Pain Measures is recommended as a framework for assessing pain in patients who are nonverbal (Herr et al., 2011; McCaffery et al., 2011). The key components of the hierarchy require the nurse to (1) attempt to obtain self-report, (2) consider underlying pathology or conditions and procedures that might be painful (e.g., surgery), (3) observe behaviors, (4) evaluate physiologic indicators, and (5) conduct an analgesic trial. [Chart 9-3](#) provides detailed information on each component of the Hierarchy of Pain Measures.

When patients cannot self-report their pain, some observational tools may be used to help with clinical decision making. Some of these assign a score by observing behaviors that tend to be associated with pain. These observational scores are not considered equivalent to a patient's self-reported pain intensity score, however.

It is imperative to remember that behaviors can indicate the presence of pain, but the absence of behavior does not indicate the absence of pain. Patients who are not moving or making any sounds may still be experiencing intense pain. For instance, older adults with moderate and severe dementia frequently have comorbid disorders that may cause pain (Mueller, Schumacher, Holzer, et al., 2017). Accurately assessing and treating their pain can be difficult to achieve (see [Chart 9-4](#) Nursing Research Profile: Evaluation of a Tool to Assess Pain in Older Adults with Dementia). The following tools are examples of validated measures that are appropriate for different populations of patients unable to self-report their pain (Fry & Elliott, 2018; Kochman, Howell, Sheridan, et al., 2017; Rijkenberg, Stilma, Bosman, et al., 2017; Schofield & Abdulla, 2018).

Chart 9-3

Hierarchy of Pain Measures

1. Attempt to obtain the patient's self-report, the single most reliable indicator of pain. Do not assume that a patient cannot provide a report of pain; many patients who are cognitively impaired are able to use a self-report tool if simple actions are taken.
 - Try using standard pain assessment tools (see text).
 - Increase the size of the font and other features of the scale.
 - Present the tool in vertical format (rather than the frequently used horizontal).
 - Try using alternative words, such as "ache," "hurt," and "sore," when discussing pain.
 - Ensure eyeglasses and hearing aids are functioning.
 - Ask about pain in the present.
 - Repeat instructions and questions more than once.
 - Allow ample time to respond.
 - Remember that head nodding and eye blinking or squeezing the eyes tightly can also be used to signal presence of pain and sometimes used to rate intensity.
 - Ask patients who are intubated and who are awake and oriented to point to a number on the numerical scale if they are able.
 - Repeat instructions and show the scale each time pain is assessed.
2. Consider the patient's condition or exposure to a procedure that is thought to be painful. If appropriate, assume pain is present and document as such when approved by institution policy and procedure. As an example, pain should be assumed to be present in a patient who is unresponsive, mechanically ventilated, and critically ill due to trauma.
3. Observe behavioral signs, for example, facial expressions, crying, restlessness, and changes in activity. A pain behavior in one patient may not be in another. Try to identify pain behaviors that are unique to the patient ("pain signature"). Many behavioral pain assessment tools are available that will yield a pain behavior score and may help determine whether pain is present. However, it is important to remember that a behavioral score is not the same as a pain intensity score. Behavioral tools are used to help identify the presence of pain, but the pain intensity is unknown if the patient is unable to provide it.
 - A surrogate who knows the patient well (e.g., parent, spouse, or caregiver) may be able to provide information about underlying painful pathology or behaviors that may indicate pain.
4. Evaluate physiologic indicators with the understanding that they are the *least* sensitive indicators of pain and may signal the existence of conditions other than pain or a lack of it (e.g., hypovolemia, blood loss). Patients quickly adapt physiologically despite pain and may have normal or below normal vital signs in the presence of severe pain. The overriding principle

is that the absence of an elevated blood pressure or heart rate does not mean the absence of pain.

5. Conduct an analgesic trial to confirm the presence of pain and to establish a basis for developing a treatment plan if pain is thought to be present. An analgesic trial involves the administration of a low dose of nonopioid or opioid and observing patient response. The initial low dose may not be enough to elicit a change in behavior and should be increased if the previous dose was tolerated, or another analgesic agent may be added. If behaviors continue despite optimal analgesic doses, other possible causes should be investigated. In patients who are completely unresponsive, no change in behavior will be evident and the optimized dose of the analgesic agent should be continued.

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Adapted from Pasero, C. (2009). Challenges in pain assessment. *Journal of PeriAnesthesia Nursing*, 24(1), 50–54; Pasero, C., & McCaffery, M. (2011). *Pain assessment and pharmacologic management*. St. Louis, MO: Mosby-Elsevier.

Chart 9-4 NURSING RESEARCH PROFILE

Evaluation of a Tool to Assess Pain in Older Adults with Dementia

Mueller, G., Schumacher, P., Holzer, E., et al. (2017). The inter-rater reliability of the observation instrument for assessing pain in elderly with dementia: An investigation in the long-term care setting. *Journal of Nursing Measurement*, 25(3), E173–E184.

Purpose

The aim of this study was to examine the interrater reliability of the German version of the observation instrument for assessing pain in older adults with dementia called the BISAD for the German (Beobachtungsinstrument für das Schmerzassessment bei alten Menschen mit Demenz). This is a tool that is commonly used to assess pain among older adults with moderate and severe dementia who reside in long-term care facilities in both Germany and Austria; however, interrater reliability was not previously done to ensure its validity.

Design

This study used a quantitative multicenter-descriptive cross-sectional design with a convenience sample of 71 participants who resided in one of three nursing homes in Austria. The nursing participants consisted of 46 registered nurses who had been working at one of the same three nursing homes for at least 2 y. Nurse participants were paired to independently evaluate a resident participant within the same hour using the eight-item BISAD observational pain assessment tool.

Findings

Although modest agreement between raters was noted, the absolute concordance of the total was only 25.32%. The analysis of interrater reliability was low and did not support reliability of the items in the BISAD. There was agreement that pain was greater with movement than when the participant residents were at rest.

Nursing Implications

Findings of this study are important since they indicate the items used in the BISAD tool are not reliable for assessing pain in older adult residents with moderate and severe dementia in nursing homes in Austria, although it is commonly used. This is an important finding since use of this tool with that population could yield inaccurate data upon which nurses could base pain management care. One interesting aspect of this study was that behaviors did demonstrate greater pain with activity than when at rest. Finally, the authors note that there may be cultural and language issues that affected the results and encourage a translated version of the tool be assessed in long-term care facilities where English is the primary language.

- FLACC: indicated for use in young children. Scores are assigned after assessing *Facial expression, Leg movement, Activity, Crying, and Consolability*, with each of these five categories assigned scores from 0 to 2, yielding a total composite score of 0 to 10. Scores of “0” are interpreted as reflecting that the patient is relaxed and comfortable, scores of “1” to “3” are interpreted as consistent with mild discomfort, scores from “4” to “6” are considered consistent with moderate pain, and scores from “7” to “10” are considered consistent with severe discomfort or pain.
- PAINAD (*Pain Assessment IN Advanced Dementia*): indicated for use in adults with advanced dementia who are not able to verbalize their needs. Patterned after the FLACC, this tool was developed by the U.S. Department of Veterans Affairs for patients who have dementia.
- CPOT (*Critical Care Pain Observation Tool*): indicated for use in patients in critical-care units who cannot self-report pain, whether or not they may be intubated. It is also patterned after the FLACC.

Reassessing Pain

Following initiation of the pain management plan, pain is reassessed and documented on a regular basis to evaluate the effectiveness of treatment. At a minimum, pain should be reassessed with each new report of pain and before and after the administration of analgesic agents (McCaffery et al., 2011). The frequency of reassessment depends on the stability of the patient and the timing of peak effect of the medication administered, which is generally between 15 and 30 minutes following parenteral administration and between 1 and 2 hours following oral administration (Chou, Gordon, de Leon-Casasola, et al., 2016). For example, in the postanesthesia care unit (PACU), reassessment may be necessary as often as every 10 minutes when pain is unstable during intravenous (IV) opioid **titration** but may be done an hour following administration of oral medication in patients with satisfactory and stable pain control the day following surgery.



Veterans Considerations

Nurses should be aware that research has demonstrated a high prevalence of pain associated disorders, including arthritis, fibromyalgia, headaches and generalized abdominal, back, and joint pain in veteran populations (Nahin, 2017). In addition, reports of severe pain are more common in military veterans compared to nonveterans, especially in those who served, more recently, in conflicts in Iraq and Afghanistan (Nahin, 2017). In particular, younger veterans (18 to 39 years of age) and male veterans report significantly

higher levels of pain compared to matched age groups and men in the general public (Nahin, 2017). As a result, it is important for the nurse to determine during assessment if a patient has served in the U.S. military, to recognize that this group may have unique needs related to their service, and to advocate for multimodal and multidisciplinary approaches to help veterans better cope with pain. For example, Groessi, Liu, Change, and colleagues (2017) found that yoga was a safe and beneficial intervention in helping veterans to reduce pain and disability, while taking fewer opioid medications.

Pain Management

Achieving optimal pain relief is best viewed on a continuum, with the primary objective being to provide both effective and safe analgesia (Pozek, De Ruyter, & Khan, 2018). The quality of pain control should be addressed whenever patient care is passed on from one clinician to another, such as at change of shift and transfer from one clinical area to another. Optimal pain relief is the responsibility of *every* member of the health care team and begins with titration of the analgesic agent, followed by continued prompt assessment, analgesic agent administration, and nonpharmacologic interventions during the course of care to safely achieve pain intensities that allow patients to meet their functional goals with relative ease.

Although it may not always be possible to achieve a patient's pain intensity goal within the short time the patient is in an area like the PACU or emergency department, this goal provides direction for ongoing analgesic care. Important information to provide during transfer report is the patient's comfort–function goal, how close the patient is to achieving it, what has been done thus far to achieve it (analgesic agents and doses and/or nonpharmacologic interventions), and how well the patient has tolerated administration of the analgesic agent (adverse effects). There is growing interest among both clinicians and researchers in linking pain management to functional goals. One effort in this work is the Clinically Aligned Pain Assessment (CAPA) Tool, which is used to assess various degrees of comfort, pain control, function, and sleep (Topham & Drew, 2017). Pain management interventions should improve and not inhibit progress toward healing and rehabilitation.

Pharmacologic Management of Pain: Multimodal Analgesia

Pain is a complex phenomenon involving multiple underlying mechanisms that requires more than one analgesic agent to manage it safely and effectively. The recommended approach for the treatment of all types of pain in all age groups is called **multimodal analgesia or multimodal pain management**. A

multimodal regimen intentionally and simultaneously combines medications with different underlying mechanisms, along with nonpharmacologic interventions, which allows for lower doses of each of the medications in the treatment plan, reducing the potential for adverse effects. Furthermore, multimodal analgesia can result in comparable or greater pain relief with fewer adverse effects than can be achieved with any single analgesic agent (Beverly, Kaye, Ljungqvist, et al., 2017; Blackburn, 2018).

Routes of Administration

Oral is the preferred route of analgesic administration and should be used whenever feasible (Chou et al., 2016). Medications administered via the oral route are generally best tolerated, easiest to administer, and most cost-effective. When the oral route is not possible, such as when patients cannot swallow, are NPO (nothing by mouth), or nauseated, other routes of administration are used. For example, patients with cancer pain who are unable to swallow may take analgesic agents by the transdermal, rectal, or subcutaneous route of administration (Burchum & Rosenthal, 2019).

In the immediate postoperative period, the IV route is most often the first-line route of administration for analgesic delivery, and patients are transitioned to the oral route as tolerated (see [Chapter 16](#) for the management of postoperative pain).

The rectal route of analgesic administration is an alternative route when oral or IV analgesic agents are not an option (e.g., for palliative purposes during end-of-life care). The rectum allows passive diffusion of medications and absorption into the systemic circulation. This route can be less expensive and does not involve the skill and expertise required of the parenteral route of administration. Limitations are that medication absorption can be unreliable and depends on many factors including rectal tissue health and administrator technique. Some patients may be resistant to or fearful of rectal administration. The rectal route is contraindicated in patients who are neutropenic or thrombocytopenic because of potential rectal bleeding. Diarrhea, perianal abscess or fistula, and abdominoperineal resection are also relative contraindications (Burchum & Rosenthal, 2019).

The topical route of administration is used for both acute and chronic pain. For example, the nonopioid diclofenac is available in patch and gel formulations for application directly over painful areas. Local anesthetic creams, such as eutectic mixture or emulsion of local anesthetics and lidocaine cream 4%, can be applied directly over the injection site prior to painful needle stick procedures; the lidocaine patch 5% is often used for well-localized types of neuropathic pain, such as postherpetic neuralgia. It is important to distinguish between topical and transdermal medication delivery. Although both routes require the medication to cross the stratum corneum to produce analgesia, transdermal delivery requires absorption into the systemic

circulation to achieve effects, whereas topical agents produce effects in the tissues immediately under the site of application (referred to as targeted peripheral analgesia). Compounding pharmacies may be consulted to custom blend antispasmodic agents, such as topical morphine or gabapentin, for topical application at the painful site.

A more invasive method used to manage pain is accomplished using **neuraxial** analgesia, which involves administering medication in the epidural or subarachnoid space (American Society of Regional Anesthesia and Pain Medicine, 2016). Delivery of analgesic agents by the neuraxial route is accomplished by inserting a needle into the subarachnoid space (for intrathecal [spinal] analgesia) or the epidural space, and either injecting the analgesic medication directly, or threading a catheter through the needle to enable bolus dosing or continuous administration (Conlin, Grant, & Wu, 2018; Hernandez, Grant, & Wu, 2018). Intrathecal catheters for acute pain management are used most often for providing anesthesia or a single bolus dose of an analgesic agent. Implanted intrathecal pumps deliver very small amounts of medication in a constant infusion for treatment of end-of-life pain or persistent pain (Jamison, Cohen, & Rosenow, 2018). Temporary epidural catheters for acute pain management are removed after 2 to 4 days. Epidural analgesia is administered by clinician-given bolus, continuous infusion (basal rate), and patient-controlled epidural analgesia (PCEA). The most common opioids given intraspinally are morphine, fentanyl, and hydromorphone. These are often combined with a local anesthetic, most often ropivacaine or bupivacaine (Jamison et al., 2018). The multimodal use of local anesthetics with opioids improves analgesia and produces an **opioid dose–sparing effect**.

A pain management technique that involves the use of an indwelling catheter is the continuous peripheral nerve block (also called *perineural anesthesia*), whereby an initial local anesthetic block is established and followed by the placement of a catheter or catheters through which an infusion of local anesthetic, usually ropivacaine or bupivacaine, is infused continuously to the targeted site of innervation. The effect of local anesthetic is dose dependent: at lower doses, the smaller sensory nerve fibers are affected before the larger motor fibers. Patients thus medicated are able to walk but have well controlled pain (Burchum & Rosenthal, 2019; Ilfeld & Mariano, 2018).

Dosing Regimen

Achieving, then maintaining, optimal pain management that is safe, effective, and progresses toward realistic functional goals requires patient education with continuing reassessment of analgesic effect and development of any untoward effects (Chou et al., 2016). Accomplishing these goals may require the mainstay analgesic agent to be given on a scheduled around-the-clock (ATC) basis, rather than PRN (as needed) to maintain stable analgesic blood levels when pain is continuous (Eksterowicz & DiMaggio, 2018). ATC dosing

regimens are designed to control pain for patients who report pain being present 12 hours or more during a 24-hour period. PRN dosing of analgesic agents is appropriate for intermittent pain, such as prior to painful procedures and for BTP (pain that “breaks through” the pain being managed by the mainstay analgesic agent), for which supplemental doses of analgesia are provided (Palat, 2018).

Patient-Controlled Analgesia

Patient-controlled analgesia (PCA) is an interactive method of pain management that allows patients to treat their pain by self-administering doses of analgesic agents (Burchum & Rosenthal, 2019). It is used to manage all types of pain by multiple routes of administration, including oral, IV, subcutaneous, epidural, and perineural (Fernandes, Hernandes, de Almeida, et al., 2017). Current guidelines from the APS, the American Society of Regional Anesthesia and Pain Medicine, and the American Society of Anesthesiologists strongly recommended IV PCA for postoperative pain management when it is necessary to use the parenteral route to deliver analgesic medications (Chou et al., 2016). A PCA infusion device is programmed so that the patient can press a button (pendant) to self-administer a dose of an analgesic agent (PCA dose) at a set time interval (demand or lockout) as needed. Patients who use PCAs must be able to understand the relationships among pain, pushing the PCA button or taking the analgesic agent, and pain relief, and must be cognitively and physically able to use any equipment that is necessary to administer the therapy (ECRI Institute Patient Safety Organization, 2017).

A basal rate (continuous infusion) may be used for patients who are opioid tolerant, and when PCEA is used. It is discouraged for patients who are opioid naïve and receiving IV PCA due to the risk of oversedation with subsequent respiratory depression (Chou et al., 2016; ECRI Institute Patient Safety Organization, 2017). Essential to the safe use of a basal rate with PCA is close monitoring by nurses of sedation and respiratory status and prompt decreases in opioid dose (e.g., discontinue basal rate) if increased sedation is detected (Pasero, Quinn, Portenoy, et al., 2011).

The primary benefit of PCA is that it recognizes that only the patient can feel the pain and only the patient knows how much analgesic will relieve it. This reinforces that PCA is for patient use only and that unauthorized activation of the PCA device by anyone other than the patient (PCA by proxy) should be discouraged (Burchum & Rosenthal, 2019).



Quality and Safety Nursing Alert

Staff, family, and other visitors should be instructed to contact the nurse if they have concerns about pain control rather than activating the PCA device for the patient.

However, for some patients who are candidates for PCA but unable to use the PCA equipment, the nurse or a capable family member may be authorized to manage the patient's pain using PCA equipment. This is referred to as Authorized Agent Controlled Analgesia; guidelines are available for the safe administration of this therapy (Cooney, Czarnecki, Dunwoody, et al., 2013).

Analgesic Medications

Analgesic medications are categorized into three main groups: (1) nonopioid antispasmodic agents, which include acetaminophen and NSAIDs; (2) opioid antispasmodic agents, which include, among others, morphine, hydromorphone, fentanyl, and oxycodone; and (3) co-analgesic agents (also referred to as **adjuvant analgesic agents**). The co-analgesic agents comprise the largest group and include various agents with unique and widely differing mechanisms of action. Examples are local anesthetics, some anticonvulsants, and some antidepressants (APS, 2016).

Nonopioid Analgesic Agents

Acetaminophen and NSAIDs comprise the group of nonopioid analgesic agents (refer to earlier discussion of the two categories of NSAIDs; see Fig. 9-2).

Indications and Administration

Nonopioid medications are analgesic agents used for a wide variety of painful conditions. They are appropriate alone for mild to some moderate nociceptive pain (e.g., from surgery, trauma, or osteoarthritis) and are added to opioids, local anesthetics, and/or anticonvulsants as part of a multimodal analgesic regimen for more severe nociceptive pain (APS, 2016; Chou et al., 2016; Comerford & Durkin, 2020). Since acetaminophen and NSAIDs have different mechanisms of action, they may be administered concomitantly (Chou et al., 2016). Although there is no research supporting staggering the two medications, it may be helpful for some patients. Unless contraindicated, surgical patients should routinely be given acetaminophen and an NSAID in scheduled doses throughout the postoperative course, which can be initiated preoperatively (Chou et al., 2016).

Nonopioids are often combined in a single tablet with opioids, such as oxycodone or hydrocodone, and are very popular for the treatment of mild to moderate acute pain. They are traditionally a common choice after invasive pain management therapies are discontinued and for pain treatment after

hospital discharge and dental surgery when an opioid is prescribed. Many people with persistent pain also take a combination nonopioid–opioid analgesic agent; however, it is important to remember that these combination medications are not appropriate for severe pain of any type because the maximum daily dose of the nonopioid limits the escalation of the opioid dose (Burchum & Rosenthal, 2019; Comerford & Durkin, 2020).

Acetaminophen is versatile in that it can be given by multiple routes of administration, including oral, rectal, and IV. Oral acetaminophen has a long history of safety in recommended doses in all age groups. It is a useful addition to multimodal treatment plans for postoperative pain (Wick, Grant, & Wu, 2017). Findings from one research study suggest that patients who receive scheduled acetaminophen with PRN opioids will use less opioids than if they receive PRN acetaminophen plus opioids (Valentine, Carvalho, Lazo, et al., 2015). These results were supported in a more recent study evaluating opioid use among women who underwent Cesarean deliveries (Holland, Bateman, Cole, et al., 2019).

IV acetaminophen is approved for the treatment of pain and fever and is given by a 15-minute infusion in single or repeated doses. It may be given alone for mild to moderate pain or in combination with opioid analgesic agents for more severe pain. The results of several research studies have been inconsistent regarding the opioid sparing effects of IV acetaminophen (Nelson & Wu, 2018). Recommended dosing is 1000 mg every 6 hours for a maximum of 4000 mg in adult patients (Comerford & Durkin, 2020).

A benefit of the NSAID group is the availability of a wide variety of agents for administration via noninvasive routes. Ibuprofen, naproxen, and celecoxib are the most widely used oral NSAIDs in the United States. When rectal formulations are unavailable, an intact oral tablet or a crushed tablet in a gelatin capsule may be inserted into the rectum. The rectal route may require higher doses than the oral route to achieve similar analgesic effects (Pasero et al., 2011). Diclofenac can be prescribed in patch and gel form for topical administration, and an intranasal patient-controlled formulation of ketorolac has been approved for the treatment of postoperative pain.

IV formulations of ketorolac and ibuprofen are available for acute pain treatment. Both have been shown to produce excellent analgesia alone for moderate nociceptive pain, and significant opioid dose–sparing effects when given as part of a multimodal analgesia plan for more severe nociceptive pain (Comerford & Durkin, 2020; Williams, 2018).

Adverse Effects of Nonopioid Analgesic Agents

Acetaminophen is widely considered one of the safest, best tolerated, and most cost effective of the analgesic agents (APS, 2016; Williams, 2018). Its most serious complication is hepatotoxicity (liver damage) as a result of overdose. In the healthy adult, a maximum daily dose below 4000 mg is rarely associated

with liver toxicity. Nevertheless, one manufacturer of oral acetaminophen voluntarily changed its dosing recommendations in 2011, calling for a maximum daily dose of 3000 mg (Shiffman, Battista, Kelly, et al., 2018). In 2014, the U.S. Food and Drug Administration (FDA) recommended that health care professionals stop prescribing and pharmacists stop dispensing prescription combination medication products that contain more than 325 mg of acetaminophen per tablet, capsule, or other dosage unit in order to reduce the risk of hepatotoxicity (FDA, 2014). Acetaminophen does not increase bleeding time and has a low incidence of gastrointestinal (GI) adverse effects, making it the analgesic agent of choice in many individuals with comorbidities. There are two potential interactions with acetaminophen that warrant caution. Acetaminophen should be avoided when consuming alcohol because the combination can result in serious liver damage; acetaminophen also should be avoided when warfarin is prescribed because it can inhibit metabolism of warfarin resulting in toxicity with bleeding risk (Burchum & Rosenthal, 2019).

NSAIDs have considerably more adverse effects than acetaminophen, with gastric toxicity and ulceration being the most common (Comerford & Durkin, 2020). The primary underlying mechanism of NSAID-induced gastric ulceration is the inhibition of COX-1, which leads to a reduction in GI-protective prostaglandins (see Fig. 9-2). This is a systemic (rather than local) effect and can occur regardless of the route of administration of the NSAID. Risk factors include advanced age (older than 60 years), presence of prior ulcer disease, and cardiovascular (CV) disease and other comorbidities (Williams, 2018). In patients with elevated risks, the use of a COX-2 selective NSAID (e.g., celecoxib) or the least ulcerogenic nonselective NSAID (e.g., ibuprofen) plus a proton pump inhibitor is recommended; however, there are risks with proton pump inhibitors as well (Gwee, Goh, Lima, et al., 2018). Proton pump inhibitors may decrease the absorption of some other medications such as itraconazole and rilpivirine (Burchum & Rosenthal, 2019). As with all medications, it is important to frequently reassess the need for continued use and to discontinue when appropriate. GI adverse effects are also related to the dose and duration of NSAID therapy; the higher the NSAID dose and the longer the duration of NSAID use, the higher the risk of GI toxicity (Williams, 2018). A principle of nonopioid analgesic use is to administer the lowest dose for the shortest time necessary (Pasero, Portenoy, & McCaffery, 2011).

All NSAIDs carry a risk of CV adverse effects through prostaglandin inhibition, and the risk is increased with COX-2 inhibition, whether it is produced by a COX-2 selective NSAID (e.g., celecoxib) or by NSAIDs that are nonselective inhibitors of both COX-1 and COX-2 (e.g., ibuprofen, naproxen, and ketorolac). Findings from a recent meta-analysis suggest that the risk of acute myocardial infarction is greatest during the first month of treatment with NSAIDs, with this risk developing during the first week of

treatment (Bally, Dendukuri, Rich, et al., 2017). All patients prescribed NSAIDs should receive the lowest effective dose for the shortest time period to decrease risks.

NSAIDs can negatively impact renal function, and are associated with diminished renal prostaglandin formation, interstitial nephritis, reduced secretion of renin, and greater reabsorption of water and sodium (APS, 2016). NSAID-induced renal toxicity can occur, but is relatively rare in otherwise healthy adults who are given NSAIDs for short-term pain management (e.g., in the perioperative period); however, individuals with acute or chronic volume depletion or hypotension rely on prostaglandin synthesis to maintain adequate renal blood flow, and NSAID inhibition of prostaglandin synthesis in such patients can cause acute kidney injury (Burchum & Rosenthal, 2019). Attention to adequate hydration is essential when administering NSAIDs to prevent this complication (Pasero et al., 2011).

Most nonselective NSAIDs increase bleeding time through inhibition of COX-1. This is both medication and dose related, so the lowest dose of nonopioids with minimal or no effect on bleeding time should be used in patients having procedures with high risk for bleeding. Options include acetaminophen, celecoxib, choline magnesium trisalicylate, salsalate, and nabumetone (APS, 2016; Burchum & Rosenthal, 2019).

Opioid Analgesic Agents

Although it is often used, the term *narcotic* is inaccurate and considered obsolete when discussing the use of **opioids** for pain management, in part because it is a term used loosely by law enforcement and the media to refer to various substances of potential abuse, which include opioids as well as cocaine and other illicit substances. Legally, controlled substances classified as narcotics include opioids, cocaine, and others. The accurate term, when discussing these agents in the context of pain management, is *opioid analgesics* (Burchum & Rosenthal, 2019).

Opioid analgesic agents are divided into two major groups: (1) **mu agonist** opioids (also called *morphinelike medications*) and (2) **agonist–antagonist** opioids. The mu agonist opioids comprise the larger of the two groups and include morphine, hydromorphone, hydrocodone, fentanyl, oxycodone, and methadone, among others. The agonist–antagonist opioids include buprenorphine, nalbuphine, and butorphanol (APS, 2016).

Opioid analgesic agents exert their effects by interacting with opioid receptor sites located throughout the body, including in the peripheral tissues, GI system, and CNS; they are abundant in the dorsal horn of the spinal cord. There are three major classes of opioid receptor sites involved in analgesia: the mu, delta, and kappa. The pharmacologic differences in the various opioids are the result of their interaction with these opioid receptor types (Burchum & Rosenthal, 2019; Sheth, Holtsman, & Mahajan, 2018). When an opioid binds

to the opioid receptor sites, it produces analgesia as well as unwanted effects, such as constipation, nausea, sedation, and respiratory depression (Arthur & Hui, 2018).

The opioid analgesic agents that are designated as first line (e.g., morphine, hydromorphone, fentanyl, and oxycodone) belong to the mu opioid agonist class because they bind primarily to the mu-type opioid receptors. The agonist–antagonist opioids are designated as “mixed” because they bind to more than one opioid receptor site. They bind as **agonists**, producing analgesia, at the kappa opioid receptor sites, and as weak antagonists at the mu opioid receptor sites. Their propensity to antagonize the effects of mu opioid analgesic agents limits their usefulness in pain management (Burchum & Rosenthal, 2019). They should be avoided in patients receiving long-term mu opioid therapy because their use may trigger severe pain and opioid **withdrawal** syndrome characterized by rhinitis, abdominal cramping, nausea, agitation, and restlessness.

Antagonists (e.g., naloxone, naltrexone, naloxegol) are medications that also bind to opioid receptors but produce no analgesia. If an antagonist is present, it competes with opioid molecules for binding sites on the opioid receptors and has the potential to block analgesia and other effects. Antagonists are used most often to reverse adverse effects, such as respiratory depression (Burchum & Rosenthal, 2019). Antagonists have been incorporated in the manufacture of some opioids in an effort to deter abuse of the opioid (Li, 2019)

Administration

Safe and effective use of opioid analgesic agents requires the development of an individualized treatment plan based on a comprehensive pain assessment, which includes clarifying the goals of treatment and discussing options with the patient and the family when appropriate (Chou et al., 2016; Sheth et al., 2018). Goals are periodically reevaluated, and changes made depending on patient response and in some cases disease progression.

Many factors are considered when determining the appropriate opioid analgesic agent for the patient with pain. These include the unique characteristics of the various opioids and patient factors, such as pain intensity, age, coexisting disease, current medication regimen and potential medication interactions, prior treatment outcomes, and patient preference (APS, 2016; Sheth et al., 2018). In all cases, a multimodal approach that may rely on the selection of appropriate analgesic agents from the nonopioid, opioid, and co-analgesic agent groups is recommended to manage all types of pain (APS, 2016; Li, 2019). [Chart 9-5](#) lists the key considerations when developing an opioid pain treatment plan.

Titration of the opioid dose is usually required at the start and throughout the course of treatment when opioids are given. Whereas patients with cancer

pain most often are titrated upward over time for progressive pain, patients with acute pain, particularly postoperative pain, are eventually titrated downward and discontinued as pain resolves (Chou et al., 2016; FDA, 2017; Sheth et al., 2018). The dose and analgesic effect of mu agonist opioids have no **ceiling effect**, although the dose may be limited by adverse effects. The absolute dose given is based on a balance between pain relief and tolerability of adverse effects. The goal of titration is to use the smallest dose that provides satisfactory pain relief with the fewest adverse effects (Sheth et al., 2018). The time at which the dose can be increased is determined by the onset and peak effects of the opioid and its formulation.

Chart 9-5

Use of Opioids

- Perform a comprehensive assessment that addresses pain, comorbidities, and functional status.
- Develop an individualized treatment plan that includes specific goals related to pain intensity, activities (function/quality of life), and adverse effects (e.g., pain intensity rating of 3 on a 0–10 numerical rating scale to ambulate accompanied by minimal or no sedation).
- Use multimodal analgesia (e.g., add acetaminophen and NSAID; anticonvulsant in patients at risk for persistent postsurgical pain).
- Assess for presence preoperatively of underlying persistent pain in surgical patients and optimize its treatment.
- Consider **preemptive analgesic agents** before surgery, particularly for those at risk for severe postoperative pain or a persistent postsurgical pain syndrome.
- Provide analgesic agents prior to all painful procedures.
- Medication selection
 - Consider diagnosis, condition, or surgical procedure, current or expected pain intensity, age, presence of major organ dysfunction or failure, and presence of coexisting disease.
 - Consider pharmacologic issues (e.g., accumulation of metabolites and effects of concurrent medications).
 - Consider prior treatment outcomes and patient preference.
 - Be aware of available routes of administration (oral, transdermal, rectal, intranasal, IV subcutaneous, perineural, intraspinal) and formulations (e.g., short acting, modified release).
 - Be aware of cost differences.
- Route of administration selection
 - Use least invasive route possible.
 - Consider convenience and patient's ability to adhere to the regimen.
 - Consider staff's (or patient's or caregiver's) ability to monitor and provide care required.
- Dosing and titration
 - Consider previous dosing requirement and relative analgesic potencies when initiating therapy.
 - Use equianalgesic dose chart ([Table 9-3](#)) to determine starting dose with consideration of patient's current status (e.g., sedation and respiratory status) and comorbidities (e.g., medical frailty), and then titrate until adequate analgesia is achieved or dose-limiting adverse effects are encountered.
 - Use appropriate dosing schedule (e.g., around-the-clock for continuous pain; PRN for intermittent pain).
 - When dose is safe but additional analgesia is desired, titrate upward as prescribed by 25% for slight increase, 50% for moderate

- increase, and 100% for considerable increase in analgesia.
- Provide supplemental doses for breakthrough pain; consider PCA if appropriate.
 - Treatment of adverse effects
 - Be aware of the prevalence and impact of opioid adverse effects.
 - Remember that most opioid adverse effects are dose dependent; always consider decreasing the opioid dose as a method of treating or eliminating an adverse effect; adding nonopioid analgesic agents for additive analgesia facilitates this approach.
 - Use a preventive approach in the management of constipation, including for patients receiving short-term opioid treatment.
 - Prevent respiratory depression by monitoring sedation levels and respiratory status frequently and decreasing the opioid dose as soon as increased sedation is detected.
 - Monitoring
 - Continually and consistently evaluate the plan on the basis of the specific goals identified at the outset and assess pain intensity, adverse effects, and activity levels.
 - Make necessary modifications to treatment plan to maintain efficacy and safety.
 - Tapering and cessation of treatment
 - If a decrease in dose or cessation of treatment is appropriate, do so in accordance with decreased pain intensity and after evaluation of functional outcomes.
 - Be aware of the potential for withdrawal syndrome (rhinitis, abdominal cramping, diarrhea, restlessness, agitation) and need for tapering schedule in patients who have been receiving opioid therapy for more than a few days.

IV, intravenous; NSAID, nonsteroidal anti-inflammatory drug; PCA, patient-controlled analgesia; PRN, as needed.

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Equianalgesia. The term *equianalgesia* means approximately “equal analgesia.” An equianalgesic chart provides a list of doses of analgesic agents, both oral and parenteral (IV, subcutaneous, and intramuscular), that are approximately equal to each other in ability to provide pain relief. Equianalgesic conversion of doses is developed from the ratio representing the difference in the potency of the two medications (Treillet, Laurent, & Hadjat, 2018). The information is used to help ensure that patients are not overdosed or underdosed when they are switched from one opioid or route of administration to another. It requires a series of calculations based on the daily dose of the current opioid to determine the equianalgesic dose of the opioid to

which the patient is to be switched. Several excellent guidelines are available to assist in calculating equianalgesic doses (Burchum & Rosenthal, 2019) (see [Table 9-3](#)). Equianalgesic tools available in electronic health records enable all clinicians at any facility to easily convert analgesic dosages (APS, 2016).

TABLE 9-3 Equianalgesic Dose Chart for Common mu Opioid Analgesic Agents

- *Equianalgesic* means approximately the same pain relief.
- The equianalgesic chart is a guideline for selecting doses for patients who are opioid-naïve. Doses and intervals between doses are titrated according to individuals' responses.
- The equianalgesic chart is helpful when switching from one medication or route of administration to another.

Opioid	Oral	Parentral	Comments
Morphine	30 mg	10 mg	Standard for comparison; first-line opioid via multiple routes of administration; once- and twice-daily oral formulations; clinically significant metabolites
Fentanyl	No formulation	100 mcg IV 100 mcg/h of transdermal fentanyl is approximately equal to 4 mg/h of IV morphine; 1 mcg/h of transdermal fentanyl is approximately equal to 2 mg/24 h of oral morphine	First-line opioid via IV, transdermal, and intraspinal routes; available in oral transmucosal and buccal formulations for breakthrough pain in patients who are opioid-tolerant; no clinically relevant metabolites
Hydrocodone	30 mg (not recommended)	No formulation	Available only in combination with acetaminophen and as such is appropriate only for mild to some moderate pain
Hydromorphone	7.5 mg	1.5 mg	First-line opioid via multiple routes of administration; once-daily oral formulation; clinically significant metabolites noted with long-term and high-dose infusion
Oxycodone	20 mg	No formulation in the United States	Short-acting and twice-daily oral formulations
Oxymorphone	10 mg	1 mg	Parentral and short-acting and twice-daily oral formulations

IV, intravenous.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer; Pasero, C., & McCaffery, M. (2011). *Pain assessment and pharmacologic management*. St. Louis, MO: Mosby-Elsevier.

Formulation terminology. The terms *short acting*, *immediate release*, and *normal release* have been used interchangeably to describe oral opioids that have an onset of action of approximately 30 minutes and a relatively short duration of 3 to 4 hours. The term *immediate release* is misleading because none of the oral analgesic agents have an immediate onset of analgesia; *short acting* is preferred. The terms *modified release*, *extended release*, *sustained release*, *controlled release*, and *long acting* are used to describe opioids that are formulated to release over a prolonged period of time. For the purposes of this chapter, the term *modified release* will be used when discussing these opioid formulations.

Substance Use Disorder, Physical Dependence, and Tolerance

In 2013 the American Psychological Association (APA) renamed *addiction* as *substance use disorder* (SUD) (Lo Coco, Melchiori, Oiendi, et al., 2019). SUD includes a number of subcategories, including *opioid use disorder*. The terms *physical dependence* and *tolerance* often are confused with *substance use disorder*, previously understood as *addiction*; thus, clarification of definitions is important (Burchum & Rosenthal, 2019).

- **Physical dependence** is a normal response that occurs with repeated administration of the opioid, with intensity and duration dependent upon the half-life of the medication and how long it has been used. It is manifested by the occurrence of withdrawal symptoms when the opioid is suddenly stopped or rapidly reduced, or an antagonist such as naloxone is given. Withdrawal symptoms may be suppressed by the natural, gradual reduction of the opioid as pain decreases or by gradual, systematic reduction, referred to as tapering (Burchum & Rosenthal, 2019). Withdrawal occurs with prolonged use of opioids, regardless of whether the use of opioids is prescribed for pain management or because of SUD (Sheth et al., 2018).
- **Tolerance** is also a normal physiologic response that can occur with regular administration of an opioid and consists of a decrease in one or more effects of the opioid (e.g., decreased analgesia, sedation, or respiratory depression). Although it may occur in conjunction with SUD, it cannot be equated with SUD. It may be treated with increases in dose to attain the previous effect. With the exception of constipation, tolerance to the opioid adverse effects develops with regular daily dosing of opioids over several days (Burchum & Rosenthal, 2019; Sheth et al., 2018).

- **Substance Use Disorder (SUD)** was historically known as addiction or addictive disease, and defined as a chronic, relapsing, treatable neurologic disease. The APA has since described SUD as the impaired use of a substance, such as opioids, even while experiencing major problems, characterized by impaired control over use, compulsive use, continued use despite harm, and craving for the substance. With SUD, use of the opioid is for nontherapeutic reasons and is thus independent of pain relief. The development and characteristics of SUD are influenced by genetic, psychosocial, and environmental factors (Auriacombe, Serre, Denis, et al., 2019; Lo Coco et al., 2019; Sheth et al., 2018).
- **Withdrawal** occurs when a medication or substance to which the body has become dependent is abruptly reduced or discontinued. This is true of prescribed medications as well as illicitly obtained substances. Withdrawal is exhibited by a cascade of unpleasant symptoms including anxiety, nausea, vomiting, rhinitis, sneezing, chills, hot flashes, abdominal cramping, tremors, diaphoresis, hyperreflexia, diarrhea, piloerection, and/or insomnia (APS, 2016; Burchum & Rosenthal, 2019; Sheth et al., 2018).
- *Pseudoaddiction* is a mistaken diagnosis of substance use disorder that occurs when a patient's pain is not well controlled; the patient may begin to manifest symptoms suggestive of SUD. In an effort to obtain adequate pain relief, the patient may respond with demanding behavior, escalating demands for more or different medications, and repeated requests for opioids on time or before the prescribed interval between doses has elapsed. Pain relief typically eliminates these behaviors and is often accomplished by increasing opioid doses or decreasing intervals between doses (Sheth et al., 2018; Weissman & Haddox, 1989).

Pain management specialists have increasingly come to realize that the progression from prescribed opioid use to the disease of opioid SUD is poorly understood and complex. The National Institute on Drug Abuse (2014) estimated that the rates of SUD among patients with chronic pain vary widely, from 3% to 40%. A government effort to address the issue of SUD is the *Comprehensive Addiction and Recovery Act of 2016* which provides prevention, treatment, and rehabilitative support (Burchum & Rosenthal, 2019). There is real concern for adequately treating the 2 million Americans who are living with opioid SUD and the 50 million people who are living with chronic pain (National Institutes of Health [NIH], 2019). The patients in both groups need and deserve to receive informed, evidence-based, compassionate nursing care.

Opioid naïve versus opioid tolerant. Patients are often characterized as being either *opioid naïve* or *opioid tolerant*. Whereas an **opioid naïve** person

has not recently taken enough opioid on a regular basis to become tolerant to the effects of an opioid, an **opioid tolerant** person has taken an opioid long enough at doses high enough to develop tolerance to many of the effects, including analgesia and sedation. There is no set time for the development of tolerance, and there is great individual variation, with some not developing tolerance at all. By convention, most clinicians consider a patient who has taken opioids regularly for approximately 7 or more days to be opioid tolerant (Pasero et al., 2011).

Opioid-Induced Hyperalgesia

Opioid-induced hyperalgesia (OIH) is a paradoxical situation in which increasing doses of an opioid result in increasing sensitivity to pain. The incidence of clinically significant OIH has not been determined; however, it is a serious consequence of opioid administration. At this time, it is not possible to predict who will develop OIH as a result of opioid exposure, and the mechanisms underlying OIH are largely unknown. In general, OIH is thought to be the result of changes in the central and peripheral nervous systems that produce increased transmission of nociceptive signals (APS, 2016; Higgins, Smith, & Matthews, 2018; Ringkamp et al., 2018; Spofford & Hurley, 2018).

Some experts characterize OIH and analgesic tolerance as “opposite sides of the coin” (Pasero et al., 2011). In tolerance, increasing doses of opioid are needed to provide the same level of pain relief because opioid exposure induces neurophysiologic changes that reverse analgesia; in OIH, opioid exposure induces neurophysiologic changes that produce pain or increase sensitivity to noxious input (APS, 2016). In other words, tolerance may be inferred clinically when opioid treatment leads to decreased sensitivity to opioid analgesia over time (in the absence of another process that would explain this), whereas OIH may be inferred clinically when opioid treatment leads to increased pain or sensitivity to pain. Patients with OIH may report an increase in pain that increases with increased dosing of opioids (APS, 2016). Recent research findings have reported the onset of postoperative OIH following intraoperative administration of opioids (Spofford & Hurley, 2018). OIH is an area in which additional research is much needed.

Select Opioid Analgesic Agents

Morphine is the standard against which all other opioid medications are compared. It is used worldwide, particularly for cancer pain, and its use is established by extensive research and clinical experience. It is available in a wide variety of short-acting and modified-release oral formulations and is given by multiple routes of administration. It was the first medication to be given intraspinally and remains a first-line choice for long-term **intraspinal** analgesia. It is the only opioid uniquely formulated to produce analgesia for up to 48 hours following epidural administration for acute pain management (extended-release epidural morphine). Morphine is a **hydrophilic** medication

(readily absorbed in aqueous solution), which accounts for its slow onset and long duration of action when compared with other opioid analgesic agents ([Tables 9-3](#) and [9-4](#)). It has two principal, clinically significant **metabolites**: morphine-3-glucuronide (M3G) and morphine-6-glucuronide (M6G). M6G may be responsible for some of the analgesic effect of morphine; accumulation of M3G can produce neurotoxicity, which necessitates switching the patient to a different opioid (Burchum & Rosenthal, 2019; Conlin et al., 2018; Howard & Brant, 2019; Sheth et al., 2018).

TABLE 9-4 Characteristics of Select First-Line Opioid Analgesic Agents^a

Opioid	Onset (Minutes)	Peak (Minutes)	Duration (Hours)
Morphine	30 (PO)	60–120 (PO)	4–12 (PO)
	5 (IV)	20 (IV)	4–5 (IV)
Fentanyl	5–15 (OT)	20–30 (OT)	2–5 (OT)
	1–2 (IV)	3–5 (IV)	1/2–1 (IV)
Hydromorphone	15–30 (PO)	30–60 (PO)	4–5 (PO)
	10–15 (IV)	15–30 (IV)	2–3 (IV)

^aCharacteristics do not apply to modified-release formulations.

IV, intravenous; OT, oral transmucosal; PO, oral.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer; Pasero, C., & McCaffery, M. (2011). *Pain assessment and pharmacologic management*. St. Louis, MO: Mosby-Elsevier.

Fentanyl, in contrast to morphine, is a **lipophilic** (readily absorbed in fatty tissues) opioid and as such has a fast onset and short duration of action (see [Tables 9-3](#) and [9-4](#)). These characteristics make it the most commonly used IV opioid when rapid analgesia is desired, such as for the treatment of severe, escalating acute pain, and for procedural pain when a short duration of action is desirable. This medication is a good choice for patients with end-organ failure because it has no clinically relevant metabolites. It also produces minimal hemodynamic adverse effects; thus, fentanyl is often preferred in patients who are hemodynamically unstable, such as the critically ill (Chou et al., 2016; Conlin et al., 2018; Howard & Brant, 2019).

Its lipophilicity makes fentanyl ideal for medication delivery by transdermal patch for long-term opioid administration and by the oral transmucosal and buccal routes for BTP treatment in patients who are opioid tolerant. Following application of the transdermal patch, a subcutaneous depot of fentanyl is established in the skin near the patch. After absorption from the depot into the systemic circulation, the medication distributes to fat and muscle. When the first patch is applied, 12 to 18 hours are required for clinically significant analgesia to be obtained; attention must be paid to providing adequate supplemental analgesia during that time. Conversely, when the patch is removed, the serum levels of fentanyl remain for a minimum of 16 hours, so it

is important to not administer additional long-acting opioids during that time. Another important caution is that heat (e.g., heating pads, hot water blankets, hot tubs, fever) may increase the rate of absorption leading to serious adverse events. The patch is changed every 48 to 72 hours depending on patient response. It is important to note that the patch is not appropriate for treating acute pain or rapidly changing pain (Burchum & Rosenthal, 2019; Howard & Brant, 2019; Sheth et al., 2018).

Hydromorphone is less hydrophilic than morphine but less lipophilic than fentanyl, which contributes to an onset and duration of action that is intermediate between morphine and fentanyl (see Tables 9-3 and 9-4). This medication is often used as an alternative to morphine, especially for acute pain because the two medications produce similar analgesia and have comparable adverse effect profiles. It is a first- or second-choice opioid (after morphine) for postoperative pain management via IV PCA and is available in a once-daily modified-release oral formulation for chronic pain management. Accumulation of its neuroexcitatory metabolite hydromorphone-3-glucuronide (H3G) may occur with high-dose, long-term infusion therapy, which would necessitate a switch to another opioid (Burchum & Rosenthal, 2019; Sheth et al., 2018).

Oxycodone is available in the United States for administration by the oral route only and is used to treat all types of pain. Single-entity short-acting and modified-release oxycodone formulations are used most often for moderate to severe cancer pain and in some patients with moderate to severe noncancer pain (see Table 9-3). When it is combined with acetaminophen, the dose of oxycodone is limited by the acetaminophen dose to avoid exceeding the maximum daily dose of that agent. Oxycodone has been used successfully as part of a multimodal treatment plan for postoperative pain as well (Burchum & Rosenthal, 2019; Sheth et al., 2018).

Oxymorphone has been available for many years in parenteral formulation and more recently in short-acting and modified-release oral tablets for the treatment of moderate to severe chronic pain (see Table 9-3). It must be taken on an empty stomach (1 hour before or 2 hours after a meal), and coingestion of alcohol at the time of dosing must be avoided because food and alcohol can increase the serum concentration of the medication up to 300% (Burchum & Rosenthal, 2019; Comerford & Durkin, 2020; Sheth et al., 2018).

Hydrocodone is commercially available only in combination with nonopioids (e.g., with acetaminophen or ibuprofen), which limits its use to the treatment of mild to some moderate pain (see Table 9-3). It is one of the more commonly prescribed analgesic agents in the United States; however, its prescription for the treatment of persistent pain (except for breakthrough dosing) should be carefully evaluated because of its ceiling on **efficacy** and safety concerns inherent in the nonopioid constituent. In the rare situation when a patient can only tolerate hydrocodone and needs a dose higher than is

possible with the acetaminophen or ibuprofen combination, hydrocodone alone can be obtained from a compounding pharmacy with a prescription. In 2014, the U.S. Drug Enforcement Agency changed hydrocodone from a schedule III to the more restrictive schedule II classification to reduce abuse of this pain medication (Federal Register, 2014). It is now also available as two extended-release products (Burchum & Rosenthal, 2019; Comerford & Durkin, 2020).

Methadone is a unique synthetic opioid analgesic medication that may have advantages over other opioids in carefully selected patients. In addition to being a mu opioid, it is an antagonist at the NMDA receptor site and thus has the potential to produce analgesic effects as a second- or third-line option for some neuropathic pain states. It may be used as an alternative when it is necessary to switch a patient to a new opioid because of inadequate analgesia or unacceptable adverse effects. The use of conventional equianalgesic dose conversion is not recommended when switching patients to and from methadone. Extensive guidelines on how to safely accomplish this are available elsewhere. In 2014, the APS, in conjunction with the Heart Rhythm Society and the College on Problems of Drug Dependence, issued clinical practice guidelines to encourage safe use of methadone (APS, 2016).

Methadone is usually given orally but has also been given by virtually every other route of administration. Although it has no active metabolites, methadone has a very long and highly variable **half-life** (5 to 100-plus hours; average is 20 hours), which makes it a good choice for the treatment of SUD; patients must be watched closely for excessive sedation, a sign of medication accumulation during this time period. (The medication is described as “long acting” because of its exceptionally long half-life.) When methadone is used to treat opioid use disorder, it is dosed once daily and is not intended to manage pain; acute pain management with other analgesic medications is needed in addition to daily methadone dosing. Other limitations are its propensity to interact with a large number of medications and prolong the QTc interval on the electrocardiogram (ECG). Some medications (e.g., clarithromycin and some antifungal medications) that inhibit CYP3A4, the enzyme that metabolizes methadone, should be avoided as they can inadvertently increase methadone levels in the blood. Despite these characteristics, methadone can be an effective and safe medication when prescribed by providers who have an appreciation of the unique characteristics of the medication and who are experienced in prescribing it (APS, 2016; Burchum & Rosenthal, 2019; Comerford & Durkin, 2020; Sheth et al., 2018).

Dual-Mechanism Analgesic Agents

The dual-mechanism analgesic agents tramadol and tapentadol bind weakly to the mu opioid receptor site and block the reuptake (resorption) of the inhibitory neurotransmitters serotonin and norepinephrine at central synapses in the spinal cord and brain stem of the modulatory descending pain pathway

(APS, 2016; Holtsman & Hale, 2018). This makes these neurotransmitters more available to counteract pain. Dual-mechanism analgesic agents have been described as providing automatic “built-in” multimodal analgesia because a single tablet produces an effect on more than one analgesic action site (Varrassi, Hanna, Macheras, et al., 2017). The underlying mechanisms of tapentadol and tramadol differ in that tramadol blocks the reuptake of both serotonin and norepinephrine, but tapentadol blocks the reuptake of only norepinephrine. This is pertinent because norepinephrine may play a more significant role than serotonin in the endogenous analgesia pathways. Serotonin may be the more powerful mediator of depression; low serotonin levels are associated with depression. This helps explain why selective serotonin reuptake inhibitors (SSRIs), such as fluoxetine and paroxetine, which block only serotonin, are effective for the treatment of depression but not pain (APS, 2016; Burchum & Rosenthal, 2019; Comerford & Durkin, 2020; Holtsman & Hale, 2018).

Tramadol is used for both acute and chronic pain and is available in oral short-acting and modified-release formulations, including a short-acting tablet in combination with acetaminophen. It has demonstrated good efficacy for the treatment of neuropathic pain (Comerford & Durkin, 2020). The medication can lower seizure threshold and interact with other medications that block the reuptake of serotonin, such as the SSRIs, putting the patient at risk for serotonin syndrome, characterized by agitation, diarrhea, heart and blood pressure changes, and loss of coordination (Holtsman & Hale, 2018).

Tapentadol is available in short-acting and modified-release oral formulations. This medication has been shown to produce dose-dependent analgesia comparable to oxycodone. Major benefits are that it has no active metabolites and a significantly more favorable adverse effect profile (particularly GI) compared with opioid analgesic agents. These characteristics make tapentadol an attractive alternative to traditional oral opioid analgesic agents for many patients with pain (Comerford & Durkin, 2020; Holtsman & Hale, 2018).

Opioids to Avoid

Codeine is a prodrug, which means it is pharmacologically inactive when given. It must be metabolized to morphine for the patient to experience pain relief. It is estimated that 5% to 10% of patients lack the enzymatic ability to convert codeine to morphine via the CYP2D6 metabolic pathway, meaning that in this population of patients, codeine is an ineffective analgesic agent. In contrast, codeine has been associated with overdoses in some children due to rapid metabolism of the medication to morphine.

Meperidine has either been removed from or severely restricted on hospital formularies for the treatment of pain in an effort to improve patient safety. However, it is an accepted practice to use it in low doses (12.5 to 25 mg IV) to

treat *rigors* (shivering) associated with general anesthesia. A major limitation to the use of meperidine is its active metabolite, normeperidine, which is a CNS stimulant and can cause delirium, irritability, tremors, myoclonus, and generalized seizures. Concern for meperidine's neurotoxicity risks has dramatically reduced its use (APS, 2016; Burchum & Rosenthal, 2019; Comerford & Durkin, 2020; Sheth et al., 2018).

Adverse Effects of Opioid Analgesic Agents

The most common adverse effects of opioids are constipation, nausea, vomiting, pruritus, hypotension, and sedation. Respiratory depression, while less common, is the most serious and feared of the opioid adverse effects (Pasero, 2009). The risk of respiratory depression is increased when other medications that have depressive effects on the CNS, such as benzodiazepines (e.g., diazepam), alcohol, and barbiturates, are used concurrently with opioids. In surgical patients, postoperative ileus can become a major complication as well (Burchum & Rosenthal, 2019; Comerford & Durkin, 2020; Sheth et al., 2018). Morphine lowers blood pressure by dilating peripheral arterioles and veins. In the presence of dehydration or with concomitant use of hypotensive medications, orthostatic hypotension may result (Burchum & Rosenthal, 2019). Long-term use of opioids may result in opioid-induced androgen deficiency and sleep disordered breathing (Chowdhuri & Javaheri, 2017; Hsieh, DiGiorgio, Fakunle, et al., 2018; Nagappa, Weingarten, Montandon, et al., 2017).

Opioids can result in delayed gastric emptying, slowed bowel motility, and decreased peristalsis, all of which result in slow-moving, hard stool that is difficult to pass. Risk is elevated with advanced age, and immobility; however, it is an almost universal opioid adverse effect (i.e., tolerance rarely develops). Constipation is a primary reason people stop taking their pain medication, which underscores the importance of taking a preventive approach and aggressive management if symptoms are detected. Prevention includes reminding patients to take a daily stool softener plus mild peristaltic stimulant for as long as they are taking opioids (APS, 2016; Burchum & Rosenthal, 2019).

Postoperative nausea and vomiting (PONV) occur following opioid administration due to medulla chemoreceptor trigger zone stimulation. PONV are among the most unpleasant of the adverse effects associated with surgery, and can have a negative impact on patient outcomes, and increase the need for nursing intervention. Guidelines from the American Society of PeriAnesthesia Nurses (ASPAN) recommend that all patients be evaluated for PONV risk, risk factors be reduced if possible, multimodal analgesia be provided (so that no opioid or the lowest effective opioid dose can be given), and prophylactic treatment (e.g., dexamethasone and a serotonin receptor antagonist, such as ondansetron, at the end of surgery) be given to patients with moderate risk

(APS, 2016; ASPAN, 2006; Gan, Diemunsch, Habib, et al., 2014). More aggressive interventions should be utilized in patients with high risk (Pasero et al., 2011). (See [Chapter 16](#) for further discussion of PONV.)

Pruritus is an adverse effect of opioids, not an allergic reaction to them. Although antihistamines such as diphenhydramine are commonly used, and patients may report being less bothered by itching after taking an antihistamine, this may be the result of sedating effects. Any additional coadministered sedating medication can be problematic in people already at risk for excessive sedation, such as postoperative patients, because this can lead to life-threatening respiratory depression (APS, 2016). Loratadine and cetirizine are considered nonsedating histamines and might be selected. Often the most effective, safest, and least expensive treatment of pruritus is opioid dose reduction. In fact, simply decreasing the opioid dose is sufficient to eliminate or make most of the adverse effects tolerable for many patients. Opioid rotation is another possible treatment. Nonsedating analgesic agents can be added to facilitate this approach. Parenteral low dose nalbuphine, an agonist–antagonist opioid, has been reported to be superior to placebo, control, antihistamines, and naloxone in the treatment of pruritus caused by neuraxial opioids (APS, 2016).

In addition to dose reduction strategies, most opioid pain treatment plans include prescriptions for medications that can be used to treat adverse effects should they occur. Recent research with animals demonstrated successful prevention of opioid induced pruritis through administration of capsaicin (Melo, Basso, Iftinca, et al., 2018). Nonpharmacologic interventions may also be effective, such as the application of a cool damp cloth over affected areas to help relieve the discomfort of pruritus.

With the exception of constipation, as patients become opioid tolerant, an accompanying tolerance to the opioid adverse effects develops. It is reassuring for patients receiving long-term opioid therapy to know that most of the adverse effects will subside with regular daily doses of opioids over several days.

Sedation and respiratory depression. Most patients experience sedation at the beginning of opioid therapy and whenever the opioid dose is increased significantly. If left untreated, excessive sedation can progress to clinically significant respiratory depression. Like other opioid adverse effects, sedation and respiratory depression are dose related. In most cases (exceptions may apply at the end of life), nurses should promptly reduce opioid doses or stop titration whenever advancing sedation is detected to prevent respiratory depression (Pasero, 2009; Pasero et al., 2011). In some patients (e.g., those with obstructive sleep apnea, pulmonary dysfunction), monitoring with capnography is warranted (Jarzyna, Junquist, Pasero, et al., 2011). When supplemental oxygen is needed to maintain the patient’s oxygen saturation, pulse oximetry may not detect hypoventilation. Patients receiving opioid

therapy and supplemental oxygen can benefit from capnography, which reflects the adequacy of ventilation and airflow, in conjunction with monitoring of sedation and respiratory function (Gupta & Edwards, 2018).



Quality and Safety Nursing Alert

Opioid-induced respiratory depression is dose related and preceded by increasing sedation. Prevention of clinically significant opioid-induced respiratory depression begins with the administration of the lowest effective opioid dose, careful titration, close monitoring of sedation and respiratory function and status (i.e., rate, depth, regularity, excursion) throughout therapy, and prompt dose reduction when advancing sedation is detected (Nagappa et al., 2017).

The knowledge that excessive sedation precedes opioid-induced respiratory depression reinforces that systematic sedation assessment is an essential aspect of the care of patients receiving opioid therapy (Gupta & Edwards, 2018; Pasero, 2009). Nursing assessment of sedation is convenient, inexpensive, and takes minimal time to perform. A simple, easy-to-understand sedation scale, developed for the assessment of *unintended* sedation, which includes what should be done at each level of sedation, is recommended to enhance accuracy and consistency of assessment, better monitor trends, and improve communication among members of the health care team (Garcia & McMullan, 2019; Jarzyna et al., 2011; Pasero, 2009; Pasero et al., 2011; Quinlan-Colwell, Thear, Miller-Baldwin, et al., 2017). [Chart 9-6](#) presents a widely used sedation scale.

Chart 9-6

Pasero Opioid-Induced Sedation Scale with Interventions

Each level of sedation is followed by the appropriate action in italics.

S = Sleep, easy to arouse

Acceptable; no action necessary; may increase opioid dose if needed

1 = Awake and alert

Acceptable; no action necessary; may increase opioid dose if needed

2 = Slightly drowsy, easily aroused

Acceptable; no action necessary; may increase opioid dose if needed

3 = Frequently drowsy, arousable, drifts off to sleep during conversation

Unacceptable; monitor respiratory status and sedation level closely until sedation level is stable at less than 3 and respiratory status is satisfactory; decrease opioid dose 25–50%¹ or notify primary² or anesthesia provider for orders; consider administering a nonsedating, opioid-sparing nonopioid, such as acetaminophen or an NSAID, if not contraindicated; ask patient to take deep breaths every 15–30 min.

4 = Somnolent, minimal, or no response to verbal and physical stimulation

Unacceptable; stop opioid; consider administering naloxone^{3,4}; call Rapid Response Team (Code Blue); stay with patient, stimulate, and support respiration as indicated by patient status; notify primary² or anesthesia provider; monitor respiratory status and sedation level closely until sedation level is stable at less than 3 and respiratory status is satisfactory.

¹Opioid analgesic agent prescriptions or a hospital protocol should include the expectation that a nurse will decrease the opioid dose if a patient is excessively sedated.

²For example, the primary provider, nurse practitioner, advanced practice nurse, or physician assistant responsible for the pain management prescription.

³For adults experiencing respiratory depression, mix 0.4 mg of naloxone and 10 mL of normal saline in syringe and administer this dilute solution very slowly (0.5 mL over 2 min) while observing the patient's response (titrate to effect).

⁴Hospital protocols should include the expectation that a nurse will administer naloxone to any patient suspected of having life-threatening opioid-induced sedation and respiratory depression.

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Respiratory depression is assessed on the basis of what is normal for a particular person and is usually described as clinically significant when there is a decrease in the rate, depth, and regularity of respirations from baseline, rather than just by a specific number of respirations per minute (Pasero et al., 2011). There are many risk factors for opioid-induced respiratory depression, including older age (65 years of age or older), obesity, obstructive sleep apnea, and preexisting pulmonary dysfunction, or other comorbidities (APS, 2016;

Jarzyna et al., 2011). Risk is elevated during the first 24 hours following surgery and in patients who require a high dose of opioid in a short period of time (e.g., more than 10 mg of IV morphine or equivalent in the PACU). Patients who receive regularly scheduled opioids often develop tolerance to this effect in approximately 1 week (APS, 2016; Nagappa et al., 2017).

A comprehensive respiratory assessment constitutes more than counting a patient's respiratory rate (Pasero, 2009). A proper assessment requires watching the rise and fall of the patient's chest to determine rate, depth, and regularity of respirations. Listening to the sound of the patient's respirations is critical as well—snoring indicates airway obstruction and must be attended to promptly with repositioning and, depending on severity, a request for respiratory therapy consultation and further evaluation (Pasero, 2009; Pasero et al., 2011).

In most cases (exceptions may apply at the end of life), the opioid antagonist naloxone is promptly given IV to reverse clinically significant opioid-induced respiratory depression (Burchum & Rosenthal, 2019). The goal is to reverse only the sedation and respiratory depressant effects of the opioid. To this end, it should be diluted and titrated very slowly to prevent severe pain and other adverse effects, which can include hypertension, tachycardia, ventricular arrhythmias, pulmonary edema, and cardiac arrest (APS, 2016) (see [Chart 9-6](#), footnote 3, for correct technique). Sometimes more than one dose of naloxone is necessary, because naloxone has a shorter duration of action (1 hour in most patients) than most opioids. In particular, this is true with transdermal fentanyl for which reversal requires repeated doses or an infusion of naloxone to insure appropriate reversal (Burchum & Rosenthal, 2019).

Co-Analgesic Medications

The **co-analgesic agents** comprise the largest group of analgesic agents, which offers many options. Medication selection and dosing is based on both experience and evidence-based guideline recommendations. There is considerable variability among individuals in their response to co-analgesic agents, including to agents within the same class; often a “trial and error” strategy is used in the outpatient setting. Treatment in the outpatient setting often is primarily for patients who have a neuropathic component of pain and involves the use of low initial doses and gradual dose escalation to allow tolerance to the adverse effects. Patients must be forewarned in this setting that the onset of analgesia is likely to require time to achieve analgesic benefit (Pasero, Polomano, Portenoy, et al., 2011). Following is a brief overview of the most commonly used co-analgesic agents.

Local Anesthetics

Local anesthetics have a long history of safe and effective use for all types of pain management. Local anesthetics are sodium channel blockers that affect

the formation and propagation of action potentials. They are given by various routes of administration and are generally well tolerated by most individuals (Pasero et al., 2011). Injectable and topical local anesthetics are commonly used for procedural pain treatment. Local anesthetics are added to opioid analgesic agents and other agents to be given intraspinally for the treatment of both acute and chronic pain. They are also infused for continuous peripheral nerve blocks, primarily after surgery (Chou et al., 2016).

The lidocaine patch 5% is placed directly over or adjacent to the painful area for absorption into the tissues directly below. This medication produces minimal systemic absorption and adverse effects. The patch is left in place for 12 hours and then removed for as long as 12 hours (12 hours on, 12 hours off regimen). This application process is repeated as needed for continuous analgesia (Burchum & Rosenthal, 2019). The medication is FDA approved for the neuropathic pain syndrome and postherpetic neuralgia; however, research suggests that it is effective and safe for a variety of other painful neuropathic conditions (APS, 2016). Lidocaine is also available in topical preparations as cream, gel, solution, ointment, and aerosol (APS, 2016; Burchum & Rosenthal, 2019). More recent liposomal formulations of local anesthetics such as bupivacaine, which can be instilled in a surgical wound, are reported to have longer duration of action, supporting improved analgesia (Shah, Votta-Velis, & Borgeat, 2018).

Allergy to local anesthetics is rare. Cardiac and CNS adverse effects are dose related (Burchum & Rosenthal, 2019). CNS signs of systemic toxicity include ringing in the ears, metallic taste, irritability, and seizures. Signs of cardiotoxicity include circumoral tingling and numbness, bradycardia, cardiac arrhythmias, and CV collapse (Pasero et al., 2011).

Membrane Stabilizer Anticonvulsant Medications

The anticonvulsant calcium channel blockers gabapentin and pregabalin are first-line analgesic agents for neuropathic pain (Peterson et al., 2018). They are increasingly being added to postoperative pain treatment plans to address the neuropathic component of surgical pain and can be considered as part of a multimodal approach for postoperative analgesia (Chou et al., 2016). Although further research is needed, their addition has been shown to improve analgesia, allow lower doses of other analgesic agents, and help prevent persistent neuropathic postsurgical pain syndromes, such as phantom limb, post thoracotomy, posthernia, and postmastectomy pain. They may be effective in improving the acute pain associated with burn injuries and as treatment for the neuropathic pain and pruritis following major burn injuries (Griggs, Goverman, Bittner, et al., 2017; Kaul, Amin, Rosenberg, et al., 2018; Wang, Beekman, Hew, et al., 2018). Analgesic anticonvulsant therapy is initiated with low doses and titrated according to patient response. Initial doses of gabapentin may not provide analgesia; titration to effective dosing may take up

to 2 months. Pregabalin has a more rapid onset of action with expected maximum effect typically reached in 2 weeks. Primary adverse effects of anticonvulsants are sedation and dizziness, which are usually transient and most notable during the titration phase of treatment (Peterson et al., 2018).

Antidepressant Medications

From an analgesic perspective, antidepressant co-analgesic medications are divided into two major groups: the tricyclic antidepressants (TCAs) and the serotonin and norepinephrine reuptake inhibitors (SNRIs). Evidence-based guidelines recommend the TCAs desipramine and nortriptyline and the SNRIs duloxetine and venlafaxine as first-line options for neuropathic pain treatment (Cruccu & Truini, 2017). Their delayed onset of action makes them inappropriate for acute pain treatment. Analgesic antidepressant therapy is initiated with low doses and titrated according to patient response (Comerford & Durkin, 2020; Issa, Marshall, & Wasan, 2018).

Primary adverse effects of TCAs are dry mouth, sedation, dizziness, mental clouding, weight gain, and constipation. Orthostatic hypotension is a potentially serious TCA adverse effect. The most serious adverse effect is cardiotoxicity, and patients with significant heart disease are at high risk. SNRIs are thought to have a more favorable adverse effect profile and to be better tolerated than the TCAs. Due to the side effects, including delirium and confusion, amitriptyline is not indicated for use in older adults (Aguiar, Costa, da Costa, et al., 2019; Burchum & Rosenthal, 2019). The 2015 Beers Criteria® identified amitriptyline as potentially inappropriate for prescription among older adults (Fick, Semla, Steinman, et al., 2019) (see [Chapter 8](#) for further information on the Beers Criteria). The most common SNRI adverse effects are nausea, headache, sedation, insomnia, weight gain, impaired memory, sweating, and tremors (Comerford & Durkin, 2020).

Ketamine

Ketamine is a dissociative anesthetic with dose-dependent analgesic, sedative, and amnestic properties (Burchum & Rosenthal, 2019). As an NMDA antagonist, it blocks the binding of glutamate at the NMDA receptors and thus prevents the transmission of pain to the brain via the ascending pathway (see [Fig. 9-1B](#), inset). At high doses, this medication can produce psychomimetic effects (e.g., hallucinations, dreamlike feelings); however, these are minimized when low doses are given. A benefit of the medication is that it does not produce respiratory depression. Ketamine is given most often by the IV route but can also be given by the oral, rectal, intranasal, and subcutaneous routes. In addition to intraoperative and procedural use, ketamine has been used for the treatment of persistent neuropathic pain, but its adverse effect profile makes it less favorable than other analgesic agents for long-term therapy. It is, however, increasingly used as a third-line analgesic agent for **refractory** acute pain among patients who are very opioid-tolerant or for patients who are not able to

be treated with opioids. It does have a potential for abuse (APS, 2016; Burchum & Rosenthal, 2019).

Gerontologic Considerations

Older adults often live with chronic pain, yet physiologic changes and comorbidities make management more complicated among them (HHS, 2019). Older adults are often sensitive to the effects of co-analgesic agents that produce sedation and other CNS effects, such as antidepressants and anticonvulsants. Since they are also at risk for undertreatment of pain, therapy should be initiated with low doses, and titration should proceed slowly with systematic assessment of patient response (Burchum & Rosenthal, 2019).

Older adults are also at increased risk for NSAID-induced GI toxicity. Acetaminophen should be used for mild pain and is recommended as first line for musculoskeletal pain (e.g., osteoarthritis). If an NSAID is needed for inflammatory pain, it is recommended that a COX-2 selective NSAID (if not contraindicated by an increased CV risk) or the nonselective NSAID least likely to cause a peptic ulcer should be used. The addition of a proton pump inhibitor to NSAID therapy, or opioid analgesic agents rather than an NSAID, is recommended for high-risk patients. The American Geriatric Society (AGS) recommends using extreme caution when prescribing NSAIDs among older adults (Jones, Ehrhardt, Ripoll, et al., 2016). NSAIDs are safest when used for short-term pain flares that may occur during transient worsening in severity of chronic diseases or conditions (e.g., osteoarthritis, fibromyalgia, low back pain). A number of NSAIDs are available in topical formulations which may be preferred for older adults (Burchum & Rosenthal, 2019; Hargas, 2017; HSS, 2019; Sowa, Weiner, & Camacho-Soto, 2018).

Age is considered an important factor to consider when selecting an opioid dose. The starting opioid dose should be reduced by 25% to 50% in adults older than 70 years because they are more sensitive to opioid adverse effects than younger adults; the number of subsequent doses is based on patient response (American Geriatrics Society [AGS], 2009; Burchum & Rosenthal, 2019).

Use of Placebos

A **placebo** is “any sham medication or procedure designed to be void of any known therapeutic value” (Lang, Christopher, Emmott, et al., 2018, p. 55). A saline injection is one example of a placebo. Administration of a medication at a known subtherapeutic dose (e.g., 0.10 mg of morphine in an adult) is also considered a placebo.

Placebos only are appropriately used as controls in research evaluating the effects of a new medication. The new substance or treatment is compared with the effects of a placebo and must show more favorable effects than placebos to

warrant further investigation or marketing of the substance or treatment (Enck, Klosterhalfen, & Weimer, 2017). When a person responds to a placebo in accordance with its intent, it is called a *positive placebo response* (Arnstein, Broglie, Wuhrman, et al., 2011). Individuals who participate in placebo-controlled research must be able to give informed consent or have a guardian who can provide informed consent.

TABLE 9-5 Nonpharmacologic Methods of Pain Management

Type	Examples	Nursing Considerations
Physical modalities	Proper body alignment; application of heat and/or cold; massage; transcutaneous electrical nerve stimulation (TENS); acupuncture; physical therapy; and aqua therapy	Be aware that some of these methods require a prescription in the inpatient setting, as inappropriate use can cause harm (e.g., burns or frostbite from extreme temperatures and prolonged thermal application).
Cognitive and behavioral methods	Relaxation breathing; distraction; listening, singing, or rhythmic tapping to music; imagery; humor; pet therapy; prayer; meditation; hypnosis	Prior to use, evaluate patient's cognitive ability to learn and perform necessary activities.
Movement therapy	Yoga, T'ai chi	Prior to use, evaluate patient's physical ability to perform necessary activities.
Biologically based therapies	Taking herbs, vitamins, and proteins; aromatherapy; diet modifications	Evaluate use to identify potential adverse effects.
Energy therapies	Therapeutic touch, Reiki, and healing touch	Obtain patient's permission before using intervention.

Adapted from National Center for Complementary and Integrative Health (NCCIH). (2015). *Complementary, alternative, or integrative health: What's in a name?* Retrieved on 11/9/2019 at: www.nccih.nih.gov/health/integrative-health#types; O'Conner-Von, S., Heck, C. R., & Peltier, C. H. (2018). *Complementary and integrative therapies for pain management*. In M. L. Czarnecki, & H. N. Turner (Eds.). *Core curriculum for pain management nursing* (3rd ed.). St. Louis, MO: Elsevier.

Chart 9-7

Considerations in Selecting and Using Nonpharmacologic Methods

- Do the patient, family, and health care team understand the relationship between nonpharmacologic pain management and antispasmodic agents? Patients who have been taking analgesic agents may mistakenly assume that when clinicians suggest a nonpharmacologic method, the purpose is to reduce the use or dose of analgesic agents. All involved must understand that nonpharmacologic methods are used to complement—not replace—pharmacologic methods.
- Does the patient understand the limitations of nonpharmacologic methods? Nonpharmacologic methods are valuable as comfort measures; however, not all such measures relieve pain and should not be promoted as such.
- Is the patient interested in using a nonmedication method, and have any been tried previously? If so, what happened? Is the patient using nonmedication methods because of unfounded fears about analgesic agents? Willingness and interest are important for successful use of nonpharmacologic methods; however, patients may fear taking analgesic agents that clearly are indicated for their pain, such as nonsteroidal anti-inflammatory drugs (NSAIDs) for an inflammatory painful condition. Such fears should be explored, and accurate information and appropriate treatment provided. Alternately, the reasons a patient refuses to use a nonpharmacologic method also should be explored, but the patient's right to refuse must be respected.
- What are the patient's preferences and coping styles? Encouraging patients to choose from a variety of techniques allows them to match the technique to their individual and cultural preferences. If none of the choices appeal to the patient, the patient's right to refuse use should be respected.
- Does the patient have the physical and cognitive abilities necessary for using the nonpharmacologic method? Does the patient have sufficient energy to learn and perform any tasks involved? For example, physical and mental fatigue can interfere with the use of distraction and relaxation imagery techniques. Does the patient want to dedicate the necessary time required for the nonpharmacologic method? For example, those who do not find a 20-min self-sustained relaxation technique appealing may be more suited for passive application of cold or heat.
- Do others (e.g., family, friends) want to be involved in helping the patient? Is the method a potential vehicle for improving relationships between the patient and others? For example, a method that patients cannot do for themselves, such as massage, may be a burden to some caregivers in the home, whereas others may welcome that opportunity to be physically close to a loved one.

- Are support materials and patient education resources available? Whenever possible, verbal, written, and in some cases, online or video education should be provided.

Adapted from McCaffery, M. (2002). What is the role of nondrug methods in the nursing care of patients with acute pain? *Pain Management Nursing*, 3(3), 77–80; McCaffery, M., & Pasero, C. (1999). *Pain: Clinical manual*. St. Louis, MO: Mosby.

Placebos should never be used clinically in a deceitful manner and without informed consent. It is disrespectful and harmful to use them. Pain relief resulting from a placebo is mistakenly believed to invalidate a patient's report of pain. This typically results in the patient being deprived of pain-relief measures, despite research showing that many patients who have obvious physical stimuli for pain (e.g., abdominal surgery) report pain relief after placebo administration. The reason for this is a mystery, but it is one of the many reasons that pain guidelines, position papers, nurse practice acts, and hospital policies nationwide agree that there are no individuals for whom and no condition for which placebos are the recommended treatment. The deceptive use of placebos has both ethical and legal implications, violates the nurse–patient relationship, and inevitably deprives patients of appropriate assessment and treatment (Arnstein et al., 2011; Enck et al., 2017; Lang et al., 2018).

Nonpharmacologic Methods of Pain Management

Most individuals use self-management strategies to deal with their health issues and promote well-being. According to national health information, it is estimated that American adults spent \$30.2 billion on complementary health practices to treat painful conditions (O'Conner-Von, Heck, & Peltier, 2018). Nonpharmacologic complementary and alternative interventions include using natural products (e.g., herbs or botanicals, vitamins, probiotics) or using mind and body practices (e.g., acupuncture, behavior-based therapies, chiropractic manipulation, massage therapy, spirituality, yoga, T'ai chi) (HHS, 2019). **Table 9-5** lists examples of select complementary and alternative therapies.

Nonpharmacologic therapies are usually effective alone for mild to some moderate-intensity pain. They should not be a replacement or alternative but complement pharmacologic therapies as part of a multimodal approach for more severe pain. The effectiveness of nonpharmacologic methods can be unpredictable, and although not all will relieve pain, they offer many benefits to patients with pain. For example, research suggests that nonpharmacologic methods can facilitate relaxation and reduce anxiety and stress. Many patients find that the use of nonpharmacologic methods helps them cope better with

their pain and feel greater control over the pain experience (HHS, 2019; O’Conner-Von et al., 2018).

Several nonpharmacologic methods can be used in the clinical setting to provide comfort and pain relief for all types of pain; however, time is often limited in this setting for implementation of these methods. Nurses play an important role in providing them and educating patients about their use. Many of the methods are relatively easy for nurses to incorporate into daily clinical practice and may be used individually or in combination with other nonpharmacologic therapies to facilitate patient-centered care utilizing a multimodal approach (HHS, 2019; O’Conner-Von et al., 2018). [Chart 9-7](#) provides points for nurses to consider before using nonpharmacologic methods.

Unfolding Patient Stories: Stan Checketts • Part 1



Stan Checketts, a 52-year-old man, is in the emergency department with severe abdominal pain. The provider has prescribed buprenorphine 0.3-mg IV push for pain. Explain the assessments performed by the nurse prior to, during, and following the administration of the medication. Also consider nonpharmacologic interventions the nurse can incorporate to assist with his pain relief. (Stan Checketts’ story continues in [Chapter 41](#).)

Care for Stan and other patients in a realistic virtual environment: **vSim** (theopoint.lww.com/vSimMedicalSurgical). Practice documenting these patients’ care in DocuCare (theopoint.lww.com/DocuCareEHR).

Nursing Implications of Pain Management

The provision of optimal pain management requires a collaborative approach between patients with pain, their families, and members of the health care team. Everyone involved must share common goals, a common knowledge base, and a common language with regard to the antispasmodic agents and nonpharmacologic methods used to manage pain. Whether nurses provide care in the home, hospital, or any other setting, they are in a unique position to coordinate a comprehensive, evidence-based approach to meet the needs of people with pain. [Chart 9-8](#) provides a plan of nursing care for the patient with pain.

Chart 9-8



PLAN OF NURSING CARE

Care of the Patient with Acute Pain

NURSING DIAGNOSIS: Acute Pain**GOAL:** Achievement and maintenance of patient's comfort–function goal

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Perform and document a comprehensive pain assessment.<ol style="list-style-type: none">a. Use a reliable and valid tool to determine pain intensity.b. Accept the patient's report of pain.c. Assist the patient in establishing a comfort–function goal.d. Apply the Hierarchy of Pain Measures in patients who are unable to report their pain.2. Administer analgesic agents as prescribed.3. Offer and educate patient how to use appropriate nonpharmacologic interventions.4. Reassess for degree of pain relief and presence of adverse effects at peak effect time of intervention.5. Obtain additional prescriptions as needed.6. Prevent and treat adverse effects.	<ol style="list-style-type: none">1. The comprehensive pain assessment is the foundation of the pain treatment plan; documentation ensures communication between team members.<ol style="list-style-type: none">a. The use of valid and reliable tools helps ensure accuracy and consistency in assessment.b. Accepting the patient's report of pain is the undisputed standard of pain assessment.c. The comfort–function goal links function to pain control and provides direction for necessary adjustments in the treatment plan to maximize function.d. The use of the Hierarchy of Pain Measures provides a process to ensure pain	<ul style="list-style-type: none">• If able, provides information about the pain• Expresses understanding of the link between function and pain control and establishes a realistic comfort–function goal• Reports a pain intensity that allows participation in important functional activities• If not able to report pain, demonstrates behaviors that indicate pain relief and participation in important functional activities• Expresses satisfaction with the use of nonpharmacologic methods• Tolerates pharmacologic and nonpharmacologic interventions without adverse effects

7. Educate patient and family about the effects of analgesic agents and the goals of care; explain how adverse effects will be prevented and treated; address fears of substance use disorder.
- treatment in the patient who cannot report pain.
- Demonstrates an understanding of the treatment plan and goals of care
2. Pharmacologic interventions are the cornerstone of pain management.
 3. Nonpharmacologic methods are used to supplement pharmacologic interventions.
 4. Reassessment permits evaluation of both the effectiveness and safety of interventions.
 5. Prescriptions for additional analgesic agents or adjustment in dose are often needed to maximize pain control.
 6. Adverse effects are prevented whenever possible and promptly treated to reduce patient discomfort and prevent harm.
 7. An understanding of the treatment plan and goals of care educates patients and their families how to partner with the health care team to optimize pain control.

CRITICAL THINKING EXERCISES

1  ebp An 88-year-old woman arrives in the emergency department following a motor vehicle crash in which she was the restrained front seat passenger. She repeatedly says “owie” and her daughter reports that this is her mom’s way of expressing pain. Her daughter also reports that her mother was recently diagnosed with dementia after family members began noticing increasing forgetfulness and confusion at home. When asked to rate the intensity of her pain, the patient replies “owie, yes” but she is not able to easily assign a number to her pain. What pain assessment tool would be most helpful in assessing this patient’s pain and in evaluating the effectiveness of interventions? What is the strength of the evidence supporting the assessment tool you selected?

2  pq A 62-year-old woman who has chronic abdominal pain following multiple surgeries resulting from an abusive domestic situation is admitted to the medical floor for reports of worsening pain at home. She reports pain and nausea but denies diarrhea or vomiting. In addition to the 50-mcg fentanyl transdermal patch she has used to control pain at home for the last 2 years, the primary provider has prescribed: 1 mg intravenous hydromorphone every 2 hours PRN pain; promethazine 25 mg IV every 4 hours PRN nausea; lorazepam 0.5 mg IV every 6 hours PRN anxiety; and zolpidem 1.75 mg sublingual nightly prior to sleep. The patient asks you to give her all her medications at the same time so that she can sleep. She asserts that “the nurses always give me the medication that way when I am in the hospital.” You are concerned that all these medications are central nervous system depressants and that given together, they could lead to oversedation and possibly result in respiratory depression. How would you prioritize administering the medications that she is requesting and your ongoing assessments to ensure adequate symptom relief while maintaining patient safety and minimizing the risks of adverse outcomes?

3  ipc A 28-year-old man is hospitalized with a diagnosis of an epidural abscess. He reports a 5-year history of daily intravenous self-administration of heroin. He tells you that the oxycodone his primary provider prescribed is “not doing anything” and his pain “is 100/10.” How will you collaborate with other members of the interprofessional health care team to manage his pain? Using a multimodal approach for analgesia, what other interventions, both pharmacologic and nonpharmacologic, can the team incorporate into his plan of care to better manage his severe pain?

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*Asterisk indicates nursing research.

**Double asterisk indicates classic reference.

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Resources

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- American Chronic Pain Association, www.theacpa.org
- American Pain Foundation (APF), www.painfoundation.org
- American Society for Pain Management Nursing (ASPMN), www.aspmn.org
- City of Hope Pain & Palliative Care Resource Center, www.cityofhope.org/
- National Center for Complementary and Integrative Health (NCCIH),
www.nccih.nih.gov
- Pain Treatment Topics, www.pain-topics.org

10 Fluid and Electrolytes

LEARNING OUTCOMES

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

1. Differentiate between osmosis, diffusion, filtration, and active transport.
2. Describe the role of the kidneys, lungs, and endocrine glands in regulating the body's fluid composition and volume.
3. Plan effective care of patients with the following imbalances: fluid volume deficit and fluid volume excess, sodium deficit (hyponatremia) and sodium excess (hypernatremia), and potassium deficit (hypokalemia) and potassium excess (hyperkalemia).
4. Describe the cause, clinical manifestations, management, and nursing interventions for the following imbalances: calcium deficit (hypocalcemia) and calcium excess (hypercalcemia), magnesium deficit (hypomagnesemia) and magnesium excess (hypermagnesemia), phosphorus deficit (hypophosphatemia) and phosphorus excess (hyperphosphatemia), and chloride deficit (hypochloremia) and chloride excess (hyperchloremia).
5. Explain the roles of the lungs, kidneys, and chemical buffers in maintaining acid–base balance; and compare metabolic as well as respiratory acidosis and alkalosis with regard to causes, clinical manifestations, diagnosis, and management.
6. Interpret arterial blood gas measurements.

NURSING CONCEPTS

Acid–Base
Cellular Regulation
Fluids and Electrolytes
Metabolism

GLOSSARY

acidosis: an acid–base imbalance characterized by an increase in H⁺ concentration (decreased blood pH) (A low arterial pH due to increased H⁺ concentration or reduced bicarbonate concentration is called *metabolic acidosis*; a low arterial pH due to increased PCO₂ is called *respiratory acidosis*.)

active transport: physiologic pump that uses energy to move fluid or electrolytes from one region to another

alkalosis: an acid–base imbalance characterized by a reduction in H⁺ concentration or increase in bicarbonate concentration (increased blood pH) (A high arterial pH with either decreased H⁺ ion concentration or increased bicarbonate concentration is called *metabolic alkalosis*; a high arterial pH due to reduced PCO₂ is called *respiratory alkalosis*.)

colloid: a fluid containing particles that are nonsoluble and evenly distributed throughout the solution

colloid oncotic pressure: osmotic pressure created by the protein (mainly albumin) in the bloodstream (*synonym:* colloidal osmotic pressure)

crystalloid: a fluid containing soluble mineral ions and water in solution

diffusion: the process by which solutes move from an area of higher concentration to one of lower concentration; does not require expenditure of energy

homeostasis: maintenance of a constant internal equilibrium in a biologic system

hydrostatic pressure: the pressure created by the weight of fluid against the wall that contains it. In the body, hydrostatic pressure in blood vessels results from the weight of fluid itself and the force resulting from cardiac contraction (*synonym:* hydraulic pressure)

hypertonic solution: a solution with an osmolality higher than that of serum

hypotonic solution: a solution with an osmolality lower than that of serum

isotonic solution: a solution with the same osmolality as blood

osmolality: the number of milliosmoles (the standard unit of osmotic pressure) per kilogram of solvent; expressed as milliosmoles per kilogram (mOsm/kg). (The term *osmolality* is used more often than *osmolarity* to evaluate serum and urine.)

osmolarity: the number of milliosmoles (the standard unit of osmotic pressure) per liter of solution; expressed as milliosmoles per liter (mOsm/L); describes the concentration of solutes or dissolved particles

osmosis: the process by which fluid moves across a semipermeable membrane from an area of low solute concentration to an area of high solute concentration; the process continues until the solute concentrations are equal on both sides of the membrane

tonicity: fluid tension or the effect that osmotic pressure of a solution with impermeable solutes exerts on cell size because of water movement across the cell membrane

Fluid and electrolyte balance are dependent on dynamic processes that are crucial for life and **homeostasis** (the maintenance of a constant internal equilibrium in a biologic system). Potential and actual disorders of fluid and electrolyte balance occur in every setting, with every disorder, and with a variety of changes that affect healthy people as well as those who are ill.

Nurses need to understand the physiology of fluid and electrolyte balance and acid–base balance to anticipate, identify, and respond to imbalances. Maintaining the balance of fluid and electrolytes is crucial to the care of patients of every age and in every clinical setting.

Fundamental Concepts

Basic concepts of chemistry are involved in fluid and electrolyte balance and imbalance. A solution is a mixture of solvent, which is a fluid medium, and solutes, which are particles. Blood is composed of blood cells that are suspended in plasma. The blood cells include erythrocytes, leukocytes, and platelets. Plasma is composed of 92% water, which is a solvent that contains solutes including proteins (mainly albumin), glucose, lipoproteins, and mineral ions, termed electrolytes.

Amount and Composition of Body Fluids

Approximately 60% of a typical adult's weight consists of fluid (water and electrolytes) (Fig. 10-1). Factors that influence the amount of body fluid are age, gender, and body fat. In general, younger people have a higher percentage of body fluid than older adults, and men have proportionately more body fluid than women. The skeleton has low water content. Muscle, skin, and blood contain the highest amounts of water (Norris, 2019).

Body fluid is located in two fluid compartments: the intracellular space (fluid in the cells) and the extracellular space (fluid outside the cells). Approximately two thirds of body fluid is in the intracellular fluid (ICF) compartment. Approximately one third is in the extracellular fluid (ECF) compartment (Norris, 2019).

The ECF compartment is further divided into the intravascular (blood), interstitial, and transcellular fluid spaces:

- The intravascular space (the fluid within the blood vessels) contains plasma, the effective circulating volume. Approximately 3 L of the average 6 L of blood volume in adults is made up of plasma. The remaining 3 L is made up of the blood cells: erythrocytes, leukocytes, and thrombocytes (platelets).
- The interstitial space contains the fluid that surrounds the cell and totals about 11 to 12 L in an adult. Lymph is an interstitial fluid.
- The transcellular space is the smallest division of the ECF compartment and contains approximately 1 L. Examples of transcellular fluids include cerebrospinal, pericardial, synovial, intraocular, and pleural fluids, sweat, and digestive secretions.

As the next section describes, the ECF transports electrolytes; it also carries other substances, such as enzymes and hormones.

Body fluid normally moves between the two major compartments (i.e., ICF and ECF) in an effort to maintain equilibrium between the spaces. A semipermeable membrane surrounding each of the cells allows certain fluids and electrolytes to move between the ICF and ECF. Loss of fluid from the ICF or ECF can disrupt equilibrium. The body constantly works to keep fluid and electrolytes in a homeostatic balance. If too much fluid moves from ICF to ECF, cellular dehydration can occur. If too much fluid moves from ECF to ICF, cell swelling can occur (Papadakis, McPhee, & Rabow, 2019).

Sometimes fluid is not lost from the body but is unavailable for use by either the ICF or ECF. Loss of ECF into a space that does not contribute to equilibrium between the ICF and the ECF is referred to as a third-space fluid shift, or third spacing. Third-space fluid accumulates within membrane-bound spaces in the body such as the peritoneal cavity and pleural space. Examples of third-spaced fluid include ascites, pleural effusion, pericardial effusion, and angioedema (Papadakis et al., 2019).

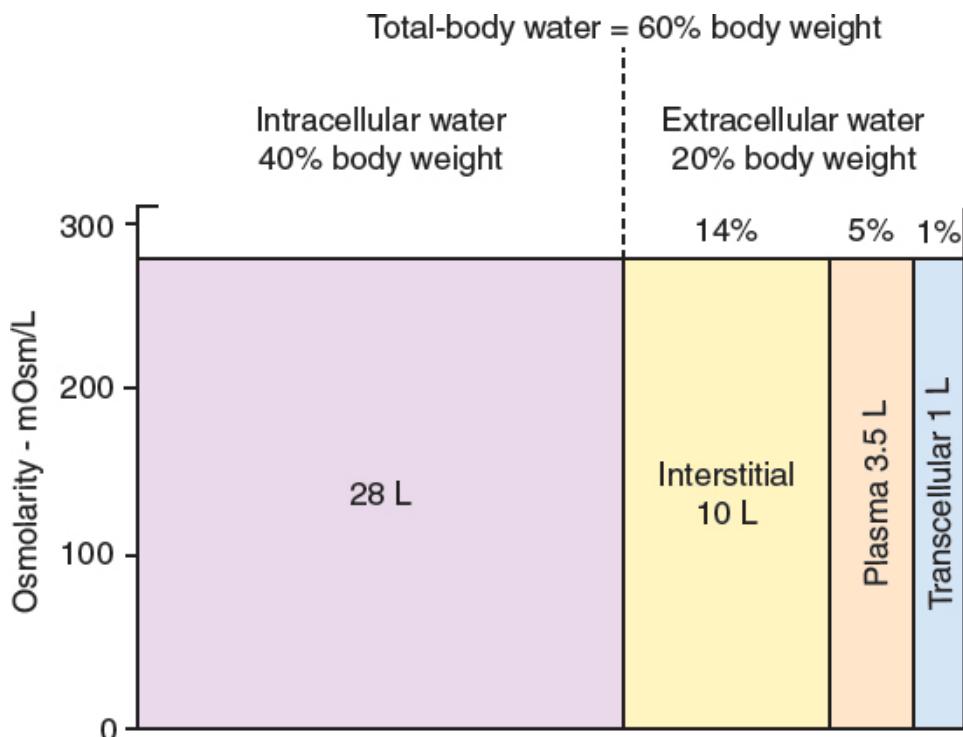


Figure 10-1 • Approximate sizes of body compartments in a 70-kg adult. Reprinted with permission from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed., Fig. 8-4, p. 162). Philadelphia, PA: Wolters Kluwer.

Early evidence of a third-space fluid shift is a decrease in urine output despite adequate fluid intake. Urine output decreases because fluid shifts out of the intravascular space; the kidneys then receive less blood and attempt to compensate by decreasing urine output. Other signs and symptoms of third spacing that indicate an intravascular fluid volume deficit (FVD) include increased heart rate, decreased blood pressure, decreased central venous pressure, edema, increased body weight, and imbalances in fluid intake and output (I&O). Third-space fluid is reabsorbed back into the bloodstream over a period of a few days to a few weeks. However, the acute volume depletion must be restored to prevent further complications (Sterns, 2017a).

Electrolytes

Electrolytes in body fluids are active chemicals (cations that carry positive charges and anions that carry negative charges). The major cations in body fluid are sodium, potassium, calcium, magnesium, and hydrogen ions. The major anions are chloride, bicarbonate, phosphate, sulfate, and negatively charged protein ions.

These chemicals unite in varying combinations. Therefore, electrolyte concentration in the body is expressed in terms of milliequivalents (mEq) per

liter, a measure of chemical activity, rather than in terms of milligrams (mg), and a unit of weight. More specifically, a milliequivalent is defined as being equivalent to the electrochemical activity of 1 mg of hydrogen. In a solution, cations and anions are equal in milliequivalents per liter.

Electrolyte concentrations in the ICF differ from those in the ECF, as reflected in [Table 10-1](#). Because special techniques are required to measure electrolyte concentrations in the ICF, it is customary to measure the electrolytes in the most accessible portion of the ECF—namely, the plasma (Norris, 2019).

Sodium ions, which are positively charged, far outnumber the other cations in the ECF. Because sodium concentration affects the overall concentration of the ECF, sodium is important in regulating the volume of body fluid. Water follows movement of sodium in the body fluids. Retention of sodium is associated with fluid retention, and excessive loss of sodium is usually associated with decreased volume of body fluid.

As shown in [Table 10-1](#), one of the major electrolytes in the ICF is potassium. The ECF has a low concentration of potassium, and patients can tolerate only small changes in potassium concentration. Changes in potassium within the ECF can cause cardiac rhythm disturbances and hyperkalemia can cause cardiac arrest. Therefore, the release of large stores of intracellular potassium, typically caused by trauma to the cells and tissues, can be extremely dangerous.

The body expends a great deal of energy maintaining the high extracellular concentration of sodium and the high intracellular concentration of potassium. It does so by means of a cell membrane pump that exchanges sodium and potassium ions, termed the sodium–potassium pump (see discussion later in this chapter).

Osmosis, Osmolality, and Osmolarity

When two different solutions are separated by a membrane that is semipermeable; fluid shifts from the region of less concentrated solution to a more concentrated solution, until the solutions are of equal concentration. This diffusion of water caused by fluid and solute concentration gradients is known as **osmosis** ([Fig. 10-2](#)). Osmolality and osmolarity are terms that describe the concentration of solutes or dissolved particles in a solution. **Osmolality** is the number of milliosmoles of solute (the standard unit of osmotic pressure) per kilogram of solvent; it is expressed as milliosmoles per kilogram (mOsm/kg). **Osmolarity** is the number of milliosmoles (the standard unit of osmotic pressure) per liter of solution; it is expressed as milliosmoles per liter (mOsm/L). The term *osmolality* is used more often than *osmolarity* to evaluate the solutes in the blood or urine (Emmett & Palmer, 2018a).



Regulation of Fluid within the Body Compartments

Normal movement of fluids through the capillary wall into the tissues depends on Starling's Laws of Capillary Forces. Capillary forces are the two forces at every capillary membrane: hydrostatic pressure (also called hydraulic pressure) and osmotic pressure. **Hydrostatic pressure** is the pressure exerted by fluid on the walls of the blood vessel, and osmotic pressure is the pressure exerted by the solutes within the plasma. Hydrostatic pressure pushes fluid out of the capillary toward the ICF. Osmotic pressure pulls fluid into the capillary from the ICF. These forces oppose each other at every capillary membrane and balance each other out in healthy (homeostatic) conditions. The direction of fluid movement depends on the differences in the two opposing forces of hydrostatic and osmotic pressure (Fig. 10-3). If hydrostatic pressure is greater than osmotic pressure, then the movement of fluid is from ECF toward the ICF.

TABLE 10-1 Concentrations of Extracellular and Intracellular Electrolytes in Adults

Electrolyte	Extracellular Concentration ^a		Intracellular Concentration ^a	
	Conventional Units	SI Units (mmol/L)	Conventional Units	SI Units (mmol/L)
Sodium	135–145 mEq/L	135–145	10–14 mEq/L	10–14
Potassium	3.5–5.0 mEq/L	3.5–5.0	140–150 mEq/L	140–150
Chloride	98–106 mEq/L	98–106	3–4 mEq/L	3–4
Bicarbonate	24–31 mEq/L	24–31	7–10 mEq/L	7–10
Calcium	8.8–10.5 mg/dL	2.2–2.6	<1 mEq/L	<0.25
Phosphorus	2.5–4.5 mg/dL	0.8–1.45	Variable	Variable
Magnesium	1.8–3.6 mg/dL	0.75–1.07	40 mEq/kg ^b	20

^aValues may vary among laboratories, depending on the method of analysis used.

^bValues vary among various tissues and with nutritional status.

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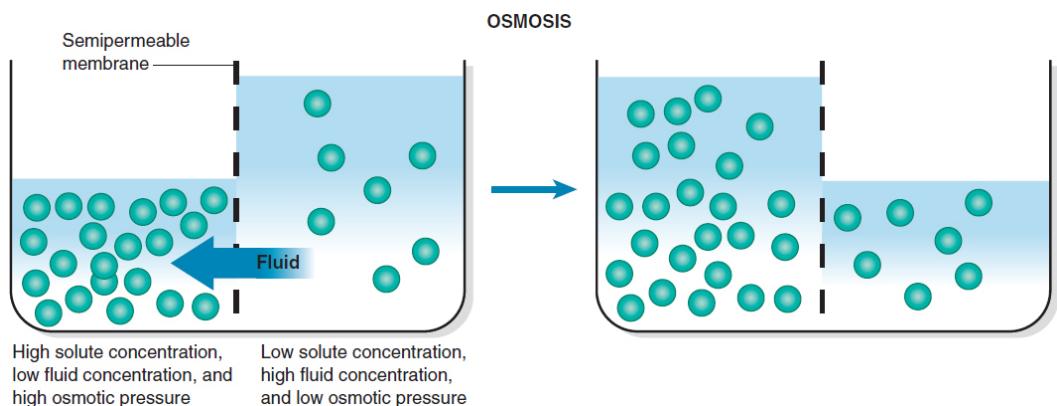


Figure 10-2 • Osmosis: when two different solutions are separated by a semi-permeable fluid shifts from the region of less concentrated solution to a more concentrated solution, until the solutions are of equal concentration.

Oncotic Pressure

Osmotic pressure specifically exerted by the albumin within the bloodstream is termed **colloid oncotic pressure** or **colloid osmotic pressure**. A **colloid** is fluid consisting of nonsoluble substances that are evenly distributed within a solvent. Blood is an example of a colloid solution. It is a mixture of blood cells and plasma which contains water, proteins, enzymes, and other solutes.

Crystalloid versus Colloid Solutions

Fluid replacement in the body depends on what type of fluid is lost. If a large amount of blood is lost, for example, the preferred treatment is replacement with matching blood (commonly packed red blood cells). However, crystalloid or colloid solutions can be used to temporarily replace blood or replenish fluid losses from the body. **Crystalloid** solutions are mineral ions dissolved in water. Examples include normal saline (0.9% NaCl), half normal saline (0.45% NaCl), and lactated Ringer's solution (Plasma-Lyte). Crystalloid solutions are commonly used to replace fluid in hypovolemia. Examples of colloid solutions include albumin solutions, hyperoncotic starch, and dextran. Colloid solutions are commonly used as temporary blood replacement until the correct type of blood is available for infusion (Mandel & Palevsky, 2019).

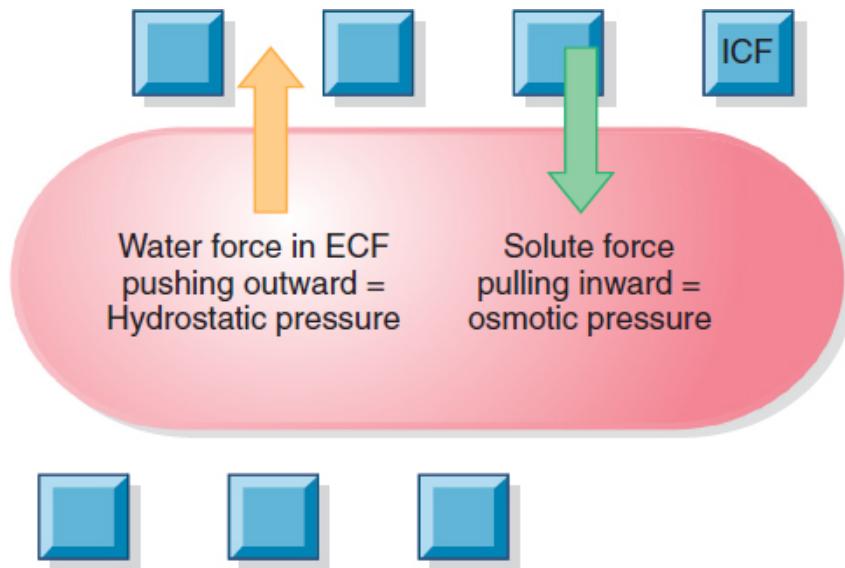


Figure 10-3 • The forces of osmotic and hydrostatic pressure constantly oppose each other. ECF, extracellular fluid; ICF, intracellular fluid.

Tonicity

Tonicity is the ability of solutes to cause an osmotic driving force that promotes water movement from one compartment to another. Movement of water is either from ICF to ECF or ECF to ICF. The tonicity of a solution can be used to drive water movement between compartments to change the state of cellular hydration and cell size. Tonicity most commonly refers to the NaCl content of the solution. The tonicity of a solution is determined by how it compares to physiologic fluid which is 0.9% NaCl (Sterns, 2017b). Fluids can be isotonic, hypotonic, or hypertonic compared to physiologic fluid of 0.9% NaCl.

Tonicity commonly pertains to intravenous (IV) solutions. IV solutions of different tonicities can be infused into the bloodstream to produce movement of water from one compartment to the other.

Isotonic solutions are composed of 0.9% NaCl; the same sodium and chloride concentration as the bloodstream and the same water concentration as the bloodstream. Isotonic solutions do not provoke water movement between ICF or ECF compartments. Isotonic solutions expand the plasma volume of the blood (Sterns, 2017b).

Hypotonic solutions are composed of less sodium chloride concentration compared to the blood; for example, 0.45% NaCl or 0.225% NaCl. Hypotonic solutions contain less solute but more water than the bloodstream. IV hypotonic solution infusions can be used to move water from the ECF into the ICF. IV hypotonic solutions can be used to hydrate a patient as they contain high water concentration (Sterns, 2017b).

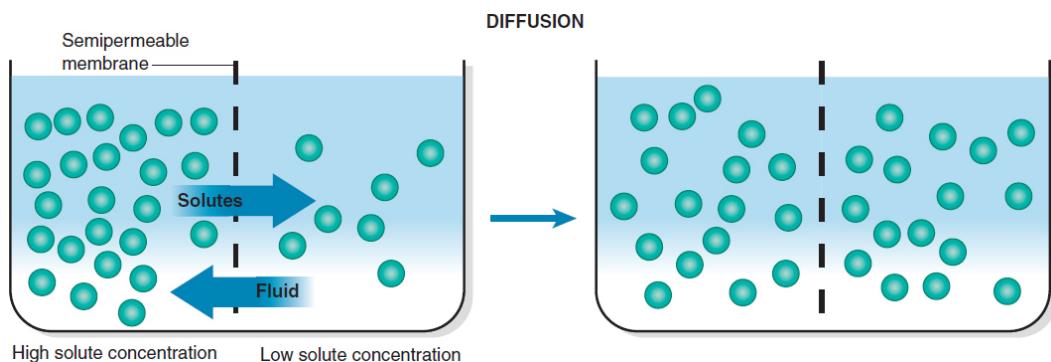


Figure 10-4 • Diffusion: movement of solutes from an area of greater concentration to an area of lesser concentration, leading ultimately to equalization of the solute concentrations.

Hypertonic solutions are composed of greater concentration of NaCl compared to blood (e.g., 3% NaCl). Hypertonic solutions contain more solute concentration and less water than the bloodstream. IV hypertonic solution can be infused into the bloodstream to pull water from the ICF into the ECF. The movement of water from ICF to ECF will cause dehydration of the cells. This is useful in disorders of severe edema; particularly cerebral edema, which requires immediate treatment. Sodium, glucose, and mannitol are examples of solutes capable of affecting water movement from ICF to ECF. Mannitol, a nonresorbable sugar alcohol, in water is an IV solution that can be used to move water from ICF to ECF rapidly. IV mannitol can induce a condition termed osmotic diuresis; it is most commonly used to decrease cerebral edema (Brater & Ellison, 2019). (See discussion of parenteral therapy later in this chapter.)

Osmotic diuresis is the increase in urine output caused by the excretion of solutes, such as glucose or mannitol. In high concentrations, glucose or mannitol can act as solutes within the bloodstream. These solutes exert a force that pulls water out of the ICF and brings it into the ECF (bloodstream). The water then is filtered out of the bloodstream at the kidneys and excreted into the urine. The urine contains extra water that is derived from the ICF; this increased urine volume is called diuresis (Brater & Ellison, 2019).

Diffusion

Diffusion is the natural tendency of a substance to move from an area of higher concentration to one of lower concentration (Fig. 10-4). It occurs through the random movement of ions and molecules. Examples of diffusion are the exchange of oxygen (O_2) and carbon dioxide (CO_2) between the pulmonary capillaries and alveoli and the tendency of sodium to move from the ECF compartment, where the sodium concentration is high, to the ICF, where its concentration is low (Hall, 2016).

Filtration

Hydrostatic pressure in the capillaries tends to filter fluid out of the intravascular compartment into the interstitial fluid. Movement of water and solutes occurs from an area of high hydrostatic pressure to an area of low hydrostatic pressure. The kidneys filter approximately 180 L of plasma per day. Another example of filtration is the passage of water and electrolytes from the capillary bed to the interstitial fluid; in this instance, the hydrostatic pressure results from the pumping action of the heart (Hall, 2016).

Sodium–Potassium Pump



The sodium concentration is greater in the ECF than in the ICF; because of this, sodium tends to enter the cell by diffusion. To maintain cellular function, more potassium needs to be inside the cell and more sodium needs to be outside the cell. The sodium–potassium pump maintains the electrolyte gradient of high extracellular Na^+ compared to low intracellular Na^+ , and high intracellular K^+ compared to low extracellular K^+ . The pump maintains the different concentrations of cations Na^+ and K^+ in and out of the cell. For every 3 Na^+ pumped out of the cell, 2 K^+ ions are pumped into the cell (Pirahanchi & Aeddula, 2019).

The sodium–potassium pump uses energy to maintain this electrolyte gradient and is powered by the enzyme termed Na^+K^+ -ATPase. The sodium–potassium pump performs **active transport** which actively moves sodium from the ICF into the ECF and actively moves potassium from the ECF to the ICF. Active transport is the use of energy to create movement against a concentration gradient (Hall, 2016; Norris, 2019).

Systemic Routes of Gains and Losses

Water and electrolytes are gained in various ways. Healthy people gain fluids by drinking and eating, and their daily average intake and output (I & O) of water are approximately equal ([Table 10-2](#)).

TABLE 10-2 Sources of Body Water Gains and Losses in the Adult

Intake (mL)		Output (mL)
Oral intake	Urine	1500
As water	Insensible losses	
In food	Lungs	300
Water of oxidation	Skin	500
	Feces	200
Total gain ^a	Total loss ^a	2500

^aApproximate volumes.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Kidneys

The daily urine volume excreted by the adult varies according to hydration status. A well-hydrated person excretes 1 to 2 L urine per day. A general rule is that the output is approximately 1 mL of urine per kilogram of body weight per hour (1 mL/kg/h) in all age groups (Sterns, 2017a). For example, a 70-kg adult will excrete 70 mL/h; over 24 hours this equals approximately 1680 mL of urine.

Skin

Perspiration is visible water and electrolyte loss through the skin (sweating). The chief solutes in sweat are sodium, chloride, and potassium. Actual sweat losses can vary from 0 to 1000 mL or more every hour, depending on factors such as the environmental and body temperature. Continuous water loss by evaporation through the skin (approximately 500 mL/day) occurs through perspiration, referred to as insensible water loss (Norris, 2019). Fever, high environmental temperature, and exercise greatly increase insensible water loss through the skin. Burn injury, which causes the loss of the natural skin barrier, also causes a large water loss (Sterns, 2017b).

Lungs

The lungs normally eliminate water vapor, also referred to as insensible water loss, at a rate of approximately 300 mL daily (Norris, 2019). The loss is much greater with increased respiratory rate or depth, or in a dry climate. Similar to water loss through the skin, fever, high environmental temperature, and exercise all increase insensible water loss through the lungs.

Gastrointestinal Tract

Loss of fluid from the gastrointestinal (GI) tract is about 100 to 200 mL daily. Approximately 8 L of fluid circulates through the GI system every 24 hours.

However, most of the fluid is reabsorbed into the bloodstream from the small intestine. Diarrhea and fistulas of the intestine can cause large losses of fluids (Sterns, 2018a).



Quality and Safety Nursing Alert

When fluid balance is critical, all routes of systemic gain and loss must be recorded and all volumes compared. Organs of fluid loss include the kidneys, skin, lungs, and GI tract.

Laboratory Tests for Evaluating Fluid Status

Serum osmolality primarily reflects the concentration of sodium, although blood urea nitrogen (BUN) and glucose also play a major role in determining serum osmolality. Urine osmolality is determined by urea, creatinine, and uric acid. When measured with serum osmolality, urine osmolality is the most reliable indicator of urine concentration. In healthy adults, normal serum osmolality is 275 to 290 mOsm/kg (Emmett & Palmer, 2018a). The value of osmolarity is usually within 10 mOsm of the value of osmolality (Emmett & Palmer, 2018a). Factors that increase and decrease serum and urine osmolality are identified in [Table 10-3](#). Serum osmolality may be measured directly through laboratory tests or estimated at the bedside by doubling the serum sodium level or by using the following formula:

$$\text{Na}^+ \times 2 = \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{3} = \text{Approximate value of serum osmolality}$$

Urine specific gravity measures the density of urine compared to water. It is a measure of the concentration of solutes in the urine. It is one way to assess the kidneys' ability to excrete or conserve water. The specific gravity of urine is compared to that of distilled water, which has a specific gravity of 1.000. The normal range of urine specific gravity is 1.005 to 1.030 (Van Leeuwen & Bladh, 2017). Urine with a specific gravity of 1.005 is very dilute or high in water content, whereas urine with a specific gravity of 1.030 is very concentrated or low in water content. Under normal conditions, a well-hydrated person excretes urine with a low specific gravity, whereas a dehydrated person excretes urine with high specific gravity. Urine specific gravity can be measured by sending approximately 20 mL of urine to the laboratory for urinalysis testing or assessed using a urine dipstick test. Specific gravity is a less reliable indicator of urine concentration than urine osmolality; increased glucose or protein in urine can cause a falsely elevated specific

gravity. Factors that increase or decrease urine osmolality are the same as those for urine specific gravity (Sterns, 2017a).

TABLE 10-3 Factors Affecting Serum and Urine Osmolality

Fluid	Factors Increasing Osmolality	Factors Decreasing Osmolality
Serum (275–290 mOsm/kg water)	Severe dehydration Free water loss Diabetes insipidus Hypernatremia Hyperglycemia Stroke or head injury Acute tubular necrosis Consumption of methanol or ethylene glycol (antifreeze) High ion gap metabolic acidosis Mannitol therapy Advanced liver disease Alcoholism Burns	Fluid volume excess Syndrome of inappropriate antidiuretic hormone (SIADH) Acute kidney injury Diuretic use Adrenal insufficiency Hyponatremia Overhydration Paraneoplastic syndrome associated with lung cancer
Urine (200–800 mOsm/kg water)	Fluid volume deficit SIADH Congestive heart failure Acidosis Prerenal kidney injury	Fluid volume excess Diabetes insipidus Hyponatremia Aldosteronism Pyelonephritis Acute tubular necrosis

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed.). Philadelphia, PA: Wolters Kluwer.

BUN is a laboratory value that measures the amount of urea in the bloodstream. The normal range of BUN is 10 to 20 mg/dL (3.6 to 7.2 mmol/L). BUN level can vary with renal function, amount of cellular breakdown, protein intake, and hydration status. If urine output decreases, as occurs in renal dysfunction, then urea is not excreted and accumulates in the bloodstream causing an increase. Increased BUN can also occur if water content in the bloodstream decreases due to dehydration. If a person is consuming a high protein diet, there is an increased amino acid content in the bloodstream, which in turn increases nitrogen content of the blood, thereby increasing BUN (Inker & Perrone, 2018).

Additional factors that increase BUN include GI bleeding, fever, and sepsis. Factors that decrease BUN include end-stage liver disease, a low protein diet, starvation (due to low protein), and any condition that results in expanded fluid volume which dilutes urea in the blood (e.g., pregnancy). Because of all these variables, BUN is not an optimal gauge of kidney function (Inker & Perrone, 2018).

Creatinine is a breakdown product of muscle metabolism that is almost totally cleared from the bloodstream and excreted by the kidney. It is a better indicator of renal function than BUN because it does not vary with protein intake or hydration status. The normal serum creatinine is approximately 0.7 to 1.4 mg/dL (62 to 124 mmol/L); however, its concentration depends on lean body mass and varies from person to person. Serum creatinine levels increase when renal function decreases in most people and is an accurate gauge of kidney function (Norris, 2019).

Hematocrit measures the percentage of red blood cells (RBCs) (erythrocytes) in a volume of whole blood and normally ranges from 42% to 52% in men and 35% to 47% in women. Conditions that increase the hematocrit value are dehydration and polycythemia. Dehydration causes decreased water content of the blood which concentrates the RBCs in the bloodstream. Polycythemia is a disorder in which there is an abnormally high number of RBCs made by the bone marrow, which in turn increases the number of RBCs in the bloodstream. Conversely, over hydration (which increases the volume of water within the bloodstream) will decrease the hematocrit. Anemia (which is a lack of sufficient production of RBCs by the bone marrow) causes decreased hematocrit (Schrier, 2018).

Urine sodium values change with sodium intake and the status of fluid volume: As sodium intake increases, excretion increases; as the circulating fluid volume decreases, sodium is conserved. Normal urine sodium levels range from 75 to 200 mEq/24 h (75 to 200 mmol/24 h). A random specimen usually contains more than 40 mEq/L of sodium. Urine sodium levels are used to assess volume status and are useful in the diagnosis of hyponatremia and acute kidney injury (Sterns, 2017a).

Homeostatic Mechanisms

The body is equipped with remarkable homeostatic mechanisms to keep the composition and volume of body fluid within narrow limits of normal. Organs involved in homeostasis include the kidneys, heart, lungs, pituitary gland, adrenal glands, and parathyroid glands (Norris, 2019).

Kidney Functions

Vital to the regulation of fluid and electrolyte balance, the kidneys normally filter 180 L of plasma every day in the adult and excrete 1 to 2 L of urine (Inker & Perrone, 2018). They act both autonomously and in response to hormones, such as aldosterone and antidiuretic hormone (ADH) (Norris, 2019). Major functions of the kidneys in maintaining normal fluid balance include the following:

- Regulation of ECF volume and osmolality by selective retention and excretion of body fluids

- Regulation of normal electrolyte levels in the ECF by selective electrolyte retention and excretion of hydrogen ions
- Regulation of pH of the ECF by retention and excretion of hydrogen ions
- Excretion of metabolic wastes and toxic substances (Inker & Perrone, 2018)

Given these functions, failure of the kidneys results in multiple fluid and electrolyte abnormalities.

Heart and Blood Vessel Functions

The pumping action of the heart circulates blood through the kidneys under sufficient pressure to allow for urine formation. Failure of this pumping action interferes with renal perfusion and thus with water and electrolyte regulation.

Lung Functions

The lungs are also vital in maintaining homeostasis. Through exhalation, the lungs remove approximately 300 mL of water daily in the normal adult as insensible water loss (Sterns, 2017a). Abnormal conditions, such as hyperventilation (abnormally deep respiration) or continuous coughing, increase this water loss. The lungs also play a major role in acid–base balance. Because the lungs regulate carbon dioxide (CO_2), which directly influences acid content of the bloodstream, they influence acid–base balance. When the lungs have a decrease in breathing rate, CO_2 is retained in the alveoli and bloodstream, which increases acid content of the blood. When the lungs have an increase in breathing rate, CO_2 is blown off, lost from the bloodstream, which decreases acid content of the blood (Norris, 2019).

Pituitary Functions

The hypothalamus manufactures ADH, which is stored in the posterior pituitary gland and released as needed to conserve water. ADH is secreted by the pituitary gland in reaction to dehydration or blood loss and acts at the nephrons. At the collecting duct of the nephron, ADH causes increased reabsorption of water from the tubules into the bloodstream (Norris, 2019). This increases the water content of the bloodstream (Fig. 10-5).

Physiology/Pathophysiology

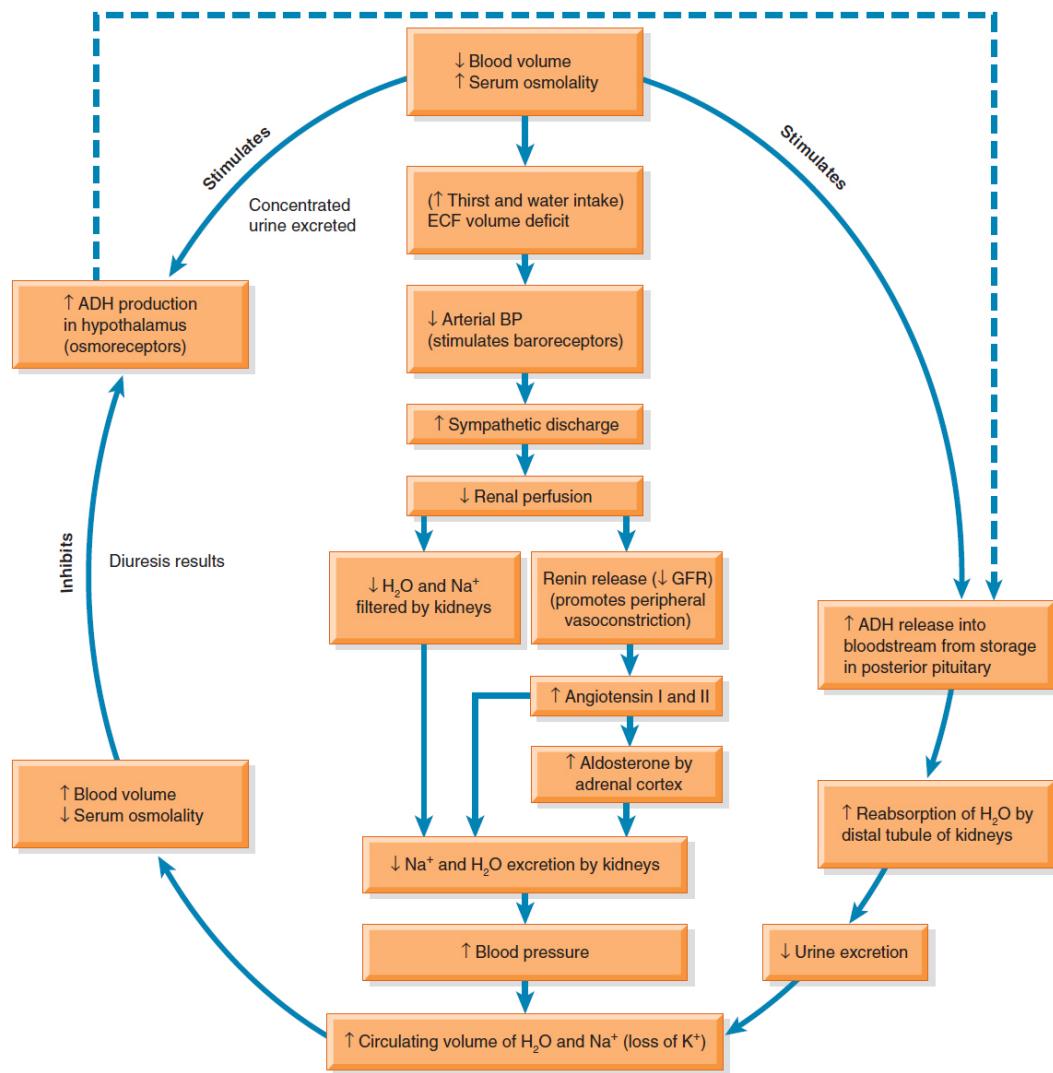


Figure 10-5 • Fluid regulation cycle. ADH, antidiuretic hormone; BP, blood pressure; ECF, extracellular fluid; GFR, glomerular filtration rate.

Adrenal Functions

Aldosterone, a mineralocorticoid secreted by the zona glomerulosa (outer zone) of the adrenal cortex, has a profound effect on fluid balance. Increased secretion of aldosterone causes sodium retention (and thus water retention) and potassium loss. Conversely, decreased secretion of aldosterone causes sodium and water loss and potassium retention.

Cortisol, another adrenocortical hormone, has less mineralocorticoid action. However, when secreted in large quantities (or administered as corticosteroid therapy), it can also produce sodium and fluid retention (Norris, 2019).

Parathyroid Functions

The parathyroid glands, embedded in the thyroid gland, regulate calcium and phosphate balance by means of parathyroid hormone (PTH). PTH influences reabsorption of calcium from the bones into the bloodstream, calcium absorption from the intestine, and calcium reabsorption into the bloodstream from the renal tubules (Norris, 2019).

Baroreceptors

The baroreceptors are located in the left atrium and the carotid and aortic arches. These receptors respond to changes in the circulating blood volume and regulate sympathetic and parasympathetic neural activity as well as endocrine activities.

As arterial pressure decreases, baroreceptors transmit fewer impulses from the carotid and the aortic arches to the vasomotor center. A decrease in impulses stimulates the sympathetic nervous system, which stimulates the sinoatrial (SA) node in the heart. The outcome is an increase in heart rate, conduction, and contractility and an increase in blood pressure. Sympathetic stimulation constricts renal arterioles, which in turn triggers renin release and stimulation of the renin–angiotensin–aldosterone system (see next section for further discussion) (Hall, 2016).

Renin–Angiotensin–Aldosterone System

When the kidneys sense low perfusion or diminished blood pressure, they secrete renin from the juxtaglomerular apparatus, which triggers the renin–angiotensin–aldosterone system. This system is one of the body's most important compensatory mechanisms in maintaining fluid balance. Renin circulates to the liver and converts angiotensinogen, a protein synthesized by the liver, into angiotensin I. Angiotensin-converting enzyme (ACE) converts angiotensin I to angiotensin II. Angiotensin II stimulates potent peripheral arterial vasoconstriction which increases arterial blood pressure. Angiotensin II also stimulates the adrenal gland to secrete aldosterone. Aldosterone increases sodium and water reabsorption at the nephron into the bloodstream. This raises blood volume and blood pressure. Aldosterone also stimulates secretion of potassium into the nephron tubules, which in turn causes potassium excretion by the kidney (Norris, 2019).

Antidiuretic Hormone and Thirst

ADH and the thirst mechanism have important roles in maintaining sodium concentration and oral intake of fluids. Oral intake is controlled by the thirst center located in the hypothalamus. As serum concentration or osmolality increases or blood volume decreases, neurons in the hypothalamus are stimulated by intracellular dehydration; thirst then occurs, and the person

increases their intake of oral fluids. When increased osmolality is sensed by the brain, the posterior pituitary is stimulated to release ADH. ADH acts at the kidney nephrons to increase water reabsorption into the bloodstream. ADH raises blood volume and decreases urine output (Hall, 2016; Norris, 2019).

Osmoreceptors

Located on the surface of the hypothalamus, osmoreceptors sense changes in sodium concentration. As osmotic pressure increases, the neurons become dehydrated and quickly release impulses to the posterior pituitary, which increases the release of ADH, which then travels in the blood to the kidneys, where it alters permeability to water, causing increased reabsorption of water and decreased urine output. The retained water dilutes the ECF and returns its concentration to normal. Restoration of normal osmotic pressure provides feedback to the osmoreceptors to inhibit further ADH release (Hall, 2016) (see Fig. 10-5).

Natriuretic Peptides

Natriuretic peptide hormones affect fluid volume and cardiovascular function through natriuresis (the excretion of sodium), direct vasodilation, and the opposition of the renin–angiotensin–aldosterone system. The most common natriuretic peptides include atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP), and *N*-terminal fragment of pro-brain natriuretic peptide (NT-pro BNP) (Chen & Colucci, 2017). ANP is synthesized, stored, and released by muscle cells of the atria of the heart. BNP and NT-pro BNP are released mainly by ventricular muscle cells of the heart. BNP and NT-pro BNP levels are often measured in the clinical diagnosis and management of heart failure (see Chapter 25). NT-pro BNP has a longer half-life than BNP, so its level remains elevated in the bloodstream for a longer period of time than BNP. Natriuretic peptides are also secreted in other disorders including renal failure, coronary heart disease, valvular heart disease, constrictive pericarditis, pulmonary hypertension, and sepsis. Natriuretic peptides decrease water and sodium in the circulatory system which in turn decreases blood pressure. Their action is directly opposite that of the renin–angiotensin–aldosterone system (Fig. 10-6).

There are two other natriuretic peptides that are not usually measured clinically. C-type natriuretic peptide (CNP) is distributed within the brain, ovary, uterus, testis, and epididymis. D-type natriuretic peptide (DNP) has structural similarities to ANP, BNP, and CNP.



Gerontologic Considerations

Normal physiologic changes of aging include reduced cardiac, renal, and respiratory function. Body fat changes, body water content decreases, and muscle mass decreases. These changes of aging may alter the older adult's responses to fluid and electrolyte changes and acid-base disturbances. Decreased respiratory function and renal function can cause impaired acid-base balance in older adults with major illness or trauma. Decreased renal function that occurs with age can also cause slightly elevated serum creatinine. Decreased muscle mass that occurs with aging leads to decreased daily breakdown of muscle, which reduces serum creatinine. Therefore, high-normal and minimally elevated serum creatinine values may indicate substantially reduced renal function in older adults (Cash & Glass, 2018).

In addition, the use of multiple medications by older adults can affect renal and cardiac function and body water content, thereby increasing susceptibility to fluid and electrolyte disturbances. Routine procedures, such as the vigorous administration of laxatives or enemas before colon x-ray studies, may produce a serious FVD, necessitating the use of IV fluids to prevent hypotension and other effects of hypovolemia.

Alterations in fluid and electrolyte balance that may produce minor changes in young and middle-aged adults may produce profound changes in older adults. In many older patients, the clinical manifestations of fluid and electrolyte disturbances may be subtle or atypical. For example, fluid deficit may not trigger thirst sensation and can lead to delirium in the older adult (see [Chapter 8](#)). Rapid infusion of an excessive volume of IV fluids can cause fluid overload and cardiac failure in older patients. These reactions are likely to occur more quickly and with the administration of smaller volumes of fluid than in healthy young and middle-aged adults because of the decreased cardiac reserve and reduced renal function that accompany aging (Cash & Glass, 2018).

Physiology/Pathophysiology

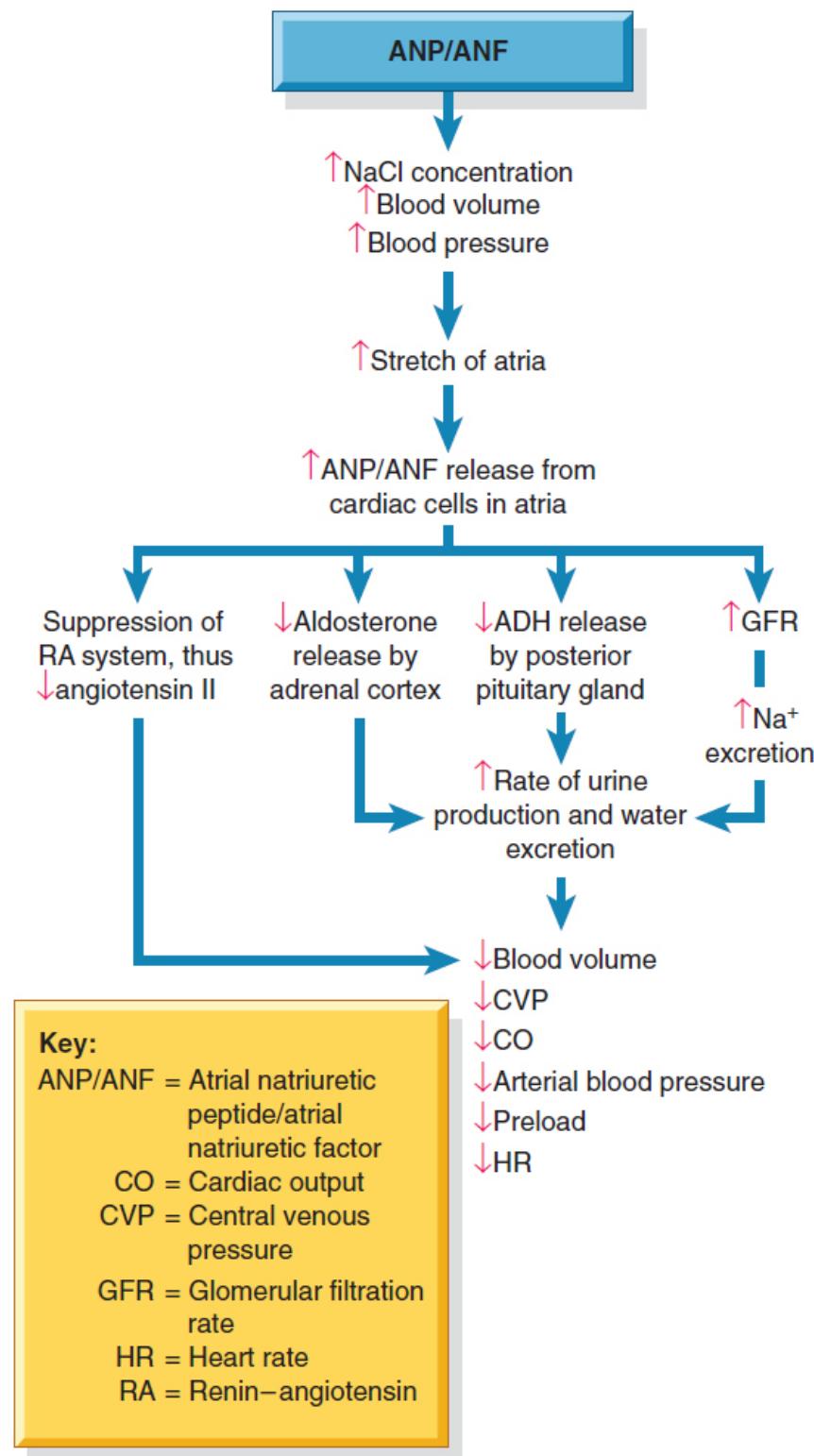


Figure 10-6 • Role of atrial natriuretic peptide in maintenance of fluid balance.

Dehydration is the rapid loss of body weight due to the loss of either water or sodium. This results in an elevated sodium concentration (Sterns, 2017c). Dehydration in older adults is common because of decreased kidney mass, decreased glomerular filtration rate, decreased renal blood flow, decreased ability to concentrate urine, inability to conserve sodium, decreased excretion of potassium, and a decrease of total-body water. Loss of subcutaneous tissue and resultant thinning of the skin occurs with aging; the dermis is dehydrated and loses strength and elasticity.

FLUID VOLUME DISTURBANCES

Hypovolemia

FVD, or hypovolemia, occurs when loss of ECF volume exceeds the intake of fluid. It occurs when water and electrolytes are lost in the same proportion as they exist in normal body fluids; thus, the ratio of serum electrolytes to water remains the same. FVD should not be confused with dehydration, which refers to loss of water alone, with increased serum sodium levels. FVD may occur alone or in combination with other imbalances. Serum electrolyte concentrations can remain normal, increase, or increase in FVD (Sterns, 2017a).

Pathophysiology

FVD results from loss of body fluids and occurs more rapidly when coupled with decreased fluid intake. FVD can also develop with a prolonged period of inadequate intake. Causes of FVD include abnormal fluid losses, such as those resulting from vomiting, diarrhea, GI suctioning, and sweating; decreased intake, as in nausea or lack of access to fluids; and third-space fluid shifts, or the movement of fluid from the vascular system to other body spaces (e.g., with edema formation in burns, ascites with liver dysfunction). Additional causes include diabetes insipidus (a decreased ability to concentrate urine due to either a deficit of ADH or nephron resistance to ADH), adrenal insufficiency, osmotic diuresis, hemorrhage, and coma (Sterns, 2017a).

Clinical Manifestations

FVD can develop rapidly, and its severity depends on the degree of fluid loss. Clinical signs and symptoms and laboratory findings are presented in [Table 10-4](#).

Assessment and Diagnostic Findings

Laboratory data used to evaluate fluid volume status include BUN and its relation to serum creatinine concentration. Normal BUN to serum creatinine concentration ratio is 10:1. A volume-depleted patient has a BUN elevated out of proportion to the serum creatinine (ratio greater than 20:1) because urea becomes concentrated in FVD (Sterns, 2017a).

The presence and cause of hypovolemia may be determined through the health history and physical examination. In addition, the hematocrit level is greater than normal because there is a decreased plasma volume, which concentrates the volume of RBCs.

Serum electrolyte changes may also exist. Potassium and sodium levels can be reduced (hypokalemia, hyponatremia) or elevated (hyperkalemia, hypernatremia).

- Hypokalemia can occur with GI and renal losses as these organs are major regulators of potassium.
- Hyperkalemia can occur with adrenal insufficiency due to aldosterone deficiency which causes lack of potassium excretion.
- Hyponatremia can occur with increased thirst and ADH release, which increases water content of the bloodstream.
- Hypernatremia can result from increased insensible water losses and diabetes insipidus.

TABLE 10-4 Fluid Volume Disturbances

Imbalance	Contributing Factors	Signs/Symptoms and Laboratory Findings
Fluid volume deficit (hypovolemia)	Loss of water and electrolytes, as in vomiting, diarrhea, fistulas, fever, excess sweating, burns, blood loss, gastrointestinal suction, and third-space fluid shifts; and decreased intake, as in anorexia, nausea, and inability to gain access to fluid. Diabetes insipidus and uncontrolled diabetes both contribute to a depletion of extracellular fluid volume.	Acute weight loss, ↓ skin turgor, oliguria, concentrated urine, capillary filling time prolonged, low CVP, ↓ BP, flattened neck veins, dizziness, weakness, thirst and confusion, ↑ pulse, muscle cramps, sunken eyes, nausea, increased temperature; cool, clammy, pale skin <i>Labs indicate:</i> ↑ hemoglobin and hematocrit, ↑ serum and urine osmolality and specific gravity, ↓ urine sodium, ↑ BUN and creatinine, ↑ urine specific gravity and osmolality
Fluid volume excess (hypervolemia)	Compromised regulatory mechanisms, such as kidney injury, heart failure, and cirrhosis; overzealous administration of sodium-containing fluids; and fluid shifts (i.e., treatment of burns). Prolonged corticosteroid therapy, severe stress, and hyperaldosteronism augment fluid volume excess.	Acute weight gain, peripheral edema and ascites, distended jugular veins, crackles, elevated CVP, shortness of breath, ↑ BP, bounding pulse and cough, ↑ respiratory rate, ↑ urine output <i>Labs indicate:</i> ↓ hemoglobin and hematocrit, ↓ serum and urine osmolality, ↓ urine sodium and specific gravity

BP, blood pressure; BUN, blood urea nitrogen; CVP, central venous pressure; ↓, decreased; ↑, increased.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Oliguria, the excretion of less than 400 mL urine per day in the adult, may or may not be present in hypovolemia. Urine specific gravity will change in relation to the kidneys' attempt to conserve water. If the kidney does not reabsorb water, urine contains more water, and urine specific gravity is low. If the kidney does reabsorb water, urine will be concentrated and specific gravity increases. Due to lack of ADH in diabetes insipidus, urine water content increases, which decreases urine specific gravity. Aldosterone is secreted when fluid volume is low, causing reabsorption of sodium and chloride and resulting in decreased urinary sodium and chloride. When the kidneys conserve water, urine osmolality can increase to greater than 450 mOsm/kg and urine specific gravity increases (Sterns, 2017a). Normal values for laboratory data are listed in Appendix A on [thePoint](#).

Gerontologic Considerations

Increased sensitivity to fluid and electrolyte changes in older patients requires careful physical assessment, measurement of I&O of fluids from all sources, assessment of daily weight, careful monitoring of side effects and interactions of medications, and prompt reporting and management of disturbances. In most adult patients, it is useful to monitor skin turgor to detect subtle changes. However, assessment of skin turgor is not as valid in older adults because the skin has lost elasticity; therefore, other assessment measures (e.g., slowness in filling of veins of the hands and feet) become more useful in detecting FVD (Cash & Glass, 2018; Weber & Kelley, 2018).

The nurse also performs a functional assessment of the older patient's ability to determine fluid and food needs and to obtain adequate intake in addition to assessments discussed earlier in this chapter. For example, the nurse assesses whether or not the patient is cognitively intact, able to ambulate and to use both arms and hands to reach fluids and foods, and able to swallow with an intact gag reflex. Results of this functional assessment have a direct bearing on how the patient will be able to meet their own need for fluids and foods (Weber & Kelley, 2018). During an older patient's hospital stay, the nurse provides fluids if the patient is unable to carry out self-care activities.

The nurse should also recognize that some older patients deliberately restrict their fluid intake to avoid episodes of urinary incontinence. In this situation, the nurse should identify interventions to deal with the incontinence, such as encouraging the patient to wear protective clothing or devices, to carry a urinal in the car, or to pace fluid intake to allow access to toilet facilities during the day. Older adults without cardiovascular or renal dysfunction should be reminded to drink adequate fluids, particularly in very warm or humid weather (Cash & Glass, 2018).

Medical Management

When planning the correction of fluid loss for the patient with FVD, the primary provider considers the patient's maintenance requirements and other factors (e.g., fever) that can influence fluid needs. If the deficit is not severe, the oral route is preferred, provided the patient can drink. However, if fluid losses are acute or severe, the IV route is required. Isotonic electrolyte crystalloid solutions (e.g., lactated Ringer's solution or 0.9% sodium chloride) are frequently the first-line choice to treat the hypotensive patient with FVD because they expand plasma volume. As soon as the patient becomes normotensive, a hypotonic electrolyte solution (e.g., 0.45% sodium chloride) is often used to provide both electrolytes and water for renal excretion of metabolic wastes (Sterns, 2017a; Sterns, 2017b). These and additional fluids are listed in [Table 10-5](#).

Accurate and frequent assessments of I&O, weight, vital signs, central venous pressure, level of consciousness, breath sounds, and skin color are monitored to determine when therapy should be slowed to avoid volume overload. The rate of fluid administration is based on the severity of loss and the patient's hemodynamic response to volume replacement (Sterns, 2017a; Sterns, 2017b).

If the patient with severe FVD is not excreting enough urine and is therefore oliguric, the primary provider needs to determine whether the depressed renal function is caused by reduced renal blood flow secondary to FVD (prerenal azotemia) or, more seriously, by acute tubular necrosis (intrarenal azotemia) from prolonged FVD (Norris, 2019). The test used in this situation is referred to as a fluid challenge test. During a fluid challenge test, volumes of fluid are given at specific rates and intervals while the patient's hemodynamic response to this treatment is monitored (i.e., vital signs, breath sounds, orientation status, central venous pressure, urine output) (Sterns, 2017b).

TABLE 10-5 Select Water and Electrolyte Solutions

Solution	Considerations
Isotonic Solutions 0.9% NaCl (isotonic, also called <i>normal saline</i> [NS]) Na ⁺ 154 mEq/L Cl ⁻ 154 mEq/L (308 mOsm/L) Also available with varying concentrations of dextrose (a 5% dextrose concentration is commonly used)	<ul style="list-style-type: none"> An isotonic solution that expands the extracellular fluid (ECF) volume; used in hypovolemic states, resuscitative efforts, shock, diabetic ketoacidosis, metabolic alkalosis, hypercalcemia, mild Na⁺ deficit Supplies an excess of Na⁺ and Cl⁻; can cause fluid volume excess and hyperchloremic acidosis if used in excessive volumes, particularly in patients with compromised renal function, heart failure, or edema When mixed with 5% dextrose, the resulting solution becomes temporarily hypertonic in relation to plasma and, in addition to the previously described electrolytes, provides 170 cal/L. After dextrose is metabolized it leaves an isotonic solution. Only solution that may be given with blood products Tonicity similar to plasma
Lactated Ringer's solution Na ⁺ 130 mEq/L K ⁺ 4 mEq/L Ca ⁺⁺ 3 mEq/L Cl ⁻ 109 mEq/L Lactate (metabolized to bicarbonate) 28 mEq/L (274 mOsm/L) Also available with varying concentrations of dextrose (the most common is 5% dextrose)	<ul style="list-style-type: none"> An isotonic solution that contains multiple electrolytes in roughly the same concentration as found in plasma (note that solution is lacking in Mg⁺⁺); provides 9 cal/L Used in the treatment of hypovolemia, burns, fluid lost as bile or diarrhea, and for acute blood loss replacement Lactate is rapidly metabolized into HCO₃⁻ in the body. Lactated Ringer's solution should not be used in lactic acidosis because the ability to convert lactate into HCO₃⁻ is impaired in this disorder Not to be given with a pH >7.5 because bicarbonate is formed as lactate breaks down, causing alkalosis Should not be used in kidney injury because it contains potassium and can cause hyperkalemia Tonicity similar to plasma
5% dextrose in water (D ₅ W) No electrolytes 50 g of dextrose	<ul style="list-style-type: none"> An isotonic solution that supplies 170 cal/L and free water to aid in renal excretion of solutes Used in treatment of hypernatremia, fluid loss, and dehydration Should not be used in excessive volumes in the early postoperative period (when antidiuretic hormone secretion is increased due to stress reaction) Should not be used solely in treatment of fluid volume deficit because it dilutes plasma electrolyte concentrations Contraindicated in head injury because it may cause increased intracranial pressure

- Should not be used for fluid resuscitation because it can cause hyperglycemia
- Should be used with caution in patients with renal or cardiac disease because of risk of fluid overload
- Electrolyte-free solutions may cause peripheral circulatory collapse, anuria in patients with sodium deficiency, and increased body fluid loss
- Converts to hypotonic solution as dextrose is metabolized by body. Over time, D₅W without NaCl can cause water intoxication (intracellular fluid volume excess [FVE]) because the solution is hypotonic
- Fluid therapy for an extended period of time without electrolytes may result in hypokalemia

Hypotonic Solutions

0.45% NaCl (half-strength saline)
 Na^+ 77 mEq/L
 Cl^- 77 mEq/L
(154 mOsm/L)
Also available with varying concentrations of dextrose (the most common is a 5% concentration)

- Provides Na^+ , Cl^- , and free water
- Free water is desirable to aid the kidneys in elimination of solute
- Lacking in electrolytes other than Na^+ and Cl^-
- When mixed with 5% dextrose, the solution becomes slightly hypertonic to plasma temporarily until dextrose is metabolized. It leaves a hypotonic solution after dextrose metabolism. Provides 170 cal/L
- Used to treat hypertonic dehydration, Na^+ and Cl^- depletion, and gastric fluid loss
- Not indicated for third-space fluid shifts or increased intracranial pressure
- Administer cautiously, because hypotonic solution can cause fluid shifts from vascular system into cells, resulting in cardiovascular collapse and increased intracranial pressure

Hypertonic Solutions

3% NaCl (hypertonic saline)
 Na^+ 513 mEq/L
 Cl^- 513 mEq/L
(1026 mOsm/L)
5% NaCL (hypertonic solution)
 Na^+ 855 mEq/L
 Cl^- 855 mEq/L
(1710 mOsm/L)
IV Mannitol 5–25% (hypertonic solution)
(1372 mOsm/L contained in 25% solution)

- Used to increase ECF volume, decrease cellular swelling
- Highly hypertonic solution used only in critical situations to treat hyponatremia
- Must be given slowly and cautiously, because it can cause intravascular volume overload and pulmonary edema
- Assists in removing intracellular fluid excess
- Highly hypertonic solution used to treat symptomatic hyponatremia
- Administer slowly and cautiously because it can cause intravascular volume overload and pulmonary edema
- Supplies no calories

Colloid Solutions

Dextran in NS or D₅W

Available in low-molecular-weight (Dextran 40) and high-molecular-weight (Dextran 70) forms

- Colloid solution used as volume/plasma expander for intravascular part of ECF
- Affects clotting by coating platelets and decreasing ability to clot
- Remains in circulatory system up to 24 h
- Used to treat hypovolemia in early shock to increase pulse pressure, cardiac output, and arterial blood pressure
- Improves microcirculation by decreasing red blood cell aggregation
- Contraindicated in hemorrhage, thrombocytopenia, renal disease, and severe dehydration
- Not a substitute for blood or blood products

An example of a typical fluid challenge test involves administering 100 to 200 mL of normal saline solution over 15 minutes. The goal is to provide fluids rapidly enough to attain adequate tissue perfusion without compromising the cardiovascular system. The response by a patient with FVD but normal renal function is increased urine output and an increase in blood pressure and central venous pressure.

Shock can occur when the volume of fluid lost exceeds 25% of the intravascular volume or when fluid loss is rapid (Mandel & Palevsky, 2019). (Shock and its causes and treatment are discussed in detail in [Chapter 11](#).)

Nursing Management

To assess for FVD, the nurse monitors and measures fluid I&O at least every 8 hours, and sometimes hourly. Maintaining an accurate I&O is a particular challenge with patients in critical-care settings. As FVD develops, body fluid losses exceed fluid intake through excessive urination (polyuria), diarrhea, vomiting, or other mechanisms. Once FVD has developed, the kidneys attempt to conserve body fluids, leading to a urine output of less than 1 mL/kg/h in an adult. Urine in this instance is concentrated and represents a healthy renal response.

Vital signs should be closely monitored in FVD.



Quality and Safety Nursing Alert

The nurse observes for a weak, rapid pulse and orthostatic hypotension (i.e., a decrease in systolic pressure exceeding 20 mm Hg when the patient moves from a lying to a sitting position).

A decrease in body temperature often accompanies FVD, unless there is a concurrent infection.

Skin and tongue turgor are monitored on a regular basis. In a healthy person, pinched skin immediately returns to its normal position when released (Weber & Kelley, 2018). This elastic property, referred to as turgor, is partially dependent on interstitial fluid volume. In a person with FVD, the skin flattens more slowly after the pinch is released. In a person with severe FVD, the skin may remain elevated for many seconds. Tissue turgor is best measured by pinching the skin over the sternum, dorsal surface of the hand, inner aspects of the thighs, or forehead. It is important to recognize skin turgor is normally diminished in old age. Tongue turgor is not affected by age, and evaluating this may be more valid than evaluating skin turgor (Sterns, 2017a). In a normal person, the tongue has one longitudinal furrow. In the person with FVD, there are additional longitudinal furrows and the tongue is smaller because of fluid loss. The degree of oral mucous membrane moisture is also assessed; a dry mouth may indicate either FVD or mouth breathing.



Quality and Safety Nursing Alert

It is useful to monitor daily body weight when monitoring fluid volume; an acute loss of 0.5 kg (1.1 lb) represents a fluid loss of approximately 500 mL. One liter (1000 mL) of fluid weighs approximately 1 kg, or 2.2 lb. A weight loss or gain of 1–2 lb/day is mainly due to water loss or gain.

Urine concentration is monitored by measuring the urine specific gravity. In a volume-depleted patient, the urine specific gravity should be greater than 1.020, indicating healthy renal conservation of fluid. Dark amber-colored urine is highly concentrated; whereas, clear yellow urine indicates a dilute urine.

Mental function is eventually affected, resulting in confusion, lack of cognition, and delirium in severe FVD as a result of decreasing cerebral perfusion. Behavioral changes are particularly evident in older adults with FVD. Decreased peripheral perfusion can result in cold extremities. In patients with relatively normal cardiopulmonary function, a low central venous pressure is indicative of hypovolemia (Sterns, 2017a). Patients with acute cardiopulmonary decompensation require more extensive hemodynamic monitoring of pressures in both sides of the heart to determine whether hypovolemia exists. Hemodynamic monitoring is particularly important in critically ill patients (Mandel & Palevsky, 2019) (see Chapters 11 and 21).

Preventing Hypovolemia

To prevent FVD, the nurse identifies patients at risk and takes measures to minimize fluid losses. For example, if the patient has diarrhea, measures should be implemented to control diarrhea and replacement fluids given. This includes administering antidiarrheal medications and small volumes of oral fluids at frequent intervals.

Correcting Hypovolemia

When possible, oral fluids are given to help correct FVD, with consideration given to the patient's likes and dislikes. The type of fluid the patient has lost is also considered, and fluids most likely to replace the lost electrolytes are appropriate. If the patient is reluctant to drink because of oral discomfort, the nurse assists with frequent mouth care and provides nonirritating fluids. The patient may be offered small volumes of oral rehydration solutions (e.g., Rehydralyte, Elete, and Cytomax). These solutions provide fluid, glucose, and electrolytes in concentrations that are easily absorbed. If nausea is present, an antiemetic may be needed before oral fluid replacement can be tolerated (Sterns, 2017a; Sterns, 2017b).

If the deficit cannot be corrected by oral fluids, therapy may need to be initiated by an alternative route (enteral or parenteral) until adequate circulating blood volume and renal perfusion are achieved. Isotonic fluids are prescribed to increase ECF volume (Sterns, 2017b).

Hypervolemia

Fluid volume excess (FVE), or hypervolemia, refers to an expansion of the ECF caused by the abnormal retention of water and sodium in approximately the same proportions in which they normally exist in the ECF. It is most often secondary to an increase in the total-body sodium content, which, in turn, leads to an increase in total-body water. This can be referred to as an isotonic accumulation of fluids. Because there is isotonic retention of body substances, the serum sodium concentration remains essentially normal.

Pathophysiology

FVE may be related to simple fluid overload or diminished function of the homeostatic mechanisms responsible for regulating fluid balance. Contributing factors can include heart failure, kidney dysfunction, and cirrhosis of the liver. Another contributing factor is consumption of excessive amounts of table or other sodium salts. Excessive administration of sodium-containing fluids in a patient with impaired regulatory mechanisms may predispose them to a serious FVE as well (Sterns, 2018a).

Clinical Manifestations

Clinical manifestations of FVE result from expansion of the ECF and may include edema, distended jugular veins, and crackles (abnormal lung sounds due to interstitial pulmonary fluid). In patients who are ambulatory, edema is most evident in the ankles; in patients who are supine, edema occurs over the sacrum (Weber & Kelley, 2018). Further discussion of clinical signs and symptoms and laboratory findings can be found in [Table 10-4](#).

Assessment and Diagnostic Findings

Laboratory data useful in diagnosing FVE include BUN and hematocrit levels. In FVE, both of these values may be decreased because of plasma dilution. In chronic kidney disease, both serum osmolality and the sodium level are decreased due to excessive retention of water. The urine sodium level is increased if the kidneys are attempting to excrete excess volume. A chest x-ray may reveal pulmonary congestion in FVE. Hypervolemia occurs when aldosterone is chronically stimulated—for example, in conditions such as cirrhosis, heart failure, and nephrotic syndrome. Aldosterone increases both sodium and water reabsorption into the bloodstream from the nephron; therefore, the urine sodium level is normal in these conditions (Emmett & Palmer, 2019; Sterns, 2018a).

Medical Management

Management of FVE is directed at the causes, and if related to excessive administration of sodium-containing fluids, discontinuing the infusion may be all that is needed. Symptomatic treatment consists of administering diuretics and restricting fluids and sodium. Diuretics are medications that reduce sodium and water reabsorption at the nephron and thereby enhance water loss via the kidneys (Brater & Ellison, 2019).

Pharmacologic Therapy

Diuretics are prescribed when dietary restriction of sodium alone is insufficient to reduce edema. The choice of diuretic is based on the severity of the hypervolemic state, the degree of impairment of renal function, and the potency of the diuretic. Thiazide diuretics block sodium and water reabsorption into the bloodstream at the distal tubule of the nephron, where 5% to 10% of sodium is normally reabsorbed. This leads to a small amount of sodium and water loss via the urine. Loop diuretics, such as furosemide, bumetanide, or torsemide, can cause a greater loss of both sodium and water because they block sodium reabsorption in the ascending limb of the loop of Henle, where 20% to 30% of filtered sodium is normally reabsorbed.

Generally, thiazide diuretics, such as hydrochlorothiazide, are prescribed for mild to moderate hypervolemia and loop diuretics for severe hypervolemia (Brater & Ellison, 2019).

Electrolyte imbalances may result from side effects of diuretics. Hypokalemia can occur with all diuretics except those that inhibit aldosterone. Potassium supplements can be prescribed with diuretics to avoid this complication. Hyperkalemia can occur with diuretics that inhibit aldosterone (e.g., spironolactone, a potassium-sparing diuretic), especially in patients with decreased renal function. Hyponatremia occurs with diuresis due to increased release of ADH secondary to reduction in circulating volume. Decreased magnesium levels occur with administration of loop and thiazide diuretics due to decreased reabsorption and increased excretion of magnesium by the kidney (Brater & Ellison, 2019; Vallerand & Sanoski, 2019).

Azotemia (increased nitrogen levels in the blood) can occur with FVE when urea and creatinine are not excreted due to decreased perfusion by the kidneys and decreased excretion of waste, as occurs in renal failure. High uric acid levels (hyperuricemia) can also occur from increased reabsorption and decreased excretion of uric acid by the kidneys.

Dialysis

If renal function is so severely impaired that pharmacologic agents cannot act efficiently, other modalities are considered to remove sodium and fluid from the body. Hemodialysis or peritoneal dialysis may be used to remove nitrogenous wastes and control potassium and acid–base balance, and to remove sodium and fluid. Continuous renal replacement therapy may also be required (see [Chapter 48](#) for a discussion of these treatment modalities).

Nutritional Therapy

Treatment of FVE usually involves dietary restriction of sodium. An average daily diet not restricted in sodium contains 6 to 15 g of salt, whereas low sodium diets can range from a mild restriction (less than 2000 mg/day) to as little as 250 mg of sodium per day, depending on the patient's needs. A mild sodium-restricted diet allows only light salting of food (about half the usual amount) in cooking and at the table, and no addition of salt to commercially prepared foods that are already seasoned. Foods high in sodium must be avoided. It is the sodium salt (sodium chloride) rather than sodium itself that contributes to edema. Therefore, patients are instructed to read food labels carefully to determine salt content (Olendzki, 2017).

Because about half of ingested sodium is in the form of seasoning, seasoning substitutes can play a major role in decreasing sodium intake. Lemon juice, onions, and garlic are excellent substitute flavorings, although some patients prefer salt substitutes. Most salt substitutes contain potassium

and must therefore be used cautiously by patients taking potassium-sparing diuretics (e.g., spironolactone, triamterene, amiloride). These substitutes should not be used in conditions associated with potassium retention, such as advanced kidney disease. Salt substitutes containing ammonium chloride can be harmful to patients with liver damage (Olendzki, 2017; Vallerand & Sanoski, 2019).

In some communities, drinking water may contain too much sodium for a sodium-restricted diet. Depending on its source, water may contain as little as 1 mg or more than 1500 mg of sodium per quart. Patients may need to use distilled water if the local water supply is very high in sodium. Bottled water can have a sodium content that ranges from 0 to 1200 mg/L; therefore, if sodium is restricted, the label must be carefully examined for sodium content before purchasing and drinking bottled water. Also, patients on sodium-restricted diets should be cautioned to avoid water softeners that add sodium to water in exchange for other ions, such as calcium. Protein intake may be increased in patients who are malnourished or who have low serum protein levels in an effort to increase capillary oncotic pressure. Increasing oncotic pressure in the bloodstream will pull fluid out of the tissues into vessels for excretion by the kidneys (Sterns, 2018a).

Nursing Management

To assess for FVE, the nurse measures I&O at regular intervals to identify excessive fluid retention. The patient is weighed daily, and rapid weight gain is noted. Breath sounds are assessed at regular intervals in at-risk patients, particularly if parenteral fluids are being given. The nurse monitors the degree of edema in the most dependent parts of the body, such as the feet and ankles in ambulatory patients and the sacral region in patients confined to bed. Pitting edema is assessed by pressing a finger into the affected part, creating a pit or indentation that is evaluated on a scale of 1+ (minimal) to 4+ (severe) (see Chapter 25, Fig. 25-2). Peripheral edema is monitored by measuring the circumference of the extremity with a tape measure marked in millimeters (Weber & Kelley, 2018).



Quality and Safety Nursing Alert

An acute weight gain of 1 kg (2.2 lb) is equivalent to a gain of approximately 1 L of fluid.

Preventing Hypervolemia

Specific interventions vary with the underlying condition and the degree of FVE. However, most patients require sodium-restricted diets in some form,

and adherence to the prescribed diet is encouraged. Patients are instructed to avoid over-the-counter (OTC) medications without first checking with a health care provider, because they may contain sodium (e.g., Alka-Seltzer). If fluid retention persists despite adherence to a prescribed diet, hidden sources of sodium, such as the water supply or use of water softeners, should be considered.

Detecting and Controlling Hypervolemia

It is important to detect FVE before the condition becomes severe. Interventions include promoting rest, restricting sodium intake, monitoring parenteral fluid therapy, and administering appropriate medications.

Regular rest periods may be beneficial, because bed rest favors diuresis of fluid. The mechanism is related to diminished venous pooling and the subsequent increase in effective circulating blood volume and renal perfusion. Sodium and fluid restriction should be instituted as indicated. Because most patients with FVE require diuretics, the patient's response to these agents is monitored. The rate of parenteral fluids and the patient's response to these fluids are also closely monitored (Frandsen & Pennington, 2018). If dyspnea or orthopnea is present, the patient is placed in a semi-Fowler position to promote lung expansion. The patient is turned and repositioned at regular intervals because edematous tissue is more prone to skin breakdown than normal tissue. Because conditions predisposing to FVE are likely to be chronic, patients are taught to monitor their response to therapy by documenting fluid I&O and body weight changes. The importance of adhering to the treatment regimen is emphasized.



Educating Patients About Edema

Because edema is a common manifestation of FVE, patients need to recognize its symptoms and understand its importance. The nurse gives special attention to edema when instructing the patient with FVE. Edema can occur as a result of increased capillary fluid pressure, decreased capillary oncotic pressure, or increased interstitial oncotic pressure, causing expansion of the interstitial fluid compartment (Hall, 2016). Edema can be localized (e.g., in the ankle, as in rheumatoid arthritis) or generalized (as in cardiac failure and kidney injury) (Sterns, 2018b). Severe generalized edema is called *anasarca*.

Edema occurs when there is a change in the capillary membrane, increasing the formation of interstitial fluid or decreasing the removal of interstitial fluid. Sodium retention is a frequent cause of the increased ECF volume. Burns and infection are examples of conditions associated with increased interstitial fluid volume. Obstruction to lymphatic outflow, a plasma albumin level less than 1.5 to 2 g/dL, or a decrease in plasma oncotic pressure contributes to increased

interstitial fluid volume. If there is decreased cardiac output as in heart failure, the kidneys sense low perfusion and secrete renin which triggers the renin–angiotensin–aldosterone system that increases sodium and water retention (Sterns, 2018b). A thorough medication history is necessary to identify any medications that could cause edema, such as nonsteroidal anti-inflammatory drugs (NSAIDs), estrogens, corticosteroids, and antihypertensive agents (Vallerand & Sanoski, 2019).

Ascites is a type of edema in which fluid accumulates in the peritoneal cavity; it results from heart failure, nephrotic syndrome, cirrhosis, and some malignant tumors. The patient commonly reports shortness of breath and a sense of pressure because of pressure on the diaphragm.

The goal of treatment is to preserve or restore the circulating intravascular fluid volume. Thus, in addition to treating the cause of the edema, other treatments may include diuretic therapy, restriction of fluids and sodium, elevation of the extremities, application of anti-embolic stockings, paracentesis (pulling fluid out of the peritoneal cavity using a needle and syringe), dialysis, and continuous renal replacement therapy in cases of kidney injury or life-threatening fluid volume overload (Sterns, 2018b).

ELECTROLYTE IMBALANCES



Disturbances in electrolyte balances are common in clinical practice and may need to be corrected based on history, physical examination findings, and laboratory values (with comparison to previous values).

Sodium Imbalances

Sodium (Na^+) is the most abundant electrolyte in the ECF; its concentration ranges from 135 to 145 mEq/L (135 to 145 mmol/L), and it is the primary determinant of ECF volume and osmolality. Sodium has a major role in controlling water distribution throughout the body, because it does not easily cross the plasma membrane and because of its abundance and high concentration in the body. Sodium is regulated by ADH, thirst, and the renin–angiotensin–aldosterone system. A loss or gain of sodium is usually accompanied by a loss or gain of water. Sodium also functions in establishing the electrochemical state necessary for muscle contraction and the transmission of nerve impulses (Hall, 2016).

The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) may be associated with sodium imbalance. When there is a decrease in the circulating plasma osmolality, blood volume, or blood pressure, ADH (also

called arginine vasopressin [AVP]) is released from the posterior pituitary. Oversecretion of ADH can cause SIADH. Patients at risk for SIADH include older adults; those who have had brain surgery or have a brain tumor, pulmonary malignancy, or acquired immune deficiency syndrome (AIDS); those on mechanical ventilation; and those taking selective serotonin reuptake inhibitors (SSRIs) (Sterns, 2017d). (SIADH is discussed in more detail in [Chapter 45](#).)

Sodium imbalance can develop under simple or complex circumstances. The two most common sodium imbalances are sodium deficit and sodium excess ([Table 10-6](#)).

TABLE 10-6 Sodium Imbalances

Imbalance	Contributing Factors	Signs/Symptoms and Laboratory Findings
Sodium deficit (hyponatremia) Serum sodium <135 mEq/L	Loss of sodium, as in use of diuretics, loss of GI fluids, renal disease, and adrenal insufficiency. Gain of water, as in excessive administration of D ₅ W and water supplements for patients receiving hypotonic tube feedings; disease states associated with SIADH, such as head trauma and oat-cell lung tumor; medications associated with water retention (oxytocin and certain tranquilizers); and psychogenic polydipsia. Hyperglycemia and heart failure cause a loss of sodium.	Anorexia, nausea and vomiting, headache, lethargy, dizziness, confusion, muscle cramps and weakness, muscular twitching, seizures, papilledema, dry skin, ↑ pulse, ↓ BP, weight gain, edema <i>Labs indicate:</i> ↓ serum and urine sodium, ↓ urine specific gravity and osmolality
Sodium excess (hypernatremia) Serum sodium >145 mEq/L	Fluid deprivation in patients who cannot respond to thirst, hypertonic tube feedings without adequate water supplements, diabetes insipidus, heatstroke, hyperventilation, watery diarrhea, burns, and diaphoresis. Excess corticosteroid, sodium bicarbonate, and sodium chloride administration, and saltwater nonfatal drowning victims.	Thirst, elevated body temperature, swollen dry tongue and sticky mucous membranes, hallucinations, lethargy, restlessness, irritability, simple partial or tonic-clonic seizures, pulmonary edema, hyperreflexia, twitching, nausea, vomiting, anorexia, ↑ pulse, and ↑ BP <i>Labs indicate:</i> ↑ serum sodium, ↓ urine sodium, ↑ urine specific gravity and osmolality, ↓ CVP

BP, blood pressure; CVP, central venous pressure; ↓, decreased; D₅W, dextrose 5% in water; GI, gastrointestinal; ↑, increased; SIADH, syndrome of inappropriate secretion of antidiuretic hormone.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

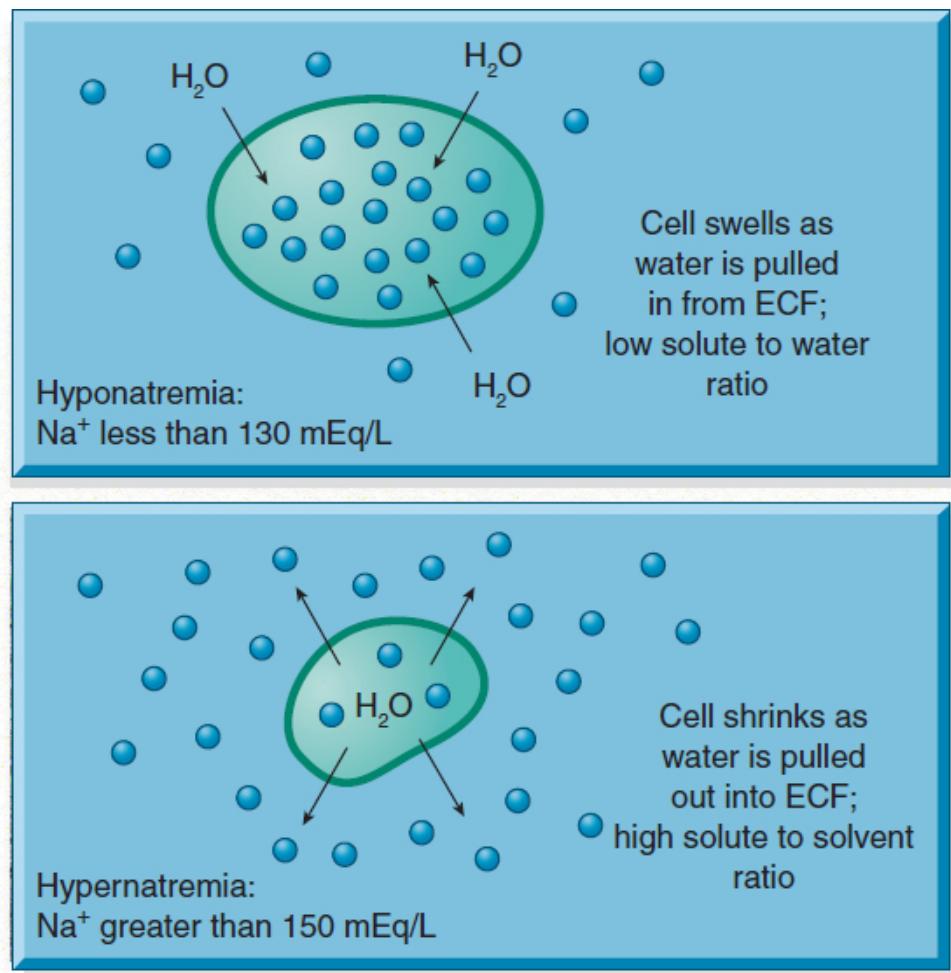


Figure 10-7 • Effect of extracellular sodium level on cell size.

Sodium Deficit (Hyponatremia)

Hyponatremia refers to a serum sodium level that is less than 135 mEq/L (135 mmol/L) (Sterns, 2017e). Hyponatremia can present as an acute or chronic form. Acute hyponatremia is commonly the result of a fluid overload in a surgical patient. This is a dilutional hyponatremia because the excess water dilutes the sodium in the bloodstream. Chronic hyponatremia is seen more frequently in patients outside the hospital setting, has a longer duration, and has less serious neurologic sequelae. Another type of hyponatremia is exercise-associated hyponatremia, which is more frequently found in women and those of smaller stature. It can occur during extreme temperatures, because of excessive fluid intake before exercise, or prolonged exercise that results in excess loss of sodium through perspiration (Apostu, 2014; McDermott, Anderson, Armstrong, et al., 2017).

Pathophysiology

Hyponatremia primarily occurs due to an imbalance of water rather than sodium. Checking the urine sodium value can assist in differentiating renal from nonrenal causes of hyponatremia. Low sodium in the urine occurs as the nephrons of the kidney retain sodium to compensate for nonrenal fluid loss (i.e., vomiting, diarrhea, sweating). High sodium concentration in the urine is associated with renal salt wasting that occurs in renal dysfunction or diuretic use. In dilutional hyponatremia, the ECF volume has excess water but there is no edema, and the excess water dilutes the sodium (Sterns, 2017e).

A deficiency of aldosterone, as occurs in adrenal insufficiency, also predisposes to sodium deficiency. Lack of aldosterone causes lack of sodium and water reabsorption into the bloodstream at the nephrons. In addition, the use of certain medications, such as anticonvulsants (e.g., carbamazepine, oxcarbazepine, levetiracetam), SSRIs (e.g., fluoxetine, sertraline, paroxetine), or desmopressin acetate, have side effects that increase the risk of hyponatremia (Liamis, Megapanou, Elisaf, et al., 2019).

Clinical Manifestations

Clinical manifestations of hyponatremia depend on the cause, magnitude, and speed with which the deficit occurs. Poor skin turgor, dry mucosa, headache, decreased saliva production, orthostatic fall in blood pressure, nausea, vomiting, and abdominal cramping can occur. Neurologic changes, including altered mental status, status epilepticus, and coma, are related to the cellular swelling and cerebral edema associated with hyponatremia. As the extracellular sodium level decreases, the cellular fluid becomes relatively more concentrated and pulls water into the cells (Fig. 10-7). In general, patients with an acute decrease in serum sodium levels have more cerebral edema and higher mortality rates than do those with more slowly developing hyponatremia. Acute decreases in sodium, developing in less than 48 hours, may be associated with cerebral edema. Cerebral edema can lead to compression of brain stem structures and brain herniation. Chronic decreases in sodium, developing over 48 hours or more, can occur in status epilepticus and other neurologic conditions (Sterns, 2017e).

Clinical features of hyponatremia associated with sodium loss and water gain include anorexia, muscle cramps, and a feeling of exhaustion. The severity of symptoms increases with the degree of hyponatremia and the speed with which it develops. When the serum sodium level decreases to less than 115 mEq/L (115 mmol/L), signs of increasing intracranial pressure, such as lethargy, confusion, muscle twitching, focal weakness, hemiparesis, papilledema, seizures, and death, may occur (Sterns, 2017e).

Assessment and Diagnostic Findings

Targeted assessment includes the history and physical examination with a focused neurologic examination; evaluation of signs and symptoms as well as laboratory test results; identification of current IV fluids, if applicable; and a review of all medications the patient is taking. Regardless of the cause of hyponatremia, the serum sodium level is less than 135 mEq/L; in SIADH, it may be lower than 100 mEq/L (100 mmol/L). Serum osmolality is usually decreased. When hyponatremia is due to lack of sodium ingestion, the urinary sodium content is less than 20 mEq/L (20 mmol/L) and the specific gravity is low (1.002 to 1.004). However, when hyponatremia is due to SIADH, the urinary sodium content is greater than 20 mEq/L, and the urine specific gravity is usually greater than 1.012. Although the patient with SIADH retains water abnormally there is no peripheral edema; instead, fluid accumulates inside the cells. This phenomenon sometimes manifests as pitting edema (Sterns, 2017d).

Medical Management

The key to treating hyponatremia is an assessment that focuses on the clinical symptoms of the patient and signs of hyponatremia (including laboratory values). As a general rule, treating the underlying condition will bring the sodium level back to normal.

Sodium Replacement

The most common treatment for hyponatremia is careful administration of sodium by mouth, nasogastric tube, or a parenteral route. For patients who can eat and drink, sodium is easily replaced, because sodium is consumed abundantly in a normal diet. For those who cannot consume sodium, lactated Ringer's solution or isotonic saline (0.9% sodium chloride) solution may be prescribed. Serum sodium must not be increased by more than 12 mEq/L in 24 hours to avoid neurologic damage due to demyelination (Jain, Phadke, Chauhan, et al., 2018). This condition may occur when the serum sodium concentration is overcorrected (exceeding 140 mEq/L) too rapidly or in the presence of hypoxia or anoxia. It may produce lesions that show symmetric myelin destruction affecting all fiber tracts that can present with altered cognition and decreased alertness, ataxia, paraparesis, dysarthria, horizontal gaze paralysis, pseudobulbar palsy, and coma. The usual daily sodium requirement in adults is approximately 100 mEq, provided there are not excessive losses (Sterns, 2020). Select water and electrolyte solutions are described in [Table 10-5](#).

In SIADH, the administration of hypertonic saline solution alone cannot change the plasma sodium concentration. Excess sodium would be excreted rapidly in highly concentrated urine. With the addition of the diuretic furosemide, urine is not concentrated and isotonic urine is excreted to effect a change in water balance. In patients with SIADH, in whom water restriction is

difficult, lithium can antagonize the osmotic effect of ADH on the nephrons' collecting ducts (Sterns, 2017d).

Water Restriction

In patients with normal or excess fluid volume, hyponatremia is usually treated effectively by restricting fluid. However, if neurologic symptoms are severe (e.g., seizures, delirium, coma), or in patients with traumatic brain injury, it may be necessary to administer small volumes of a hypertonic sodium solution with the goal of alleviating cerebral edema. Incorrect use of these fluids is extremely dangerous, because 1 L of 3% sodium chloride solution contains 513 mEq of sodium and 1 L of 5% sodium chloride solution contains 855 mEq of sodium. The recommendation for hypertonic saline administration in patients with craniocerebral trauma is 3% saline between 0.10 and 1.0 mL/kg of body weight per hour (Sterns, 2017d).



Quality and Safety Nursing Alert

In patients with hyponatremia, highly hypertonic sodium solutions (2–23% sodium chloride) should be administered slowly. The patient needs close monitoring, because only small volumes are needed to elevate the serum sodium concentration.

Pharmacologic Therapy

AVP receptor antagonists (also called ADH receptor antagonists) are pharmacologic agents that treat hyponatremia by blocking the effect of ADH at the nephron, which in turn allows diuresis to occur and leads to water excretion. Use of IV conivaptan HCl, an AVP receptor antagonist, is limited to the treatment of hospitalized patients. It may be a useful therapy for those patients with moderate to severe symptomatic hyponatremia but is contraindicated in patients with seizures, delirium, or coma, which warrants the use of hypertonic saline. Tolvaptan is an oral medication indicated for clinically significant hypervolemic and euvolemic hyponatremia that must be initiated and monitored in the hospital setting (Frandsen & Pennington, 2018; Vallerand & Sanoski, 2019).

Nursing Management

The nurse needs to identify and monitor patients at risk for hyponatremia. The nurse monitors I&O as well as daily body weight. I&O can be used to identify excess water input or lack of sufficient water output.

The nurse needs to get a thorough history to identify if the patient is a performance athlete. Performance athletes (i.e., marathon runners) may use

salt tablets to compensate for loss of sodium with sweating, hoping to decrease sodium loss during prolonged exercise; however, there is no evidence that this practice works and it is not recommended (Hew-Butler, Loi, Pani, et al., 2017).

Hyponatremia is a frequently overlooked cause of confusion in older patients, who are at increased risk because of decreased renal function and subsequent inability to excrete excess fluids. Administration of prescribed and OTC medications that cause sodium loss or water retention is often the predisposing factor. A diminished sense of thirst or decreased ability to access food or fluids may also contribute to the problem (Cash & Glass, 2018).

Detecting and Controlling Hyponatremia

Early detection and treatment of hyponatremia are necessary to prevent serious consequences. For patients at risk, the nurse closely monitors fluid I&O as well as daily body weight. It is also necessary to monitor laboratory values (i.e., sodium) and be alert for GI manifestations such as anorexia, nausea, vomiting, and abdominal cramping. The nurse must be alert for central nervous system changes, such as lethargy, confusion, muscle twitching, and seizures. Neurologic signs are associated with very low sodium levels that have fallen rapidly because of fluid overloading. Serum sodium is monitored very closely in patients who are at risk for hyponatremia; when indicated, urine sodium and specific gravity are also monitored.

For a patient with abnormal losses of sodium who can consume a general diet, the nurse encourages foods and fluids with high sodium content to control hyponatremia. For example, broth made with one beef cube contains approximately 900 mg of sodium; 8 oz of tomato juice contains approximately 700 mg of sodium. The nurse also needs to be familiar with the sodium content of parenteral fluids (see [Table 10-5](#)).

If the primary cause of hyponatremia is water retention, it is safer to restrict fluid intake than to administer sodium. In normovolemia or hypervolemia, administration of sodium predisposes a patient to fluid volume overload. In severe hyponatremia, the aim of therapy is to elevate the serum sodium level only enough to alleviate neurologic signs and symptoms. It is generally recommended that the serum sodium concentration be increased to no greater than 125 mEq/L (125 mmol/L) with a hypertonic saline solution (Sterns, 2020).



Quality and Safety Nursing Alert

When administering fluids to patients with cardiovascular disease, the nurse assesses for hemodynamic signs of circulatory overload (e.g., cough, dyspnea, jugular venous distention, dependent edema, 1–2 lb weight gain in 24 h). The lungs should be auscultated for crackles as this can indicate pulmonary edema.

For the patient taking lithium, the nurse observes for lithium toxicity, particularly when sodium is lost. In such instances, supplemental salt and fluid are given. Because diuretics promote sodium loss, the patient taking lithium is instructed not to use diuretics without close medical supervision. For all patients on lithium therapy, normal salt and oral fluid intake (approximately 6- to 15-g sodium and 2.5- to 3.0-L fluid/day) should be encouraged and a sodium restricted diet should be avoided (Frandsen & Pennington, 2018; Vallerand & Sanoski, 2019).

Excess water supplements are avoided in patients receiving isotonic or hypotonic enteral feedings, particularly if abnormal sodium loss occurs or water is being abnormally retained (as in SIADH). Actual fluid needs are determined by evaluating fluid I&O, urine specific gravity, and serum sodium levels.

Sodium Excess (Hypernatremia)

Hypernatremia is a serum sodium level higher than 145 mEq/L (145 mmol/L). It can be caused by a gain of sodium in excess of water or by a loss of water in excess of sodium. It can occur in patients with normal fluid volume or in those with FVD or FVE. With water loss, the patient loses more water than sodium; as a result, the serum sodium concentration increases and the increased concentration pulls fluid out of the cell. This is both an extracellular and an intracellular FVD. In sodium excess, the patient ingests or retains more sodium than water (Mushin & Mount, 2018; Sterns, 2017e).

Chart 10-1

Dysnatremia

Dysnatremia is lack of normal sodium level in the bloodstream; either hypernatremia or hyponatremia. In performance athletes who lose excessive water via perspiration, sodium can become concentrated in the bloodstream causing hypernatremia. This can lead to the following life-threatening conditions:

- Encephalopathy
- Confusion
- Disorientation
- Stupor

In performance athletes who lose excess sodium via perspiration during exercise, this can lead to excess sodium depletion from the bloodstream causing hyponatremia. This can also manifest as the following:

- Confusion
- Disorientation
- Stupor

Testing of the blood and urine can differentiate hyponatremia from hypernatremia, determine the severity of the dysnatremia, and guide appropriate therapy.

Adapted from Apostu, M. (2014). A strategy for maintaining fluid and electrolyte balance in aerobic effort. *Procedia—Social and Behavioral Sciences*, 117(2014), 323—328; Hew-Butler, T., Loi, V., Pani, A., et al. (2017). Exercise-induced hyponatremia: 2017 update. *Frontiers in Medicine (Lausanne)*, 4, 21.

Pathophysiology

A common cause of hypernatremia is fluid deprivation in patients who do not respond to thirst. Most often affected are patients who are very old, very young, or cognitively impaired. Administration of hypertonic enteral feedings without adequate water supplements leads to hypernatremia, as does watery diarrhea and greatly increased insensible water loss through the lungs or skin (e.g., hyperventilation, burns). In addition, diabetes insipidus, which is a lack of ADH due to posterior pituitary dysfunction, can lead to lack of adequate reabsorption of water into the bloodstream at the level of the nephron. This leads to inadequate water volume in the bloodstream which leads to hypernatremia if the patient does not respond to thirst, or if fluids are excessively restricted (Sterns, 2017c).

Less common causes of hypernatremia are heatstroke, nonfatal drowning in seawater (which contains a sodium concentration of approximately 500 mEq/L), and malfunction of hemodialysis or peritoneal dialysis systems. IV

administration of hypertonic saline or excessive use of sodium bicarbonate also causes hypernatremia. Exertional dysnatremia can occur in performance athletes (Apostu, 2014) ([Chart 10-1](#)).

Clinical Manifestations

The clinical manifestations of hypernatremia are due to increased plasma osmolality caused by an increase in plasma sodium concentration. Water moves out of the cell into the ECF, resulting in cellular dehydration (Norris, 2019) (see [Fig. 10-7](#)). Clinical signs and symptoms as well as laboratory findings can be found in [Table 10-6](#). Dehydration (resulting in hypernatremia) is often overlooked as the cause of mental status and behavioral changes in older patients (Cash & Glass, 2018). Body temperature may increase mildly, but it returns to normal after the hypernatremia is corrected.

A primary characteristic of hypernatremia is thirst. Thirst is a strong defender of normal serum sodium levels in healthy people. Because of thirst, hypernatremia does not occur unless the person is unconscious or cannot access water. However, those who are ill and older adults may have an impaired thirst mechanism (Cash & Glass, 2018).

Assessment and Diagnostic Findings

In hypernatremia, the serum sodium level exceeds 145 mEq/L (145 mmol/L) and the serum osmolality exceeds 300 mOsm/kg (300 mmol/L). The urine specific gravity and urine osmolality are increased as the kidneys attempt to conserve water (provided the water loss is from a route other than the kidneys). Patients with diabetes insipidus do not reabsorb water into the bloodstream at the nephron. These patients consequently develop excess urine output, dehydration, and hypernatremia. Without ADH, these patients excrete very dilute urine with a urine osmolality less than 250 mOsm/kg (Emmett & Palmer, 2018a).

Medical Management

Treatment of hypernatremia consists of a gradual lowering of the serum sodium level by the infusion of a hypotonic solution (e.g., 0.45% sodium chloride) or an isotonic nonsaline solution (e.g., dextrose 5% in water [D₅W]). D₅W can be used when water needs to be replaced without sodium. However, hypotonic sodium chloride solution (0.45% NaCl) is thought to be safer than D₅W because it allows a gradual reduction in the serum sodium level. Gradual reduction in serum sodium decreases the risk of cerebral edema. Hypotonic sodium chloride solution (0.45% NaCl) is the IV solution of choice in severe hyperglycemia with hypernatremia. A rapid reduction in the serum sodium

level that occurs with D₅W temporarily decreases the plasma osmolality below that of the fluid in the brain tissue, causing dangerous cerebral edema. Alternatively in hypernatremia, diuretics can be prescribed to treat the excess sodium (Sterns & Hoorn, 2019).

There is no consensus about the exact rate at which serum sodium levels should be reduced. As a general rule, the serum sodium level is reduced at a rate no faster than 0.5 to 1 mEq/L/h to allow sufficient time for readjustment through diffusion across fluid compartments. Desmopressin acetate, a synthetic ADH, may be prescribed to treat diabetes insipidus if it is the cause of hypernatremia (Bichet, 2017; Mushin & Mount, 2018).

Nursing Management

Fluid losses and gains are carefully monitored in patients who are at risk for hypernatremia. The nurse should assess for abnormal losses of water or low water intake and for large gains of sodium, as might occur with ingestion of OTC medications that have a high sodium content (e.g., Alka-Seltzer). In addition, the nurse obtains a medication history, because some prescription medications have a high sodium content. The nurse also notes the patient's thirst or elevated body temperature and evaluates it in relation to other clinical signs and symptoms. The patient is monitored closely for changes in behavior, such as restlessness, disorientation, and lethargy (Sterns, 2017c; Sterns & Hoorn, 2019).

Preventing Hypernatremia

The nurse attempts to prevent hypernatremia by providing oral fluids at regular intervals, particularly in patients who are unable to perceive or respond to thirst. If fluid intake remains inadequate or the patient is unconscious, the nurse consults with the primary provider to plan an alternative route for intake, either by enteral feedings or by the parenteral route. If enteral feedings are used, sufficient water should be given to keep the serum sodium and BUN within normal limits. As a rule, the higher the osmolality of the enteral feeding, the greater is the need for water supplementation (Emmett & Palmer, 2018a; Sterns & Hoorn, 2019). Some herbal medications can also increase serum sodium levels.

For patients with diabetes insipidus, adequate water intake must be ensured. If the patient is alert and has an intact thirst mechanism, merely providing access to water may be sufficient. If the patient has a decreased level of consciousness or other disability interfering with adequate fluid intake, parenteral fluid replacement may be prescribed. This therapy can be anticipated in patients with neurologic disorders, particularly in the early postoperative period (Bichet, 2017).

Correcting Hypernatremia

When parenteral fluids are necessary for managing hypernatremia, the nurse monitors the patient's response to the infusion of fluids by reviewing serial serum sodium levels and by observing for changes in neurologic status, such as confusion, disorientation, and possible decreased level of consciousness (Mushin & Mount, 2018). With a gradual decrease in the serum sodium level, neurologic status should improve. However, too rapid reduction in the serum sodium level renders the plasma temporarily hypoosmotic compared to the intracellular fluid within the brain cells. This can cause fluid in the plasma to shift into the brain cells which can produce the dangerous state of cerebral edema (Hutto & French, 2017).

Potassium Imbalances

Potassium (K^+) is the major intracellular electrolyte; in fact, 98% of the body's potassium is inside the cells. The remaining 2% is in the ECF and is important to neuromuscular and cardiac function. Potassium influences both skeletal and cardiac muscle activity. For example, alterations in K^+ concentration can change myocardial irritability and rhythm. Under the influence of the sodium-potassium pump, potassium is constantly being pumped into the cells. The normal serum potassium concentration ranges from 3.5 to 5 mEq/L (3.5 to 5 mmol/L), and even minor variations are significant (Norris, 2019). Potassium imbalances are commonly associated with various diseases, injuries, medications (e.g., NSAIDs and ACE inhibitors), and acid-base imbalances (Mount, 2017a). The two types of potassium imbalances are potassium deficit and potassium excess ([Table 10-7](#)).

TABLE 10-7 Potassium Imbalances

Imbalance	Contributing Factors	Signs/Symptoms
Potassium deficit (hypokalemia) Serum potassium $<3.5 \text{ mEq/L}$	Diarrhea, vomiting, gastric suction, corticosteroid administration, hyperaldosteronism, carbenicillin, amphotericin B, bulimia, osmotic diuresis, alkalosis, starvation, diuretics, and digoxin toxicity	Fatigue, anorexia, nausea and vomiting, muscle weakness, polyuria, decreased bowel motility, ventricular asystole or fibrillation, paresthesias, leg cramps, ↓ BP, ileus, abdominal distention, hypoactive reflexes. <i>ECG</i> : flattened T waves, prominent U waves, ST depression, prolonged PR interval
Potassium excess (hyperkalemia) Serum potassium $>5.0 \text{ mEq/L}$	Pseudohyperkalemia, oliguric kidney injury, use of potassium-conserving diuretics in patients with renal insufficiency, metabolic acidosis, Addison disease, crush injury, burns, stored bank blood transfusions, rapid IV administration of potassium, and certain medications such as ACE inhibitors, NSAIDs, cyclosporine	Muscle weakness, tachycardia → bradycardia, arrhythmias, flaccid paralysis, paresthesias, intestinal colic, cramps, abdominal distention, irritability, anxiety. <i>ECG</i> : tall tented T waves, prolonged PR interval and QRS duration, absent P waves, ST depression

ACE, angiotensin-converting enzyme; BP, blood pressure; ↓, decreased; ECG, electrocardiogram; →, followed by; IV, intravenous; NSAIDs, nonsteroidal anti-inflammatory drugs.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

To maintain potassium balance, the renal system must function, because 80% of the potassium excreted daily leaves the body by way of the kidneys; the other 20% is lost through the bowel and in sweat. The kidneys regulate potassium balance by adjusting the amount of potassium that is excreted in the urine. As serum potassium levels increase, so does the potassium level in the renal tubular cell. A concentration gradient occurs, favoring the movement of potassium into the renal tubule and excretion of potassium in the urine. Aldosterone also increases the excretion of potassium by the kidney. Because the kidneys do not conserve potassium as well as they conserve sodium, potassium may still be lost in urine in the presence of a potassium deficit (Norris, 2019).

Potassium Deficit (Hypokalemia)

Hypokalemia (serum potassium level below 3.5 mEq/L [3.5 mmol/L]) usually indicates a deficit in total potassium stores. However, it may occur in patients

with normal potassium stores: When **alkalosis** (high blood pH) is present, a temporary shift of serum potassium into the cells occurs (see later discussion).

Pathophysiology

Potassium-losing diuretics, such as the thiazides and loop diuretics, can induce hypokalemia. Other medications that can lead to hypokalemia include corticosteroids, sodium penicillin, and amphotericin B (Vallerand & Sanoski, 2019). GI loss of potassium is another common cause of potassium depletion. Vomiting and gastric suction frequently lead to hypokalemia, because potassium is lost when gastric fluid is lost and because potassium is lost through the kidneys in response to metabolic alkalosis. Because relatively large amounts of potassium are contained in intestinal fluids, potassium deficit occurs frequently with diarrhea, which may contain as much potassium as 30 mEq/L. Potassium deficit also occurs from prolonged intestinal suctioning, recent ileostomy, and villous adenoma (a tumor of the intestinal tract characterized by excretion of potassium-rich mucus) (Mount, 2017b).

Alterations in acid–base balance have a significant effect on potassium distribution due to shifts of hydrogen and potassium ions between the cells and the ECF. Respiratory or metabolic alkalosis promotes the transcellular shift of potassium and can have a variable and unpredictable effect on serum potassium. For example, hydrogen ions move out of the cells into the bloodstream in alkalotic states to help correct the high pH, and potassium ions move into the cells to maintain an electrically neutral state (Mount, 2017c) (see later discussion of acid–base balance).

Aldosterone from the adrenal gland acts on the nephron to increase sodium and water reabsorption into the bloodstream. It simultaneously secretes potassium into the renal tubules which in turn is excreted in the urine. In hyperaldosteronism, potassium is constantly secreted into the nephron tubule fluid which leads to loss of potassium into the urine. Hyperaldosteronism causes renal potassium wasting and can lead to severe potassium depletion. Primary hyperaldosteronism is seen in patients with adrenal adenomas (tumors). Secondary hyperaldosteronism occurs in patients with cirrhosis, nephrotic syndrome, heart failure, or malignant hypertension (Dick, Queiroz, Bernardi, et al., 2018).

Insulin promotes the entry of potassium into cells from the bloodstream; therefore, patients with persistent insulin hypersecretion may experience hypokalemia. Patients receiving high carbohydrate parenteral nutrition will have increased secretion of insulin. This will cause the shift of potassium into the cells from the bloodstream, causing hypokalemia. In diabetic ketoacidosis (DKA), potassium moves out of the cell since H⁺ ions are high; during this acute phase it seems as though the patient has hyperkalemia. With insulin

treatment of DKA, potassium moves back into the cells, causing hypokalemia (Palmer & Clegg, 2016a).

Patients who are not able to eat a normal diet for a prolonged period are at risk for hypokalemia. This may occur in debilitated older adults and in patients with alcoholism or anorexia nervosa. In addition to poor intake, people with bulimia frequently experience increased potassium loss through self-induced vomiting and overuse of laxatives, diuretics, and enemas. These patients may also be deficient in magnesium. Magnesium depletion also causes renal potassium loss and must be corrected first; otherwise, urine loss of potassium will continue (Mount, 2017d).

Clinical Manifestations

Potassium deficiency can result in widespread derangements in physiologic function. Severe hypokalemia can cause death through cardiac or respiratory arrest. Clinical signs develop when the potassium level decreases to less than 3 mEq/L (3 mmol/L) (Mount, 2017b). Clinical signs and symptoms can be found in [Table 10-7](#). If prolonged, hypokalemia can lead to an inability of the kidneys to concentrate urine, causing dilute urine (resulting in polyuria, nocturia) and excessive thirst. Potassium depletion suppresses the release of insulin and results in glucose intolerance (Palmer & Clegg, 2016a).

Assessment and Diagnostic Findings

In hypokalemia, the serum potassium concentration is less than the lower limit of normal, which is 3.5 mEq/L. Electrocardiographic (ECG) changes can include flat T waves or inverted T waves or both, suggesting ischemia, and depressed ST segments ([Fig. 10-8](#)). An elevated U wave is specific to hypokalemia.

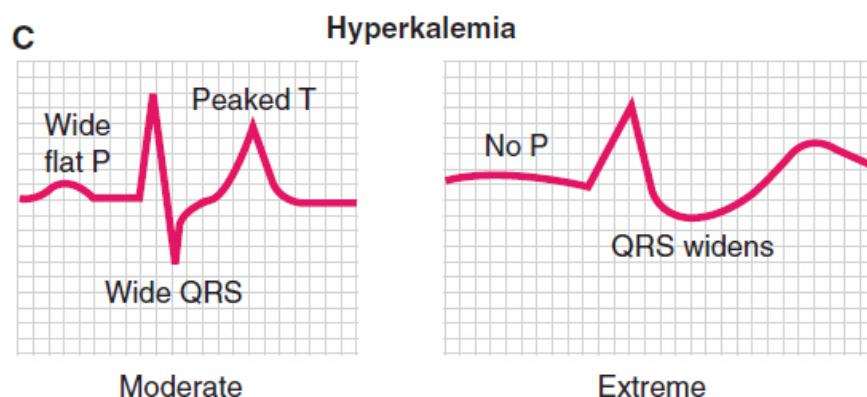
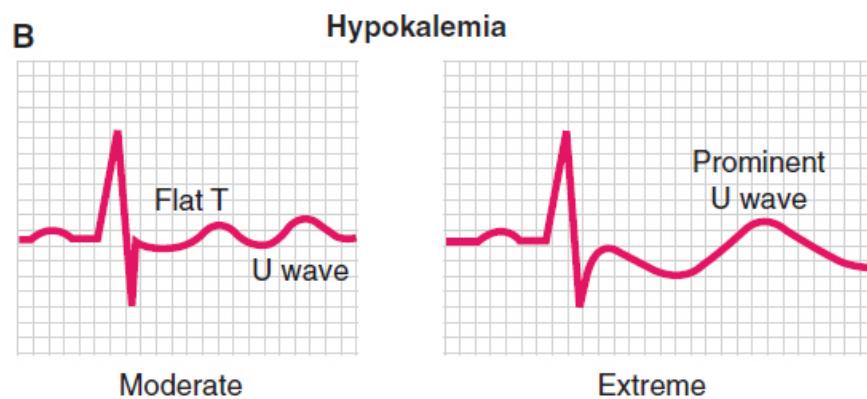
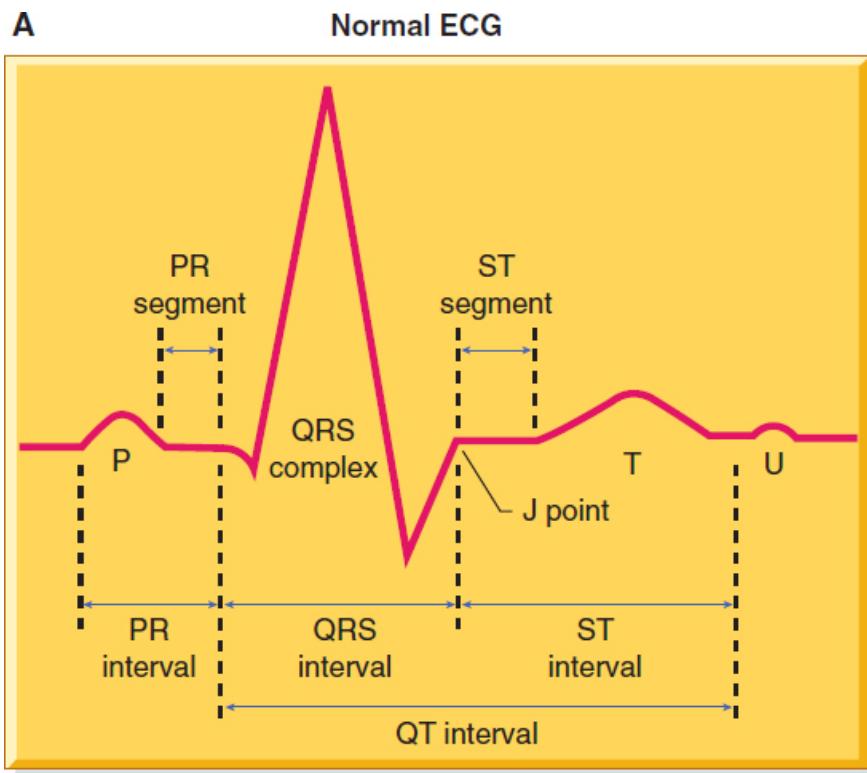


Figure 10-8 • Effect of potassium on the electrocardiogram (ECG).
A. Normal tracing. **B.** Hypokalemia: serum potassium level below

normal. **Left:** Flattening of the T wave and the appearance of a U wave. **Right:** Further flattening with prominent U wave. **C.** Hyperkalemia: serum potassium level above normal. **Left:** Moderate elevation with wide, flat P wave; wide QRS complex; and peaked T wave. **Right:** ECG changes seen with extreme potassium elevation: widening of QRS complex and absence of P wave.



Quality and Safety Nursing Alert

Hypokalemia increases sensitivity to digitalis, predisposing the patient to digitalis toxicity at lower digitalis levels.

Metabolic alkalosis is commonly associated with hypokalemia. This is discussed further in the section on acid–base disturbances in this chapter.

The source of the potassium loss is usually evident from a careful history. However, if the cause of the loss is unclear, a 24-hour urinary potassium excretion test can be performed to distinguish between renal and extrarenal loss. Urinary potassium excretion exceeding 20 mEq/day with hypokalemia suggests that renal potassium loss is the cause.

Medical Management

If hypokalemia cannot be prevented by conventional measures such as increased intake in the daily diet or by oral potassium supplements for deficiencies, then it is treated cautiously with IV replacement therapy (Mount, 2017b). Potassium loss must be corrected daily; administration of 40 to 60 mEq/day of potassium is adequate in the adult if there are no abnormal losses of potassium.

For patients who are at risk for hypokalemia, a diet containing sufficient potassium should be provided. Dietary intake of potassium in the average adult is 50 to 100 mEq/day. Foods high in potassium include most fruits and vegetables, legumes, whole grains, milk, and meat (Palmer & Clegg, 2016b).

When dietary intake is inadequate for any reason, oral or IV potassium supplements may be prescribed. Many salt substitutes contain 50 to 60 mEq of potassium per teaspoon and may be sufficient to prevent hypokalemia. If oral administration of potassium is not feasible, the IV route is indicated. The IV route is mandatory for patients with severe hypokalemia (e.g., serum level of 2 mEq/L). Although potassium chloride (KCl) is usually used to correct potassium deficits, potassium acetate or potassium phosphate may be prescribed (Mount, 2017e; Vallerand & Sanoski, 2019).

Nursing Management

Because hypokalemia can be life-threatening, the nurse needs to monitor for its early presence in patients at risk. Fatigue, anorexia, muscle weakness, decreased bowel motility, paresthesias, and arrhythmias are signals that warrant assessing the serum potassium concentration. When available, the ECG may provide useful information (Mount, 2017b). For example, patients receiving digitalis who are at risk for potassium deficiency should be monitored closely for signs of digitalis toxicity, because hypokalemia potentiates the action of digitalis.

Preventing Hypokalemia

The nurse helps prevent hypokalemia by encouraging patients at risk to eat foods rich in potassium (when the diet allows). Consumption of foods high in potassium should be encouraged; examples include bananas, melon, citrus fruits, fresh and frozen vegetables (avoid canned vegetables), lean meats, milk, and whole grains. If the hypokalemia is caused by abuse of laxatives or diuretics, patient education may help alleviate the problem. Part of the health history and assessment should be directed at identifying problems that are amenable to prevention through education. Careful monitoring of fluid I&O is necessary, because 40 mEq of potassium is lost for every liter of urine output. The ECG is monitored for changes, and arterial blood gas (ABG) values are checked for elevated bicarbonate and pH levels.

Correcting Hypokalemia

The oral route is ideal to treat mild to moderate hypokalemia because oral potassium supplements are well absorbed. Care should be exercised when administering potassium, particularly in older adults, who have lower lean body mass and total-body potassium levels and therefore lower potassium requirements. In addition, because of the physiologic loss of renal function with advancing years, potassium may be retained more readily in older than in younger people (Cash & Glass, 2018).



Quality and Safety Nursing Alert

Oral potassium supplements can produce small bowel lesions; therefore, the patient must be assessed for and cautioned about abdominal distention, pain, or GI bleeding.

Administering Intravenous Potassium

Potassium should be given only after adequate urine output has been established. A decrease in urine volume to less than 20 mL/h for 2 consecutive hours is an indication to stop the potassium infusion and notify the primary

provider. Potassium is primarily excreted by the kidneys; when oliguria occurs, potassium administration can cause the serum potassium concentration to rise to dangerous levels (Mount, 2017e).



Quality and Safety Nursing Alert

Potassium is never given by IV push or intramuscularly to avoid replacing potassium too quickly. Potassium is extremely irritating to tissues. IV potassium must be given using an infusion pump.

Each health care facility has its own policy for the administration of potassium, which must be consulted. Administration of IV potassium is done with extreme caution using an infusion pump with the patient monitored by continuous ECG. Caution must be used when selecting a premixed solution of IV fluid containing KCl, as the concentrations range from 10 to 40 mEq/100 mL. Renal function should be monitored through BUN and serum creatinine levels and urine output if the patient is receiving potassium replacement. During replacement therapy, the patient should be monitored for signs of worsening hypokalemia as well as hyperkalemia.

Potassium Excess (Hyperkalemia)

Hyperkalemia (serum potassium level greater than 5 mEq/L [5 mmol/L]) seldom occurs in patients with normal renal function. In older adults, there is an increased risk of hyperkalemia due to decreases in renin and aldosterone as well as an increased number of comorbid cardiac conditions. Like hypokalemia, hyperkalemia is often caused by iatrogenic (treatment-induced) causes. Although hyperkalemia is less common than hypokalemia, it is usually more dangerous because cardiac arrest is more frequently associated with high serum potassium levels (Mount, 2017a).

Pathophysiology

Major causes of hyperkalemia are decreased renal excretion of potassium, rapid administration of potassium, and movement of potassium from the ICF compartment to the ECF compartment. Hyperkalemia is commonly seen in patients with untreated kidney injury, particularly those in whom potassium levels increase as a result of infection or excessive intake of potassium in food or medications (Mount, 2017a). Patients with hypoaldosteronism or Addison disease are at risk for hyperkalemia because of a lack of aldosterone. Lack of aldosterone activity at the nephron causes inadequate sodium and water reabsorption into the bloodstream and inadequate excretion of potassium in the

urine. Therefore, deficient adrenal hormones lead to sodium loss and potassium retention (Norris, 2019).

Medications have been identified as a probable contributing factor in more than 60% of hyperkalemic episodes. Medications commonly implicated are KCl, heparin, ACE inhibitors, NSAIDs, beta-blockers, cyclosporine, tacrolimus, and potassium-sparing diuretics (Comerford & Durkin, 2020). Potassium regulation is compromised in acute and chronic kidney disease, with a glomerular filtration rate less than 10% to 20% of normal (Mount, 2017c).

Improper use of potassium supplements predisposes all patients to hyperkalemia, especially if salt substitutes are used. Not all patients receiving potassium-losing diuretics require potassium supplements, and patients receiving potassium-conserving diuretics should not receive supplements.



Quality and Safety Nursing Alert

Potassium supplements are extremely dangerous for patients who have impaired renal function and thus decreased ability to excrete potassium. Even more dangerous is the IV administration of potassium to such patients, because serum levels can rise very quickly. It is possible to exceed the renal tolerance of any patient with rapid IV potassium administration, as well as when large amounts of oral potassium supplements are ingested.

In **acidosis** (low blood pH), potassium moves out of the cells and into the ECF. This occurs as hydrogen ions enter the cells to buffer the pH of the ECF (see later discussion).

An elevated ECF potassium level should be anticipated when extensive tissue trauma has occurred, as in burns, crushing injuries, or severe infections. Similarly, it can occur with lysis of malignant cells after chemotherapy (i.e., tumor lysis syndrome). Any disorder that causes high amounts of cellular lysis or deterioration can cause hyperkalemia (Mount, 2017a).

Pseudohyperkalemia (a false hyperkalemia) has several causes, including the improper collection or transport of a blood sample, a traumatic venipuncture, and use of a tight tourniquet around an exercising extremity while drawing a blood sample, producing hemolysis of the sample before analysis. Other causes include marked leukocytosis (white blood cell count exceeding 200,000/mm³) and thrombocytosis (platelet count exceeding 1 million/mm³); drawing blood above a site where potassium is infusing; and familial pseudohyperkalemia, in which potassium leaks out of the RBCs while the blood is awaiting analysis. Lack of awareness of these causes of pseudohyperkalemia can lead to aggressive treatment of a nonexistent

hyperkalemia, resulting in serious lowering of serum potassium levels. Therefore, measurements of grossly elevated levels of potassium in the absence of clinical manifestations (e.g., normal ECG) should be verified by retesting (Mount, 2017a).

Clinical Manifestations

Clinical signs and symptoms can be found in [Table 10-7](#). The most important consequence of hyperkalemia is its effect on the myocardium. Cardiac effects of elevated serum potassium are usually not significant when the level is less than 7 mEq/L (7 mmol/L); however, they are almost always present when the level is 8 mEq/L (8 mmol/L) or greater. As the plasma potassium level rises, disturbances in cardiac conduction occur. The earliest changes, often occurring at a serum potassium level greater than 6 mEq/L (6 mmol/L), are peaked, narrow T waves; ST-segment depression; and a shortened QT interval. If the serum potassium level continues to increase, the PR interval becomes prolonged and is followed by disappearance of the P waves. Finally, there is decomposition and widening of the QRS complex (see [Fig. 10-7](#)). Ventricular arrhythmias and cardiac arrest may occur (Mount, 2017a).

Assessment and Diagnostic Findings

Serum potassium levels and ECG changes are crucial to the diagnosis of hyperkalemia, as discussed previously. ABG analysis may reveal either a metabolic or a respiratory acidosis. These are discussed further in the section on acid–base disturbances in this chapter. Correcting the acidosis helps correct the hyperkalemia.

Medical Management

In disorders involving potassium level changes, an ECG should be obtained immediately. Shortened repolarization and peaked T waves are seen initially in hyperkalemia. To verify results, a repeat serum potassium level should be obtained from a vein that is not concomitantly infusing an IV solution containing potassium (Mount, 2017e).

In nonacute situations, restriction of dietary potassium and potassium-containing medications may correct the imbalance. For example, eliminating the use of potassium-containing salt substitutes in a patient who is taking a potassium-conserving diuretic may be all that is needed to deal with mild hyperkalemia.

Administration, either orally or by retention enema, of cation exchange resins (e.g., sodium polystyrene sulfonate) may be necessary. The use of cation exchange resins requires normal bowel function. For instance, cation exchange

resins cannot be used if the patient has a paralytic ileus (i.e., absence of peristalsis in the intestine), because intestinal perforation can occur. Sodium polystyrene sulfonate binds with potassium and then is eliminated in the feces. Other cations in the GI tract can also be depleted which can cause hypomagnesemia and hypocalcemia. Sodium polystyrene sulfonate may also cause sodium retention and fluid overload and should be used with caution in patients with heart failure (Frandsen & Pennington, 2018; Mount, 2017e).

Patiromer sorbitex calcium is another oral agent that is a potassium-removing resin used to treat hyperkalemia. It exchanges calcium for potassium in the lower intestine, thereby increasing fecal excretion of potassium. Side effects include GI intolerance, hypomagnesemia, and edema (Depret, Peacock, Liu, et al., 2019).

Emergency Pharmacologic Therapy

If serum potassium levels are dangerously elevated, it may be necessary to administer IV calcium gluconate. Within minutes after administration, calcium antagonizes the action of hyperkalemia on the heart but does not reduce the serum potassium concentration. Calcium chloride and calcium gluconate are not interchangeable; calcium gluconate contains 4.5 mEq of calcium, and calcium chloride contains 13.6 mEq of calcium. Therefore, caution is required when using calcium preparations to reduce potassium levels (Ashurst, Sergent, & Sergent, 2016).

Monitoring the blood pressure is essential to detect hypotension, which may result from the rapid IV administration of calcium gluconate. The ECG should be continuously monitored during administration; the appearance of bradycardia is an indication to stop the infusion. The myocardial protective effects of calcium last about 30 minutes. Extra caution is required if the patient has received an accelerated dose of a digitalis-based cardiac glycoside to reach a desired serum digitalis level rapidly as parenteral administration of calcium sensitizes the heart to digitalis and may precipitate digitalis toxicity (Ashurst et al., 2016).

IV administration of sodium bicarbonate may be necessary in severe metabolic acidosis to alkalinize the plasma, shift potassium into the cells, and furnish sodium to antagonize the cardiac effects of potassium. Effects of this therapy begin within 30 to 60 minutes and may persist for hours; however, they are temporary. Circulatory overload and hypernatremia can occur when large amounts of hypertonic sodium bicarbonate are given. Bicarbonate therapy should be guided by the bicarbonate concentration or calculated base deficit obtained from blood gas analysis or laboratory measurement (Mount, 2017c).

IV administration of regular insulin and a hypertonic dextrose solution causes a temporary shift of potassium into the cells. Glucose and insulin therapy have an onset of action within 30 minutes and lasts for several hours.

Loop diuretics, such as furosemide, increase excretion of water by inhibiting sodium, potassium, and chloride reabsorption in the ascending loop of Henle and distal renal tubule (Ashurst et al., 2016).

Beta-2 agonists, such as albuterol, are highly effective in decreasing potassium; however, their use is not without risk as they can cause tachycardia and chest discomfort (Depret et al., 2019; Long, Warix, & Koyfman, 2018). Beta-2 agonists, administered intravenously or via nebulizer, move potassium into the cells and may be used in the absence of ischemic cardiac disease. Their use is a stopgap measure that only temporarily protects the patient from hyperkalemia.

If the hyperkalemic condition is not transient, removal of potassium from the body can also be done through peritoneal dialysis, hemodialysis, or other forms of renal replacement therapy.

Nursing Management

Patients at risk for potassium excess (e.g., those with kidney disease) need to be identified and closely monitored for signs of hyperkalemia. The nurse monitors I&O and observes for signs of muscle weakness and arrhythmias. When measuring vital signs, an apical pulse should be taken. The presence of paresthesias and GI symptoms such as nausea and intestinal cramping should be noted. Serum potassium levels, as well as BUN, serum creatinine, serum glucose, and ABG values, should be monitored for patients at risk for developing hyperkalemia.

Preventing Hyperkalemia

Measures should be taken to prevent hyperkalemia in patients at risk, when possible, by encouraging the patient to adhere to the prescribed potassium restriction. Potassium-rich foods to be avoided include many fruits and vegetables, legumes, whole-grain breads, lean meat, milk, eggs, coffee, tea, and cocoa. Conversely, foods with minimal potassium content include butter, margarine, cranberry juice or sauce, ginger ale, gumdrops or jelly beans, hard candy, root beer, sugar, and honey. Labels of cola beverages must be checked carefully because some are high in potassium and some are not (McDonough & Youn, 2017).

Correcting Hyperkalemia

It is possible to exceed the tolerance for potassium if given rapidly by the IV route. Therefore, careful monitoring is necessary when administering potassium solutions. Particular attention is paid to the solution's concentration and rate of administration. IV administration should only be via an infusion pump (Mount, 2017e).

The nurse must caution patients to use salt substitutes sparingly if they are taking other supplementary forms of potassium or potassium-conserving diuretics. In addition, potassium-conserving diuretics, potassium supplements, and salt substitutes should not be given to patients with kidney injury (Mount, 2017e).

Calcium Imbalances

More than 99% of the body's calcium (Ca^{++}) is located in the skeletal system; it is a major component of bones and teeth. About 1% of skeletal calcium is rapidly exchangeable with blood calcium, and the rest is more stable and only slowly exchanged. The small amount of calcium located outside the bone circulates in the serum, partly bound to protein and partly ionized. Calcium plays a major role in transmitting nerve impulses and helps regulate muscle contraction and relaxation, including cardiac muscle. Calcium is instrumental in activating enzymes that stimulate many essential chemical reactions in the body, and it also plays a role in blood coagulation. Because many factors affect calcium regulation, both hypocalcemia and hypercalcemia are relatively common disturbances (Norris, 2019) ([Table 10-8](#)).

The normal adult total serum calcium level is 8.8 to 10.4 mg/dL (2.2 to 2.6 mmol/L) (Fischbach & Fischbach, 2018). Calcium exists in plasma in three forms: ionized, bound, and complex. Approximately 50% of the serum calcium exists in a physiologically active ionized form that is important for neuromuscular activity and blood coagulation; this is the only physiologically and clinically significant form. The normal ionized serum calcium level is 4.5 to 5.1 mg/dL (1.1 to 1.3 mmol/L). Less than half of the plasma calcium is bound to serum proteins, primarily albumin. The remainder is combined with nonprotein anions: phosphate, citrate, and carbonate (Hogan & Goldfarb, 2018).

TABLE 10-8 Calcium Imbalances

Imbalance	Contributing Factors	Signs/Symptoms and Laboratory Findings
Calcium deficit (hypocalcemia) Serum calcium <8.8 mg/dL	Hypoparathyroidism (may follow thyroid surgery or radical neck dissection), malabsorption, pancreatitis, alkalosis, vitamin D deficiency, massive subcutaneous infection, generalized peritonitis, massive transfusion of citrated blood, chronic diarrhea, decreased parathyroid hormone, diuretic phase of acute kidney injury, ↑ PO ₄ , fistulas, burns, alcoholism	Numbness, tingling of fingers, toes, and circumoral region; positive Trousseau sign and Chvostek sign; seizures, carpopedal spasms, hyperactive deep tendon reflexes, irritability, bronchospasm, anxiety, impaired clotting time, ↓ prothrombin, diarrhea, ↓ BP. <i>ECG:</i> prolonged QT interval and lengthened ST <i>Labs indicate:</i> ↓ Mg ⁺⁺
Calcium excess (hypercalcemia) Serum calcium >10.4 mg/dL	Hyperparathyroidism, malignant neoplastic disease, prolonged immobilization, overuse of calcium supplements, vitamin D excess, oliguric phase of acute kidney injury acidosis, corticosteroid therapy, thiazide diuretic use, increased parathyroid hormone, and digoxin toxicity	Muscular weakness, constipation, anorexia, nausea and vomiting, polyuria and polydipsia, dehydration, hypoactive deep tendon reflexes, lethargy, deep bone pain, pathologic fractures, flank pain, calcium stones, hypertension. <i>ECG:</i> shortened ST segment and QT interval, bradycardia, heart blocks

BP, blood pressure; ↓, decreased; ECG, electrocardiogram; ↑, increased.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Calcium is absorbed from foods in the presence of normal gastric acidity and vitamin D. It is excreted primarily in the feces, with the remainder excreted in the urine. The serum calcium level is controlled by PTH and calcitonin. As ionized serum calcium decreases in the bloodstream, the parathyroid glands secrete PTH. This, in turn, increases calcium absorption from the GI tract, increases calcium reabsorption from the renal tubule, and releases calcium from the bone. The resultant increase in calcium ion concentration in the bloodstream then suppresses PTH secretion. When calcium increases excessively, the thyroid gland secretes calcitonin, which inhibits calcium reabsorption from bone and decreases the serum calcium concentration (Norris, 2019).

Calcium Deficit (Hypocalcemia)

Hypocalcemia (serum calcium value lower than 8.8 mg/dL [2.20 mmol/L]) occurs in a variety of clinical situations. A patient may have a total-body calcium deficit (as in osteoporosis) but a normal serum calcium level. Older adults and those with disability have an increased risk of hypocalcemia because immobility, particularly lack of weight-bearing activity, increases bone resorption (Cash & Glass, 2018; Goltzman, 2017).

Pathophysiology

The parathyroid glands are instrumental in regulating blood and body calcium levels. Several factors can cause hypocalcemia, including primary hypoparathyroidism and surgical hypoparathyroidism. Surgical hypoparathyroidism is more common as a result of unintentional trauma or devascularization of the parathyroid glands (Kazaure & Sosa, 2018). Not only is hypocalcemia associated with thyroid and parathyroid surgery, but it can also occur after radical neck dissection and is most likely in the first 24 to 48 hours after surgery. Transient hypocalcemia can occur with massive administration of citrated blood (i.e., massive hemorrhage and shock), because citrate can combine with ionized calcium and temporarily remove it from the circulation (Goltzman, 2017).

Inflammation of the pancreas causes the breakdown of proteins and lipids. It is thought that calcium ions combine with the fatty acids released by lipolysis, forming soaplike compounds. As a result of this process, hypocalcemia occurs and is common in pancreatitis. Hypocalcemia may also be related to excessive secretion of glucagon from the inflamed pancreas, which results in increased secretion of calcitonin from the thyroid gland.

Hypocalcemia is common in patients with acute kidney injury, because these patients frequently have elevated serum phosphate levels. Hyperphosphatemia usually causes a reciprocal drop in the serum calcium level. Other causes of hypocalcemia include inadequate vitamin D consumption, magnesium deficiency, medullary thyroid carcinoma, low serum albumin levels, alkalosis, and alcohol abuse. Medications predisposing to hypocalcemia include aluminum-containing antacids, aminoglycosides, caffeine, cisplatin, corticosteroids, mithramycin, phosphates, isoniazid, loop diuretics, and proton pump inhibitors (Goltzman, 2017).

Clinical Manifestations

Tetany, the most characteristic manifestation of hypocalcemia and hypomagnesemia, refers to the entire symptom complex induced by increased neural excitability. Clinical signs and symptoms are caused by spontaneous discharges of both sensory and motor fibers in peripheral nerves and are outlined in [Table 10-8](#).

Chvostek sign (Fig. 10-9A) consists of twitching of muscles innervated by the facial nerve in response to tapping of the muscle just below the zygomatic arch. Trousseau sign (Fig. 10-9B) can be elicited by inflating a blood pressure cuff on the upper arm to about 20 mm Hg above systolic pressure; within 2 to 5 minutes, carpal spasm will occur as ischemia of the ulnar nerve develops (Goltzman, 2019a).

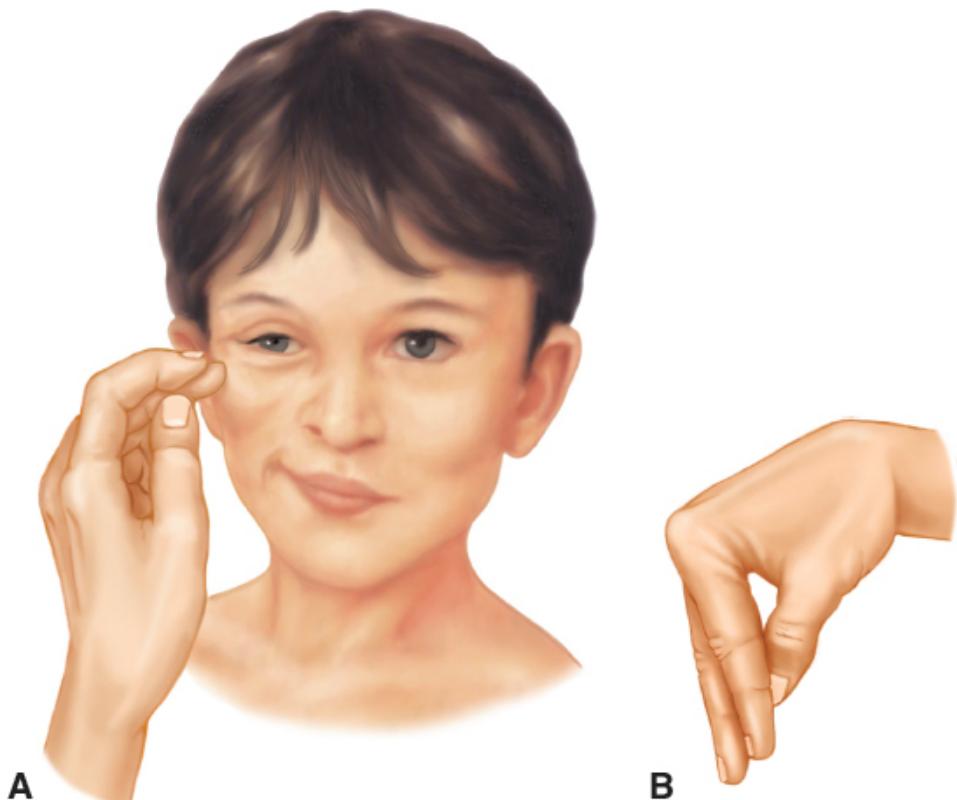


Figure 10-9 • **A.** Chvostek sign: a contraction of the facial muscles elicited in response to light tap over the facial nerve in front of the ear. **B.** Trousseau sign: a carpopedal spasm induced by inflating a blood pressure cuff above systolic blood pressure. Adapted from Bullock, B. A., & Henze, R. J. (2000). *Focus on pathophysiology* (p. 173). Philadelphia, PA: Lippincott Williams & Wilkins.

Hypocalcemia can cause seizures because low calcium levels increase irritability of the central and peripheral nervous systems. Other changes associated with hypocalcemia include mental changes such as depression, impaired memory, confusion, delirium, and hallucinations. A prolonged QT interval is seen on the ECG due to prolongation of the ST segment, and torsades de pointes, a type of ventricular tachycardia, may occur. Respiratory effects with decreasing calcium include dyspnea and laryngospasm. Signs and symptoms of chronic hypocalcemia include hyperactive bowel sounds, dry and brittle hair and nails, and abnormal clotting (Goltzman, 2019a).

Osteoporosis is associated with prolonged low intake of calcium and represents a total-body calcium deficit, even though serum calcium levels are usually normal. This disorder occurs in millions of Americans and is most common in postmenopausal women. It is characterized by loss of bone mass, which causes bones to become porous and brittle and therefore susceptible to fracture (Black & Rosen, 2016) (see [Chapter 36](#) for further discussion of osteoporosis).

Assessment and Diagnostic Findings

When evaluating serum calcium levels, the serum albumin level and the arterial pH must also be considered. Because abnormalities in serum albumin levels may affect interpretation of the serum calcium level, it may be necessary to calculate the corrected serum calcium if the serum albumin level is abnormal. For every decrease in serum albumin of 1 g/dL below 4 g/dL, the total serum calcium level is underestimated by approximately 0.8 mg/dL (Hogan & Goldfarb, 2018). [Chart 10-2](#) displays a quick method that nurses can use to calculate the corrected serum calcium level.

Clinicians often discount a low serum calcium level in the presence of a similarly low serum albumin level. The ionized calcium level is usually normal in patients with reduced total serum calcium levels and concomitant hypoalbuminemia. When the arterial pH increases (alkalosis), more calcium becomes bound to protein. As a result, the ionized portion decreases. Symptoms of hypocalcemia may occur with alkalosis. Acidosis has the opposite effect—that is, less calcium is bound to protein and therefore more exists in the ionized form. However, relatively small changes in serum calcium levels occur in these acid–base abnormalities (Yu & Stubbs, 2019).

Chart 10-2

Calculating Corrected Serum Calcium Level

Abnormalities in serum albumin levels may affect interpretation of the serum calcium level. Below is a method for calculating the corrected serum calcium level if the serum albumin level is abnormal.

Quick Calculation Method

$$\begin{aligned} & \text{Measured total serum Ca}^{++} \text{ level (mg/dL)} + 0.8 \\ & \quad \times (4.0 - \text{Measured albumin level [g/dL]}) \\ & = \text{Corrected total calcium concentration (mg/dL)} \end{aligned}$$

Example Calculation

A patient's reported serum albumin level is 2.5 g/dL; the reported serum calcium level is 10.5 mg/dL. First, the decrease in serum albumin level from normal (i.e., the difference from the normal albumin concentration of 4 g/dL) is calculated: $4 \text{ g/dL} - 2.5 \text{ g/dL} = 1.5 \text{ g/dL}$. Next, the following ratio is calculated:

$$\begin{aligned} 0.8 \text{ mg/dL} : 1 \text{ g/dL} &= X \text{ mg/dL} : 1.5 \text{ mg/dL} \\ X &= 0.8 \times 1.5 \text{ mg/dL} \\ X &= 1.2 \text{ mg/dL calcium} \end{aligned}$$

Finally, 1.2 mg/dL is added to 10.5 mg/dL (the reported serum calcium level) to obtain the corrected total serum calcium level: $1.2 \text{ mg/dL} + 10.5 \text{ mg/dL} = 11.7 \text{ mg/dL}$.

Ideally, the ionized level of calcium should be measured in the laboratory. However, in many laboratories, only the total calcium level is reported; therefore, the concentration of the ionized fraction must be estimated by simultaneous measurement of the serum albumin level. Magnesium and phosphorus levels need to be assessed to identify possible causes of decreased calcium (Yu & Stubbs, 2019).

Medical Management

Emergency Pharmacologic Therapy

Acute symptomatic hypocalcemia is life-threatening and requires prompt treatment with IV administration of a calcium salt. Parenteral calcium salts include calcium gluconate and calcium chloride (Duval, Bach, Masson, et al., 2018; Goltzman, 2019b). Although calcium chloride produces a significantly higher ionized calcium level than calcium gluconate does, it is not used as often because it is more irritating and can cause sloughing of tissue if it infiltrates.

IV administration of calcium is particularly dangerous in patients receiving digitalis-derived medications, because calcium ions exert an effect similar to that of digitalis and can cause digitalis toxicity, with adverse cardiac effects. The IV site that delivers calcium must be observed often for any evidence of infiltration because of the risk of extravasation and resultant cellulitis or necrosis. A 0.9% sodium chloride solution should not be used with calcium because it increases renal calcium loss. Solutions containing phosphates or bicarbonate should not be used with calcium because they cause precipitation when calcium is added. The nurse must clarify with the primary provider and pharmacist which calcium salt to administer, because calcium gluconate yields 4.5 mEq of calcium and calcium chloride provides 13.6 mEq of calcium. Calcium replacement can cause orthostatic hypotension; therefore, the patient should remain in bed during IV infusion, and blood pressure is monitored (Duval et al., 2018).



Quality and Safety Nursing Alert

Too rapid IV administration of calcium can cause cardiac arrest, preceded by bradycardia. Therefore, calcium should be diluted in D₅W and given as a slow IV bolus or a slow IV infusion using an infusion pump. A 0.9% sodium chloride solution should not be used when administering calcium.

Nutritional Therapy

Vitamin D therapy may be instituted to increase calcium absorption from the GI tract; otherwise, the amount of calcium absorbed may not satisfy the body's calcium requirement. In addition, aluminum hydroxide, calcium acetate, or calcium carbonate antacids may be prescribed to decrease elevated phosphorus levels before treating hypocalcemia in the patient with chronic kidney disease. Increasing the dietary intake of calcium to at least 1000 to 1500 mg/day in the adult is recommended. Calcium supplements must be given in divided doses of no higher than 500 mg to promote calcium absorption. Calcium-containing foods include milk products; green, leafy vegetables; canned salmon; canned sardines; and fresh oysters. Hypomagnesemia can also cause tetany; if the tetany responds to IV calcium, then a low magnesium level is considered as a possible cause in chronic kidney dysfunction (Goltzman, 2019b; Rosen & Drezner, 2019).

Nursing Management

It is important to assess for hypocalcemia in at-risk patients. Seizure precautions are initiated if hypocalcemia is severe. The status of the airway is

closely monitored because laryngospasm can occur. Safety precautions are taken, as indicated, if confusion is present (Duval et al., 2018).

The nurse must educate the patient with hypocalcemia about foods that are rich in calcium. The nurse must also advise the patient to consider calcium supplements if sufficient calcium is not consumed in the diet. Such supplements should be taken in divided doses with meals. Alcohol and caffeine in high doses inhibit calcium absorption, and moderate cigarette smoking increases urinary calcium excretion. The patient is also cautioned to avoid the overuse of laxatives and antacids that contain phosphorus, because their use decreases calcium absorption (Rosen & Drezner, 2019).

Calcium Excess (Hypercalcemia)

Hypercalcemia (serum calcium value greater than 10.4 mg/dL [2.6 mmol/L]) can affect many organ systems. Symptoms depend on the degree of hypercalcemia and the rate of rise of calcium in the bloodstream. Mild hypercalcemia usually causes no apparent symptoms if the rise in calcium level is chronic over a long period of time. Moderate hypercalcemia can also be well tolerated. Marked symptoms occur when rise in calcium level is acute (Shane, 2019b).

Pathophysiology

The most common causes of hypercalcemia are malignancies and hyperparathyroidism. Malignant tumors can produce hypercalcemia by various mechanisms. The most common malignancies that are associated with hypercalcemia are breast, lung, renal, and multiple myeloma. In hyperparathyroidism, excessive PTH secretion causes increased release of calcium from the bones and increased intestinal and renal absorption of calcium. Calcifications of soft tissue occur when the calcium–phosphorus product ($\text{serum calcium} \times \text{serum phosphorus}$) exceeds 70 mg/dL. Calcium levels are inversely related to phosphorus levels (Shane, 2019a).

Bone calcium is lost during immobilization, and sometimes this causes elevation of total (and especially ionized) calcium in the bloodstream. However, symptomatic hypercalcemia from immobilization is rare; when it does occur, it is limited to people with high calcium turnover rates (e.g., adolescents during a growth spurt). Most cases of hypercalcemia secondary to immobility occur after severe or multiple fractures or spinal cord injury (Shane, 2019a).

Thiazide diuretics can cause a slight elevation in serum calcium levels because they potentiate the action of PTH on the kidneys, reducing urinary calcium excretion. Vitamin A and D intoxication, as well as chronic lithium use and theophylline toxicity, can cause calcium excess.

Hypercalcemia reduces neuromuscular excitability because it suppresses activity at the myoneural junction. Decreased tone in smooth and striated muscle may cause symptoms such as muscle weakness, incoordination, anorexia, and constipation. Lethal arrhythmias (e.g., ventricular fibrillation) can occur when the serum calcium level is about 18 mg/dL (4.5 mmol/L). Calcium enhances the inotropic effect of digitalis; therefore, hypercalcemia aggravates digitalis toxicity (Shane, 2019a).

Clinical Manifestations

Clinical signs and symptoms can be found in [Table 10-8](#). The symptoms of hypercalcemia are proportional to the degree of elevation of the serum calcium level. The more severe symptoms tend to appear when the serum calcium level climbs rapidly and may be as high as 16 mg/dL (4 mmol/L) or higher. However, some patients become profoundly disturbed with serum calcium levels of only 12 mg/dL (3 mmol/L). These symptoms resolve as serum calcium levels return to normal after treatment (Shane, 2019b).

Hypercalcemic crisis refers to an acute rise in the serum calcium level. Severe thirst and polyuria are often present. Other findings may include muscle weakness, intractable nausea, abdominal cramps, severe constipation, diarrhea, peptic ulcer symptoms, and bone pain. Lethargy, confusion, and coma may also occur. This condition is dangerous and may result in cardiac arrest. Emergency treatment with calcitonin is indicated (Shane & Berenson, 2019) (see later discussion under Pharmacologic Therapy).

Assessment and Diagnostic Findings

In hypercalcemia, the serum calcium level is greater than 10.4 mg/dL (2.6 mmol/L). Cardiovascular changes may include a variety of arrhythmias (e.g., heart blocks) and shortening of the QT interval and ST segment. The PR interval is sometimes prolonged. The double-antibody PTH test may be used to differentiate between primary hyperparathyroidism and malignancy as a cause of hypercalcemia: PTH levels are increased in primary or secondary hyperparathyroidism and suppressed in malignancy. X-rays may reveal bone changes if the patient has hypercalcemia secondary to a malignancy, bone cavitations, or urinary calculi. Urine calcium can be normal or elevated in hyperparathyroidism and hypercalcemia caused by malignancy (Shane, 2019b).

Medical Management

Therapeutic aims include decreasing the serum calcium level and reversing the process causing the hypercalcemia. Treating the underlying cause (e.g.,

chemotherapy for a malignancy, partial parathyroidectomy for hyperparathyroidism) is essential.

Pharmacologic Therapy

To treat hypercalcemia, measures include administering fluids to dilute serum calcium and promote its excretion by the kidneys, mobilizing the patient, and restricting dietary calcium intake. IV administration of 0.9% sodium chloride solution temporarily dilutes the serum calcium level and increases urinary calcium excretion by inhibiting tubular reabsorption of calcium. Administering IV phosphate can cause a reciprocal drop in serum calcium. Furosemide is often used in conjunction with administration of a saline solution; in addition to causing diuresis, furosemide increases calcium excretion. Although often overlooked, fluids and medications that contain calcium and dietary sources of calcium should be halted (Shane & Berenson, 2019).

Calcitonin can be used to lower the serum calcium level and is particularly useful for patients with heart disease or acute kidney injury who cannot tolerate large sodium loads. Calcitonin reduces bone resorption, increases the deposition of calcium and phosphorus in the bones, and increases urinary excretion of calcium and phosphorus. Although several forms are available, calcitonin derived from salmon is commonly used. Skin testing for allergy to salmon calcitonin may be necessary before the hormone is given. Systemic allergic reactions are possible because this hormone is a protein; resistance to the medication may develop later because of antibody formation. Calcitonin is administered by intramuscular injection or intranasal spray rather than subcutaneously, because patients with hypercalcemia have poor perfusion of subcutaneous tissue (Shane & Berenson, 2019).

For patients with cancer, treatment is directed at controlling the condition by surgery, chemotherapy, or radiation therapy. Corticosteroids may be used to decrease bone turnover and tubular reabsorption for patients with sarcoidosis, myelomas, lymphomas, and leukemias; patients with solid tumors are less responsive. Some bisphosphonates (e.g., pamidronate disodium, ibandronate sodium) inhibit osteoclast activity. IV forms of bisphosphonates can cause fever, transient leukopenia, eye inflammation, nephrotic syndrome, and jaw osteonecrosis (Shane & Berenson, 2019).

Mithramycin, a cytotoxic antibiotic, inhibits bone resorption and thus lowers the serum calcium level. This agent must be used cautiously because it has significant side effects, including thrombocytopenia, nephrotoxicity, rebound hypercalcemia when discontinued, and hepatotoxicity. Inorganic phosphate salts can be given orally or by nasogastric tube (in the form of Phospho-Soda or Neutra-Phos), rectally (as retention enemas), or IV. IV phosphate therapy is used with extreme caution in the treatment of hypercalcemia, because it can cause severe calcification in various tissues,

hypotension, tetany, and acute kidney injury (Frandsen & Pennington, 2018; Vallerand & Sanoski, 2019).

Nursing Management

The nurse must monitor for hypercalcemia in at-risk patients. Interventions such as increasing patient mobility and encouraging fluids can help prevent hypercalcemia, or at least minimize its severity. Hospitalized patients at risk should be encouraged to ambulate as soon as possible. Those who are outpatients and receive home care are educated about the importance of frequent ambulation (Daly, 2017).

When encouraging oral fluids, the nurse considers the patient's likes and dislikes. Fluids containing sodium should be given unless contraindicated, because sodium assists with calcium excretion. Patients are encouraged to drink 2.8 to 3.8 L (3 to 4 quarts) of fluid daily (Sterns, 2017b). Adequate fiber in the diet is encouraged to offset the tendency for constipation. Safety precautions are implemented, as necessary, when altered mental status is present. The patient and family are informed that these mental changes are reversible with treatment. Increased calcium increases the effects of digitalis; therefore, the patient on digitalis should be frequently assessed for signs and symptoms of digitalis toxicity. Because ECG changes (premature ventricular contractions, paroxysmal atrial tachycardia, and heart block) can occur, the cardiac rate and rhythm are monitored for any abnormalities (Shane & Berenson, 2019; Vallerand & Sanoski, 2019).

Magnesium Imbalances

Magnesium (Mg^{++}) is an abundant intracellular cation. It acts as an activator for many intracellular enzyme systems and plays a role in both carbohydrate and protein metabolism. The normal serum magnesium level is 1.8 to 2.6 mg/dL (0.74 to 1.07 mmol/L) (Fischbach & Fischbach, 2018). Approximately one third of serum magnesium is bound to protein; the remaining two thirds exist as free cations—the active component (Mg^{++}). Magnesium balance is important in neuromuscular function. Because magnesium acts directly on the myoneural junction, variations in the serum level affect neuromuscular irritability and contractility. For example, an excess of magnesium diminishes the excitability of the muscle cells, whereas a deficit increases neuromuscular irritability and contractility. Magnesium produces its sedative effect at the neuromuscular junction, probably by inhibiting the release of the neurotransmitter acetylcholine. It also increases the stimulus threshold in nerve fibers. Magnesium imbalances are magnesium deficit and magnesium excess (Norris, 2019) (Table 10-9).

Magnesium also affects the cardiovascular system, acting peripherally to produce vasodilation and decreased peripheral resistance. Most magnesium in the body is stored within bone, whereas magnesium ions in the blood are bound to protein, such as albumin, or free as Mg⁺⁺ ions (Yu, 2019a).

TABLE 10-9 Magnesium Imbalances

Imbalance	Contributing Factors	Signs/Symptoms
Magnesium deficit (hypomagnesemia) Serum magnesium <1.8 mg/dL	Chronic alcoholism, hyperparathyroidism, hyperaldosteronism, diuretic phase of acute kidney injury, malabsorptive disorders, diabetic ketoacidosis, refeeding after starvation, parenteral nutrition, chronic laxative use, diarrhea, acute myocardial infarction, heart failure, decreased serum K ⁺ and Ca ⁺⁺ and certain pharmacologic agents (e.g., gentamicin, cisplatin, cyclosporine)	Neuromuscular irritability, positive Trousseau sign and Chvostek sign, insomnia, mood changes, anorexia, vomiting, increased tendon reflexes, and ↑ BP. ECG: PVCs, flat or inverted T waves, depressed ST segment, prolonged PR interval, and widened QRS
Magnesium excess (hypermagnesemia) Serum magnesium >2.6 mg/dL	Oliguric phase of acute kidney injury (particularly when magnesium- containing medications are given), adrenal insufficiency, excessive IV magnesium administration, diabetic ketoacidosis, and hypothyroidism	Flushing, hypotension, muscle weakness, drowsiness, hypoactive reflexes, depressed respirations, cardiac arrest and coma, diaphoresis. ECG: tachycardia → bradycardia, prolonged PR interval and QRS, peaked T waves

BP, blood pressure; ECG, electrocardiogram; →, followed by; ↑, increased; IV, intravenous; PVCs, premature ventricular contractions.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Magnesium Deficit (Hypomagnesemia)

Hypomagnesemia refers to a below-normal serum magnesium concentration and is frequently associated with hypokalemia and hypocalcemia. Magnesium is similar to calcium in two aspects: (1) it is the ionized fraction of magnesium that is primarily involved in neuromuscular activity and other physiologic processes, and (2) magnesium levels should be evaluated in combination with albumin levels. Because about 30% of magnesium is protein bound, principally to albumin, a decreased serum albumin level can reduce the measured total magnesium concentration; however, it does not reduce the ionized plasma magnesium concentration. Magnesium is essential for the

function of the Na^+/K^+ pump, and low Mg^{++} levels have effects on intracellular influxes of K^+ and myocardial ion fluxes (Norris, 2019; Yu, 2019a).

Pathophysiology

An important route of magnesium loss is the GI tract; such loss can occur with nasogastric suction, diarrhea, or fistulas. Because fluid from the lower GI tract has a higher concentration of magnesium (10 to 14 mEq/L) than fluid from the upper tract (1 to 2 mEq/L), losses from diarrhea and intestinal fistulas are more likely to induce magnesium deficit than are those from gastric suction. Although magnesium losses are relatively small in nasogastric suction, hypomagnesemia occurs if losses are prolonged and magnesium is not replaced through IV infusion. Because the distal small bowel is the major site of magnesium absorption, any disruption in small bowel function (e.g., intestinal resection or inflammatory bowel disease) can lead to hypomagnesemia. Hypomagnesemia is a common yet often overlooked imbalance in acutely and critically ill patients. Studies suggest that up to 65% of patients in intensive care are magnesium deficient (Upala, Jaruvongvanich, Wijarnpreecha, et al., 2016). Hypomagnesemia may occur with withdrawal from alcohol and administration of tube feedings or parenteral nutrition (Yu, 2019a).

Chronic alcohol abuse is a major cause of symptomatic hypomagnesemia in the United States. The serum magnesium level should be measured at least every 2 or 3 days in patients undergoing withdrawal from alcohol. The level may be normal on admission but may decrease as a result of metabolic changes, such as the intracellular shift of magnesium associated with IV glucose administration (Yu, 2019a).

During nutritional replacement, the major cellular electrolytes move from the serum to intracellular compartments of newly synthesized cells. Therefore, if the enteral or parenteral feeding formula is deficient in magnesium content, serious hypomagnesemia can occur. Because of this, serum magnesium levels should be measured at regular intervals in patients who are receiving parenteral or enteral feedings, especially those who have undergone a period of starvation (Yu, 2019a).

Other causes of hypomagnesemia include the administration of aminoglycosides, cyclosporine, cisplatin, diuretics, digitalis, proton pump inhibitors, and amphotericin, as well as the rapid administration of citrated blood, especially to patients with renal or hepatic disease. Magnesium deficiency often occurs in DKA, secondary to increased renal excretion during osmotic diuresis and shifting of magnesium into the cells with insulin therapy (Yu, 2019a). A magnesium IV solution can be used to counteract seizures due to preeclampsia or eclampsia, the cardiac arrhythmia torsades de pointes, asthma, and hypertension (Vallerand & Sanoski, 2019).

Clinical Manifestations

Clinical signs and symptoms can be found in [Table 10-9](#). Some clinical manifestations of hypomagnesemia are due directly to the low serum magnesium level; others are due to secondary changes in potassium and calcium metabolism. Symptoms do not usually occur until the serum magnesium level has dropped to less than 1.8 mEq/L (0.75 mmol/L). Chvostek and Troussseau signs (see earlier discussion) occur, in part, because of accompanying hypocalcemia (Norris, 2019).

Hypomagnesemia may be accompanied by marked alterations in psychological status. Apathy, depressed mood, apprehension, and extreme agitation have been noted, as well as ataxia, dizziness, insomnia, and confusion. At times, delirium, auditory or visual hallucinations, and frank psychoses may occur (Yu & Yarlagadda, 2019).

Magnesium deficiency can disturb the ECG by prolonging the QRS, depressing the ST segment, and predisposing to cardiac arrhythmias, such as premature ventricular contractions, supraventricular tachycardia, torsades de pointes, and ventricular fibrillation. Increased susceptibility to digitalis toxicity is associated with low serum magnesium levels. Patients receiving digoxin are also likely to be receiving diuretic therapy, predisposing them to renal loss of magnesium. Concurrent hypokalemia and hypocalcemia must be addressed in addition to hypomagnesemia. These electrolyte disturbances are difficult to correct until magnesium has been replenished. Additionally, hypocalcemia can be worsened by isolated treatment of hypomagnesemia with IV magnesium sulfate because sulfate binds ionized calcium (Yu & Yarlagadda, 2019).

Assessment and Diagnostic Findings

On laboratory analysis, the serum magnesium level is less than 1.8 mg/dL (0.74 mmol/L). Urine magnesium may help identify the cause of magnesium depletion, and levels are measured after a loading dose of magnesium sulfate is given. Additional diagnostic techniques (nuclear magnetic resonance spectroscopy and the ion-selective electrode) are sensitive and direct means of measuring ionized serum magnesium levels (Yu, 2019b).

Medical Management

Mild magnesium deficiency can be corrected by diet alone. Principal dietary sources of magnesium include green leafy vegetables, beans, lentils, white potatoes, wheat bran, dry roasted almonds, and peanut butter (National Institutes of Health [NIH], 2019).

If necessary, magnesium salts can be given orally in an oxide or gluconate form to replace continuous losses but can produce diarrhea. Patients receiving

parenteral nutrition require magnesium in the IV solution to prevent hypomagnesemia

Vital signs must be assessed frequently during magnesium administration to detect changes in cardiac rate or rhythm, hypotension, and respiratory distress. Monitoring urine output is essential before, during, and after magnesium administration as this is how Mg⁺⁺ is excreted; the primary provider is notified if urine volume decreases to less than 100 mL over 4 hours. Calcium gluconate must be readily available to treat hypocalcemic tetany or hypermagnesemia.



Quality and Safety Nursing Alert

Inadvertent overdosage of IV magnesium can result in serious patient harm and death. Whenever a patient is prescribed IV magnesium, a second nurse should independently double check the IV magnesium prescription, including dose calculations, and check infusion pump settings. Milligrams (mg) and grams (g) are not equivalent to milliequivalent (mEq) dosages.

Nursing Management

The nurse should be aware of patients at risk for hypomagnesemia and observe them for its signs and symptoms. Patients receiving digitalis are monitored closely, because a deficit of magnesium can predispose them to digitalis toxicity. If hypomagnesemia is severe, seizure precautions are implemented. Other safety precautions are instituted, as indicated, if confusion is observed. Patients should be screened for dysphagia (difficulty in swallowing), as this may occur in those with magnesium depletion.

Patient education plays a major role in treating magnesium deficit. The patient is educated about sources of magnesium-rich foods, including green vegetables, nuts, legumes, bananas, and oranges.

Magnesium Excess (Hypermagnesemia)

Hypermagnesemia (serum magnesium level higher than 2.6 mg/dL [1.07 mmol/L]) is a rare electrolyte abnormality, because the kidneys efficiently excrete magnesium (Norris, 2019). A serum magnesium level can appear falsely elevated if blood specimens are allowed to hemolyze or are drawn from an extremity with a tourniquet that was applied too tightly.

Pathophysiology

The most common cause of hypermagnesemia is kidney injury (Yu & Gupta, 2019). In fact, most patients with advanced kidney injury have at least a slight elevation in serum magnesium levels. This condition is aggravated when such patients receive magnesium to control seizures.

Hypermagnesemia can occur in patients with untreated DKA when catabolism causes the release of cellular magnesium that cannot be excreted because of profound fluid volume depletion and resulting oliguria. A surplus of magnesium can also result from excessive magnesium given to treat hypertension of pregnancy or to treat hypomagnesemia. Increased serum magnesium levels can also occur in adrenocortical insufficiency, Addison disease, or hypothermia.

Excessive use of magnesium-based antacids or laxatives and medications that decrease GI motility, including opioids and anticholinergics, can also increase serum magnesium levels. Decreased elimination of magnesium or its increased absorption due to intestinal hypomotility from any cause can contribute to hypermagnesemia. Lithium intoxication can also cause an increase in serum magnesium levels. Extensive soft tissue injury or necrosis as with trauma, shock, sepsis, cardiac arrest, or severe burns can also result in hypermagnesemia (Yu & Gupta, 2019).

Clinical Manifestations

Acute elevation of the serum magnesium level depresses the central nervous system as well as the peripheral neuromuscular junction. Clinical signs and symptoms can be found in [Table 10-9](#). The respiratory center is depressed when serum magnesium levels exceed 10 mEq/L (5 mmol/L). Coma, atrioventricular heart block, and cardiac arrest can occur when the serum magnesium level is greatly elevated and not treated. High levels of magnesium also result in platelet clumping and delayed thrombin formation (Yu & Gupta, 2019).

Assessment and Diagnostic Findings

In hypermagnesemia, the serum magnesium level is greater than 2.6 mg/dL (1.07 mmol/L). Increased potassium and calcium are present concurrently. As creatinine clearance decreases to less than 3.0 mL/min, the serum magnesium levels increase. ECG findings may include a prolonged PR interval, tall T waves, a widened QRS, and a prolonged QT interval, as well as an atrioventricular block (Norris, 2019).

Medical Management

Hypermagnesemia can be prevented by avoiding the administration of magnesium to patients with kidney injury and by carefully monitoring seriously ill patients who are receiving magnesium salts. In patients with severe hypermagnesemia, all parenteral and oral magnesium salts are discontinued. In emergencies, such as respiratory depression or defective cardiac conduction, ventilatory support and IV elemental calcium as a magnesium antagonist are indicated. In addition, hemodialysis with a magnesium-free dialysate can reduce the serum magnesium to a safe level within hours. Administration of loop diuretics (e.g., furosemide) and sodium chloride or lactated Ringer's IV solution enhances magnesium excretion in patients with adequate renal function. IV calcium antagonizes the cardiovascular and neuromuscular effects of magnesium (Yu & Gupta, 2019).

Nursing Management

Patients at risk for hypermagnesemia should be monitored closely. If hypermagnesemia is suspected, the nurse should monitor the vital signs, noting hypotension and shallow respirations. The nurse should be aware that arrhythmias, bradycardia, and heart block can occur. The nurse also observes for decreased deep tendon reflexes (DTRs), muscle weakness, and changes in the level of consciousness. Medications that contain magnesium are not given to patients with compromised renal function, and patients with kidney injury are cautioned to check with their primary providers before taking OTC supplements. Administration of fluids and diuretics are often used in treatment, and monitoring of I&O is important. Hemodialysis may be necessary in severe hypermagnesemia, particularly if cardiovascular or neurologic manifestations are present. Hemodialysis is also necessary in patients with severe hypermagnesemia and kidney injury (Yu & Gupta, 2019).

Phosphorus Imbalances

Phosphorus (HPO_4^{2-}) is a critical constituent of all body tissues and is plentiful in the average diet of people living in developed countries. Intake of phosphorus usually exceeds body requirements and the kidney excretes the excess. Phosphorus is essential to the function of muscle and RBCs; the formation of adenosine triphosphate (ATP) and of 2,3-diphosphoglycerate, which facilitates the release of oxygen from hemoglobin; and the maintenance of acid–base balance, as well as the nervous system and the intermediary metabolism of carbohydrate, protein, and fat. It is a major component of the cell structure, phospholipids, nucleotides, and nucleic acids (DNA and RNA). It provides structural support to bones and teeth. Phosphorus is the primary anion of the ICF and requires active transport mechanisms to maintain its

presence inside the cells. Most intracellular phosphorus is bound to proteins and lipids. About 85% of phosphorus is located in bones and teeth, 14% in soft tissue, and less than 1% in the ECF (Norris, 2019).

TABLE 10-10 Phosphorus Imbalances

Imbalance	Contributing Factors	Signs/Symptoms
Phosphorus deficit (hypophosphatemia)	Refeeding after starvation, alcohol withdrawal, diabetic ketoacidosis, respiratory and metabolic alkalosis, ↓ magnesium, ↓ potassium, hyperparathyroidism, vomiting, diarrhea, hyperventilation, vitamin D deficiency associated with malabsorptive disorders, burns, acid-base disorders, parenteral nutrition, and diuretic and antacid use	Paresthesias, muscle weakness, bone pain and tenderness, chest pain, confusion, cardiomyopathy, respiratory failure, seizures, tissue hypoxia, and increased susceptibility to infection, nystagmus
Phosphorus excess (hyperphosphatemia)	Acute kidney injury and chronic kidney disease, excessive intake of phosphorus, vitamin D excess, respiratory and metabolic acidosis, hypoparathyroidism, volume depletion, leukemia/lymphoma treated with cytotoxic agents, increased tissue breakdown, rhabdomyolysis	Tetany, tachycardia, anorexia, nausea and vomiting, muscle weakness, signs and symptoms of hypocalcemia; hyperactive reflexes; soft tissue calcifications in lungs, heart, kidneys, and cornea

↓, decreased.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

The normal serum phosphorus level is 2.7 to 4.5 mg/dL (0.87 to 1.45 mmol/L) in adults (Fischbach & Fischbach, 2018). Phosphorus homeostasis is maintained through absorption and secretion in the GI tract, filtration and absorption in the kidneys, and shifts into and out of bone. PTH and vitamin D assist in phosphate homeostasis by varying phosphate reabsorption in the proximal tubule of the kidney. PTH allows the shift of phosphate from bone to plasma. Phosphorus deficit and phosphorus excess are less common electrolyte imbalances (Table 10-10).

Phosphorus Deficit (Hypophosphatemia)

Hypophosphatemia is indicated by a value below 2.7 mg/dL (0.87 mmol/L). Although it often indicates phosphorus deficiency, hypophosphatemia may

occur under a variety of circumstances in which total-body phosphorus stores are normal. Phosphorus deficiency can also occur as an abnormally low content of phosphorus in lean tissues without evidence of low phosphate in the bloodstream. Hypophosphatemia can be due to lack of sufficient intake, excess excretion (renal phosphate wasting), shift of phosphorus from extracellular to intracellular spaces, or by decreased intestinal absorption of phosphorus (Yu & Stubbs, 2019).

Pathophysiology

Hypophosphatemia rarely occurs due to inadequate intake, as there are many dietary sources of phosphate (e.g., meats, dairy products, beans). However, phosphate can become depleted in GI malabsorption disorders such as chronic diarrhea, Crohn's disease, or celiac disease. Hypophosphatemia can also occur in anorexia, bulimia, and alcoholism. Vitamin D deficit can cause low phosphate levels in the bloodstream. Vitamin D regulates intestinal absorption of calcium and phosphate ion; therefore, a deficiency of vitamin D can cause both decreased calcium and phosphorus levels. These deficiencies can lead to osteomalacia (softened and brittle bones) in the adult (Norris, 2019).

Hypophosphatemia can occur if there is high intake of antacids—particularly those containing calcium, magnesium, or aluminum. Excess phosphorus binding by antacids may decrease the phosphorus available from the diet to an amount lower than required to maintain serum phosphorus balance. The degree of hypophosphatemia depends on the amount of phosphorus in the diet compared to the dose of antacid (Yu & Stubbs, 2019).

Hypophosphatemia can also occur during the administration of calories to patients who have had severe protein–calorie malnutrition. This syndrome can be induced in any person with severe malnutrition (e.g., patients with anorexia nervosa or alcoholism, older patients who are debilitated and unable to eat). Feeding a patient who is nutritionally deprived stimulates a large insulin release that can cause shift of phosphate from the extracellular to the intracellular compartment. Also, marked hypophosphatemia can develop in patients who are malnourished who receive parenteral nutrition that does not contain sufficient phosphorus (Aubry, Friedli, Schetz, et al., 2018).

Other causes of hypophosphatemia include heatstroke, prolonged intense hyperventilation, alcohol withdrawal, DKA, respiratory alkalosis, hepatic encephalopathy, and major thermal burns. Hyperparathyroidism can also cause increased urinary losses of phosphorus leading to hypophosphatemia. Loss of phosphorus through the kidneys also occurs with acute volume expansion, osmotic diuresis, the use of carbonic anhydrase inhibitors (acetazolamide), and some malignancies.

Respiratory alkalosis can cause a decrease in phosphorus in the bloodstream because of an intracellular shift of phosphorus. Respiratory alkalosis,

commonly caused by extreme hyperventilation, can reduce serum phosphate concentrations to very low levels and is a common cause of marked hypophosphatemia in patients who are hospitalized.

Some genetic disorders, such as Fanconi syndrome and X-linked hypophosphatemic rickets, cause renal phosphate wasting. Acquired oncogenic osteomalacia, a paraneoplastic syndrome that occurs with some types of cancer, can also cause low phosphate levels. Hypophosphatemia is also a frequent complication of renal transplantation (Yu & Stubbs, 2019).

Clinical Manifestations

Most of the signs and symptoms of phosphorus deficiency result from a deficiency of ATP, 2,3-diphosphoglycerate, or both (Yu & Stubbs, 2019). ATP deficiency impairs cellular energy resources; diphosphoglycerate deficiency impairs oxygen delivery to tissues, resulting in generalized weakness and neurologic manifestations. Clinical signs and symptoms can be found in [Table 10-10](#).

Muscle damage may develop as the ATP level in the muscle tissue declines. Clinical manifestations are muscle weakness, which may be subtle or profound and may affect any muscle group; muscle pain; and at times acute rhabdomyolysis (breakdown of skeletal muscle). Weakness of the diaphragm and intercostal muscles may greatly impair ventilation. Bone pain, altered mental status, seizures, focal neurologic signs, and heart failure can occur with severe hypophosphatemia (Yu & Stubbs, 2019).

Assessment and Diagnostic Findings

On laboratory analysis, the serum phosphorus level is less than 2.7 mg/dL (0.87 mmol/L). When reviewing laboratory results, the nurse should keep in mind that glucose or insulin administration causes a decrease in the serum phosphorus level. Acute hypophosphatemia can occur during the treatment of DKA due to high doses of insulin. Elevated PTH levels that occur as a result of hyperparathyroidism can lower blood levels of phosphate. A 24-hour urine collection for phosphorus can be done if renal-phosphate wasting is suspected. Fanconi syndrome causes loss of glucose, amino acids, uric acid, and phosphate in the urine. X-rays and bone density studies may show skeletal changes of reduced bone mineralization caused by osteomalacia or rickets. A technetium (Tc99m) sestamibi scan of the neck can be done to check for hyperparathyroidism. CT scan, MRI, or indium-111 octreotide scan may show oncogenic osteomalacia (decreased bone density due to cancer) (Sharma & Castro, 2019).

Medical Management

Prevention of hypophosphatemia is the goal. In patients at risk for hypophosphatemia, serum phosphate levels should be closely monitored and correction initiated before deficits become severe. Adequate amounts of phosphorus should be added to parenteral solutions, and attention should be paid to the phosphorus levels in enteral feeding solutions.

Medical management of hypophosphatemia depends on the etiology, and treatment of the cause is essential. Severe hypophosphatemia is dangerous and requires prompt attention. Oral phosphate supplements are usually adequate for mild hypophosphatemia. Sources of phosphorus include dairy foods, meats, and beans. Oral supplements may be sufficient to treat renal-wasting disorders, malabsorption, or oncogenic osteomalacia until an exact etiology is found. Vitamin D may also be necessary to enhance absorption of phosphorus and calcium.

Aggressive IV phosphorus correction is usually limited to the patient whose serum phosphorus levels decrease to less than 1 mg/dL (0.3 mmol/L). Possible effects of IV administration of phosphorus include tetany from hypocalcemia and calcifications in tissues (blood vessels, heart, lung, kidney, eyes) from hyperphosphatemia.

Recently a monoclonal antibody-type drug, burosomab, has been approved for patients with renal phosphate wasting disorders and hypophosphatemic rickets. This drug has been found to normalize phosphate levels, improve bone mineralization, and heal fractures in rickets (Perwad & Portale, 2019).

If hyperparathyroidism is caused by parathyroid tumor, surgery may be necessary. A parathyroid tumor, termed an adenoma, can cause hyperparathyroidism with resulting demineralization of bone (osteomalacia). Patients with hyperparathyroidism can also be given calcimimetic medications, such as cinacalcet, which activate the calcium receptor in the gland and decrease PTH secretion (Wang & Yuan, 2019).

Patients with oncogenic osteomalacia should be treated for the cancer causing this syndrome. If malabsorption is the cause of hypophosphatemia, treatment of the GI disorder is necessary. Because patients with osteomalacia are at high risk for fracture, fall precautions should be observed. If hypophosphatemia is caused by an eating disorder, the patient should obtain behavioral counseling and nutritional therapy.

Nursing Management

The nurse should identify patients at risk for hypophosphatemia and monitor them. Because patients who are malnourished and receiving parenteral nutrition are at risk when calories are introduced too aggressively, preventive measures involve gradually introducing the solution to avoid rapid shifts of phosphorus into the cells. Vitamin D and calcium levels should also be monitored as phosphate levels are influenced by these levels. Vitamin D

supplementation may be needed to enhance calcium and phosphate absorption (Aubry et al., 2018).

In patients requiring correction of phosphorus losses, the nurse frequently monitors serum phosphorus levels and documents and reports early signs of hypophosphatemia (apprehension, confusion, change in level of consciousness). If the patient experiences mild hypophosphatemia, foods such as milk and milk products, organ meats, beans, nuts, fish, poultry, and whole grains should be encouraged. With moderate hypophosphatemia, supplements such as Neutra-Phos capsules, K-Phos, and Fleet Phospho-Soda may be prescribed (Vallerand & Sanoski, 2019; Yu & Stubbs, 2019).

For patients with severe hypophosphatemia, IV preparations of phosphorus are available as sodium or potassium phosphate. The rate of phosphorus administration should not exceed 3 mmol/h, and the site should be carefully monitored because tissue sloughing and necrosis can occur with infiltration. Serum calcium and phosphate blood levels should be monitored every 6 hours with IV therapy.

Phosphorus Excess (Hyperphosphatemia)

Hyperphosphatemia is a serum phosphorus level that exceeds 4.5 mg/dL (1.45 mmol/L). Abnormally high levels of phosphate in the bloodstream can arise from excessive intake of phosphorus, decreased excretion of phosphorus, or a disorder that shifts intracellular phosphate into the extracellular space.

Pathophysiology

Various conditions can lead to hyperphosphatemia; however, the most common is kidney injury, which diminishes urinary phosphate excretion (Stubbs & Yu, 2019). When renal insufficiency progresses to 40% to 50% of renal function, hyperphosphatemia can occur. Other causes include increased intake or a shift of phosphate from the intracellular to extracellular space. Conditions such as excessive vitamin D intake, administration of total parenteral nutrition, chemotherapy for neoplastic disease, hypoparathyroidism, pseudohypoparathyroidism, metabolic or respiratory acidosis, DKA, acute hemolysis, high phosphate intake, profound muscle necrosis, and increased phosphorus absorption may also lead to this phosphorus imbalance. Hypoparathyroidism causes hyperphosphatemia by failure of the kidneys to inhibit renal reabsorption of phosphate. The kidneys reabsorb excessive phosphate into the bloodstream. Pseudohypoparathyroidism is a disorder caused by end-organ resistance to PTH. Excessive use of phosphate-containing laxatives or enemas can also lead to hyperphosphatemia. False elevation of phosphate in the bloodstream can occur with elevated protein or bilirubin levels, dyslipidemia, or hemolysis. The primary complication of increased

phosphorus is metastatic calcification in the organs, soft tissues, joints, and arteries. Arterial calcification is a risk factor for myocardial infarction, stroke, and peripheral arterial disease (Norris, 2019; Stubbs & Yu, 2019).

Clinical Manifestations

Clinical signs and symptoms can be found in [Table 10-10](#). Most symptoms result from decreased calcium levels and soft tissue calcifications. The most important short-term consequence is tetany (severe muscle cramping). Because of the reciprocal relationship between phosphorus and calcium, a high serum phosphorus level tends to cause a low serum calcium concentration. Hypocalcemia causes neuromuscular irritability and muscle spasms (Bove-Fenderson & Mannstadt, 2018).

The major long-term consequence is soft tissue calcification, which occurs mainly in patients with a reduced glomerular filtration rate. High serum levels of inorganic phosphorus promote precipitation of calcium phosphate in nonosseous sites, which can decrease urine output, impair vision, and produce palpitations. Skin and soft tissue deposits of calcium can cause pruritus (Marcucci, Cianferotti, & Brandi, 2018).

Assessment and Diagnostic Findings

On laboratory analysis, the serum phosphorus level exceeds 4.5 mg/dL (1.5 mmol/L). The serum calcium level is useful also for diagnosing the primary disorder and assessing the effects of treatments. X-rays may show skeletal changes with abnormal bone development. PTH levels are decreased in hypoparathyroidism. BUN and creatinine levels are used to assess renal function (Marcucci et al., 2018).

Medical Management

Phosphate intake should be reduced and phosphate binders can be given with meals to reduce hyperphosphatemia. When possible, treatment is directed at the underlying disorder. For example, hyperphosphatemia may be related to volume depletion or respiratory or metabolic acidosis. Correction of these disorders may normalize the blood level of phosphate.

Calcium carbonate or calcium citrate are phosphate binders that can be used to lower blood phosphate levels. However, careful monitoring of calcium is essential as hypercalcemia can result. Phosphate binding resins that do not contain calcium include sevelamer and lanthanum. Sucroferric oxyhydroxide can also be used particularly in patients who require iron supplementation. Forced diuresis with a loop diuretic or saline diuresis can be used in patients

with normal renal function. Hemodialysis can also lower phosphorus (Carfagna, Del Vecchio, Pontoriero, et al., 2018).

Nursing Management

The nurse monitors patients at risk for hyperphosphatemia. If a low phosphorus diet is prescribed, the patient is instructed to avoid phosphorus-rich foods, such as hard cheeses, cream, nuts, meats, whole-grain cereals, dried fruits, dried vegetables, kidneys, sardines, and dairy foods (Shimada, Shutto-Uchita, & Yamabe, 2019). When appropriate, the nurse instructs the patient to avoid phosphate-containing laxatives and enemas. When administering phosphate binders, calcium levels should be monitored as well. During diuresis the nurse monitors urine output. The nurse also educates the patient about recognizing the signs of hypocalcemia, such as muscle cramping (Goltzman, 2019a).

Chloride Imbalances

Chloride (Cl^-) is the major anion of the ECF compartment. It maintains cellular integrity by providing water balance and maintains acid–base balance. Sodium and chloride make up the electrolytes that exert osmotic pressure. Chloride is also contained in gastric and pancreatic juices, sweat, bile, and saliva. Chloride is produced in the stomach, where it combines with hydrogen to form hydrochloric acid. Chloride control depends on the intake of chloride and the excretion and reabsorption of its ions in the kidneys. A small amount of chloride is lost in the feces (Norris, 2019).

The normal serum chloride level is 97 to 107 mEq/L (97 to 107 mmol/L). Inside the cell, the chloride level is 4 mEq/L. The serum level of chloride reflects a change in dilution or concentration of the ECF and does so in direct proportion to the sodium concentration. Serum osmolality parallels chloride levels as well. Aldosterone secretion increases sodium reabsorption, thereby increasing chloride reabsorption (Hall, 2016).

The choroid plexus, which secretes cerebrospinal fluid in the brain, depends on sodium and chloride to attract water to form the fluid portion of the cerebrospinal fluid.

Bicarbonate has an inverse relationship with chloride. As chloride moves from plasma into the RBCs (called the *chloride shift*), bicarbonate moves back into the plasma. Hydrogen ions are formed, which then help release oxygen from hemoglobin (Emmett & Szerlip, 2017a). When the level of one of these three electrolytes (sodium, bicarbonate, or chloride) is disturbed, the other two are also affected. Chloride assists in maintaining acid–base balance and works as a buffer in the exchange of oxygen and CO_2 in RBCs (Fischbach &

Fischbach, 2018). Chloride is primarily obtained from the diet as table salt. The chloride imbalances are chloride deficit and chloride excess ([Table 10-11](#)).

Chloride Deficit (Hypochloremia)

Hypochloremia is a serum chloride level below 97 mEq/L (97 mmol/L).

Pathophysiology

Hypochloremia can occur with GI tube drainage, gastric suctioning, gastric surgery, and severe vomiting and diarrhea. Administration of chloride-deficient IV solutions, low sodium intake, decreased serum sodium levels, DKA, chronic respiratory acidosis, massive blood transfusions, diuretic therapy, excessive sweating, burns, SIADH, and fever may cause hypochloremia. Administration of aldosterone, ACTH, corticosteroids, bicarbonate, excess diuretics, or laxatives decreases serum chloride levels as well. As chloride decreases (usually because of volume depletion), sodium and bicarbonate ions are retained by the kidney to balance the loss. Bicarbonate accumulates in the ECF, which raises the pH and can lead to hypochloremic metabolic alkalosis (Emmett & Szerlip, 2017a).

Clinical Manifestations

The signs and symptoms of hypochloremia are outlined in [Table 10-11](#). The signs and symptoms of hyponatremia, hypokalemia, and metabolic alkalosis may also be present. Metabolic alkalosis is a disorder that results in a high pH and a high serum bicarbonate level as a result of excess alkali intake or loss of hydrogen ions. With compensation, the partial pressure of CO₂ in arterial blood (PaCO₂) increases to 50 mm Hg. Hyperexcitability of muscles, tetany, hyperactive DTRs, weakness, twitching, and muscle cramps may result. Hypokalemia can cause hypochloremia, resulting in cardiac arrhythmias. In addition, because low chloride levels parallel low sodium levels, a water excess may occur. Hyponatremia can cause seizures and coma (Squiers, 2017).

TABLE 10-11 Chloride Imbalances

Imbalance	Contributing Factors	Signs/Symptoms and Laboratory Findings
Chloride deficit (hypochloremia) Serum chloride <96 mEq/L	Addison disease, reduced chloride intake or absorption, untreated diabetic ketoacidosis, chronic respiratory acidosis, excessive sweating, vomiting, gastric suction, diarrhea, sodium and potassium deficiency, metabolic alkalosis; loop, osmotic, or thiazide diuretic use; overuse of bicarbonate, rapid removal of ascitic fluid with a high sodium content, IV fluids that lack chloride (dextrose and water), draining fistulas and ileostomies, heart failure, cystic fibrosis	Agitation, irritability, tremors, muscle cramps, hyperactive deep tendon reflexes, hypertonicity, tetany, slow shallow respirations, seizures, arrhythmias, coma <i>Labs indicate:</i> ↓ serum chloride, ↓ serum sodium, ↑ pH, ↑ serum bicarbonate, ↑ total carbon dioxide content, ↓ urine chloride level, ↓ serum potassium
Chloride excess (hyperchloremia) Serum chloride >108 mEq/L	Excessive sodium chloride infusions with water loss, head injury (sodium retention), hypernatremia, kidney injury, corticosteroid use, dehydration, severe diarrhea (loss of bicarbonate), respiratory alkalosis, administration of diuretics, overdose of salicylates, sodium polystyrene sulfonate, acetazolamide, phenylbutazone and ammonium chloride use, hyperparathyroidism, metabolic acidosis	Tachypnea, lethargy, weakness, deep rapid respirations, decline in cognitive status, ↓ cardiac output, dyspnea, tachycardia, pitting edema, arrhythmias, coma <i>Labs indicate:</i> ↑ serum chloride, ↑ serum potassium and sodium, ↓ serum pH, ↓ serum bicarbonate, normal anion gap, ↑ urinary chloride level

↓, decreased; ↑, increased; IV, intravenous.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health state* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Assessment and Diagnostic Findings

In addition to the chloride level, sodium and potassium levels are also evaluated, because these electrolytes are lost along with chloride. ABG analysis identifies the acid–base imbalance, which is usually metabolic alkalosis. The urine chloride level, which is also measured, decreases in hypochloremia.

Medical Management

Treatment involves correcting the cause of hypochloremia and the contributing electrolyte and acid–base imbalances. Normal saline (0.9% sodium chloride) or half-strength saline (0.45% sodium chloride) solution is given by IV to replace the chloride. If the patient is receiving a diuretic (loop, osmotic, or thiazide), it may be discontinued or another diuretic prescribed (Squiers, 2017).

Ammonium chloride, an acidifying IV agent, may be prescribed to treat metabolic alkalosis; the dosage depends on the patient’s weight and serum chloride level. This agent is metabolized by the liver, and its effects last for about 3 days. Its use should be avoided in patients with impaired liver or renal function (Emmett & Szerlip, 2017b).

Nursing Management

The nurse monitors the patient’s I&O, ABG values, and serum electrolyte levels. Changes in the patient’s level of consciousness and muscle strength and movement are reported to the primary provider promptly. Vital signs are monitored, and respiratory assessment is carried out frequently. The nurse provides and educates the patient about foods with high chloride content, which include tomato juice, bananas, dates, eggs, cheese, milk, salty broth, canned vegetables, and processed meats. A person who drinks free water (water without electrolytes) or bottled water and excretes large amounts of chloride needs instruction to avoid drinking this kind of water (Squiers, 2017).

Chloride Excess (Hyperchloremia)

Hyperchloremia exists when the serum level of chloride exceeds 107 mEq/L (107 mmol/L). Hypernatremia, bicarbonate loss, and metabolic acidosis can occur with high chloride levels.

Pathophysiology

High serum chloride levels are almost exclusively a result of iatrogenically induced hyperchloremic metabolic acidosis, stemming from excessive

administration of chloride relative to sodium, most commonly as 0.9% normal saline solution, 0.45% normal saline solution, or lactated Ringer's solution. This condition can also be caused by the loss of bicarbonate ions via the kidney or the GI tract with a corresponding increase in chloride ions. Chloride ions in the form of acidifying salts accumulate, and acidosis occurs with a decrease in bicarbonate ions. Head trauma, increased perspiration, excess adrenocortical hormone production, and decreased glomerular filtration can lead to a high serum chloride level (Squiers, 2017).

Clinical Manifestations

The signs and symptoms of hyperchloremia are the same as those of metabolic acidosis: hypervolemia and hypernatremia. Tachypnea, weakness, lethargy, deep and rapid respirations, diminished cognitive ability, and hypertension occur. If untreated, hyperchloremia can lead to a decrease in cardiac output, arrhythmias, and coma. A high chloride level is accompanied by a high sodium level and fluid retention (Squiers, 2017).

Assessment and Diagnostic Findings

The serum chloride level is 108 mEq/L (108 mmol/L) or greater, the serum sodium level is greater than 145 mEq/L (145 mmol/L), the serum pH is less than 7.35, and the serum bicarbonate level is less than 22 mEq/L (22 mmol/L). Urine chloride levels are elevated.

Medical Management

Correcting the underlying cause of hyperchloremia and restoring electrolyte, fluid, and acid–base balance are essential. Hypotonic IV solutions may be given to restore balance. Lactated Ringer's solution may be prescribed to convert lactate to bicarbonate in the liver, which increases the bicarbonate level and corrects the acidosis. IV sodium bicarbonate may be given to increase bicarbonate levels, which leads to the renal excretion of chloride ions because bicarbonate and chloride compete for combination with sodium. Diuretics may be given to eliminate chloride as well. Sodium, chloride, and fluids are restricted (Squiers, 2017).

Nursing Management

Monitoring vital signs, ABG values, and I&O is important to assess the patient's status and the effectiveness of treatment. Assessment findings related to respiratory, neurologic, and cardiac systems are documented, and changes are discussed with the primary provider. The nurse educates the patient about

the diet that should be followed to manage hyperchloremia and maintain adequate hydration.



Acid–Base Disturbances

Acid–base disturbances are commonly encountered in clinical practice, especially in critical-care units. Identification of the specific acid–base imbalance is important in ascertaining the underlying cause of the disorder and determining appropriate treatment.

Plasma pH is an indicator of hydrogen ion (H^+) concentration and measures the acidity or alkalinity of the blood. Homeostatic mechanisms keep pH within a normal range (7.35 to 7.45). These mechanisms consist of buffer systems, the kidneys, and the lungs. The H^+ concentration is extremely important: The greater the concentration, the more acidic the solution and the lower the pH. The lower the H^+ concentration, the more alkaline the solution and the higher the pH (Larkin & Zimmanck, 2015; Norris, 2019).

Buffer systems prevent major changes in the pH of body fluids by removing or releasing H^+ ; they can act quickly to prevent excessive changes in H^+ concentration. Hydrogen ions are buffered by both intracellular and extracellular buffers. The body's major extracellular buffer system is the bicarbonate–carbonic acid buffer system, which is assessed when ABGs are measured. Normally, there are 20 parts of bicarbonate (HCO_3^-) to 1 part of carbonic acid (H_2CO_3). If this ratio is altered, the pH will change. It is the ratio of HCO_3^- to H_2CO_3 that is important in maintaining pH, not absolute values (Larkin & Zimmanck, 2015; Norris, 2019).

CO_2 is a potential acid; when dissolved in water, it becomes carbonic acid ($CO_2 + H_2O = H_2CO_3$). Therefore, when CO_2 is increased, the carbonic acid content is also increased. When the CO_2 level decreases, carbonic acid decreases. If either bicarbonate or carbonic acid is increased or decreased so that the 20:1 ratio is no longer maintained, acid–base imbalance results (Larkin & Zimmanck, 2015; Theodore, 2019).

Less important buffer systems in the ECF include the inorganic phosphates and the plasma proteins. Intracellular buffers include proteins, organic and inorganic phosphates, and, in RBCs, hemoglobin (Norris, 2019).

The kidneys regulate the bicarbonate level in the ECF; they can regenerate bicarbonate ions as well as reabsorb them from the renal tubular cells. In respiratory acidosis and most cases of metabolic acidosis, the kidneys excrete hydrogen ions and conserve bicarbonate ions to help restore balance. In respiratory and metabolic alkalosis, the kidneys retain hydrogen ions and excrete bicarbonate ions to help restore balance. The kidneys cannot

compensate for the metabolic acidosis created by kidney injury. Renal compensation for imbalances is relatively slow (a matter of hours or days) (Larkin & Zimmanck, 2015; Norris, 2019).

The lungs, under the control of the medulla, control the CO₂ and thus the carbonic acid content of the ECF. They do so by adjusting ventilation in response to the amount of CO₂ in the arterial blood. A rise in the partial pressure of CO₂ in arterial blood (designated PaCO₂) is a powerful stimulant to respiration. Of course, the partial pressure of oxygen in arterial blood (designated PaO₂) also influences respiration. However, its effect is not as marked as that produced by the PaCO₂ (Theodore, 2019).

In metabolic acidosis, the lungs compensate by raising respiratory rate, causing greater elimination of CO₂ (to reduce the acid load). In metabolic alkalosis, the lungs compensate by decreasing respiratory rate, causing CO₂ to be retained (to increase the acid load).

In order to understand acid–base imbalances, it is important to comprehend the chemical reaction that occurs in the bloodstream to maintain homeostasis. The bicarbonate buffer system is demonstrated by the following chemical equation:



TABLE 10-12 Normal Values for Arterial and Mixed Venous Bloods

Parameter	Arterial Blood	Mixed Venous Blood
pH	7.35–7.45	7.32–7.42
PCO ₂	35–45 mm Hg	38–52 mm Hg
PO ₂ ^a	>80 mm Hg	24–48 mm Hg
HCO ₃ ⁻	22–26 mEq/L	19–25 mEq/L
Base excess/deficit	±2 mEq/L	±5 mEq/L
Oxygen saturation (SaO ₂ %)	>94%	65–75%

^aAt altitudes of 3000 feet and higher; age dependent.

Adapted from Fischbach, F., & Fischbach, M. (2018). *A manual of laboratory and diagnostic tests* (10th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

In this chemical equation, carbon dioxide (CO₂) and water form carbonic acid (H₂CO₃) which dissociates into H⁺ and HCO₃⁻. It is also important to recognize that the major organs that control acid–base balance are the lungs, in control of CO₂, and the kidneys, which can excrete or reabsorb the ions needed to balance the pH. ABGs are laboratory tests that can provide values of blood pH, PaCO₂, PaO₂, and HCO₃⁻. ABGs are used to diagnose acid–base imbalances of the bloodstream. The saturation of Hgb with oxygen can also be

measured by pulse oximetry and designated as SaO₂. Normal range of SaO₂ is 95% to 100% (Theodore, 2019) ([Table 10-12](#)).

If the lungs slow down breathing rate, a condition termed hypoventilation, CO₂ will increase. Hypoventilation increases CO₂ retention. Normal PaCO₂ range is 35 to 45 mm Hg. Hypoventilation can increase the PaCO₂ greater than 45 mm Hg. Increased CO₂ will move the chemical equation to the right, which produces more H⁺ ions and an acidotic bloodstream. The normal pH of the bloodstream is 7.35 to 7.45. An acidotic bloodstream has a pH less than 7.35 (Larkin & Zimmanck, 2015; Norris, 2019).

If the lungs increase breathing rate, a condition called hyperventilation, CO₂ is lost via the lungs. If CO₂ decreases it moves the equation to the left, pulling H⁺ out of the bloodstream and creating an alkalotic bloodstream. An alkalotic bloodstream has a pH greater than 7.45 (Larkin & Zimmanck, 2015; Norris, 2019).

If H⁺ ions increase in the bloodstream due to lactic acid accumulation, DKA, toxicity, or other source, it will push the equation to the left and increase CO₂ to be eliminated by the lungs. The lungs will increase breathing rate to rid the body of CO₂ in attempt to compensate for the high acid (Larkin & Zimmanck, 2015; Theodore, 2019).

If H⁺ ions decrease in the bloodstream, it will pull the equation to the right using up some CO₂ to create more H⁺. The lungs will slow breathing rate in an attempt to compensate by accumulating CO₂, which will produce H⁺ (Larkin & Zimmanck, 2015; Theodore, 2019).

Acute and Chronic Metabolic Acidosis (Base Bicarbonate Deficit)

Metabolic acidosis is a common clinical disturbance characterized by a low pH due to an increased H⁺ concentration and a low plasma bicarbonate concentration. Metabolic acidosis can occur by a gain of hydrogen ions or a loss of bicarbonate ions in the bloodstream. It can be divided clinically into two forms, according to the values of the serum anion gap: high anion gap metabolic acidosis and normal anion gap metabolic acidosis. The anion gap refers to the difference between the sum of all measured positively charged electrolytes (cations) and the sum of all negatively charged electrolytes (anions) in blood. Because the sum of measured cations is typically greater than the sum of measured anions in the bloodstream, there normally exists a disparity with predominance of cations; this is referred to as the anion gap. The anion gap reflects unmeasured anions (phosphates, sulfates, and proteins) in plasma that replace bicarbonate in metabolic acidosis. Measuring the anion

gap is necessary when analyzing conditions of metabolic acidosis as it can help determine the cause of the acidosis (Emmett & Szerlip, 2018).

The anion gap can be calculated by either of the following equations:

$$\text{Anion gap} = \text{Na}^+ + \text{K}^+ - (\text{Cl}^- + \text{HCO}_3^-)$$

$$\text{Anion gap} = \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$$

Potassium is often omitted from the equation because of its low level in the plasma; therefore, the second equation is used more often than the first (Theodore, 2019).

The normal value for an anion gap is 8 to 12 mEq/L (8 to 12 mmol/L) without potassium in the equation. If potassium is included in the equation, the normal value for the anion gap is 12 to 16 mEq/L (12 to 16 mmol/L). The unmeasured anions in the serum normally account for less than 16 mEq/L of the anion production. Metabolic acidotic conditions can be differentiated according to the anion gap; there is either a normal anion gap or high anion gap. A person diagnosed with metabolic acidosis is determined to have normal anion gap metabolic acidosis if the anion gap is within this normal range (8 to 12 mEq/L). An anion gap greater than 16 mEq (16 mmol/L) suggests excessive accumulation of unmeasured anions and would indicate high anion gap metabolic acidosis. An anion gap occurs because not all electrolytes are measured. More anions are left unmeasured than cations (Emmett & Szerlip, 2018).

Pathophysiology

Normal anion gap metabolic acidosis results from the direct loss of bicarbonate, as in diarrhea, lower intestinal fistulas, ureterostomies, use of diuretics, early renal insufficiency, excessive administration of chloride, and the administration of parenteral nutrition without bicarbonate or bicarbonate-producing solutes (e.g., lactate) (Emmett & Szerlip, 2018).

High anion gap metabolic acidosis occurs when there is an excessive accumulation of acids. High anion gap occurs in lactic acidosis, salicylate poisoning (acetylsalicylic acid), renal failure, methanol, ethylene or propylene glycol toxicity, DKA, and ketoacidosis that occurs with starvation. The high amount of hydrogen ions due to the acids present are neutralized and buffered by HCO_3^- causing the bicarbonate concentration to fall and become exhausted. Other anions in the bloodstream are called upon to neutralize the high acid in the blood. In all of these instances, abnormally high levels of anions are used to neutralize the H^+ , which increases the anion gap above normal limits (high anion gap) (Emmett & Szerlip, 2019).



Concept Mastery Alert

Metabolic acidosis is characterized by a low pH and low plasma bicarbonate concentration. Nurses need to remember that an anion gap is calculated primarily to identify the cause (pathology) of metabolic acidosis.

	Reduced Negative Gap	or Normal Anion Gap Anion	High Anion Gap
Anion gap without potassium	<8	8–12 mEq/L	>12
Anion gap with potassium	<12	12–16 mEq/L	>16
Clinical significance	Hypoproteinemia	Normal anion gap metabolic acidosis	High anion gap metabolic acidosis

Clinical Manifestations

Signs and symptoms of metabolic acidosis vary with the severity of the acidosis but include headache, confusion, drowsiness, increased respiratory rate and depth, nausea, and vomiting. Peripheral vasodilation and decreased cardiac output occur when the pH drops to less than 7. Additional physical assessment findings include decreased blood pressure, cold and clammy skin, arrhythmias, and shock. Chronic metabolic acidosis is primarily caused by chronic kidney disease because dysfunctional kidneys do not excrete acid (Emmett & Szerlip, 2018; Kovesdy, 2018).

Assessment and Diagnostic Findings

ABG measurements are used in the diagnosis of acid–base imbalances such as metabolic acidosis. Expected ABG changes include a low bicarbonate level (less than 22 mEq/L) and a low blood pH (less than 7.35). The cardinal feature of metabolic acidosis is a decrease in the serum bicarbonate level. In conditions of acidosis there is elevated H⁺ and the sodium–potassium cellular pump brings H⁺ into the cells in place of K⁺. Therefore, high K⁺ accumulates in the bloodstream in metabolic acidosis as a result of the shift of potassium out of the cells (Theodore, 2019). Later, when the acidosis is corrected and pH normalized, the cellular pump causes potassium to move back into the cells and hypokalemia may occur. Blood levels of potassium need to be closely monitored. ECG monitoring is recommended as changes of potassium in the bloodstream can cause arrhythmias (Palmer & Clegg, 2016a).

In metabolic acidosis, the lungs compensate for the high H⁺ through hyperventilation to decrease the CO₂ level, which in turn reduces H⁺ (see

carbonic acid equation). Calculation of the anion gap is helpful in determining the cause of metabolic acidosis. There are certain conditions that cause high anion gap metabolic acidosis and others that cause normal anion gap metabolic acidosis (Table 10-13).

TABLE 10-13 Summary of Single Acid–Base Disturbances and Their Compensatory Responses

Acid–Base Imbalance	Primary Disturbance	Respiratory Compensation and Predicted Response ^a	Renal Compensation and Predicted Response ^{a,b}
Metabolic acidosis	↓ pH and HCO_3^- $\text{HCO}_3^- < 22 \text{ mEq/L}$	↑ ventilation and ↓ PCO_2 <i>1 mEq/L ↓ $\text{HCO}_3^- \rightarrow 1-1.2 \text{ mm Hg} \downarrow \text{PCO}_2$</i>	↑ H^+ excretion and ↑ HCO_3^- reabsorption if no renal disease
Metabolic alkalosis	↑ pH and HCO_3^- $\text{HCO}_3^- > 26 \text{ mEq/L}$	↓ ventilation and ↑ PCO_2 <i>1 mEq/L ↑ $\text{HCO}_3^- \rightarrow 0.7 \text{ mm Hg} \uparrow \text{PCO}_2$</i>	↓ H^+ excretion and ↓ HCO_3^- reabsorption if no renal disease
Respiratory acidosis	↓ pH and ↑ PCO_2 $\text{PCO}_2 > 45 \text{ mm Hg}$	None	↑ H^+ excretion and ↑ HCO_3^- reabsorption <i>Acute: 1 mm Hg ↑ $\text{PCO}_2 \rightarrow 0.1 \text{ mEq/L} \uparrow \text{HCO}_3^-$</i> <i>Chronic: 1 mm Hg ↑ $\text{PCO}_2 \rightarrow 0.3 \text{ mEq/L} \uparrow \text{HCO}_3^-$</i>
Respiratory alkalosis	↑ pH and ↓ PCO_2 $\text{PCO}_2 < 35 \text{ mm Hg}$	None	↓ H^+ excretion and ↓ HCO_3^- reabsorption <i>Acute: 1 mm Hg ↓ $\text{PCO}_2 \rightarrow 0.2 \text{ mEq/L} \downarrow \text{HCO}_3^-$</i> <i>Chronic: 1 mm Hg ↓ $\text{PCO}_2 \rightarrow 0.4 \text{ mEq/L} \downarrow \text{HCO}_3^-$</i>

Note: Predicted compensatory responses are in *italics*.

^aIf blood values are the same as predicted compensatory values, a single acid–base disorder is present; if values are different, a mixed acid–base disorder is present.

^bAcute renal compensation refers to duration of minutes to several hours; chronic renal compensation refers to a duration of several days.

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Medical Management

Treatment is directed at correcting the metabolic imbalance. If the problem results from excessive intake of chloride, treatment is aimed at eliminating the source of the chloride. When necessary, bicarbonate is given; however, the administration of sodium bicarbonate during cardiac arrest can result in

paradoxical intracellular acidosis. Hyperkalemia may occur with acidosis and hypokalemia with reversal of the acidosis and subsequent movement of potassium back into the cells. Therefore, the serum potassium level is monitored closely, and hypokalemia is corrected as acidosis is reversed (Mount, 2017c).

In chronic metabolic acidosis, low serum calcium levels are treated before the chronic metabolic acidosis is treated to avoid tetany resulting from an increase in pH and a decrease in ionized calcium. Alkalizing agents may be given. Treatment modalities may also include hemodialysis or peritoneal dialysis (Goltzman, 2019b).

Acute and Chronic Metabolic Alkalosis (Base Bicarbonate Excess)

Metabolic alkalosis is a clinical disturbance characterized by a high pH (decreased H⁺ concentration) and a high plasma bicarbonate concentration. It is caused by a gain of bicarbonate or a loss of H⁺ (Emmett & Szerlip, 2017a; Norris, 2019).

Pathophysiology

A common cause of metabolic alkalosis is severe vomiting or gastric suction that causes loss of stomach HCl (hydrogen and chloride ions). The disorder also occurs in pyloric stenosis, in which only gastric fluid is lost. Gastric fluid has an acid pH (usually 1 to 3), and loss of this highly acidic fluid pulls H⁺ ions from the bloodstream to replenish the gastric acid. As a result, the bloodstream loses H⁺ ions and becomes alkalotic. Other situations predisposing to metabolic alkalosis include those associated with loss of potassium, such as diuretic therapy that promotes excretion of potassium (e.g., thiazides, furosemide), and ACTH secretion (as in hyperaldosteronism and Cushing's syndrome) (Emmett & Szerlip, 2017a; Norris, 2019).

Hypokalemia produces alkalosis in two ways: (1) when the bloodstream is low in K⁺, the nephrons reabsorb K⁺ into the bloodstream and secrete H⁺ into the tubule fluid which is excreted in the urine and (2) when the bloodstream is low in K⁺, intracellular potassium moves out of the cells into the ECF, and as potassium ions leave the cells, hydrogen ions must enter to maintain electroneutrality (Mount, 2017c). Excessive alkali ingestion from antacids containing bicarbonate or from the use of sodium bicarbonate during cardiopulmonary resuscitation can also cause metabolic alkalosis (Emmett & Szerlip, 2017b).

Chronic metabolic alkalosis can occur with long-term diuretic therapy (thiazides or furosemide), villous adenoma in the GI tract, external drainage of gastric fluids, significant potassium depletion, cystic fibrosis, and the chronic ingestion of milk and calcium carbonate (Emmett & Szerlip, 2017b).

Clinical Manifestations

In alkalosis, H⁺ ions are decreased in the bloodstream, leaving negatively charged proteins attracting other positive ions. Calcium (Ca⁺⁺) ions bind to these proteins. As calcium ions bind to proteins in the bloodstream, free Ca⁺⁺ ions decrease in the bloodstream and hypocalcemia develops. Alkalosis is primarily manifested by symptoms related to hypocalcemia, such as tingling of the fingers and toes, dizziness, and tetany (cramping muscles). Because it is the ionized fraction of calcium that is diminished in metabolic alkalosis, neuromuscular symptoms due to hypocalcemia are often the predominant symptoms (Emmett & Szerlip, 2017c).

In metabolic alkalosis, the lungs attempt to compensate by slowing respiratory rate, which increases CO₂ retention, and in turn increases H⁺ content of the blood (see carbonic acid equation). If the kidneys are functional, there is increased renal excretion of HCO₃⁻ and conservation of H⁺ in an attempt to reduce the alkalinity of the bloodstream. As the pH of blood increases in metabolic alkalosis, H⁺ ions are reabsorbed into the bloodstream to neutralize the blood. As the nephrons increase H⁺ ion reabsorption, they excrete K⁺, and hypokalemia develops (Larkin & Zimmanck, 2015). In hypokalemia a prominent U wave often develops on the ECG and ventricular rhythm disturbances, such as PVCs, may occur. Hypokalemia also can lead to decreased GI motility and paralytic ileus (Emmett & Szerlip, 2017c).

Assessment and Diagnostic Findings

In metabolic alkalosis, evaluation of ABGs reveals a pH greater than 7.45 and a serum bicarbonate concentration greater than 26 mEq/L (see Table 10-12 for normal values of ABGs). The PaCO₂ increases as the lungs attempt to compensate for the excess bicarbonate by retaining CO₂. This hypoventilation is more pronounced in patients who are semiconscious, unconscious, or debilitated than in patients who are alert. Because of hypoventilation the patient may develop hypoxemia (Emmett & Szerlip, 2017c).

Urine chloride levels may help identify the cause of metabolic alkalosis if the patient's history provides inadequate information. Metabolic alkalosis is the setting in which urine chloride concentration may be a more accurate estimate of fluid volume than the urine sodium concentration. Urine chloride concentrations can help to determine the source of the metabolic alkalosis. Urine chloride concentrations can be used to differentiate between vomiting, diuretic therapy, and excessive

adrenocorticosteroid secretion as the cause of the metabolic alkalosis. In patients with vomiting or cystic fibrosis, those receiving nutritional repletion, and those receiving diuretic therapy, hypovolemia and hypochloremia produce urine chloride concentrations lower than 25 mEq/L. Signs of hypovolemia are not present, and the urine chloride concentration exceeds 40 mEq/L in patients with mineralocorticoid excess or alkali loading; these patients usually have expanded fluid volume (Emmett & Palmer, 2019).

Medical Management

Treatment of both acute and chronic metabolic alkalosis is aimed at correcting the underlying acid–base disorder. Because volume depletion is commonly present with GI losses of H⁺, the patient's I&O must be monitored carefully.

Treatment includes restoring normal fluid volume by administering normal saline because continued volume depletion perpetuates the alkalosis. In patients with hypokalemia, potassium is given as KCl to replace both K⁺ and Cl⁻ losses. Proton pump inhibitors (e.g., omeprazole) are recommended to reduce the production of gastric hydrogen chloride (HCl). This decreased HCl will in turn decrease the loss of HCl with gastric suction in metabolic alkalosis. Carbonic anhydrase inhibitors (e.g., acetazolamide) are useful in treating metabolic alkalosis in patients who cannot tolerate rapid volume expansion (e.g., patients with heart failure). Carbonic anhydrase inhibitors act at the nephron to enhance bicarbonate excretion (Mehta & Emmett, 2018).

Acute and Chronic Respiratory Acidosis (Carbonic Acid Excess)

Respiratory acidosis is a clinical disorder in which the pH is less than 7.35 and the PaCO₂ is greater than 45 mm Hg. It may be either acute or chronic.

Pathophysiology

Respiratory acidosis is due to inadequate excretion of CO₂ with inadequate ventilation, resulting in elevated plasma CO₂ concentrations

and, consequently, increased levels of carbonic acid. In addition to an elevated PaCO_2 , inadequate ventilation usually causes a decrease in PaO_2 . Acute respiratory acidosis occurs in emergency situations, such as acute pulmonary edema, aspiration of a foreign object, atelectasis, pneumothorax, and overdose of sedatives, as well as in nonemergent situations, such as sleep apnea associated with severe obesity, severe pneumonia, and acute respiratory distress syndrome. Respiratory acidosis commonly occurs in patients with severe chronic obstructive pulmonary disease (COPD) when patients acutely decompensate due to respiratory infection or heart failure. Respiratory acidosis can also occur in diseases that impair respiratory muscle function and cause hypoventilation. These disorders include severe scoliosis, muscular dystrophy, multiple sclerosis, myasthenia gravis, and Guillain-Barré syndrome (Feller-Kopman & Schwartzstein, 2017).

Clinical Manifestations

Clinical signs in acute and chronic respiratory acidosis vary. Acute respiratory acidosis can occur due to sudden hypercapnia (elevated PaCO_2) that will increase pulse, blood pressure, and respiratory rate. The patient may complain of confusion, disorientation, or may exhibit diminished level of consciousness. An elevated PaCO_2 , greater than 60 mm Hg causes reflexive cerebrovascular vasodilation and increased cerebral blood flow. Ventricular fibrillation may be the first sign of respiratory acidosis in anesthetized patients (Feller-Kopman & Schwartzstein, 2017).

If respiratory acidosis is severe, intracranial pressure may increase, resulting in papilledema and dilated conjunctival blood vessels. Acidosis can cause hyperkalemia as the hydrogen ion concentration overwhelms the compensatory mechanisms. Acidosis causes H^+ ion to move into the cells, causing a shift of potassium out of the cell. The bloodstream then gains increased potassium ions (i.e., hyperkalemia) (Mount, 2017c).

Chronic respiratory acidosis occurs with pulmonary diseases such as COPD, including emphysema and chronic bronchitis; obstructive sleep apnea; and obesity. As long as the PaCO_2 does not exceed the body's ability to compensate, the patient will be asymptomatic. However, if the PaCO_2 increases rapidly, reflexive cerebral vasodilation will increase intracranial pressure, and cyanosis and tachypnea will develop. Patients with slowly progressive COPD gradually accumulate CO_2 over a

prolonged period of time (months to years) and the body becomes used to high CO₂ levels. Patients with long-term COPD may not develop symptoms of hypercapnia because compensatory renal changes have had time to occur (Feller-Kopman & Schwartzstein, 2017).



Quality and Safety Nursing Alert

If the PaCO₂ is chronically greater than 50 mm Hg, the respiratory center becomes relatively insensitive to CO₂ as a respiratory stimulant, leaving hypoxemia as the major drive for respiration. Patients with long-term COPD breathe independently based on a hypoxic drive. High oxygen concentration administration can remove the stimulus of hypoxemia. The patient can lose the independent stimulus to breathe and incur respiratory failure. Therefore, oxygen is given with extreme caution in patients with long-term COPD.

Assessment and Diagnostic Findings

In respiratory acidosis, ABG analysis reveals a pH less than 7.35, a PaCO₂ greater than 45 mm Hg, and variation in the bicarbonate level, depending on the duration of the acute respiratory acidosis. When compensation occurs over a prolonged period and renal retention of bicarbonate has fully occurred, the bicarbonate neutralizes the acidosis. Arterial pH is within the lower limits of normal (e.g., pH 7.35). Depending on the cause of respiratory acidosis, other diagnostic measures include monitoring of serum electrolyte levels, chest x-ray for determining respiratory infection or other disease, and a drug screen if an overdose is suspected. ECG monitoring is recommended to identify any cardiac involvement as a result of COPD (Feller-Kopman & Schwartzstein, 2017).

Medical Management

Treatment is directed at improving ventilation in acute and chronic respiratory acidosis. Exact measures vary according to the cause of inadequate ventilation. Pharmacologic agents are commonly used. For example, bronchodilators help reduce bronchial spasm and increase ventilation, antibiotics are used for respiratory infections, and

thrombolytics or anticoagulants are used for pulmonary emboli (Stoller, 2019).

Pulmonary physiotherapy and nebulizer treatment can be used to clear the respiratory tract of mucus and purulent drainage. Adequate hydration (2 to 3 L/day) is indicated to keep the mucous membranes moist and decrease viscosity of mucus, thereby facilitating removal of secretions. Low concentration of supplemental oxygen is given as necessary (Aboussouan, 2018).

Mechanical ventilation, used appropriately, may be necessary to improve pulmonary ventilation. PaCO_2 should be reduced slowly and gradually using a mechanical ventilator. Mechanical ventilation can cause too rapid ventilatory loss of CO_2 , which pulls H^+ out of the bloodstream too rapidly. If there is a rapid loss of H^+ the bloodstream becomes too alkalotic. The kidneys are unable to eliminate bicarbonate quickly enough to prevent alkalosis and seizures (Feller-Kopman & Schwartzstein, 2017).

Acute and Chronic Respiratory Alkalosis (Carbonic Acid Deficit)

Respiratory alkalosis is a clinical condition in which the arterial pH is greater than 7.45 and the PaCO_2 is less than 35 mm Hg. As with respiratory acidosis, acute and chronic conditions can cause this acid–base disturbance.

Pathophysiology

Respiratory alkalosis is caused by hyperventilation, which causes excessive loss or “blowing off” of CO_2 and, hence, there is a decrease in the plasma carbonic acid concentration (see carbonic acid equation). Causes include extreme anxiety such as panic disorder, hypoxemia, salicylate intoxication, gram-negative sepsis, and inappropriate ventilator settings.

Chronic respiratory alkalosis results from chronic hypocapnia which leads to decreased serum H^+ ion, resulting in alkalosis. Chronic hepatic insufficiency and cerebral tumors can cause chronic hyperventilation that leads to chronic respiratory alkalosis (Schwartzstein, Richards, Edlow, et al., 2018).

Clinical Manifestations

Clinical signs of respiratory alkalosis consist of lightheadedness and inability to concentrate due to cerebral artery vasoconstriction and decreased cerebral blood flow, numbness and tingling from decreased calcium ionization in the bloodstream, tinnitus, and sometimes loss of consciousness. Cardiac effects of respiratory alkalosis include tachycardia and ventricular and atrial arrhythmias (Schwartzstein et al., 2018).

Assessment and Diagnostic Findings

Analysis of ABGs assists in the diagnosis of both acute and chronic respiratory alkalosis. In the acute state, the pH is elevated above normal (greater than 7.45) as a result of a low PaCO₂ and a normal bicarbonate level. The kidneys take days to compensate for acid–base imbalances. Therefore, the kidneys cannot alter the bicarbonate level in the bloodstream quickly enough and medical intervention is necessary (Norris, 2019).

In the compensated state of chronic respiratory alkalosis, the kidneys have had sufficient time to lower the bicarbonate level to a near-normal level. Evaluation of serum electrolytes is indicated to identify any decrease in potassium, as hydrogen is pulled out of the cells in exchange for potassium. A decreased calcium level may occur as severe alkalosis inhibits calcium ionization, resulting in carpopedal spasms and tetany. A decreased phosphate level can occur due to alkalosis because there is increased uptake of phosphate by the cells. A toxicology screen should be performed to rule out salicylate intoxication due to aspirin poisoning (Schwartzstein et al., 2018).

Medical Management

Treatment depends on the exact underlying cause of respiratory alkalosis. If the cause is anxiety, the patient is instructed to breathe more slowly to allow CO₂ to accumulate or to breathe into a closed system (such as a paper bag or CO₂ rebreather mask). An antianxiety agent may be required to relieve hyperventilation in very anxious patients. Treatment of other causes of respiratory alkalosis is directed at correcting the underlying problem.

Mixed Acid–Base Disorders

Patients can simultaneously experience two or more independent acid–base disorders. A normal pH in the presence of changes in the PaCO_2 and plasma HCO_3^- concentration immediately suggests a mixed disorder. An example of a mixed disorder is the simultaneous occurrence of metabolic acidosis due to lactic acid accumulation and respiratory acidosis due to hypoventilation. Both of these disorders result in excessive acid accumulation in the bloodstream due to respiratory failure and cardiac arrest (Emmett & Palmer, 2018b).

Compensation

Generally, the pulmonary and renal systems compensate for each other to return the pH to normal. In a single acid–base disorder, the system not causing the problem tries to compensate by returning the ratio of bicarbonate to carbonic acid to the normal 20:1. The lungs compensate for metabolic disturbances by changing CO_2 excretion; hypoventilation accumulates CO_2 , hyperventilation causes loss of CO_2 . The kidneys compensate for respiratory disturbances by altering bicarbonate reabsorption and H^+ secretion (Norris, 2019; Theodore, 2019).

In respiratory acidosis, excess hydrogen in the blood is excreted in the urine in exchange for bicarbonate ions which are conserved. In respiratory alkalosis, the renal excretion of bicarbonate increases, and hydrogen ions are retained. In metabolic acidosis, the lungs compensate by increasing the ventilation rate and the kidneys retain bicarbonate. In metabolic alkalosis, the respiratory system compensates by decreasing ventilation to conserve CO_2 and increase the PaCO_2 , which in turn increases carbonic acid. Because the lungs respond to acid–base disorders within minutes, compensation for metabolic imbalances occurs faster than renal compensation for respiratory imbalances (Norris, 2019; Theodore, 2019).

Table 10-13 summarizes compensation effects.

Blood Gas Analysis

Blood gas analysis is often used to identify the specific acid–base disturbance and the degree of compensation that has occurred. The analysis is usually based on an arterial blood sample; however, if an arterial sample cannot be obtained, a mixed venous sample may be used

(Theodore, 2019). Results of ABG analysis provide information about alveolar ventilation, oxygenation, and acid–base balance. It is necessary to evaluate the concentrations of serum electrolytes (e.g., sodium, potassium, chloride) along with ABG data because electrolytes are commonly affected by acid–base imbalances. The health history, physical examination, previous blood gas results, and serum electrolytes should always be part of the assessment used to determine the cause of the acid–base disorder (Larkin & Zimmanck, 2015). Responding to isolated sets of blood gas results without these data can lead to serious errors in interpretation. Treatment of the underlying condition usually corrects acid–base disorders ([Chart 10-3](#)).

PARENTERAL FLUID THERAPY

When patients cannot take oral fluid or oral feedings, their status is termed NPO (*nil per os*), meaning nothing by mouth. In patients who are NPO, **parenteral fluid therapy**, also termed IV fluid therapy, is used to administer fluids. IV fluid therapy can be initiated to replace fluids in various clinical settings such as hospitals, outpatient diagnostic and surgical settings, clinics, and home health care. IV fluids can also be used to administer medications and provide nutrients.

Purpose

The choice of an IV solution depends on the purpose of its administration. Generally, IV fluids are given to achieve one or more of the following goals:

- To provide water, electrolytes, and nutrients to meet daily requirements
- To replace water and correct electrolyte deficits
- To administer medications and blood products

IV solutions contain dextrose and/or electrolytes mixed in various proportions with water. Pure, electrolyte-free water can never be given by IV because it rapidly enters RBCs and causes them to rupture (Sterns, 2017b).

Types of Intravenous Solutions

IV solutions are categorized as isotonic, hypotonic, or hypertonic, according to whether their total osmolality is the same as, less than, or greater than that of blood, respectively (see earlier discussion of osmolality and tonicity). Electrolyte solutions are considered isotonic if the total electrolyte content (anions + cations) is between 250 and 375 mEq/L, hypotonic if the total electrolyte content is less than 250 mEq/L, and hypertonic if the total electrolyte content is greater than 375 mEq/L. The nurse must also consider a solution's osmolality, keeping in mind that the osmolality of plasma is approximately 300 mOsm/L (300 mmol/L). For example, a 10% dextrose solution has an osmolality of approximately 505 mOsm/L, which is greater than the osmolality of the bloodstream (Emmett & Palmer, 2018a).

Isotonic Fluids

Fluids that are classified as isotonic have a total osmolality close to that of the ECF and do not cause cells to shrink or swell. When isotonic fluids are administered they expand the ECF volume. One liter of isotonic fluid expands the ECF by 1 L; however, isotonic solution expands the plasma component of ECF by only 0.25 L. An isotonic solution is a crystalloid solution (water containing soluble mineral salts). Plasma is a colloidal solution. A colloidal solution is a mixture of fluid containing insoluble large particles, such as proteins. Colloidal solutions exert oncotic pressure; crystalloids do not exert oncotic pressure (Siparsky, 2019; Sterns, 2018a).



Quality and Safety Nursing Alert

It is important for the nurse to recognize that in blood loss, 3 L of isotonic fluid (crystalloid solution) is needed to replace 1 L of blood (colloidal solution).

Because isotonic fluids expand the water volume in the intravascular space, patients with heart failure or hypertension who receive isotonic solutions should be carefully monitored for signs of fluid overload (Siparsky, 2019).

Chart 10-3



ASSESSMENT

Assessing Arterial Blood Gases

The following steps are recommended to evaluate arterial blood gas values. They are based on the assumption that the average values are:

$$\text{pH} = 7.35\text{--}7.45$$

$$\text{PaCO}_2 = 35\text{--}45 \text{ mm Hg}$$

$$\text{HCO}_3^- = 24 \text{ to } 27 \text{ mEq/L}$$

1. *First, note the pH.* It can be high, low, or normal, as follows:

A normal pH may indicate perfectly normal blood gases, or it may indicate a *compensated* imbalance. A compensated imbalance is one in which the body has been able to correct the pH by either respiratory or metabolic changes (depending on the primary problem). For example, a patient with primary metabolic acidosis starts out with a low bicarbonate level but a normal CO₂ level. Soon afterward, the lungs try to compensate for the imbalance by exhaling large amounts of CO₂ (hyperventilation). As another example, a patient with primary respiratory acidosis starts out with a high CO₂ level; soon afterward, the kidneys attempt to compensate by retaining bicarbonate. If the compensatory mechanism is able to restore the bicarbonate to carbonic acid ratio back to 20:1, full compensation (and thus normal pH) will be achieved.

$$\text{pH} > 7.45 \text{ (alkalosis)}$$

$$\text{pH} < 7.35 \text{ (acidosis)}$$

$$\text{pH} = 7.4 \text{ (normal)}$$

2. The next step is to determine the primary cause of the disturbance. This is done by evaluating the PaCO₂ and HCO₃⁻ in relation to the pH.

Example: pH > 7.45 (alkalosis)

- If the PaCO₂ is less than 35 mm Hg, the primary disturbance is respiratory alkalosis. (This situation occurs when a patient hyperventilates and “blows off” too much CO₂. Recall that CO₂ dissolved in water becomes carbonic acid, the acid side of the “carbonic acid–bicarbonate buffer system.”)
- If the HCO₃⁻ is greater than 27 mEq/L, the primary disturbance is metabolic alkalosis. (This situation occurs when the body gains too much bicarbonate, an alkaline substance. Bicarbonate is the basic or alkaline side of the “carbonic acid–bicarbonate buffer system.”)

Example: pH < 7.35 (acidosis)

- If the PaCO₂ is greater than 40 mm Hg, the primary disturbance is respiratory acidosis. (This situation occurs when a patient

- hypoventilates and thus retains too much CO₂, an acidic substance.)
- d. If the HCO₃⁻ is less than 24 mEq/L, the primary disturbance is metabolic acidosis. (This situation occurs when the body's bicarbonate level drops, either because of direct bicarbonate loss or because of gains of acids such as lactic acid or ketones.)
3. The next step involves determining if compensation has begun. This is done by looking at the value other than the primary disorder. If it is moving in the same direction as the primary value, compensation is under way. Consider the following gases:
- The first set (1) indicates acute respiratory acidosis without compensation (the PaCO₂ is high, the HCO₃⁻ is normal). The second set (2) indicates chronic respiratory acidosis. Note that compensation has taken place—that is, the HCO₃⁻ has elevated to an appropriate level to balance the high PaCO₂ and produce a normal pH.
- | | pH | PaCO ₂ | HCO ₃ ⁻ |
|-----|-----|-------------------|-------------------------------|
| (1) | 7.2 | 60 mm Hg | 24 mEq/L |
| (2) | 7.4 | 60 mm Hg | 37 mEq/L |
4. Two distinct acid–base disturbances may occur simultaneously. These can be identified when the pH does not explain one of the changes. When the PaCO₂ is ↑ and the HCO₃ is ↓, respiratory acidosis and metabolic acidosis coexist. When the PaCO₂ is ↓ and the HCO₃ is ↑, respiratory alkalosis and metabolic alkalosis coexist.
- Example: Metabolic and respiratory acidosis**
- a. pH 7.2 decreased pH (indicates acidosis)
 - b. PaCO₂ 52 increased pH (indicates respiratory acidosis)
 - c. HCO₃ 13 decreased HCO₃ (indicates metabolic acidosis)
5. If metabolic acidosis exists, then calculate the anion gap (AG) to determine the cause of the metabolic acidosis (AG vs. non-AG):
- $$AG = Na - (Cl + HCO_3^-)$$
- Normal AG = 10–14 mmol/L
6. Evaluate the patient to determine if the clinical signs and symptoms are compatible with the acid–base analysis.

Adapted from Fischbach, F., & Fischbach, M. (2018). *A manual of laboratory and diagnostic tests* (10th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

A solution of D₅W is unique in that it may be both isotonic and hypotonic (Hoorn, 2017). Once given, the glucose is rapidly metabolized, and this initially isotonic solution (same osmolality as serum) then disperses as a hypotonic fluid—one third extracellular and two thirds intracellular. It is essential to consider this action of D₅W, especially if the patient is at risk for increased intracranial pressure. During fluid resuscitation, this solution should not be used because hyperglycemia can result. Therefore, D₅W is used mainly to supply water and to correct an increased serum osmolality. About 1 L of D₅W provides less than 170 kcal and is a minor source of the body's daily caloric requirements (Hoorn, 2017).

Normal Saline Solution

Normal saline (0.9% sodium chloride) solution contains water, sodium, and chloride. Because the osmolality is entirely contributed by electrolytes, the solution remains within the ECF and expands the intravascular volume. For this reason, normal saline solution is often used to correct an extracellular volume deficit but is not identical to ECF. It is used with administration of blood transfusions and to replace large sodium losses, such as in burn injuries. It should not be used in heart failure, pulmonary edema, renal impairment, or sodium retention. Normal saline does not supply calories (Hoorn, 2017).

Other Isotonic Solutions

Several other solutions contain ions in addition to sodium and chloride and are somewhat similar to the ECF in composition. Lactated Ringer's solution contains potassium and calcium in addition to sodium chloride. It is used to correct dehydration, blood loss, and sodium depletion and to replace GI losses.

Hypotonic Fluids

One purpose of hypotonic solution is to replace fluid, because it is hypotonic compared with plasma. Another purpose of hypotonic solution is to provide free water. At times, hypotonic sodium solutions are used to treat hypernatremia and other hyperosmolar conditions. Half-strength saline (0.45% sodium chloride) solution is frequently used.

Hypertonic Fluids

Hypertonic fluids include 3% NaCl and IV mannitol. If a patient is sodium depleted, a hypertonic sodium IV solution might be used. If a patient is experiencing acute cerebral edema, IV mannitol is often used. Hypertonic solutions pull water from the interstitial and intracellular compartments into the bloodstream. These solutions draw water out of intracellular compartments causing cellular dehydration (Hoorn, 2017). Normal saline and lactated Ringer's solution are considered isotonic solutions. When 5% dextrose (D_5W) is added to normal saline solution or lactated Ringer's solution, the total osmolality exceeds that of the ECF. With the added dextrose, these are then considered hypertonic solutions. However, the dextrose is quickly metabolized, and after the dextrose is depleted, only the isotonic solution remains. Therefore, any effect on the intracellular compartment is temporary. Similarly, with hypotonic electrolyte solutions containing 5% dextrose, once the dextrose is metabolized, these solutions disperse as hypotonic fluids. However, higher concentrations of dextrose, such as 50% dextrose ($D_{50}W$) in water, are strongly hypertonic. Hypertonic solutions should be administered into central veins so that they can be diluted by large amounts of rapid blood flow (Hoorn, 2017).

Saline solutions are also available in osmolar concentrations greater than that of the ECF. These solutions draw water from the ICF to the ECF and cause cells to shrink. If given rapidly or in large quantity, they may cause an extracellular volume excess and precipitate circulatory overload and dehydration. As a result, these solutions must be given cautiously and usually only when the serum osmolality has decreased to dangerously low levels. Hypertonic solutions exert an osmotic pressure greater than that of the ECF (Hoorn, 2017).

Other Intravenous Therapies

When the patient is unable to tolerate food, nutritional requirements are often met using the IV route. Solutions may include high concentrations of glucose (such as 50% dextrose in water), protein, or fat to meet nutritional requirements (see [Chapter 41](#)). The IV route may also be used to administer colloids, plasma expanders, and blood products (Hoorn, 2017). Examples of blood products include whole blood, packed RBCs, fresh-frozen plasma, albumin, and cryoprecipitate (these are discussed in more detail in [Chapter 28](#)).

Many medications are also delivered by the IV route, either by continuous infusion or by intermittent bolus directly into the vein. Because IV medications enter the circulation rapidly, administration by this route is potentially hazardous. All medications can produce adverse reactions; however, medications given by the IV route can cause these reactions quickly after administration, because the medications are delivered directly into the bloodstream. Administration rates and recommended dilutions for individual medications are available in specialized texts pertaining to IV medications and in manufacturers' package inserts; these should be consulted to ensure safe IV administration of medications (Institute for Safe Medication Practices [ISMP], 2019).



Quality and Safety Nursing Alert

The nurse must assess the patient for a history of allergic reactions to medications. Although obtaining drug allergy information is important when administering any medication, it is especially critical with IV administration, because the medication is delivered directly into the bloodstream. This can trigger an immediate hypersensitivity reaction.

Nursing Management of the Patient Receiving Intravenous Therapy



In many settings, the ability to perform venipuncture to gain access to the venous system for administering fluids and medication is an expected nursing skill. This responsibility includes selecting the appropriate venipuncture site and type of cannula and being proficient in the technique of vein entry. The nurse should demonstrate competency in and knowledge of IV catheter placement according to the Nurse Practice Act applicable in their state and should follow the rules and regulations, organizational policies and procedures, and practice guidelines of that state's board of nursing (Gorski, Hadaway, Hagle, et al., 2016).

Managing Systemic Complications

Fluid Overload

Overloading the circulatory system with excessive IV fluids causes increased blood pressure and central venous pressure. Signs and symptoms of fluid overload include moist crackles on auscultation of the lungs, cough, restlessness, distended neck veins, edema, weight gain, dyspnea, and rapid, shallow respirations. Possible causes include rapid infusion of an IV solution or hepatic, cardiac, or renal disease. The risk of fluid overload and subsequent pulmonary edema is especially increased in older patients with cardiac disease; this is referred to as circulatory overload. Its treatment includes decreasing the IV rate, monitoring vital signs frequently, assessing breath sounds, and placing the patient in a high Fowler position. The primary provider is contacted immediately. This complication can be avoided by using an infusion pump and by carefully monitoring all infusions. Complications of circulatory overload include heart failure and pulmonary edema (Connelly, 2018).

Air Embolism

The risk of air embolism is rare but ever-present. It is most often associated with cannulation of central veins and directly related to the size of the embolus and the rate of entry. Air entering into central veins gets to the right ventricle, where it lodges against the pulmonary valve and blocks the flow of blood from the ventricle into the pulmonary arteries. Manifestations of air embolism include palpitations, dyspnea, continued coughing, jugular venous distention, wheezing, and cyanosis; hypotension; weak, rapid pulse; altered mental status; and chest, shoulder, and low back pain. Treatment calls for immediately clamping the cannula and replacing a leaking or open infusion system, placing the patient on the left side in the Trendelenburg position, assessing vital signs and breath sounds, and administering oxygen. Air embolism can be prevented by using locking adapters on all lines, filling all tubing completely with solution, and using an air detection alarm on an IV infusion pump. Complications of air embolism include shock and death. The amount of air necessary to induce death in humans is not known; however, the rate of entry is probably as important as the actual volume of air (Malik, Claus, Illman, et al., 2017).

Infection

Pyogenic substances in either the infusion solution or the IV administration set can cause bloodstream infections. Signs and symptoms include an abrupt temperature elevation shortly after the infusion is started, backache, headache, increased pulse and respiratory rate, nausea and vomiting, diarrhea, chills and shaking, and general malaise. Additional symptoms include erythema, edema, and induration or drainage at the insertion site. In sepsis, vascular collapse and septic shock may occur (Connelly, 2018). (See [Chapter 11](#) for a discussion of septic shock.)

Infection ranges in severity from local involvement of the insertion site to systemic dissemination of organisms through the bloodstream, as in sepsis. Measures to prevent infection are essential at the time the IV line is inserted and throughout the entire infusion (Hugill, 2017).

Managing Local Complications

Local complications of IV therapy include phlebitis, infiltration and extravasation, thrombophlebitis, hematoma, and clotting of the needle (Simin, Milutinović, Turkulov, et al., 2019). [Chart 10-4](#) provides a Nursing Research Profile about complications of peripheral IVs.

Phlebitis

Phlebitis, or inflammation of a vein, can be categorized as chemical, mechanical, or bacterial; however, two or more of these types of irritation often occur simultaneously. Chemical phlebitis can be caused by an irritating medication or solution (increased pH or high osmolality of a solution), rapid infusion rates, and medication incompatibilities. Mechanical phlebitis results from long periods of cannulation, catheters in flexed areas, catheter gauges larger than the vein lumen, and poorly secured catheters. Bacterial phlebitis can develop from poor hand hygiene, lack of aseptic technique, failure to check all equipment before use, and failure to recognize early signs and symptoms of phlebitis. Other factors include poor venipuncture technique, catheter in place for a prolonged period, and failure to adequately secure the catheter (Hugill, 2017). Phlebitis is characterized by a reddened, warm area around the insertion site or along the path of the vein, pain or tenderness at the site or along the vein, and swelling. The incidence of phlebitis increases with the length of time the IV line is in place, the composition of the fluid or medication infused (especially its pH and tonicity), catheter material, emergency insertions, the size and site of the cannula inserted,

ineffective filtration, inadequate anchoring of the line, and the introduction of microorganisms at the time of insertion (Mihala, Ray-Barruel, Chopra, et al., 2018).

Chart 10-4



NURSING RESEARCH PROFILE

Complication Rates of Peripheral IVs

Simin, D., Milutinović, D., Turkulov, V., et al. (2019). Incidence, severity and risk factors of peripheral intravenous cannula-induced complications: An observational prospective study. *Journal of Clinical Nursing*, 28(9–10), 1585–1599.

Purpose

The incidence and severity of complications of peripheral IV insertion is not known. The purpose of this study was to determine the incidence, severity, and risk factors of peripheral IV cannula-induced complications in hospitalized adult patients.

Design

This was an observational prospective study. Observations were made of 1428 IV insertions among 368 adult patients hospitalized in a tertiary medical center.

Findings

Phlebitis was the most common complication, with a rate of 44%, followed by infiltration with a rate of 16%, while the incidence of occlusion and catheter dislodgement were 7.6% and 5.6%, respectively. The risk factors for phlebitis were identified as the presence of a comorbidity (i.e., diabetes), a current infection, and an indwelling urinary catheter. The frequency of infiltration increased with the age of the patient, the number of attempts at the IV insertion in the same anatomic site, and the number of medications and IV solutions administered per day.

Nursing Implications

Medical-surgical nurses assess for all complications following IV insertion but should be particularly alert for phlebitis, which is the most common. This study provides evidence that older patients are at higher risk of infiltration and that attempting multiple IV sticks in the same anatomic site may lead to higher complication rates. The nurse should also be aware that patients who have comorbidities (such as diabetes) and those who are receiving multiple medications and IV solutions are at higher risk for IV complications.

Chart 10-5 ASSESSMENT



Assessing for Phlebitis

Grade	Clinical Criteria
0	No clinical symptoms
1	Erythema at access site with or without pain
2	Pain at access site Erythema, edema, or both
3	Pain at access site Erythema, edema, or both Streak formation Palpable venous cord
4	Pain at access site with erythema Streak formation Palpable venous cord (longer than 1 inch) Purulent drainage

Adapted from Gorski, L. A., Hadaway, L., Hagle, M., et al. (2016). Infusion therapy standards of practice. *Journal of Infusion Nursing*, 39(1 Suppl), S1–S159.

The Infusion Nurses Society (INS) has identified specific standards for assessing phlebitis (Gorski et al., 2016); these appear in [Chart 10-5](#). Phlebitis is graded according to the most severe presenting indication.

Treatment consists of discontinuing the IV line and restarting it in another site, and applying a warm, moist compress to the affected site (INS, 2016). Phlebitis can be prevented by using aseptic technique during insertion, using the appropriate-size cannula or needle for the vein, considering the composition of fluids and medications when selecting a site, observing the site hourly for any complications, anchoring the cannula or needle well, and changing the IV site according to agency policy and procedures (Mihala et al., 2018).

Infiltration and Extravasation

Infiltration is the unintentional administration of a nonvesicant solution or medication into surrounding tissue. This can occur when the IV cannula dislodges or perforates the wall of the vein. Infiltration is characterized by edema around the insertion site, leakage of IV fluid from the insertion site, discomfort and coolness in the area of infiltration, and a significant decrease in the flow rate. When the solution is particularly irritating, sloughing of tissue may result. Close monitoring

of the insertion site is necessary to detect infiltration before it becomes severe (Nickel, 2019; Simin et al., 2019).

Infiltration is usually easily recognized if the insertion area is larger than the same site of the opposite extremity but is not always so obvious. A common misconception is that a backflow of blood into the tubing proves that the catheter is properly placed within the vein. However, if the catheter tip has pierced the wall of the vessel, IV fluid will seep into tissues and flow into the vein. Although blood return occurs, infiltration may have occurred as well. A more reliable means of confirming infiltration is to apply a tourniquet above (or proximal to) the infusion site and tighten it enough to restrict venous flow. If the infusion continues to drip despite the venous obstruction, infiltration is present.

As soon as the nurse detects infiltration, the infusion should be stopped, the IV catheter removed, and a sterile dressing applied to the site after careful inspection to determine the extent of infiltration. The infiltration of any amount of blood product, irritant, or vesicant is considered the most severe (Brooks, 2018; Odom, Lowe, & Yates, 2018).

The IV infusion should be started in a new site or proximal to the infiltration site if the same extremity must be used again. A warm compress may be applied to the site if small volumes of noncaustic solutions have infiltrated over a long period, or if the solution was isotonic with a normal pH; the affected extremity should be elevated to promote the absorption of fluid. If the infiltration is recent and the solution was hypertonic or had an increased pH, a cold compress may be applied to the area (Gorski et al., 2016; Odom et al., 2018).

Infiltration can be detected and treated early by inspecting the site every hour for redness, pain, edema, blood return, coolness at the site, and IV fluid leaking from the IV site. Using the appropriate size and type of cannula for the vein prevents this complication. The use of electronic infusion devices (EIDs) does not cause an infiltration or extravasation; however, these devices will exacerbate the problem until the infusion is turned off. The Infusion Nursing Standards of Practice state that a standardized infiltration scale should be used to document the infiltration (Brooks, 2018; Gorski et al., 2016) ([Chart 10-6](#)).

Extravasation is similar to infiltration, with an inadvertent administration of vesicant or irritant solution or medication into the surrounding tissue. Medications such as vasopressors, potassium and calcium preparations, and chemotherapeutic agents can cause pain, burning, and redness at the site. Blistering, inflammation, and necrosis of tissues can occur. Older patients, comatose or anesthetized patients,

patients with diabetes, and patients with peripheral vascular or cardiovascular disease are at greater risk for extravasation; other risk factors include high pressure infusion pumps, palpable cording of vein, and fluid leakage from the insertion site. The extent of tissue damage is determined by the concentration of the medication, the quantity that extravasated, the location of the infusion site, the tissue response, and the duration of the process of extravasation (Gorski et al., 2016; Odom et al., 2018).

Chart 10-6 ASSESSMENT

Assessing for Infiltration

Grade Clinical Criteria

- | | |
|---|---|
| 0 | No clinical symptoms |
| 1 | Skin blanched, edema less than 1 inch in any direction, cool to touch, with or without pain |
| 2 | Skin blanched, edema 1 to 6 inches in any direction, cool to touch, with or without pain |
| 3 | Skin blanched, translucent, gross edema greater than 6 inches in any direction, cool to touch, mild to moderate pain, possible numbness |
| 4 | Skin blanched, translucent, skin tight, leaking, skin discolored, bruised, swollen, gross edema greater than 6 inches in any direction, deep pitting tissue edema, circulatory impairment, moderate to severe pain, infiltration of any amount of blood products, irritant, or vesicant |

Adapted from Gorski, L. A., Hadaway, L., Hagle, M., et al. (2016). Infusion therapy standards of practice. *Journal of Infusion Nursing*, 39(1 Suppl), S1–S159.

When extravasation occurs, the infusion must be stopped and the provider notified promptly. The agency's protocol to treat extravasation is initiated; the protocol may specify specific treatments, including antidotes specific to the medication that extravasated, and may indicate whether the IV line should remain in place or be removed before treatment. The protocol often specifies infiltration of the infusion site with an antidote prescribed after assessment by the provider, removal of the cannula, and application of warm compresses to sites of extravasation from alkaloids or cold compresses to sites of extravasation

from alkylating and antibiotic vesicants. The affected extremity should not be used for further cannula placement. Thorough neurovascular assessments of the affected extremity must be performed frequently (Gorski et al., 2016).

Reviewing the institution's IV policy and procedures and incompatibility charts and checking with the pharmacist before administering any IV medication, whether peripherally or centrally, are recommended to determine incompatibilities and vesicant potential to prevent extravasation. Careful, frequent monitoring of the IV site, avoiding insertion of IV devices in areas of flexion, securing the IV line, and using the smallest catheter possible that accommodates the vein help minimize the incidence and severity of this complication. In addition, when vesicant medication is given by IV push, it should be given through a side port of an infusing IV solution to dilute the medication and decrease the severity of tissue damage if extravasation occurs. Extravasation is rated as grade 4 on the infiltration scale. Complications of an extravasation may include blister formation, skin sloughing and tissue necrosis, functional or sensory loss in the injured area, and disfigurement or loss of limb (Gorski et al., 2016; Nickel, 2019).

Thrombophlebitis

Thrombophlebitis refers to the presence of a clot plus inflammation in the vein. It is evidenced by localized pain, redness, warmth, and swelling around the insertion site or along the path of the vein, immobility of the extremity because of discomfort and swelling, sluggish flow rate, fever, malaise, and leukocytosis (Brooks, 2018).

Treatment includes discontinuing the IV infusion; applying a cold compress first to decrease the flow of blood, followed by a warm compress; elevating the extremity; and restarting the line in the opposite extremity. If the patient has signs and symptoms of thrombophlebitis, the IV line should not be flushed (although flushing may be indicated in the absence of phlebitis to ensure cannula patency and to prevent mixing of incompatible medications and solutions). The catheter should be cultured after the skin around the catheter is cleaned with alcohol. If purulent drainage exists, the site is cultured before the skin is cleaned (Brooks, 2018; Hugill, 2017).

Thrombophlebitis can be prevented by avoiding trauma to the vein at the time the IV line is inserted, observing the site every hour, and checking medication additives for compatibility (Gorski et al., 2016).

Hematoma

Hematoma results when blood leaks into tissues surrounding the IV insertion site. Leakage can result if the vein wall is perforated during venipuncture, the needle slips out of the vein, a cannula is too large for the vessel, or insufficient pressure is applied to the site after removal of the needle or cannula. The signs of a hematoma include ecchymosis, immediate swelling at the site, and leakage of blood at the insertion site.

Treatment includes removing the needle or cannula and applying light pressure with a sterile, dry dressing; applying ice for 24 hours to the site to avoid extension of the hematoma; elevating the extremity to maximize venous return, if tolerated; assessing the extremity for any circulatory, neurologic, or motor dysfunction; and restarting the line in the other extremity if indicated. A hematoma can be prevented by carefully inserting the needle and by frequently monitoring patients who have a bleeding disorder, are taking anticoagulant medication, or have advanced liver disease (Nickel, 2019).

Clotting and Obstruction

Blood clots may form in the IV line as a result of kinked IV tubing, a very slow infusion rate, an empty IV bag, or failure to flush the IV line after intermittent medication or solution administrations. The signs are decreased flow rate and blood backflow into the IV tubing (Brooks, 2018).

If blood clots in the IV line, the infusion must be discontinued and restarted in another site with a new cannula and administration set. The tubing should not be irrigated or milked. Neither the infusion rate nor the solution container should be raised, and the clot should not be aspirated from the tubing. Clotting of the needle or cannula may be prevented by not allowing the IV solution bag to run dry, taping the tubing to prevent kinking and maintain patency, maintaining an adequate flow rate, and flushing the line after intermittent medication or other solution administration. In some cases, a specially trained nurse or primary provider may inject a thrombolytic agent into the catheter to clear an occlusion resulting from fibrin or clotted blood (Brooks, 2018; Gorski et al., 2016).

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

At times, IV therapy must be given in the home setting, in which case much of the daily management rests with the patient and family. Education becomes essential to ensure that the patient and family can manage the IV fluid and infusion correctly and avoid complications. Written instructions as well as demonstration and return demonstration help reinforce the key points for all of these functions (Payne, 2019).

Continuing and Transitional Care

Home infusion therapies cover a wide range of treatments, including antibiotic, analgesic, and antineoplastic medications; blood or blood component therapy; and parenteral nutrition. When direct nursing care is necessary, arrangements are made to have an infusion nurse visit the home and administer the IV therapy as prescribed. In addition to implementing and monitoring the IV therapy, the nurse carries out a comprehensive assessment of the patient's condition and continues to educate the patient and family about the skills involved in overseeing the IV therapy setup. Any dietary changes that may be necessary because of fluid or electrolyte imbalances are explained or reinforced during such sessions (Payne, 2019).

Periodic laboratory testing may be necessary to assess the effects of IV therapy and the patient's progress. Blood specimens may be obtained by a laboratory near the patient's home, or a home visit may be arranged to obtain blood specimens for analysis (Payne, 2019).

The nurse collaborates with the case manager in assessing the patient, family, and home environment; developing a plan of care in accordance with the patient's treatment plan and level of ability; and arranging for appropriate referral and follow-up if necessary. Any necessary equipment may be provided by the agency or purchased by the patient, depending on the terms of the home care arrangements. Appropriate documentation is necessary to assist in obtaining third-party payment for the service provided.

CRITICAL THINKING EXERCISES

1  ebp A 34-year-old woman comes to the walk-in clinic where you work. She is going to run her first marathon and asks about using salt tablets to compensate for loss of sodium through sweat. What is the best evidence for the use of salt tablets in performance athletes? What is the strength of the evidence base guiding your recommendation?

2  ipc An 82-year-old man with hypovolemia has been admitted to the medical unit where you work. What nursing and interprofessional assessments are indicated during your initial interactions with him? What other interprofessional services might you try to engage?

3  pq A 55-year-old woman is sent to the emergency department by her primary provider. The patient has a history of congestive heart failure. The primary provider has requested laboratory tests. Blood gas results are as follows: pH 7.46; HCO₃⁻ 26; PaCO₂ 40 mm Hg. Blood chemistry results are as follows: potassium 4.5 mEq/L; sodium 140 mEq/L; glucose 110 mg/dL. Vital signs: BP 140/92, HR 90 bpm, RR 18 bpm. What is your priority for this patient? Give your rationale. What further diagnostic testing is indicated?

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*Asterisk indicates nursing research.

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Resources

Infusion Nurses Society (INS), www.ins1.org

11 Shock, Sepsis, and Multiple Organ Dysfunction Syndrome

LEARNING OUTCOMES

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

1. Describe the pathophysiology, clinical manifestations, and collaborative management of progressive stages of various types of shock, of sepsis, and of multiple organ dysfunction syndrome.
2. Compare and contrast the pathophysiology, clinical manifestations, and collaborative management of shock states in hypovolemic, cardiogenic, and distributive shock.
3. Identify medical and nursing management priorities in treating patients across the continuum of shock.
4. Describe medical and nursing management priorities in the treatment and prevention of sepsis and septic shock.
5. Discuss the role of nurses in providing psychosocial support to patients experiencing shock, sepsis, and multiple organ dysfunction syndrome, and their families.
6. Discuss the role of nurses in providing transitional care to patients and their families after having been managed in a critical-care unit for shock, sepsis, or multiple organ dysfunction syndrome.

NURSING CONCEPTS

Cellular Regulation
Fluids and Electrolytes
Infection
Medical Emergencies
Perfusion

GLOSSARY

- anaphylactic shock:** distributive shock state resulting from a severe allergic reaction producing an acute systemic vasodilation and relative hypovolemia
- cardiogenic shock:** shock state resulting from impairment or failure of the myocardium
- colloids:** intravenous solutions that contain molecules that are too large to pass through capillary membranes
- crystalloids:** intravenous electrolyte solutions that move freely between the intravascular compartment and interstitial spaces
- cytokines:** messenger substances that may be released by a cell to create an action at that site or may be carried by the bloodstream to a distant site before being activated; (*synonyms:* biochemical mediators, inflammatory mediators)
- distributive shock:** shock state resulting from displacement of intravascular volume creating a relative hypovolemia and inadequate delivery of oxygen to the cells
- hypovolemic shock:** shock state resulting from decreased intravascular volume due to fluid loss
- multiple organ dysfunction syndrome:** presence of altered function of two or more organs in an acutely ill patient such that interventions are necessary to support continued organ function
- neurogenic shock:** shock state resulting from loss of sympathetic tone causing relative hypovolemia
- sepsis:** life-threatening organ dysfunction caused by a dysregulated host response to infection
- septic shock:** a subset of sepsis in which underlying circulatory and cellular metabolic abnormalities are profound enough to substantially increase mortality
- shock:** life-threatening physiologic condition in which there is inadequate blood flow to tissues and cells of the body
- systemic inflammatory response syndrome:** a syndrome resulting from a clinical insult that initiates an inflammatory response that is systemic, rather than localized to the site of the insult; a type of cytokine release syndrome also referred to as cytokine storm

Shock is a life-threatening condition that results from inadequate tissue perfusion. Many conditions may cause shock; irrespective of the cause, tissue hypoperfusion prevents adequate oxygen delivery to cells, leading to cell dysfunction and death. The progression of shock is neither linear nor predictable, and shock states, especially septic shock (the most life-threatening form of sepsis), comprise an area of ongoing clinical research. Nurses caring

for patients with shock and those at risk for shock must understand the underlying mechanisms of the various shock states (i.e., hypovolemic, cardiogenic, obstructive [see [Chapter 25](#)], and distributive shock [i.e., septic, neurogenic, anaphylactic]) and recognize the subtle as well as more obvious signs of each of these states. If shock is not effectively treated, **multiple organ dysfunction syndrome (MODS)** which is the presence of altered function of two or more organs in an acutely ill patient such that interventions are necessary to support continued organ function may ensue, often resulting in patient death. MODS may be a complication of any form of shock but is most commonly seen in patients with sepsis. Rapid assessment with early recognition and response to shock states and sepsis is essential to the patient's recovery.

Overview of Shock

Shock can best be defined as a clinical syndrome that results from inadequate tissue perfusion, creating an imbalance between the delivery of oxygen and nutrients needed to support cellular function (Kislitsina, Rich, Wilcox, et al., 2019; Massaro, 2018). Adequate blood flow to the tissues and cells requires an effective cardiac pump, adequate vasculature or circulatory system, and sufficient blood volume. If one of these components is impaired, perfusion to the tissues is threatened or compromised. Without treatment, inadequate blood flow to the cells results in poor delivery of oxygen and nutrients, cellular hypoxia, and cell death that progresses to organ dysfunction and eventually death.

Shock affects all body systems. It may develop rapidly or slowly, depending on the underlying cause. During shock, the body struggles to survive, calling on all its homeostatic mechanisms to restore blood flow. Any insult to the body can create a cascade of events resulting in poor tissue perfusion. Therefore, any patient with any disease state may be at risk for developing shock. The primary underlying pathophysiologic process and underlying disorder are used to classify the shock state (e.g., hypovolemic shock, cardiogenic shock, obstructive shock [see [Chapter 25](#)], distributive shock [i.e., septic, neurogenic, anaphylactic]; all discussed later in the chapter).

Regardless of the initial cause of shock, certain physiologic responses are common to all types of shock. These physiologic responses include hypoperfusion of tissues, hypermetabolism, and activation of the inflammatory response. The body responds to shock states by activating the sympathetic nervous system and mounting a hypermetabolic and inflammatory response. Failure of compensatory mechanisms to effectively restore physiologic balance is the final pathway of all shock states and results in end-organ dysfunction and death (Massaro, 2018; Seymour & Angus, 2018).

Nursing care of patients with shock requires ongoing systematic assessment. Many of the interventions required in caring for patients with shock call for close collaboration with other members of the health care team and rapid implementation of prescribed therapies. Nurses are in key positions to identify early signs of shock and anticipate rapid therapy.

Normal Cellular Function

Energy metabolism occurs within the cell, where nutrients are chemically broken down and stored in the form of adenosine triphosphate (ATP). Cells use this stored energy to perform necessary functions, such as active transport, muscle contraction, and biochemical synthesis, as well as specialized cellular functions, such as the conduction of electrical impulses. ATP can be synthesized aerobically (in the presence of oxygen) or anaerobically (in the absence of oxygen). Aerobic metabolism yields far greater amounts of ATP per mole of glucose than does anaerobic metabolism; therefore, it is a more efficient and effective means of producing energy. In addition, anaerobic metabolism results in the accumulation of the toxic end product lactic acid, which must be removed from the cell and transported to the liver for conversion into glucose and glycogen.

Pathophysiology

The pathophysiology of shock involves cellular changes, vascular responses, and changes in blood pressure.

Cellular Changes

In shock, the cells lack an adequate blood supply and are deprived of oxygen and nutrients; therefore, they must produce energy through anaerobic metabolism. This results in low-energy yields from nutrients and an acidotic intracellular environment. Because of these changes, normal cell function ceases (Fig. 11-1). The cell swells and the cell membrane becomes more permeable, allowing electrolytes and fluids to seep out of and into the cell. The sodium–potassium pump becomes impaired; cell structures, primarily the mitochondria, are damaged, and death of the cell results.

Glucose is the primary substrate required for the production of cellular energy in the form of ATP. In stress states, catecholamines, cortisol, glucagon, and inflammatory **cytokines** (i.e., biochemical or inflammatory mediators) are released, causing hyperglycemia and insulin resistance to mobilize glucose for cellular metabolism. Activation of these substances promotes gluconeogenesis, which is the formation of glucose from noncarbohydrate sources such as proteins and fats. Glycogen that has been stored in the liver is converted to

glucose through glycogenolysis to meet metabolic needs, increasing the blood glucose concentration (i.e., hyperglycemia).

Continued activation of the stress response by shock states causes a depletion of glycogen stores, resulting in increased proteolysis and eventual organ failure (Massaro, 2018). The deficit of nutrients and oxygen for normal cellular metabolism causes a buildup of metabolic end products in the cells and interstitial spaces. The clotting cascade, also associated with the inflammatory process, becomes activated, which compounds this pathologic cycle. With significant cell injury or death caused by shock, the clotting cascade is overproductive, resulting in small clots lodging in microcirculation, further hampering cellular perfusion (Seymour & Angus, 2018). This upregulation of the clotting cascade further compromises microcirculation of tissues, exacerbating cellular hypoperfusion (Seymour & Angus, 2018). Cellular metabolism is impaired, and a self-perpetuating negative situation (i.e., a positive feedback loop) is initiated.

Vascular Responses

Local regulatory mechanisms, referred to as autoregulation, stimulate vasodilation or vasoconstriction in response to biochemical mediators released by the cell, communicating the need for oxygen and nutrients (Kislitsina et al., 2019). A cytokine is a substance released by a cell or immune cells such as macrophages; the substance triggers an action at a cell site or travels in the bloodstream to a distant site, where it triggers action. Researchers are learning more every day about the physiologic actions of numerous proinflammatory and anti-inflammatory biochemical mediators that are responsible for the complex clinical presentation of shock states (Honore, Hoste, Molnár, et al., 2019).

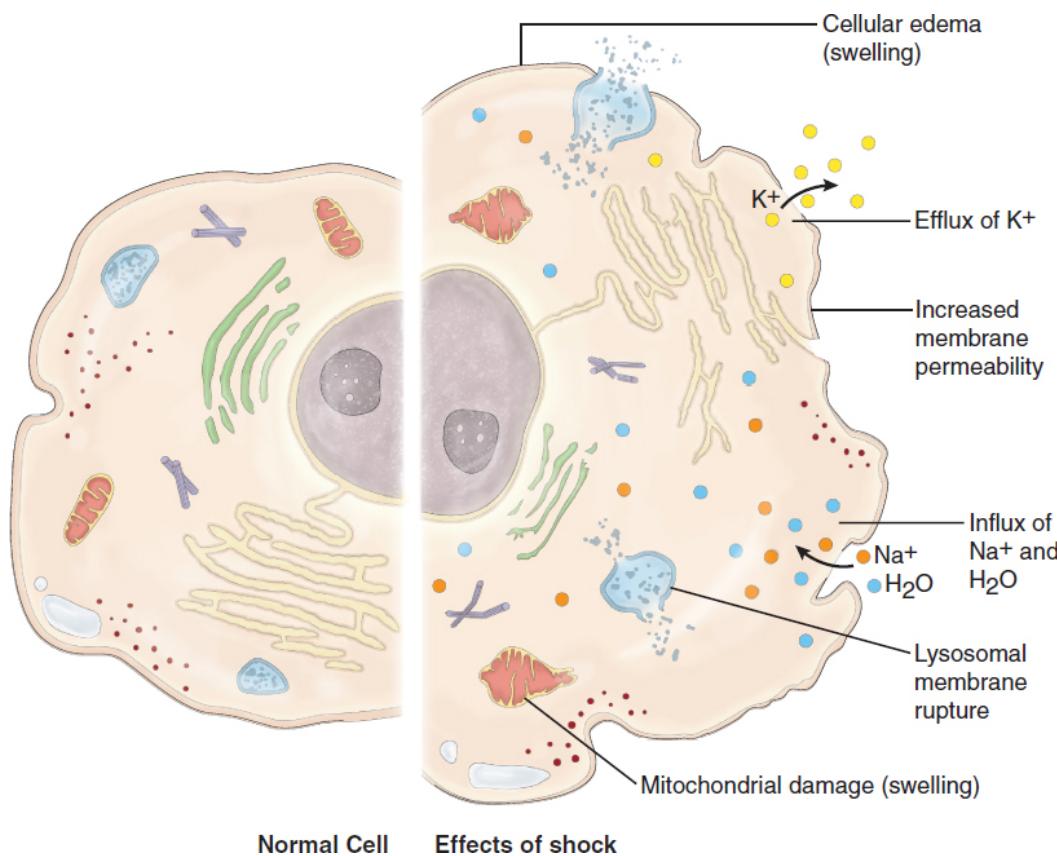


Figure 11-1 • Cellular effects of shock. The cell swells and the cell membrane becomes more permeable; fluids and electrolytes seep from and into the cell. Mitochondria and lysosomes are damaged and the cell dies.

Blood Pressure Regulation

Three major components of the circulatory system—blood volume, the cardiac pump, and the vasculature—must respond effectively to complex neural, chemical, and hormonal feedback systems to maintain an adequate blood pressure (BP) and perfuse body tissues. BP is regulated through a complex interaction of neural, chemical, and hormonal feedback systems affecting both cardiac output and peripheral resistance. This relationship is expressed in the following equation:

$$\text{Mean arterial BP} = \text{Cardiac output} \times \text{Peripheral resistance}$$

Cardiac output is a product of the stroke volume (the amount of blood ejected from the left ventricle during systole) and heart rate. Peripheral resistance is primarily determined by the diameter of the arterioles.

Tissue perfusion and organ perfusion depend on mean arterial pressure (MAP), or the average pressure at which blood moves through the vasculature. MAP must exceed 65 mm Hg for cells to receive the oxygen and nutrients needed to metabolize energy in amounts sufficient to sustain life (Hallisey &

Greenwood, 2019). True MAP can be calculated only by complex methods; however, most digital BP machines provide a MAP reading to guide clinical decisions.

BP is regulated by baroreceptors (pressure receptors) located in the carotid sinus and aortic arch. These pressure receptors are responsible for monitoring the circulatory volume and regulating neural and endocrine activities (see [Chapter 27](#) for further description). When BP drops, catecholamines (e.g., epinephrine, norepinephrine) are released from the adrenal medulla. These increase heart rate and cause vasoconstriction, restoring BP. Chemoreceptors, also located in the aortic arch and carotid arteries, regulate BP and respiratory rate using much the same mechanism in response to changes in oxygen and carbon dioxide (CO_2) concentrations in the blood. These primary regulatory mechanisms can respond to changes in BP on a moment-to-moment basis.

The kidneys regulate BP by releasing renin, an enzyme needed for the eventual conversion of angiotensin I to angiotensin II, a potent vasoconstrictor. This stimulation of the renin–angiotensin mechanism and the resulting vasoconstriction indirectly lead to the release of aldosterone from the adrenal cortex, which promotes the retention of sodium and water (i.e., hypernatremia). Hypernatremia then stimulates the release of antidiuretic hormone (ADH) by the pituitary gland. ADH causes the kidneys to retain water further in an effort to raise blood volume and BP. These secondary regulatory mechanisms may take hours or days to respond to changes in BP. The relationship between the initiation of shock and the responsiveness of primary and secondary regulatory mechanisms that compensate for deficits in blood volume, the pumping effectiveness of the heart, or vascular tone, which may result because of the shock state, is noted in [Figure 11-2](#).

Stages of Shock

Shock progresses along a continuum and can be identified as early or late, depending on the signs and symptoms and the overall severity of organ dysfunction. A convenient way to understand the physiologic responses and subsequent clinical signs and symptoms of shock is to divide the continuum into separate stages: compensatory (stage 1), progressive (stage 2), and irreversible (stage 3). The earlier that interventions are initiated along this continuum, the greater the patient's chance of survival. Current research and evidence-based practice focuses on assessing patients at greatest risk for shock and implementing early and aggressive interventions to reverse tissue hypoxia (Zhang, Hong, Smischney, et al., 2017). Current evidence suggests that the window of opportunity that increases the likelihood of patient survival occurs when aggressive therapy begins within 3 hours of identifying a shock state,

especially septic shock (Rhodes, Evans, Alhazzani, et al., 2017; Singer, Deutschman, Seymour, et al., 2016).

Physiology/Pathophysiology

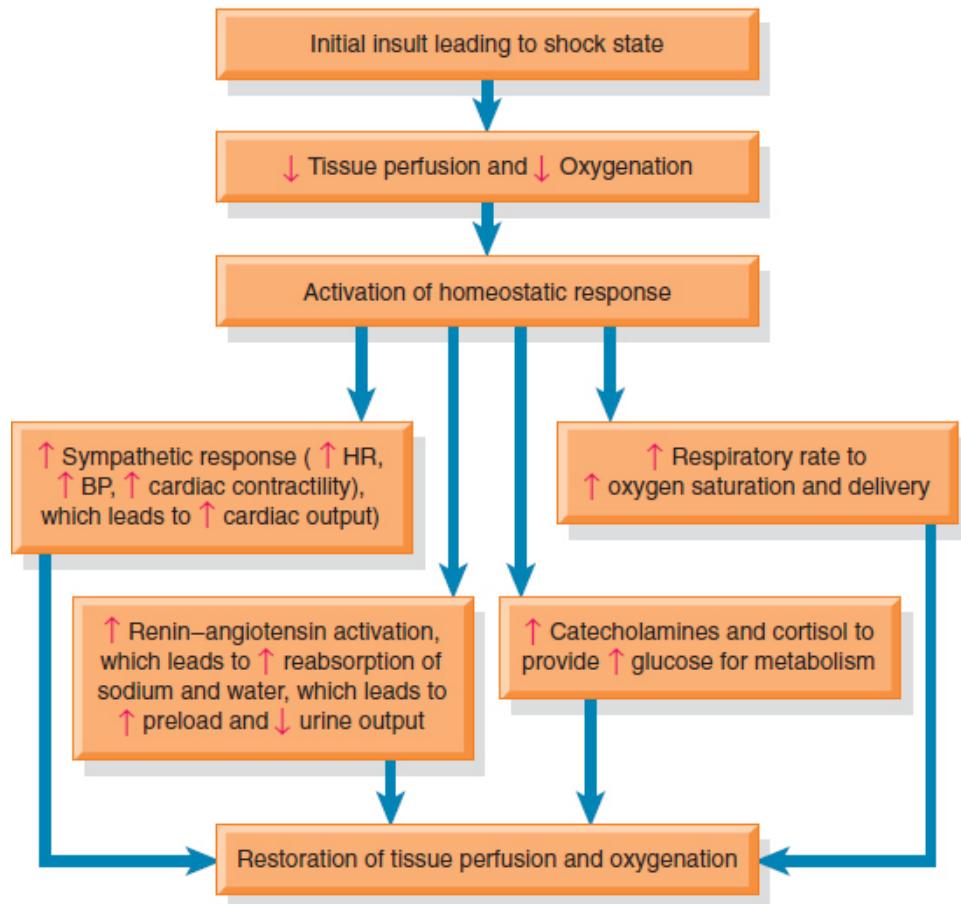


Figure 11-2 • Pathophysiologic sequence of regulatory mechanisms in shock.

Compensatory Stage

In the compensatory stage of shock, the BP remains within normal limits. Vasoconstriction, increased heart rate, and increased contractility of the heart contribute to maintaining adequate cardiac output. This results from stimulation of the sympathetic nervous system and subsequent release of catecholamines (e.g., epinephrine, norepinephrine). Patients display the often-described “fight-or-flight” response. The body shunts blood from organs such as the skin, kidneys, and gastrointestinal (GI) tract to the brain, heart, and lungs to ensure adequate blood supply to these vital organs. As a result, the skin may be cool and pale, bowel sounds are hypoactive, and urine output decreases in response to the release of aldosterone and ADH.

Clinical Manifestations

Despite a normal BP, the patient shows numerous clinical signs indicating inadequate organ perfusion ([Table 11-1](#)). The result of inadequate perfusion is anaerobic metabolism and a buildup of lactic acid, producing metabolic acidosis. The respiratory rate increases in response to the need to increase oxygen to the cells and in compensation for metabolic acidosis. This rapid respiratory rate facilitates removal of excess CO₂ but raises the blood pH and often causes a compensatory respiratory alkalosis. The patient may experience a change in effect, feel anxious, or be confused. If treatment begins in this stage of shock, the prognosis for the patient is better than in later stages.

TABLE 11-1

Clinical Findings in Stages of Shock

Finding	Stage		
	Compensatory	Progressive	Irreversible
Blood pressure	Normal	Systolic \leq 100 mm Hg; MAP \leq 65 mm Hg Requires fluids resuscitation to support blood pressure	Requires mechanical or pharmacologic support
Heart rate	>100 bpm	>150 bpm	Erratic
Respiratory status	\geq 22 breaths/min PaCO ₂ <32 mm Hg	Rapid, shallow respirations; crackles PaO ₂ <80 mm Hg PaCO ₂ >45 mm Hg	Requires intubation and mechanical ventilation and oxygenation
Skin	Cold, clammy Capillary refill \leq 3.5 s	Mottling, petechiae Capillary refill \geq 3.5 s	Jaundice
Urinary output	Decreased	<0.5 mL/kg/h	Anuric; requires dialysis
Mentation	Confusion and/or agitation	Lethargy	Unconscious
Acid-base balance	Respiratory alkalosis	Metabolic acidosis	Profound acidosis

MAP, mean arterial pressure; PaCO₂, partial pressure of arterial carbon dioxide; PaO₂, partial pressure of arterial oxygen.

Adapted from Bridges, E. (2017). Assessing patients during septic shock resuscitation. *American Journal of Nursing*, 117(10), 34–40; Makic, M. B. F., & Bridges, E. (2018).

Managing sepsis and septic shock: Current guidelines and definitions. *American Journal of Nursing*, 118(2), 34–39; Rhodes, A., Evans, L., Alhazzani, W., et al. (2017). Surviving Sepsis Campaign. (2017). Retrieved on 11/27/20 at:

www.sccm.org/getattachment/SurvivingSepsisCampaign/Guidelines/Adult-Patients/SSC-Guidelines-FAQ.pdf?lang=en-US; Simmons, J., & Ventetuolo, C. E. (2017).

Cardiopulmonary monitoring of shock. *Current Opinion*, 23(3), 223–231.



Medical Management

Medical treatment is directed toward identifying the cause of the shock, correcting the underlying disorder so that shock does not progress, and supporting those physiologic processes that thus far have responded successfully to the threat. Because compensation cannot be maintained indefinitely, measures such as fluid replacement, supplemental oxygen, and

medication therapy must be initiated to maintain an adequate BP and reestablish and maintain adequate tissue perfusion (Makic & Bridges, 2018; Rhodes et al., 2017).



Nursing Management

Early intervention along the continuum of shock is the key to improving the patient's prognosis (Makic & Bridges, 2018; Rhodes et al., 2017). The nurse must systematically assess the patient at risk for shock, recognizing subtle clinical signs of the compensatory stage before the patient's BP drops. Early interventions include identifying the cause of shock, administering intravenous (IV) fluids and oxygen, and obtaining necessary laboratory tests to rule out and treat metabolic imbalances or infection. Special considerations related to recognizing early signs of shock in the older adult patient are discussed in [Chart 11-1](#).

Monitoring Tissue Perfusion

In assessing tissue perfusion, the nurse observes for subtle changes in level of consciousness, vital signs (including pulse pressure), urinary output, skin (including assessment of capillary refill and signs of mottling), respiratory rate, and laboratory values (e.g., base deficit, lactic acid levels). In the compensatory stage of shock, serum sodium and blood glucose levels are elevated in response to the release of aldosterone and catecholamines. If infection is suspected, blood cultures should be obtained prior to administration of prescribed antibiotics; both of these interventions should be given priority in the care of the patient (Bridges, 2017; Levy, Evans, & Rhodes, 2018).

The nurse should monitor the patient's hemodynamic status and promptly report deviations to the primary provider, assist in identifying and treating the underlying disorder by continuous in-depth assessment of the patient, administer prescribed fluids and medications, and promote patient safety. Vital signs are key indicators of hemodynamic status, and BP is an indirect measure of tissue hypoxia. The nurse should report a systolic BP of 100 mm Hg or lower, or a drop in systolic BP of 40 mm Hg from baseline, or a MAP of 65 mm Hg or less (Hallisey & Greenwood, 2019; Rhodes et al., 2017). If the patient is concurrently diagnosed with an infection or if an infection is suspected, the nurse should promptly notify the primary provider if the patient exhibits any two of the three following signs (Singer et al., 2016) (see later discussion of Septic Shock):

- Respiratory rate greater than or equal to 22 breaths/min
- Altered mentation

- Systolic BP less than or equal to 100 mm Hg

Pulse pressure correlates well with stroke volume. Pulse pressure is calculated by subtracting the diastolic measurement from the systolic measurement; the difference is the pulse pressure. Normally, the pulse pressure is 30 to 40 mm Hg. Narrowing or decreased pulse pressure is an earlier indicator of shock than a drop in systolic BP (Simmons & Ventetuolo, 2017). Decreased or narrowing pulse pressure, an early indication of decreased stroke volume, is illustrated in the following example:

Chart 11-1



Recognizing Shock in Older Adults

The physiologic changes associated with aging, coupled with pathologic and chronic disease states, place older adults at increased risk for developing a state of shock and possibly multiple organ dysfunction syndrome. Older adults can recover from shock if it is detected and treated early with aggressive and supportive therapies. Nurses play an essential role in assessing and interpreting subtle changes in older adults' responses to illness.

- Medications such as beta-blocking agents (e.g., metoprolol) used to treat hypertension may mask tachycardia, a primary compensatory mechanism to increase cardiac output, during hypovolemic states.
- The aging immune system may not mount a truly febrile response (temperature greater than 38.3°C [101°F]); however, a lack of a febrile response (temperature less than 37°C [98.6°F]) or an increasing trend in body temperature should be addressed. The patient may also report increased fatigue and malaise in the absence of a febrile response.
- The heart does not function well in hypoxic states, and the aging heart may respond to decreased myocardial oxygenation with arrhythmias that may be misinterpreted as a normal part of the aging process.
- There is a progressive decline in respiratory muscle strength, maximal ventilation, and response to hypoxia. Older adults have a decreased respiratory reserve and decompensate more quickly.
- Changes in mentation may be inappropriately misinterpreted as dementia. Older adults with a sudden change in mentation should be aggressively assessed for acute delirium (hypo- and hyperdelirium states) and treated for the presence of infection and organ hypoperfusion.

Adapted from Rhodes, A., Evans, L., Alhazzani, W., et al. (2017). Surviving Sepsis Campaign: International guidelines for management of sepsis and septic shock: 2016. *Critical Care Medicine*, 45(3), 486–552; Rowe, T. A., & McKoy, J. M. (2017). Sepsis in older adults. *Infectious Disease Clinics of North America*, 31(4), 731–742.

$$\text{Systolic BP} - \text{Diastolic BP} = \text{Pulse pressure}$$

Normal pulse pressure:

$$120 \text{ mm Hg} - 80 \text{ mm Hg} = 40 \text{ mm Hg}$$

Narrowing of pulse pressure:

$$90 \text{ mm Hg} - 70 \text{ mm Hg} = 20 \text{ mm Hg}$$

Elevation of the diastolic BP with release of catecholamines and attempts to increase venous return through vasoconstriction is an early compensatory mechanism in response to decreased stroke volume, BP, and overall cardiac output.



Quality and Safety Nursing Alert

By the time BP drops, damage has already been occurring at the cellular and tissue levels. Therefore, the patient at risk for shock must be assessed and monitored closely before the BP falls.

Continuous central venous oximetry (ScvO_2) monitoring may be used to evaluate mixed venous blood oxygen saturation and severity of tissue hypoperfusion states. A central catheter is introduced into the superior vena cava (SVC), and a sensor on the catheter measures the oxygen saturation of the blood in the SVC as blood returns to the heart and pulmonary system for reoxygenation. A normal ScvO_2 value is 70% (Zhang et al., 2017). Body tissues use approximately 25% of the oxygen delivered to them during normal metabolism. During stressful events, such as shock, more oxygen is consumed and the ScvO_2 saturation is lower, indicating that the tissues are consuming more oxygen.

Interventions focus on decreasing tissue oxygen requirements and increasing perfusion to deliver more oxygen to the tissues. For instance, sedating agents may be given to lower metabolic demands, or the patient's pain may be treated with opioid, nonopioid, or sedating agents (e.g., propofol, dexmedetomidine, acetaminophen) to decrease metabolic demands for oxygen. Supplemental oxygen and mechanical ventilation may be required to increase the delivery of oxygen in the blood. Administration of IV fluids and medications supports BP and cardiac output, and the transfusion of packed red blood cells enhances oxygen transport. Monitoring tissue oxygen consumption with ScvO_2 is an invasive measure to more accurately assess tissue oxygenation in the compensatory stage of shock before changes in vital signs detect altered tissue perfusion (Rhodes et al., 2017; Zhang et al., 2017).

In the patient who has an arterial line present, arterial pulse waveform analysis or pulse contour may be used to determine the patient's stroke volume and responsiveness to IV fluid replacement to meet tissue perfusion needs (Simmons & Ventetuolo, 2017). More commonly, passive leg raising (PLR) is used to determine which patients will or will not respond to IV fluid bolus challenges. PLR involves raising the patient's legs to a 30- to 45-degree angle to increase venous return and thus cardiac output (Fig. 11-3). If the blood pressure improves with PLR, the patient will respond to additional fluids. If the blood pressure does not improve, giving more IV fluids will not improve

the patient's condition and can precipitate fluid overload (Laher, Watermeyer, Buchanan, et al., 2017; Pickett, Bridges, Kritek, et al., 2018). If the patient is mechanically ventilated, functional hemodynamic monitoring will be used to assess fluid volume needs and the patient's response to IV fluid administration (i.e., preload, cardiac output, and blood pressure). An improvement in preload, cardiac output, and blood pressure indicates a favorable response. Functional hemodynamic monitoring has replaced static measures obtained from central venous pressure (CVP) or pulmonary artery (PA) monitoring. Functional hemodynamic monitoring assesses real-time dynamic changes between the patient's breathing patterns and circulating blood volume. Assessing functional hemodynamic parameters requires the patient to be mechanically ventilated and have an invasive arterial catheter placed and connected to monitoring devices that measure fluid volume (Bridges, 2013; Hallisey & Greenwood, 2019; Simmons & Ventetuolo, 2017).

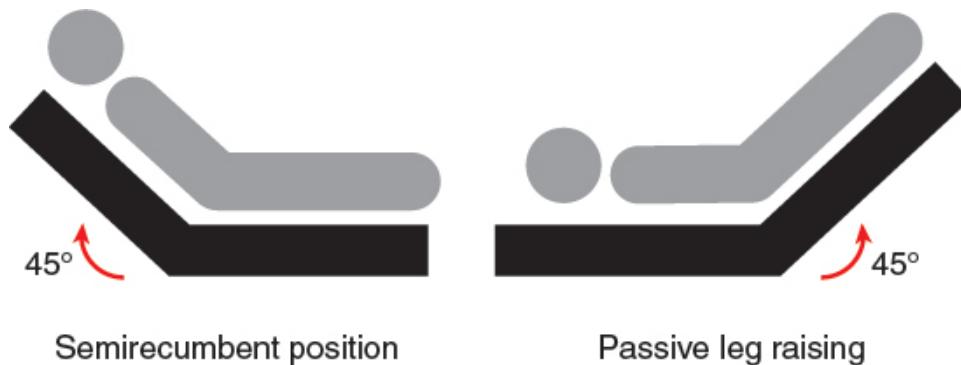


Figure 11-3 • Passive leg raise.

Although treatments are prescribed and initiated by the primary provider, the nurse usually implements them, operates and troubleshoots equipment used in treatment, monitors the patient's status during treatment, and evaluates the immediate effects of treatment. In addition, the nurse assesses the response of the family to the crisis and its treatment.

Reducing Anxiety

Patients and their families often become anxious and apprehensive when they face a major threat to health and well-being and are the focus of attention of many health care providers. Providing brief explanations about the diagnostic and treatment procedures, supporting the patient during these procedures, and providing information about their outcomes are usually effective in reducing stress and anxiety and thus promoting the patient's physical and mental well-being. Speaking in a calm, reassuring voice and using gentle touch also help ease the patient's concerns. These actions may provide comfort for patients who are critically ill and frightened (Reaza-Alarcón & Rodríguez-Martín,

2019). Evidence suggests that family members have certain needs during a health-related crisis, which include that health care professionals provide honest, consistent, and thorough communication; are present with the patient to facilitate physical and emotional support; demonstrate caring behaviors, see the patient frequently, and know the daily, short-term, and long-term plan of care for the patient (Reaza-Alarcón & Rodríguez-Martín, 2019; Wong, Redley, Digby, et al., 2019).

The nurse should advocate for family members to be present during procedures and routine patient care activities. The presence of family provides an important connection and support for the patient during a time of crisis. Clinical practice guidelines suggest that sharing decision making with the patient and the family enhances communication with the health care team, reduces patient anxiety, and improves overall satisfaction with care (Davidson, Aslakson, Long, et al., 2017).

Clarifying Advance Directives

Because shock can progress rapidly and its course may be unpredictable, it is important for the nurse to ask patients on admission if they have advance directives, including durable power of attorney for health care or living wills, or if they have had conversations with anyone about their health care wishes. When appropriate, the patient and family (with patient permission) should be approached regarding preferences for resuscitation and Physician Orders for Life-Sustaining Treatment (POLST) (see [Chapter 13](#)). The POLST helps ensure that the patient's wishes are honored if their condition deteriorates rapidly and they lose the ability to speak for themselves (Turner & Hylton, 2019).

Promoting Safety

The nurse must be vigilant for potential threats to the patient's safety, because a high anxiety level and changes in mental status may impair judgment. Patients who were previously cooperative and followed instructions may now disrupt IV lines and catheters, increasing the risk for potential complications. Close monitoring, frequent reorientation, hourly rounding, and implementing interventions to prevent falls (e.g., bed alarms) are essential.

Progressive Stage

In the second stage of shock, the mechanisms that regulate BP can no longer compensate, and the MAP falls below normal limits. Patients are clinically hypotensive; this is defined as a systolic BP of 100 mm Hg or lower, or a decrease in systolic BP of 40 mm Hg from baseline. The patient shows signs of declining mental status (Rhodes et al., 2017; Singer et al., 2016).

Pathophysiology

Although all organ systems suffer from hypoperfusion at this stage, several events perpetuate the shock syndrome. First, the overworked heart becomes dysfunctional, the body's inability to meet increased oxygen requirements produces ischemia, and biochemical mediators cause myocardial depression (Massaro, 2018). This leads to failure of the heart, even if the underlying cause of the shock is not of cardiac origin. Second, the autoregulatory function of the microcirculation fails in response to the numerous biochemical mediators released by the cells, resulting in increased capillary permeability, with areas of arteriolar and venous constriction further compromising cellular perfusion (Massaro, 2018). At this stage, the prognosis worsens. The relaxation of precapillary sphincters causes fluid to leak from the capillaries, creating interstitial edema and decreased return to the heart. In addition, the inflammatory response is activated, and proinflammatory and anti-inflammatory mediators are released, which activate the coagulation system in an effort to reestablish homeostasis (Seymour & Angus, 2018). The body mobilizes energy stores and increases oxygen consumption to meet the increased metabolic needs of the underperfused tissues and cells. Anaerobic metabolism ensues, resulting in a buildup of lactic acid and disruption of normal cell function.

Even if the underlying cause of the shock is reversed, the sequence of compensatory responses to the decrease in tissue perfusion perpetuates the shock state, and a vicious cycle ensues. The cellular reactions that occur during the progressive stage of shock are an active area of clinical research. It is believed that the body's response to shock or lack of response in this stage of shock may be the primary factor determining the patient's survival. Early recognition of shock signs and symptoms is essential to improving morbidity and mortality.

Clinical Manifestations

Chances of survival depend on the patient's general health before the shock state as well as the amount of time it takes to restore tissue perfusion. As shock progresses, organ systems decompensate (see [Table 11-1](#)).

Respiratory Effects

The lungs, which become compromised early in shock, are affected at this stage. Subsequent decompensation of the lungs increases the likelihood that mechanical ventilation will be needed. Respirations are rapid and shallow. Crackles are heard over the lung fields. Decreased pulmonary blood flow causes arterial oxygen levels to decrease and CO₂ levels to increase. Hypoxemia and biochemical mediators cause an intense inflammatory

response and pulmonary vasoconstriction, perpetuating pulmonary capillary hypoperfusion and hypoxemia. The hypoperfused alveoli stop producing surfactant and subsequently collapse. Pulmonary capillaries begin to leak, causing pulmonary edema, diffusion abnormalities (shunting), and additional alveolar collapse. This condition is called *acute lung injury* (ALI); as ALI continues, interstitial inflammation and fibrosis are common consequences, leading to acute respiratory distress syndrome (ARDS) (Mitchell & Seckel, 2018). Further explanation of ALI and ARDS, as well as their nursing management, can be found in [Chapter 19](#).

Cardiovascular Effects

A lack of adequate blood supply leads to arrhythmias and ischemia. The heart rate is rapid, sometimes exceeding 150 bpm. The patient may complain of chest pain and even suffer a myocardial infarction (MI). Levels of cardiac biomarkers (e.g., cardiac troponin I [cTn-I]) increase. In addition, myocardial depression and ventricular dilation may further impair the heart's ability to pump enough blood to the tissues to meet increasing oxygen requirements.

Neurologic Effects

As blood flow to the brain becomes impaired, mental status deteriorates. Changes in mental status occur with decreased cerebral perfusion and hypoxia. Initially, the patient may exhibit subtle changes in behavior, become agitated, confused, or demonstrate signs of delirium. (See [Chapter 61](#) for more information about delirium.) Subsequently, lethargy increases, and the patient begins to lose consciousness.

Renal Effects

When the MAP falls below 65 mm Hg (Hallisey & Greenwood, 2019), the glomerular filtration rate of the kidneys cannot be maintained, and significant changes in renal function occur. Acute kidney injury (AKI) is characterized by an increase in blood urea nitrogen (BUN) and serum creatinine levels, fluid and electrolyte shifts, acid–base imbalances, and a loss of the renal–hormonal regulation of BP. Urinary output usually decreases to less than 0.5 mL/kg/h (or less than 30 mL/h) but may vary depending on the phase of AKI. (See [Chapter 48](#) for further information about AKI.)

Hepatic Effects

Decreased blood flow to the liver impairs the ability of liver cells to perform metabolic and phagocytic functions. Consequently, the patient is less able to metabolize medications and metabolic waste products, such as ammonia and lactic acid. Metabolic activities of the liver, including gluconeogenesis and glycogenolysis, are impaired. The patient becomes more susceptible to

infection as the liver fails to filter bacteria from the blood. Liver enzymes (aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase) and bilirubin levels are elevated, and the patient develops jaundice.

Gastrointestinal Effects

GI ischemia can cause stress ulcers in the stomach, putting the patient at risk for GI bleeding. In the small intestine, the mucosa can become necrotic and slough off, causing bloody diarrhea. Beyond the local effects of impaired perfusion, GI ischemia leads to bacterial translocation and organ dysfunction, in which bacterial toxins enter the bloodstream through the lymphatic system. In addition to causing infection, bacterial toxins can cause cardiac depression, vasodilation, increased capillary permeability, and an intense inflammatory response with activation of additional biochemical mediators. The net result is interference with healthy cellular functioning and the ability to metabolize nutrients (Rhodes et al., 2017).

Hematologic Effects

The combination of hypotension, sluggish blood flow, metabolic acidosis, coagulation system imbalance, and generalized hypoxemia can interfere with normal hemostatic mechanisms. In shock states, the inflammatory cytokines activate the clotting cascade, causing deposition of microthrombi in multiple areas of the body and consumption of clotting factors. The alterations of the hematologic system, including imbalance of the clotting cascade, are linked to the overactivation of the inflammatory response (Massaro, 2018; Seymour & Angus, 2018). Disseminated intravascular coagulation (DIC) may occur either as a cause or as a complication of shock. In this condition, widespread clotting and bleeding occur simultaneously. Ecchymoses (bruises) and petechiae (bleeding) may appear in the skin. Coagulation times (e.g., prothrombin time, activated partial thromboplastin time) are prolonged. Clotting factors and platelets are consumed and require replacement therapy to achieve hemostasis. (Further discussion of DIC appears in [Chapter 29](#).)



Medical Management

Specific medical management in the progressive stage of shock depends on the type of shock, its underlying cause, and the degree of decompensation in the organ systems. Medical management specific to each type of shock is discussed later in this chapter. Although medical management in the progressive stage differs by type of shock, some medical interventions are common to all types. These include the use of appropriate IV fluids and medications to restore tissue perfusion by the following methods:

- Supporting the respiratory system
- Optimizing intravascular volume
- Supporting the pumping action of the heart
- Improving the competence of the vascular system

Other aspects of management may include early enteral nutritional support, targeted hyperglycemic control with IV insulin and use of antacids, histamine-2 (H₂) blockers, or antipeptic medications to reduce the risk of GI ulceration and bleeding.

Tight glycemic control (i.e., maintaining serum glucose close to the normal parameters of 80 to 100 mg/dL) is not recommended in patients who are critically ill because this therapy has been found to result in adverse patient outcomes (Griesdale, DeSouza, VanDam, et al., 2009). Current evidence suggests that maintaining serum glucose less than 180 mg/dL with insulin therapy and close monitoring is indicated in the management of the patient who is critically ill (American Diabetes Association, 2019; Rhodes et al., 2017).



Quality and Safety Nursing Alert

Glycemic control is linked to outcomes in the patient in shock. Although tight glycemic control is not indicated, evidence shows that maintaining serum glucose less than 180 mg/dL is linked to best outcomes.



Nursing Management

Nursing care of patients in the progressive stage of shock requires expertise in assessing and understanding shock and the significance of changes in assessment data. Early interventions are essential to the survival of patients; therefore, suspecting that a patient may be in shock and reporting subtle changes in assessment are imperative. Patients in the progressive stage of shock are cared for in the intensive care setting to facilitate close monitoring (hemodynamic monitoring, electrocardiographic [ECG] monitoring, arterial blood gases, serum electrolyte levels, physical and mental status changes); rapid and frequent administration of various prescribed medications and fluids; and possibly interventions with supportive technologies, such as mechanical ventilation, dialysis (e.g., continuous renal replacement therapy), and intra-aortic balloon pump (IABP).

Working closely with other members of the health care team, the nurse carefully documents treatments, medications, and fluids that are given; the

time, dosage or volume, and patient responses are recorded. In addition, the nurse coordinates care, including the scheduling of diagnostic procedures that may occur at the bedside, and communication among health care personnel, family members, and the patient.

Preventing Complications

The nurse monitors the patient for early signs of complications to help reduce potential risks to the patient. Monitoring includes evaluating blood levels of medications, observing invasive vascular lines and catheters for signs of infection, and checking neurovascular status if arterial lines are inserted, especially in the lower extremities. Simultaneously, the nurse promotes the patient's safety and comfort by ensuring that all procedures, including invasive procedures and arterial and venous punctures, are carried out using correct aseptic techniques and that venous and arterial puncture and infusion sites are maintained with the goal of preventing infection. Nursing interventions that reduce the incidence of ventilator-associated pneumonia (VAP) must also be implemented. These include frequent oral care with a toothbrush, aseptic suction technique, turning, elevating the head of the bed at least 30 degrees to prevent aspiration, and implementing daily interruption of sedation as prescribed to evaluate patient readiness for extubation (Mitchell, Russo, Cheng, et al., 2019). See [Chart 19-6](#) for an overview of the evidence-based ("bundled") interventions aimed at preventing VAP. Positioning and repositioning of the patient to promote comfort and maintain skin integrity are essential.

The nurse must also be vigilant in assessing for acute delirium, characterized by an acute change in mental status, inattention, disorganized thinking, and altered level of consciousness. Delirium is potentially preventable (Devlin, Skrobik, Gelians, et al., 2018; Makic, 2018). Patients who are critically ill and have delirium have longer mechanical ventilation support needs, experience higher functional decline, and have higher rates of morbidity and mortality than those without delirium. Furthermore, they are at a higher risk of developing postintensive care syndrome (PICS), which manifests as new or worsening impairments in the patient's physical, cognitive, or mental status after a critical illness has resolved and that persists beyond the acute hospitalization (Devlin et al., 2018). Delirium should be assessed at a minimum each shift using a standardized delirium assessment tool, such as the Confusion Assessment Method (CAM)-ICU (Critical Illness, Brain Dysfunction, and Survivorship (CIBS) Center, 2020). The CAM-ICU is a modified version of the CAM, specifically designed for use in patients who are critically ill (see [Chapter 8, Chart 8-7](#) for discussion of the CAM). Nursing interventions that can prevent delirium include engaging the patient in frequent reorientation activities (e.g., to date, time, place), assessing and treating pain, promoting sleep, providing early mobilization activities, and limiting sedation,

especially sedation with benzodiazepines (e.g., lorazepam) (Devlin et al., 2018; Makic, 2018).

Promoting Rest and Comfort

Efforts are made to minimize the cardiac workload by reducing the patient's physical activity and treating pain and anxiety. Because promoting patient rest and comfort is a priority, the nurse performs essential nursing activities in blocks of time, allowing the patient to have periods of uninterrupted sleep, which may prevent acute delirium, as noted previously (Devlin et al., 2018). To conserve the patient's energy, the nurse should protect the patient from temperature extremes (e.g., excessive warmth or cold, shivering), which can increase the metabolic rate and oxygen consumption and thus the cardiac workload.

Supporting Family Members

Because patients in shock receive intense attention by the health care team, families may be overwhelmed and frightened. Family members may be reluctant to ask questions or seek information for fear that they will be in the way or will interfere with the attention given to the patient. The nurse should make sure that the family is comfortably situated and kept informed about the patient's status. Often, families need encouragement from the health care team to get some rest; family members are more likely to take this advice if they feel that the patient is being well cared for and that they will be notified of any significant changes in the patient's status. A visit from the hospital chaplain may be comforting and provides some attention to the family while the nurse concentrates on the patient. Ensuring patient- and family-centered care is central to the delivery of high-quality care. This helps meet the emotional well-being as well as the physiologic needs of the patient and the family (Wong et al., 2019).

Irreversible Stage

The irreversible (or refractory) stage of shock represents the point along the shock continuum at which organ damage is so severe that the patient does not respond to treatment and cannot survive. Despite treatment, BP remains low. Renal and liver dysfunction, compounded by the release of biochemical mediators, creates an acute metabolic acidosis. Anaerobic metabolism contributes to a worsening lactic acidosis. Reserves of ATP are almost totally depleted, and mechanisms for storing new supplies of energy have been destroyed. Respiratory system dysfunction prevents adequate oxygenation and ventilation despite mechanical ventilatory support, and the cardiovascular system is ineffective in maintaining an adequate MAP for tissue perfusion.

Multiple organ dysfunction progressing to complete organ failure has occurred, and death is imminent. Multiple organ dysfunction can occur as a progression along the shock continuum or as a syndrome unto itself and is described in more detail later in this chapter.



Medical Management

Medical management during the irreversible stage of shock is similar to interventions and treatments used in the progressive stage. Although the patient may have progressed to the irreversible stage, the judgment that the shock is irreversible can only be made after the patient has failed to respond to treatment. Strategies that may be experimental (e.g., investigational medications, such as immunomodulation therapy) may be tried to reduce or reverse the severity of shock.



Nursing Management

As in the progressive stage of shock, the nurse focuses on carrying out prescribed treatments, monitoring the patient, preventing complications, protecting the patient from injury, and providing comfort. Offering brief explanations to the patient about what is happening is essential even if there is no certainty that the patient hears or understands what is being said. Simple comfort measures, including reassuring touches, should continue to be provided despite the patient's nonresponsiveness to verbal stimuli (Wong et al., 2019).

As it becomes obvious that the patient is unlikely to survive, the family needs to be informed about the prognosis and likely outcome. Opportunities should be provided throughout the patient's care for the family to see, touch, and talk to the patient. Close friends or spiritual or religious advisors may be of comfort to the family members in dealing with the inevitable death of their loved one.

During this stage of shock, the family may misinterpret the actions of the health care team. They have been told that nothing has been effective in reversing the shock and that the patient's survival is very unlikely, yet they find primary providers and nurses continuing to work feverishly on the patient. Distraught, grieving families may interpret this as a chance for recovery when none exists, and family members may become angry when the patient dies. Conferences with all members of the health care team and the family promote better understanding by the family of the patient's prognosis and the purpose for management interventions. Engaging palliative care specialists can be beneficial in developing a plan of care that maximizes comfort and effective

symptom management as well as assisting the family with difficult decisions (Ivany & Aitken, 2019). During these conferences, it is essential to explain that the equipment and treatments being provided are intended for patient comfort and do not suggest that the patient will recover. Family members should be encouraged to express their views of life-support measures. In some cases, ethics committees may be consulted to assist families and health care teams make complex end-of-life decisions (Ivany & Aitken, 2019; Turner & Hylton, 2019).

General Management Strategies in Shock

As described previously and in the discussion of types of shock to follow, management in all types and all phases of shock includes the following:

- Support of the respiratory system with supplemental oxygen and/or mechanical ventilation to provide optimal oxygenation (see [Chapter 19](#))
- Fluid replacement to restore intravascular volume
- Vasoactive medications to restore vasomotor tone and improve cardiac function
- Nutritional support to address the metabolic requirements that are often dramatically increased in shock

Therapies described in this section require collaboration among all members of the health care team.

Fluid Replacement

Fluid replacement, also referred to as fluid resuscitation, is given in all types of shock. The type of fluids administered, and the speed of delivery vary; however, fluids are given to improve cardiac and tissue oxygenation, which in part depends on flow. The fluids given may include **crystalloids** (electrolyte solutions that move freely between intravascular compartment and interstitial spaces), **colloids** (large-molecule IV solutions), and blood components (packed red blood cells, fresh-frozen plasma, and platelets).

Crystalloid and Colloid Solutions

In emergencies, the “best” fluid is often the fluid that is readily available. Fluid resuscitation should be initiated early in shock to maximize intravascular volume. Isotonic crystalloid solutions are often selected because they contain the same concentration of electrolytes as the extracellular fluid and, therefore, can be given without altering the concentrations of electrolytes in the plasma. IV crystalloids commonly used for resuscitation in hypovolemic shock include

0.9% sodium chloride solution (normal saline) and lactated Ringer's solution. Lactated Ringer's is an electrolyte solution containing the lactate ion, which should not be confused with lactic acid. The lactate ion is converted to bicarbonate, which helps buffer the overall acidosis that occurs in shock. Lactated Ringer's solution more closely resembles plasma and is considered a more appropriate first choice solution over 0.9% normal saline (de-Madaria, Herrera-Marante, Gonzalez-Camacho, et al., 2018). While normal saline is an isotonic solution, large infusions may cause hypernatremia, hypokalemia, and hyperchloremic metabolic acidosis (de-Madaria et al., 2018; Sethi, Owyang, Meyers, et al., 2018). Hypertonic crystalloid solution, often 3% sodium chloride, does not improve patient outcomes and may result in unintended complications and is not recommended as a fluid for resuscitation (Bauer, MacLaren, & Erstad, 2019). A disadvantage of using isotonic crystalloid solutions is that some of the volume given is lost to the interstitial compartment and some remains in the intravascular compartment. This occurs as a consequence of cellular permeability that occurs during shock. Diffusion of crystalloids into the interstitial space means that more fluid may need to be given than the amount lost to support tissue perfusion (Lewis, Pritchard, Evans, et al., 2018).

Care must be taken when rapidly administering isotonic crystalloids to avoid both underresuscitating and overresuscitating the patient in shock. Insufficient fluid replacement is associated with a higher incidence of morbidity and mortality from lack of tissue perfusion, whereas excessive fluid administration can cause systemic and pulmonary edema that progresses to ALI (see [Chapter 19](#)), intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS), and MODS (see later discussion).

ACS is a serious complication that may occur when large volumes of fluid are given. It may also occur after trauma, abdominal surgery, pancreatitis, or sepsis (Harrell & Miller, 2017). In ACS, fluid leaks into the intra-abdominal cavity, increasing pressure that is displaced onto surrounding vessels and organs. Venous return, preload, and cardiac output are compromised. The pressure also elevates the diaphragm, making it difficult to breathe effectively. The renal and GI systems also begin to show signs of dysfunction (e.g., decreased urine output, absent bowel sounds, intolerance of tube feeding). Abdominal compartment pressure can be measured. Normally, it is 0 to 5 mm Hg, and a pressure of 12 mm Hg is considered to be indicative of IAH (Harrell & Miller, 2017). If ACS is present, interventions that usually include surgical decompression are necessary to relieve the pressure.

Generally, IV colloidal solutions are similar to plasma proteins, in that they contain molecules that are too large to pass through capillary membranes. Colloids expand intravascular volume by exerting oncotic pressure, thereby pulling fluid into the intravascular space, increasing intravascular volume. In addition, colloids have a longer duration of action than crystalloids, because

the molecules remain within the intravascular compartment longer. Typically, if colloids are used to treat tissue hypoperfusion, albumin is the agent prescribed. Albumin is a plasma protein; an albumin solution is prepared from human plasma and is heated during production to reduce its potential to transmit disease. The disadvantage of albumin is its high cost compared to crystalloid solutions. Resuscitation with colloid solutions has not reduced the risk of morbidity or death compared to resuscitation with crystalloid solutions; moreover, colloids can be considerably more expensive than crystalloid solutions (Annane, Siami, Jaber, et al., 2013; Lewis et al., 2018).



Quality and Safety Nursing Alert

With all colloidal solutions, side effects include the rare occurrence of anaphylactic reactions. Nurses must monitor patients closely.

Complications of Fluid Administration

Close monitoring of the patient during fluid replacement is necessary to identify side effects and complications. The most common and serious side effects of fluid replacement are cardiovascular overload, pulmonary edema, and ACS. The patient receiving fluid replacement must be monitored frequently for adequate urinary output, changes in mental status, skin perfusion, and changes in vital signs. Lung sounds are auscultated frequently to detect signs of fluid accumulation. Adventitious lung sounds, such as crackles, may indicate pulmonary edema and ALI and ARDS.



Quality and Safety Nursing Alert

When administering large volumes of crystalloid solutions, the nurse must monitor the lungs for adventitious sounds, signs and symptoms of interstitial edema, work of breathing (i.e., increasing effort required for the patient to breathe, depth of breathing, respiratory rate), and changes in oxygen saturation.

Often, a CVP line is inserted (typically into the subclavian or jugular vein) and is advanced until the tip of the catheter rests near the junction of the SVC and the right atrium. CVP measurements have traditionally been used to assess preload in the right side of the heart and fluid responsiveness during shock states. However, research confirms there is no direct relationship between CVP, circulating blood volume, and fluid responsiveness; thus, CVP is no longer used as a reliable measure to guide fluid replacement therapy (Laher et al., 2017; Marik, Baram, & Vahid, 2008; Marik & Cavallazzi, 2013;

Simmons & Ventetuolo, 2017). CVP devices provide access for large volumes of fluid replacement, and administration of blood products and vasoactive agents. If CVP measurements are still used to evaluate fluid needs of the patient, assessment variables such as BP, urine output, heart rate, and PLR should be considered when interpreting the CVP for clinical decisions (Laher et al., 2017; Simmons & Ventetuolo, 2017). Some CVP catheters allow the monitoring of intravascular measures and venous oxygen levels. Assessment of venous oxygenation (venous oxygen saturation SvO_2 , or $ScvO_2$ with a CVP line) may be helpful in evaluating the adequacy of intravascular volume (Rhodes et al., 2017; Rivers, McIntyre, Morro, et al., 2005). Hemodynamic monitoring with arterial lines may be implemented to allow close monitoring of the patient's BP and tissue perfusion. A pulmonary artery catheter may be inserted to assist with closer monitoring of a patient's cardiac status as well as response to therapy. Advances in noninvasive or minimally invasive technology (e.g., esophageal Doppler, arterial pulse contour analysis, cardiac output devices, intrathoracic impedance monitoring) provide additional hemodynamic monitoring options (Hallisey & Greenwood, 2019; Laher et al., 2017; Zhang et al., 2017). (For additional information about hemodynamic monitoring, see [Chapter 21](#).)

Placement of central lines for fluid administration and monitoring requires collaborative practice between the provider and the nurse to ensure that all measures to prevent central line-associated bloodstream infection (CLABSI) are implemented. Several interventions aimed at preventing CLABSI should be implemented collaboratively while the central line is being placed as well as during ongoing nursing management of the central line itself. [Chart 11-2](#) describes the evidence-based (“bundled”) interventions that have been found to reduce CLABSI.

Vasoactive Medication Therapy

Vasoactive medications are given in all forms of shock to improve the patient's hemodynamic stability when fluid therapy alone cannot maintain adequate MAP. Specific medications are selected to correct the particular hemodynamic alteration that is impeding cardiac output. These medications help increase the strength of myocardial contractility, regulate the heart rate, reduce myocardial resistance, and initiate vasoconstriction.

Vasoactive medications are selected for their action on receptors of the sympathetic nervous system. These receptors are known as alpha-adrenergic and beta-adrenergic receptors. Beta-adrenergic receptors are further classified as beta-1 and beta-2 adrenergic receptors. When alpha-adrenergic receptors are stimulated, blood vessels constrict in the cardiorespiratory and GI systems, skin, and kidneys. When beta-1 adrenergic receptors are stimulated, heart rate and myocardial contraction increase. When beta-2 adrenergic receptors are

stimulated, vasodilation occurs in the heart and skeletal muscles, and the bronchioles relax. The medications used in treating shock consist of various combinations of vasoactive medications to maximize tissue perfusion by stimulating or blocking the alpha- and beta-adrenergic receptors.

When vasoactive medications are given, vital signs must be monitored frequently (at least every 15 minutes until stable, or more often if indicated). Vasoactive medications should be given through a central venous line, because infiltration and extravasation of some vasoactive medications can cause tissue necrosis and sloughing (Maclaren, Mueller, & Dasta, 2019). Individual medication dosages are usually titrated by the nurse, who adjusts drip rates on the basis of the prescribed dose and target outcome parameter (e.g., BP, heart rate) and the patient's response. Dosages are changed to maintain the MAP at a physiologic level that ensures adequate tissue perfusion (usually greater than 65 mm Hg).



Quality and Safety Nursing Alert

Vasoactive medications should never be stopped abruptly, because this could cause severe hemodynamic instability, perpetuating the shock state.

Dosages of vasoactive medications must be tapered. When vasoactive medications are no longer needed or are necessary to a lesser extent, the infusion should be weaned with frequent monitoring of BP (e.g., every 15 minutes). **Table 11-2** presents some of the commonly prescribed vasoactive medications used in the treatment of shock.

Nutritional Support

Nutritional support is an important aspect of care for critically ill patients. Increased metabolic rates during shock increase energy requirements and therefore caloric requirements. Patients in shock may require more than 3000 calories daily. The release of catecholamines early in the shock continuum causes rapid depletion of glycogen stores. Nutritional energy requirements are then met by breaking down lean body mass. In this catabolic process, skeletal muscle mass is broken down even when the patient has large stores of fat or adipose tissue. Loss of skeletal muscle greatly prolongs the patient's recovery time.

Parenteral or enteral nutritional support should be initiated as soon as possible. Enteral nutrition is preferred, promoting GI function through direct exposure to nutrients and limiting infectious complications associated with parenteral feeding (Reintam, Blaser, Starkopf, et al., 2017). Implementation of

an evidence-based enteral feeding protocol that is tolerant of increased gastric residual volumes ensures the delivery of adequate nutrition to patients who are critically ill (Wang, Ding, Fang, et al., 2019). Gastric residual volume does not predict a patient's risk of aspiration (Wang et al., 2019). Implementing early enteral nutrition has been found to promote gut-mediated immunity, reduce metabolic response to stress, and improve overall patient morbidity and mortality (Reintam et al., 2017). (See [Chapter 39](#) for further discussion of monitoring gastric residual volumes.)

Chart 11-2

Collaborative Practice Interventions to Prevent Central Line–Associated Bloodstream Infections (CLABSIs)

Current best practices can include the implementation of specific evidence-based bundle interventions that when used together (i.e., as a “bundle”) improve patient outcomes. This chart outlines specific parameters for the central line bundled collaborative interventions that have been found to reduce central line–associated bloodstream infections (CLABSI).

What are the five key elements of the central line bundle?

- Hand hygiene
- Maximal sterile barrier precautions during line insertion (see later discussion)
- Chlorhexidine skin antisepsis
- Optimal catheter site selection with avoidance of using the femoral vein for central venous access in adult patients
- Daily review of line necessity, with prompt removal of unnecessary lines

When should hand hygiene be performed in the care of a patient with a central line?

- All clinicians who provide care to the patient should adhere to good hand hygiene practices, particularly:
 - Before and after palpating the catheter insertion site
 - With all dressing changes to the intravascular catheter access site
 - When hands are visibly soiled or contamination of hands is suspected
 - Before donning and after removing gloves

What changes can be made to improve hand hygiene?

- Implement a central line procedure checklist that requires that clinicians perform hand hygiene as an essential step in care.
- Post signage stating the importance of hand hygiene.
- Have soap and alcohol-based hand sanitizers prominently placed to facilitate hand hygiene practices.
- Model hand hygiene practices.
- Provide patient and family education and engage family in hand hygiene practices during visitation.

What are maximal sterile barrier precautions?

- These are implemented during central line insertion:
 - For the primary provider, this means strict adherence with wearing a cap, mask, sterile gown, and sterile gloves. The cap should cover all hair, and the mask should cover the nose and the mouth tightly. The nurse should also wear a cap and a mask.
 - For the patient, this means covering the patient from head to toe with a sterile drape, with a small opening for the site of insertion. If

a full-size drape is not available, two drapes may be applied to cover the patient, or the operating room may be consulted to determine how to procure full-size sterile drapes, because these are routinely used in surgical settings.

- Nurses should be empowered to enforce use of a central line checklist to be sure that all processes related to central line placement are properly executed for every line placed.

Which antiseptic should be used to prepare the patient's skin for central line insertion?

- Chlorhexidine skin antisepsis has been proven to provide better skin antisepsis than other antiseptic agents, such as povidone–iodine solution.
- An alcohol chlorhexidine antiseptic should be applied using a back-and-forth friction scrub for at least 30 s; this should not be wiped or blotted dry.
- The antiseptic solution should be allowed time to dry completely before the insertion site is punctured/accessed (approximately 2 min).

What nursing interventions are essential to reduce the risk of infection?

- Maintaining sterile technique when changing the central line dressing
- Always performing hand hygiene before manipulating or accessing the line ports
- Wearing clean gloves before accessing the line port
- Performing a 15- to 30-s “hub scrub” using chlorhexidine or alcohol and friction in a twisting motion on the access hub (reduces biofilm on the hub that may contain pathogens)
- Using chlorhexidine-containing dressings in patients older than 2 mo
- Consider using antiseptic-containing port protectors to cover connectors

When should central lines be discontinued?

- Assessment for removal of central lines should be included as part of the nurse's daily goal sheets.
- The time and date of central line placement should be recorded and evaluated by staff to aid in decision making.
- The need for the central line access should be reviewed as part of multidisciplinary rounds.
- During these rounds, the “line day” should be stated to remind everyone how long the central line has been in place (e.g., “Today is line day 6”).
- An appropriate time frame for regular review of the necessity for a central line should be identified, such as weekly, when central lines are placed for long-term use (e.g., chemotherapy, extended antibiotic administration).

Quality improvement processes that trend CLABSI rates and the adherence with CLABSI bundle prevention strategies have been found to effectively engage the multidisciplinary team in achieving goals to reduce infections related to central lines.

Adapted from Institute for Healthcare Improvement (IHI). (2012). How-to guide: Prevent central line-associated bloodstream infection. Retrieved on 9/10/2019 at: www.ihi.org/resources/Pages/Tools/HowtoGuidePreventCentralLineAssociatedBloodstreamInfection.aspx; Marschall, J., Mermel, L. A., Fakih, M., et al. (2014). Strategies to prevent central-line associated bloodstream infections in acute care hospitals: 2014 update. *Infection Control and Hospital Epidemiology*, 35(7), 753–771.

Stress ulcers occur frequently in acutely ill patients because of the compromised blood supply to the GI tract. Therefore, antacids, H₂ blockers (e.g., famotidine), and proton pump inhibitors (e.g., lansoprazole, esomeprazole magnesium) are prescribed to prevent ulcer formation by inhibiting gastric acid secretion or increasing gastric pH.

TABLE 11-2

Select Vasoactive Agents Used in Treating Shock

Medication	Desired Action in Shock	Disadvantages
Inotropic Agents		
Dobutamine	Improve contractility,	Increase oxygen demand of the heart
Dopamine	increase stroke	
Epinephrine	volume, increase	
Milrinone	cardiac output	
Vasodilators		
Nitroglycerin	Reduce preload and	Cause hypotension
Nitroprusside	afterload, reduce oxygen demand of heart	
Vasopressor Agents		
Norepinephrine	Increase blood pressure by vasoconstriction	Increase afterload, thereby increasing cardiac workload; compromise perfusion to skin, kidneys, lungs, gastrointestinal tract
Dopamine		
Phenylephrine		
Vasopressin		
Epinephrine		
Angiotensin II		

Adapted from Annane, D., Ouanes-Besbes, L., DeBacker, D., et al. (2018). A global perspective on vasoactive agents in shock. *Intensive Care Medicine*, 44(6), 833–346; Maclaren, R., Mueller, S. W., & Dasta, J. F. (2019). Use of vasopressors and inotropes in the pharmacotherapy of shock. In J. T. DiPiro, R. L. Talbert, G. C. Yee, et al. (Eds.). *Pharmacotherapy: A pathophysiologic approach* (11th ed.). New York: McGraw-Hill Medical.

Hypovolemic Shock

Hypovolemic shock, the most common type of shock, is characterized by decreased intravascular volume. Body fluid is contained in the intracellular and extracellular compartments. Intracellular fluid accounts for about two thirds of the total body water. The extracellular body fluid is found in one of two compartments: intravascular (inside blood vessels) or interstitial (surrounding tissues). The volume of interstitial fluid is about three to four times that of intravascular fluid. Hypovolemic shock occurs when there is a reduction in intravascular volume by 15% to 30%, which represents an approximate loss of 750 to 1500 mL of blood in a 70-kg (154-lb) person (American College of Surgeons, 2018).

Pathophysiology

Hypovolemic shock can be caused by external fluid losses, as in traumatic blood loss, or by internal fluid shifts, as in severe dehydration, severe edema, or ascites ([Chart 11-3](#)). Intravascular volume can be reduced by both fluid loss and fluid shifting between the intravascular and interstitial compartments.

Chart 11-3  RISK FACTORS	
Hypovolemic Shock	
External: Fluid Losses	Internal: Fluid Shifts
Trauma	Hemorrhage
Surgery	Burns
Vomiting	Ascites
Diarrhea	Peritonitis
Diuresis	Dehydration
Diabetic ketoacidosis	Necrotizing pancreatitis
Diabetes insipidus	

The sequence of events in hypovolemic shock begins with a decrease in the intravascular volume. This results in decreased venous return of blood to the heart and subsequent decreased ventricular filling. Decreased ventricular filling results in decreased stroke volume (amount of blood ejected from the heart) and decreased cardiac output. When cardiac output drops, BP drops and tissues cannot be adequately perfused ([Fig. 11-4](#)).



Medical Management

Major goals in the treatment of hypovolemic shock are to restore intravascular volume to reverse the sequence of events leading to inadequate tissue perfusion, to redistribute fluid volume, and to correct the underlying cause of the fluid loss as quickly as possible. Depending on the severity of shock and the patient's condition, often all three goals are addressed simultaneously.

Physiology/Pathophysiology

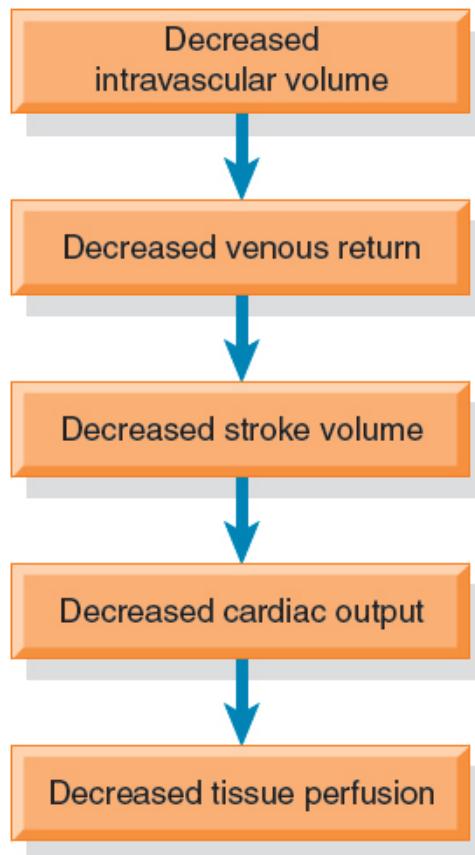


Figure 11-4 • Pathophysiologic sequence of events in hypovolemic shock.

Treatment of the Underlying Cause

If the patient is hemorrhaging, efforts are made to stop the bleeding. This may involve applying pressure to the bleeding site or surgical interventions to stop internal bleeding. If the cause of the hypovolemia is diarrhea or vomiting, medications to treat diarrhea and vomiting are given while efforts are made to identify and treat the cause. In older adult patients, dehydration may be the cause of hypovolemic shock.

Fluid and Blood Replacement

Beyond reversing the primary cause of the decreased intravascular volume, fluid replacement is of primary concern. At least two large-gauge IV lines are inserted to establish access for fluid administration. If an IV catheter cannot be quickly inserted, an intraosseous catheter may be used for access in the sternum, legs (tibia), or arms (humerus) to facilitate rapid fluid replacement (Clemency, Tanaka, May, et al., 2017). Multiple IV lines allow simultaneous

administration of fluid, medications, and blood component therapy if required. Because the goal of the fluid replacement is to restore intravascular volume, it is necessary to administer fluids that will remain in the intravascular compartment to avoid fluid shifts from the intravascular compartment into the intracellular compartment. [Table 11-3](#) summarizes the fluids commonly used in the treatment of shock.

As discussed earlier, crystalloid solutions such as lactated Ringer's solution or 0.9% sodium chloride solution are commonly used to treat hypovolemic shock, as large amounts of fluid must be given to restore intravascular volume. If hypovolemia is primarily due to blood loss, the American College of Surgeons (2018) recommends administration of 3 mL of crystalloid solution for each milliliter of estimated blood loss. This is referred to as the 3:1 rule. Colloid solutions (e.g., albumin) may also be used. Hetastarch and dextran solutions are not indicated for fluid administration because these agents interfere with platelet aggregation.

TABLE 11-3 Fluid Replacement in Shock^a

Fluids	Advantages	Disadvantages
Crystalloids		
Lactated Ringer's	Widely available; Lactate ion that helps buffer metabolic acidosis	Requires large volume of infusion; overresuscitation can result in pulmonary edema, abdominal compartment syndrome
0.9% Sodium chloride (normal saline solution)	Widely available	Requires large volume of infusion; can cause hypernatremia, hypokalemia, hyperchloremic metabolic acidosis; overresuscitation can result in pulmonary edema, abdominal compartment syndrome
Colloids		
Albumin (5%, 25%)	Rapidly expands plasma volume	Expensive; requires human donors; limited supply; can cause heart failure
Blood Products		
Plasma, packed red blood cells, and platelets	Rapidly replaces volume lost due to hemorrhage	Crossmatch type-specific blood is desired for optimal massive transfusion protocols to reduce transfusion-related complications (e.g., transfusion-related acute lung injury, hemolytic reactions, transfusion-associated circulatory overload)

^aDeliver a minimum of 30 mL/kg of crystalloid.

Adapted from Holcomb, J. B., Tiley, B. C., Baraniuk, S., et al. (2015). Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs a 1:1:2 ratio and mortality in patients with severe trauma. *JAMA*, 313(5), 471–482; Lewis, S. R., Pritchard, M. W., Evans, D. J. W., et al. (2018). Colloids versus crystalloids for fluid resuscitation in critically ill people. *The Cochrane Database of Systematic Reviews*, 8(8), CD000567;

Rhodes, A., Evans, L., Alhazzani, W., et al. (2017). Surviving Sepsis Campaign: International guidelines for management of sepsis and septic shock: 2016. *Critical Care Medicine*, 45(3), 486–552.

Blood products, which are also colloids, may need to be given, particularly if the cause of the hypovolemic shock is hemorrhage. The decision to give blood is based on the patient's lack of response to crystalloid resuscitation, the volume of blood lost, the need for hemoglobin to assist with oxygen transport, and the necessity to correct the patient's coagulopathy. Patients requiring massive transfusion respond better when blood products are given in a 1:1:1

ratio, meaning units of plasma, platelets, and packed red blood cells (Holcomb, Tiley, Baraniuk, et al., 2015).

Packed red blood cells are given to replenish the patient's oxygen-carrying capacity in conjunction with other fluids that will expand volume. Plasma and platelets are transfused to assist with coagulation and hemostasis. The need for transfusions is based on the patient's oxygenation needs and coagulation status, which are determined by vital signs, blood gas, chemistry, coagulation laboratory values, and clinical appearance.

Redistribution of Fluid

In addition to administering fluids to restore intravascular volume, positioning the patient properly assists fluid redistribution. PLR may be used to evaluate the patient's responsiveness to fluids and continued resuscitation efforts (Simmons & Ventetuolo, 2017). The nurse assesses for an improvement in the patient's vital signs, specifically a rise in the BP. Trendelenburg is not indicated as this position makes breathing difficult and does not increase BP or cardiac output (Bridges & Jarquin-Valdivia, 2005).

Pharmacologic Therapy

If fluid administration fails to reverse hypovolemic shock, then vasoactive medications that prevent cardiac failure are given. Medications are also given to reverse the cause of the dehydration. For example, insulin is given if dehydration is secondary to hyperglycemia, desmopressin is given for diabetes insipidus, antidiarrheal agents for diarrhea, and antiemetic medications for vomiting.



Nursing Management

Primary prevention of shock is an essential focus of nursing care. Hypovolemic shock can be prevented in some instances by closely monitoring patients who are at risk for fluid deficits and assisting with fluid replacement before intravascular volume is depleted. In other circumstances, nursing care focuses on assisting with treatment targeted at the cause of the shock and restoring intravascular volume.

General nursing measures include ensuring safe administration of prescribed fluids and medications and documenting their administration and effects. Volumetric IV pumps should be used to administer prescribed vasopressor medications. Another important nursing role is monitoring for complications and side effects of treatment and reporting them promptly.

Administering Blood and Fluids Safely

Administering blood transfusions safely is a vital nursing role. In emergency situations, it is important to acquire blood specimens quickly, to obtain a baseline complete blood count, and to type and crossmatch the blood in anticipation of blood transfusions. A patient who receives a transfusion of blood products must be monitored closely for adverse effects (see [Chapter 28](#)).

Fluid replacement complications can occur, especially when large volumes are given rapidly. Therefore, the nurse monitors the patient closely for cardiovascular overload and signs of difficulty breathing, a condition known as transfusion-associated circulatory overload. Transfusion-related ALI may occur and is characterized by pulmonary edema, hypoxemia, respiratory distress, and pulmonary infiltrates, usually within hours after massive transfusion (Vlaar & Kleinman, 2019). The risk of these complications is increased in older adults, in patients with preexisting cardiac disease, and with increasing number of blood products given. ACS is also a possible complication of excessive fluid resuscitation and may initially present with respiratory symptoms (difficulty breathing) and decreased urine output. Hemodynamic pressure, vital signs, arterial blood gases, serum lactate levels, hemoglobin and hematocrit levels, bladder pressure monitoring, and fluid intake and output (I&O) are among the parameters monitored. Temperature should also be monitored closely to ensure that rapid fluid resuscitation does not cause hypothermia. IV fluids may need to be warmed when large volumes are given. Physical assessment focuses on observing the jugular veins for distention and monitoring jugular venous pressure. Jugular venous pressure is low in hypovolemic shock; it increases with effective treatment and is significantly increased with fluid overload and heart failure. The nurse must monitor cardiac and respiratory status closely and report changes in BP, pulse pressure, CVP, heart rate and rhythm, and lung sounds to the primary provider.

Implementing Other Measures

Oxygen is given to increase the amount of oxygen carried by available hemoglobin in the blood. A patient who is confused may feel apprehensive with an oxygen mask or cannula in place, and frequent explanations about the need for the mask may reduce some of the patient's fear and anxiety. Simultaneously, the nurse must direct efforts to the safety and comfort of the patient.

Cardiogenic Shock

Cardiogenic shock occurs when the heart's ability to contract and to pump blood is impaired and the supply of oxygen is inadequate for the heart and the tissues. The causes of cardiogenic shock are known as either coronary or noncoronary. Coronary cardiogenic shock is more common than noncoronary

cardiogenic shock and is seen most often in patients with acute MI resulting in damage to a significant portion of the left ventricular myocardium (Wilcox, 2019). Patients who experience an anterior wall MI are at greatest risk for cardiogenic shock because of the potentially extensive damage to the left ventricle caused by occlusion of the left anterior descending coronary artery. Noncoronary causes of cardiogenic shock are related to conditions that stress the myocardium (e.g., severe hypoxemia, acidosis, hypoglycemia, hypocalcemia, tension pneumothorax) as well as conditions that result in ineffective myocardial function (e.g., cardiomyopathies, valvular damage, cardiac tamponade, arrhythmias).

Pathophysiology

In cardiogenic shock, cardiac output, which is a function of both stroke volume and heart rate, is compromised. When stroke volume and heart rate decrease or become erratic, BP falls and tissue perfusion is reduced. Blood supply for tissues and organs and for the heart muscle itself is inadequate, resulting in impaired tissue perfusion. Because impaired tissue perfusion weakens the heart and impairs its ability to pump, the ventricle does not fully eject its volume of blood during systole. As a result, fluid accumulates in the lungs. This sequence of events can occur rapidly or over a period of days ([Fig. 11-5](#)).

Clinical Manifestations

Patients in cardiogenic shock may experience the pain of angina, develop arrhythmias, complain of fatigue, express feelings of doom, and show signs of hemodynamic instability.

Physiology/Pathophysiology

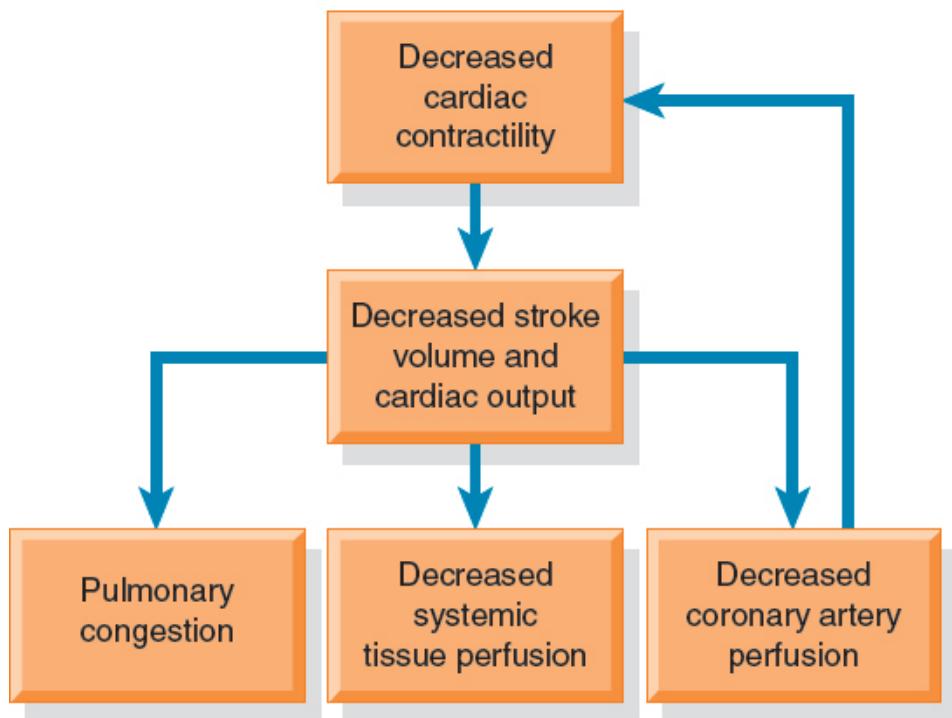


Figure 11-5 • Pathophysiologic sequence of events in cardiogenic shock.

Assessment and Diagnostic Findings

To assess the degree of myocardial damage, laboratory biomarkers for ventricular dysfunction (e.g., B-type natriuretic peptide), cardiac enzyme levels and biomarkers (cTn-I), and serum lactate are measured. In addition, a transthoracic echocardiography may be performed at the bedside, and serial 12-lead electrocardiograms are obtained (Bellumkonda, Gul, & Masri, 2018; Wilcox, 2019). Continuous ECG and ST segment monitoring is also done to closely monitor the patient for ischemic changes.



Medical Management

The goals of medical management in cardiogenic shock are to limit further myocardial damage and preserve the healthy myocardium and to improve cardiac function by increasing cardiac contractility, decreasing ventricular afterload, or both (Wilcox, 2019). In general, these goals are achieved by increasing oxygen supply to the heart muscle while reducing oxygen demands.

Correction of Underlying Causes

As with all forms of shock, the underlying cause of cardiogenic shock must be corrected. It is necessary first to treat the oxygenation needs of the heart muscle to ensure its continued ability to pump blood to other organs. In the case of coronary cardiogenic shock (e.g., acute coronary syndromes, ischemic cardiomyopathy, nonischemic cardiomyopathies, and myocarditis), the patient may require thrombolytic (fibrinolytic) therapy, a percutaneous coronary intervention, coronary artery bypass graft surgery, IABP therapy, ventricular assist device, or some combination of these treatments (Bellumkonda et al., 2018; Wilcox, 2019). In the case of noncoronary cardiogenic shock, interventions focus on correcting the underlying cause, such as replacement of an impaired cardiac valve, correction of an arrhythmia, correction of acidosis and electrolyte disturbances, or treatment of a tension pneumothorax. If the cause of the cardiogenic shock was related to a cardiac arrest, once the patient is successfully resuscitated, targeted temperature management, also called *therapeutic hypothermia*, may be initiated to actively lower the body temperature to a targeted core temperature (e.g., 32°C [89.6°F] to 36°C [96.8°F]) to preserve neurologic function (American Heart Association, 2018). (See [Chapter 23](#) for more information regarding MI.)

Initiation of First-Line Treatment

Treatment priorities for patients in cardiogenic shock are focused upon ensuring adequate oxygenation, pain control, and maintaining hemodynamic stability.

Oxygenation

In the early stages of shock, if the patient's oxygen saturation is less than 90%, supplemental oxygen is given by nasal cannula at a rate of 2 to 6 L/min (Neto, 2018). Monitoring of arterial blood gas values, pulse oximetry values, and ventilatory effort (i.e., work of breathing) helps determine whether the patient requires a more aggressive method of oxygen delivery (including noninvasive and invasive mechanical ventilation).

Pain Control

If a patient experiences chest pain, IV morphine may be given for pain relief. In addition to relieving pain, morphine may dilate the blood vessels, reducing the workload of the heart by both decreasing the cardiac filling pressure (preload) and reducing the pressure against which the heart muscle has to eject blood (afterload). However, recent evidence suggests morphine may adversely affect antiplatelet agents used to treat the ischemic event; thus, the benefits of morphine may not outweigh its risks (McCarthy, Bhambhani, Pomerantsev, et al., 2018; Neto, 2018).

Hemodynamic Monitoring

Hemodynamic monitoring is initiated to assess the patient's response to treatment. In many institutions, this is performed in the intensive care unit (ICU), where an arterial line can be inserted. The arterial line enables accurate and continuous monitoring of BP and provides a port from which to obtain frequent arterial blood samples without having to perform repeated arterial punctures. A multilumen CVP and PA catheter may be inserted to allow measurement of myocardial filling pressures, pulmonary artery pressures, cardiac output, and pulmonary and systemic resistance. (For more information, see [Chapter 21](#).)

Fluid Therapy

Appropriate fluid administration is also necessary in the treatment of cardiogenic shock. Administration of fluids must be monitored closely to detect signs of fluid overload. Incremental IV fluid boluses are cautiously given to determine optimal filling pressures for improving cardiac output.



Quality and Safety Nursing Alert

A fluid bolus should never be given rapidly, because rapid fluid administration in patients with cardiac failure may result in acute pulmonary edema.

Pharmacologic Therapy

Vasoactive medication therapy consists of multiple pharmacologic strategies to restore and maintain adequate cardiac output. In coronary cardiogenic shock, the aims of vasoactive medication therapy are improved cardiac contractility, decreased preload and afterload, and stabilized heart rate and rhythm.

Because improving contractility and decreasing cardiac workload are opposing pharmacologic actions, two types of medications may be given in combination: inotropic agents and vasodilators. Inotropic medications increase cardiac output by mimicking the action of the sympathetic nervous system, activating myocardial receptors to increase myocardial contractility (inotropic action), or increasing the heart rate (chronotropic action). These agents may also enhance vascular tone, increasing preload. Vasodilators are used primarily to decrease afterload, reducing the workload of the heart and oxygen demand. Vasodilators also decrease preload. Medications commonly combined to treat cardiogenic shock include dobutamine, nitroglycerin, and dopamine (see [Table 11-2](#)).

Dobutamine

Dobutamine produces inotropic effects by stimulating myocardial beta-receptors, increasing the strength of myocardial activity and improving cardiac output. Myocardial alpha-adrenergic receptors are also stimulated, resulting in decreased pulmonary and systemic vascular resistance (decreased afterload) (Maclaren et al., 2019).

Nitroglycerin

IV nitroglycerin in low doses acts as a venous vasodilator and therefore reduces preload. At higher doses, nitroglycerin causes arterial vasodilation and therefore reduces afterload as well. These actions, in combination with dobutamine, increase cardiac output while minimizing cardiac workload. In addition, vasodilation enhances blood flow to the myocardium, improving oxygen delivery to the weakened heart muscle (Maclaren et al., 2019; Wilcox, 2019).

Dopamine

Dopamine is a sympathomimetic agent that has varying vasoactive effects depending on the dosage. It may be used with dobutamine and nitroglycerin to improve tissue perfusion. Doses of 2 to 8 $\mu\text{g}/\text{kg}/\text{min}$ improve contractility (inotropic action), slightly increase the heart rate (chronotropic action), and may increase cardiac output. Doses that are higher than 8 $\mu\text{g}/\text{kg}/\text{min}$ predominantly cause vasoconstriction, which increases afterload and thus increases cardiac workload. Because this effect is undesirable in patients with cardiogenic shock, dopamine doses must be carefully titrated.

In severe metabolic acidosis, which occurs in the later stages of shock, metabolic acidosis must first be corrected to ensure maximum effectiveness of vasoactive medications (Maclaren et al., 2019).

Other Vasoactive Medications

Additional vasoactive agents that may be used in managing cardiogenic shock include norepinephrine, epinephrine, milrinone, vasopressin, phenylephrine, and angiotensin II. Each of these medications stimulates different receptors of the sympathetic nervous system. A combination of these medications may be prescribed, depending on the patient's response to treatment. All vasoactive medications have adverse effects, making specific medications more useful than others at different stages of shock (see [Table 11-2](#)). Diuretics such as furosemide may be given to reduce the workload of the heart by reducing fluid accumulation.

Antiarrhythmic Medications

Multiple factors, such as hypoxemia, electrolyte imbalances, and acid–base imbalances, contribute to serious cardiac arrhythmias in all patients with shock. In addition, as a compensatory response to decreased cardiac output and BP, the heart rate increases beyond normal limits. This impedes cardiac output further by shortening diastole and thereby decreasing the time for ventricular

filling. Consequently, antiarrhythmic medications are required to stabilize the heart rate. General principles regarding the administration of vasoactive medications are discussed later in this chapter. (For a full discussion of cardiac arrhythmias as well as commonly prescribed medications, see [Chapter 22](#).)

Mechanical Assistive Devices

If cardiac output does not improve despite supplemental oxygen, vasoactive medications, and fluid boluses, mechanical assistive devices are used temporarily to improve the heart's ability to pump. Intra-aortic balloon counterpulsation with an IABP is one means of providing temporary circulatory assistance (Hochman & Reyentovich, 2019). The IABP is a catheter with an inflatable balloon at the end. The catheter is usually inserted through the femoral artery and threaded toward the heart, and the balloon is positioned in the descending thoracic aorta ([Fig. 11-6](#)). The IABP uses internal counterpulsation through the regular inflation and deflation of the balloon to augment the pumping action of the heart. It inflates during diastole, increasing the pressure in the aorta during diastole and therefore increasing blood flow through the coronary and peripheral arteries. It deflates just before systole, lessening the pressure within the aorta before left ventricular contraction, decreasing the amount of resistance the heart has to overcome to eject blood and therefore decreasing left ventricular workload. The device is connected to a console that synchronizes the inflation and deflation of the balloon with the ECG or the arterial pressure (as indicators for systole and diastole). Hemodynamic monitoring is often used to determine the patient's response to the IABP. The IABP provides short-term (days) support for the failing myocardium.

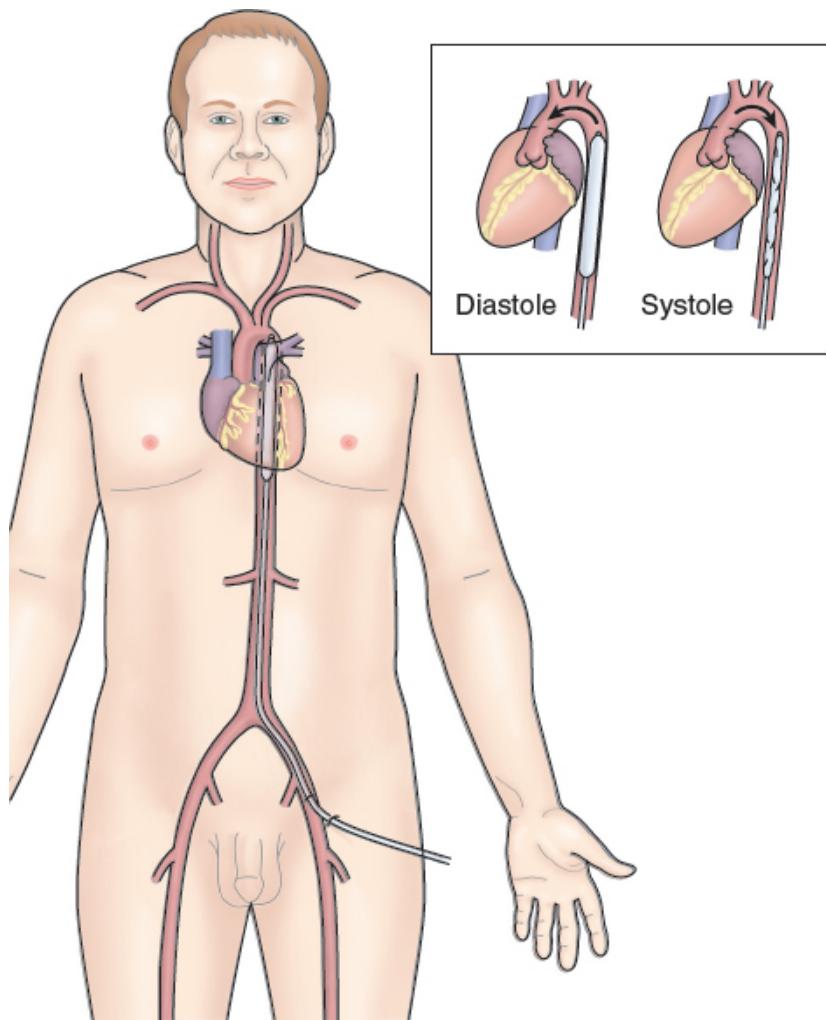


Figure 11-6 • The intra-aortic balloon pump inflates at the beginning of diastole, which results in increased perfusion of the coronary and peripheral arteries. It deflates just before systole, which results in a decrease in afterload (resistance to ejection) and in the left ventricular workload.

Other means of mechanical assistance include left and right ventricular assist devices and total temporary artificial hearts (see [Chapters 24](#) and [25](#)). Another short-term means of providing cardiac or pulmonary support to the patient in cardiogenic shock is through an extracorporeal device similar to the cardiopulmonary bypass (CPB) system used in open-heart surgery (see [Chapter 23](#)). CPB is used only in emergency situations until definitive treatment, such as heart transplantation, can be initiated.



Nursing Management

The role of the nurse managing the care of a patient with cardiogenic shock revolves around preventing its serious complications, monitoring hemodynamics, administering medications and fluids, maintaining intra-aortic counterpulsation as indicated, and promoting safety and comfort.

Preventing Cardiogenic Shock

Identifying at-risk patients early, promoting adequate oxygenation of the heart muscle, and decreasing cardiac workload can prevent cardiogenic shock. This can be accomplished by conserving the patient's energy, promptly relieving angina, and administering supplemental oxygen. Often, however, cardiogenic shock cannot be prevented. In such instances, nursing management includes working with other members of the health care team to prevent shock from progressing and to restore adequate cardiac function and tissue perfusion.

Monitoring Hemodynamic Status

A major role of the nurse is monitoring the patient's hemodynamic and cardiac status. Arterial lines and ECG monitoring equipment must be well maintained and functioning properly. The nurse anticipates the medications, IV fluids, and equipment that might be used and is ready to assist in implementing these measures. Changes in hemodynamic, cardiac, and pulmonary status and laboratory values are documented and reported promptly. In addition, adventitious breath sounds, changes in cardiac rhythm, and other abnormal physical assessment findings are reported immediately.

Administering Medications and Intravenous Fluids

The nurse plays a critical role in the safe and accurate administration of IV fluids and medications. Fluid overload and pulmonary edema are risks because of ineffective cardiac function and accumulation of blood and fluid in the pulmonary tissues. The nurse documents medications and treatments that are given as well as the patient's response to treatment.

The nurse must be knowledgeable about the desired effects as well as the side effects of medications. For example, the nurse monitors the patient for decreased BP after administering morphine or nitroglycerin. Arterial and venous puncture sites must be observed for bleeding, and pressure must be applied at the sites if bleeding occurs. IV infusions must be observed closely because tissue necrosis and sloughing may occur if vasoconstrictor medications infiltrate the tissues. When possible, vasoactive medications should be given using central IV lines (Bauer et al., 2019). Furthermore, the need for the central IV access devices should be reviewed daily to reduce the risk of CLABSI (Marschall, Mermel, Fakih, et al., 2014). The nurse must also monitor urine output, serum electrolytes, BUN, and serum creatinine levels to

detect decreased renal function secondary to the effects of cardiogenic shock or its treatment.

Maintaining Intra-Aortic Balloon Counterpulsation

The nurse plays a critical role in caring for the patient receiving intra-aortic balloon counterpulsation. The nurse makes ongoing timing adjustments of the balloon pump to maximize its effectiveness by synchronizing it with the cardiac cycle. The patient is at risk for circulatory compromise to the leg on the side where the catheter for the balloon has been inserted; therefore, the nurse must check the neurovascular status of the lower extremities frequently.

Enhancing Safety and Comfort

The nurse must take an active role in safeguarding the patient, enhancing comfort, and reducing anxiety. This includes administering medication to relieve chest pain, preventing infection at the multiple arterial and venous line insertion sites, protecting the skin, and monitoring respiratory and renal function. Proper positioning of the patient promotes effective breathing without decreasing BP and may also increase patient comfort while reducing anxiety.

Brief explanations about procedures that are being performed and the use of comforting touch often provide reassurance to the patient and the family. The family is usually anxious and benefits from opportunities to see and talk to the patient. Explanations of treatments and the patient's responses are often comforting to family members.

Family presence during a patient's critical illness has been a concern for family members, patients, nurses and other health care providers. Evidence-based clinical practice guidelines have outlined several elements that may enhance and support the patient and family experience during critical illness. Davidson and colleagues (2017) identified the importance of frequent, clear communication, an environment which encourages comfortable visitation, flexible visitation policies, including the option for family to stay overnight, and patient and family engagement in clinical decisions as mechanisms to enhance family support. A qualitative research study conducted by Wong and colleagues (2020) explored family perspectives of participation in patient care in the adult ICU. The nurse researchers interviewed and observed 30 family members. Findings demonstrated that families' perceptions of their contribution to the patient's psychosocial and emotional well-being were influenced by their engagement in care. See the Nursing Research Profile in [Chart 11-4](#).

Distributive Shock

Distributive shock occurs when intravascular volume pools in peripheral blood vessels. This abnormal displacement of intravascular volume causes a relative hypovolemia because not enough blood returns to the heart, which leads to inadequate tissue perfusion. The ability of the blood vessels to constrict helps return the blood to the heart. Vascular tone is determined both by central regulatory mechanisms, as in BP regulation, and by local regulatory mechanisms, such as tissue demands for oxygen and nutrients. Therefore, distributive shock can be caused by either a loss of sympathetic tone or a release of biochemical mediators from cells that causes vasodilation.

The varied mechanisms leading to the initial vasodilation in distributive shock provide the basis for the further subclassification of shock into three types: septic shock, neurogenic shock, and anaphylactic shock. These subtypes of distributive shock cause variations in the pathophysiologic chain of events and are explained below separately. In all types of distributive shock, massive arterial and venous dilation promotes peripheral pooling of blood. Arterial dilation reduces systemic vascular resistance. Initially, cardiac output can be high, both from the reduction in afterload (systemic vascular resistance) and from the heart muscle's increased effort to maintain perfusion despite the incompetent vasculature. Pooling of blood in the periphery results in decreased venous return. Decreased venous return results in decreased stroke volume and decreased cardiac output. Decreased cardiac output, in turn, causes decreased BP and ultimately decreased tissue perfusion. [Figure 11-7](#) presents the pathophysiologic sequence of events in distributive shock.

Chart 11-4



NURSING RESEARCH PROFILE

Family Participation in the Intensive Care Unit

Wong, P., Redley, B., Digby, R., et al. (2020). Families' perspectives of participation in patient care in an adult intensive care unit: A qualitative study. *Australian Critical Care*, 33(4), 317–325.

Purpose

The aim of this study was to describe family participation in patient care in the adult intensive care unit (ICU).

Design

This was a qualitative, descriptive observational study that enrolled a convenience sample of 30 family members who had an adult family member admitted to the ICU for more than 72 h. Researchers observed the family members' engagement in patient care activities and conducted semi-structured interviews focused on their experiences and perceptions of providing care.

Findings

Most of the family participants (77%) were female. Ages of the patient and family member were similar ranging from 28 to 73, with a median age for family members of 53. Researchers observed 193 activities in which 25% involved physical care for the patient, while the remaining 75% of activities were intangible care interventions such as communication and psychosocial/emotional care. Themes that emerged from the interviews revealed that the families felt part of the health care team and thus wanted to support the care and recovery of the loved one. Family members reported being motivated to provide care because they wanted the best care for the patient, desired to know the prognosis, treatment, and condition of the patient, and desired to help the patient recover. Lastly, family members wanted to advocate for their relatives, provide psychosocial and emotional care, and be supportive during medical treatments. The researchers observed that the physical environment was either a contributor or barrier to the family member's ability to engage in care. Equipment sometimes created physical barriers that made it hard for family to get close to the patient. Staff support and communication either encouraged or discouraged family participation in care. When nurses communicated openly, keeping families informed, family members felt more welcomed and better able to support the patient's treatment plans.

Nursing Implications

Most families wish to be engaged and to advocate for their loved ones in the treatment decision making process. The ability for family to feel part of the health care team and develop trust in the plan of care is influenced by positive relationships with the nursing and medical staff as well as the welcoming nature of the environment. Patient- and family-centered care is

facilitated when families are considered collaborative partners in the care of adult patients who are critically ill.

Physiology/Pathophysiology

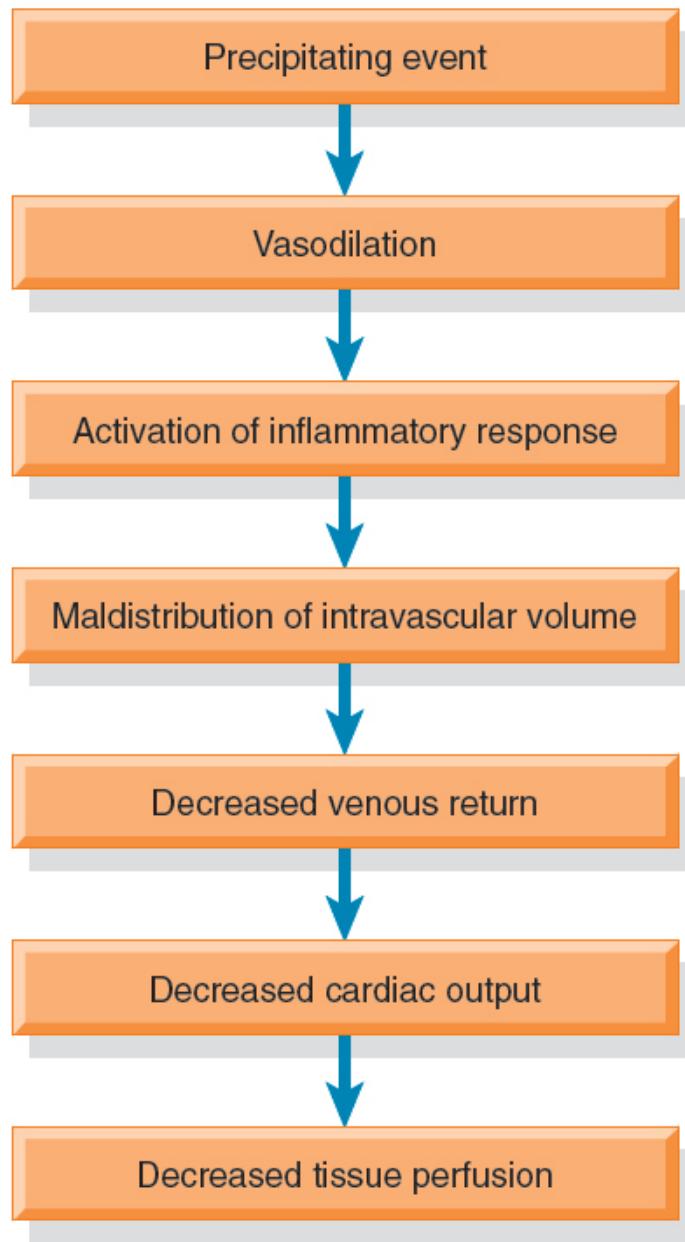


Figure 11-7 • Pathophysiologic sequence of events in distributive shock.

Sepsis and Septic Shock

Septic shock, the most common type of distributive shock, is caused by widespread infection or sepsis. According to the Third International Consensus Definitions for Sepsis and Septic Shock (*Sepsis-3*) Task Force, **sepsis** is “life-threatening organ dysfunction caused by a dysregulated host response to infection (Singer et al., 2016, p. 804),” and **septic shock** is “a subset of sepsis in which underlying circulatory and cellular metabolism abnormalities are profound enough to substantially increase mortality (Singer et al., 2016, p. 806).”

Despite the increased sophistication of antibiotic therapy, the incidence of both sepsis and septic shock has continued to rise; today sepsis and septic shock are the leading causes of death in noncoronary ICU patients. Worldwide, sepsis impacts more than 31 million people, and more than 6 million people die from it annually (Global Sepsis Alliance, 2017). The number of hospital admissions related to sepsis has increased threefold over the last decade (Surviving Sepsis Campaign, 2019). Identifying and aggressively treating the source of infection and quickly restoring tissue perfusion are important interventions that may positively influence clinical outcomes.

Hospital-acquired conditions, which may include hospital-associated infections (i.e., infections not present at the time of admission to the health care setting) in critically ill patients that may progress to septic shock, most frequently originate in the bloodstream (bacteremia), lungs (pneumonia), and urinary tract (urosepsis). Other infections include intra-abdominal infections and wound infections. Of increasing concern are bacteremias associated with intravascular catheters and indwelling urinary catheters (Centers for Disease Control and Prevention [CDC], 2019).

Additional risk factors that contribute to the growing incidence of sepsis are the increased use of invasive procedures and indwelling medical devices, the increased number of antibiotic-resistant microorganisms, and the aging population (CDC, 2019). Older adult patients are at particular risk for sepsis because of decreased physiologic reserves, an aging immune system, comorbid conditions and often nonspecific presentation of infection (Rowe & McKoy, 2017) (see [Chapter 8](#)). Other patients at risk are those undergoing surgical and other invasive procedures, especially patients who have undergone emergency surgery or multiple surgeries (Rhodes et al., 2017); those with malnutrition or immunosuppression; and those with chronic illness such as diabetes, hepatitis, chronic kidney disease, and immunodeficiency disorders (Keeley, Hine, & Nsutebu, 2017) ([Chart 11-5](#)).

The incidence of sepsis can be reduced by using strict infection control practices, beginning with thorough hand hygiene techniques (Dunne, Kingston, Slevin, et al., 2018). Other interventions include implementing programs to prevent central line infections and ventilator-associated events (e.g., aspiration) and pneumonia; ensuring early removal of invasive devices that are no longer necessary (e.g., indwelling urinary catheters); promoting

early ambulation and timely débridement of wounds; and adhering to standard precautions and infection prevention/control practices, including the use of meticulous aseptic technique and proper cleaning of equipment and the patient environment.

Chart 11-5 RISK FACTORS

Distributive Shock

Septic Shock

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

Neurogenic Shock

- Spinal cord injury
- Spinal anesthesia
- Depressant action of medications

Anaphylactic Shock

- History of medication sensitivity
- Transfusion reaction
- History of reaction to insect bites/stings
- Food allergies
- Latex sensitivity

Adapted from Keeley, A., Hine, P., & Nsutebu, E. (2017). The recognition and management of sepsis and septic shock: A guide for non-intensivists. *Postgraduate Medicine Journal*, 93(1104), 626–634; Pajno, G. B., Fernandez-Rivas, M., Arasi, S., et al. (2018). EAACI guidelines on allergen immunotherapy: IgE-mediated food allergy. *Allergy*, 73(4), 799–815; Rhodes, A., Evans, L., Alhazzani, W., et al. (2017). Surviving Sepsis Campaign: International guidelines for management of sepsis and septic shock: 2016. *Critical Care Medicine*, 45(3), 486–552; Singer, M., Deutschman, C. S., Seymour, C. W., et al. (2016). The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA*, 315(8), 801–810.

Pathophysiology

Gram-negative bacteria traditionally were the most commonly implicated microorganisms in sepsis. However, there is an increased incidence of gram-positive bacterial infections, viral infections, and fungal infections that can also cause sepsis (Keeley et al., 2017). Although a site of infection is identified in most cases, in up to 30% of patients with sepsis an identifiable source of infection is not determined (Surviving Sepsis Campaign, 2019).

When microorganisms invade body tissues, patients exhibit an immune response. This immune response provokes the activation of biochemical cytokines and mediators associated with an inflammatory response and produces a complex cascade of physiologic events that leads to poor tissue perfusion. Increased capillary permeability results in fluid seeping from the capillaries. Capillary instability and vasodilation interrupt the body's ability to provide adequate perfusion, oxygen, and nutrients to the tissues and cells. The widespread inflammatory response that occurs is called the **systemic inflammatory response syndrome** (SIRS). SIRS, which is a type of cytokine release syndrome and is often referred to as cytokine storm, results from a clinical insult that initiates an inflammatory response that is systemic, rather than localized to the site of the insult (see [Chapter 12](#) for further discussion of cytokine release syndrome). The insult may be a significant injury (e.g., multitrauma) or an infection (e.g., sepsis). A patient presenting with manifestations of SIRS may be exhibiting a protective inflammatory response to the initiating insult or may be exhibiting a response to infection, which may lead to sepsis. The clinical criteria used to identify SIRS, which include a temperature greater than 38.3°C (101°F) or less than 36°C (96.8°F), tachycardia, tachypnea, and a white blood cell (WBC) count greater than 12,000 cells/mm³, less than 4000 cells/mm³, or greater than 10% immature WBC (bands), have been found to be not helpful in diagnosing sepsis, however (Singer et al., 2016).

In addition to SIRS, proinflammatory and anti-inflammatory cytokines released during the inflammatory response activate the coagulation system, which begins to form clots regardless of whether or not bleeding is present (Seymour & Angus, 2018). This results not only in microvascular occlusions that further disrupt cellular perfusion but also in an inappropriate consumption of clotting factors. Imbalances among the inflammatory response and the clotting and fibrinolysis cascades are considered critical elements of the devastating physiologic progression that occurs in patients with sepsis.

Sepsis is an evolving process that may result in septic shock and life-threatening organ dysfunction if not recognized and treated early. In the early stage of septic shock, BP may remain within normal limits, or the patient may be hypotensive but responsive to fluids. The heart rate increases, progressing to tachycardia. Hyperthermia and fever, with warm, flushed skin and bounding pulses, are present. The respiratory rate is elevated. Urinary output may remain at normal levels or decrease. GI status may be compromised, as evidenced by

nausea, vomiting, diarrhea, or decreased gastric motility. Hepatic dysfunction is evidenced by rising bilirubin levels and worsening coagulopathies (e.g., decreasing platelet counts). Signs of hypermetabolism include increased serum glucose and insulin resistance. Subtle changes in mental status, such as confusion or agitation, may be present. The lactate level is elevated because of the maldistribution of blood. Inflammatory markers such as WBC counts, plasma C-reactive protein (CRP), and procalcitonin levels are also elevated (Rhodes et al., 2017; Seymour & Angus, 2018; Singer et al., 2016).

As sepsis progresses, tissues become less perfused and acidotic, compensation begins to fail, and the patient begins to show signs of organ dysfunction. The cardiovascular system also begins to fail, the BP does not respond to fluid resuscitation and vasoactive agents, and signs of end-organ damage are evident (e.g., AKI, pulmonary dysfunction, hepatic dysfunction, confusion progressing to nonresponsiveness). As sepsis progresses to septic shock, the BP drops and the skin becomes cool, pale, and mottled. Temperature may be normal or below normal. Heart and respiratory rates remain rapid. Urine production ceases, and multiple organ dysfunction progressing to death occurs.



Medical Management

A significant body of research has been conducted in the past few decades that is aimed at reducing the morbidity and mortality caused by sepsis and septic shock. In 1991, 2003, 2008, 2012, and again in 2016 (i.e., *Sepsis-3*), critical-care experts systematically reevaluated the body of research and provided evidence-based recommendations for the acute management of patients with sepsis and septic shock (Rhodes et al., 2017; Singer et al., 2016; Surviving Sepsis Campaign, 2019). Developing and implementing protocols, sepsis bundles, that focus on prevention and early detection and management of patients with sepsis have reduced the mortality of hospitalized patients (Kahn, Davis, Yabes, et al., 2019). *Sepsis-3* provides an overview of key concepts and principles germane to sepsis that must be understood in order for primary providers and nurses to be able to identify and manage patients with sepsis, including the following (Levy & Townsend, 2019; Singer et al., 2016):

- As the leading cause of death from infection, sepsis must be recognized and treated promptly.
- Sepsis is different from infection in that in sepsis there is a dysregulated host response with organ dysfunction. An infection may cause specific organ dysfunction without a dysregulated host response.
- Sepsis is caused by an interplay between infectious pathogens and a myriad of patient-specific risks, including genetics, age, and the

- presence of other diseases/disorders.
- The organ dysfunction that occurs with sepsis may not be readily apparent; on the contrary, a new onset of organ dysfunction may be caused by an unrecognized infectious process.

Research efforts are focusing on better identification and early aggressive treatment of patients with sepsis, rapid and effective restoration of tissue perfusion, evaluation and treatment of the patient's immune response, and treatment of dysregulation of the coagulation system that occurs with sepsis (Keeley et al., 2017; Makic & Bridges, 2018).

Correction of Underlying Causes

Current treatment of sepsis and septic shock involves rapid identification and elimination of the cause of infection. Current goals are to identify and initiate treatment for patients in early sepsis within 1 hour to optimize patient outcomes (Levy et al., 2018; Levy & Townsend, 2019). Several evidence-based screening tools can be used to help identify patients for sepsis (see later discussion of assessment tools under the section Nursing Management).

In an effort to continue to reduce deaths from sepsis, Centers for Medicare and Medicaid Services (CMS) has identified sepsis and sepsis bundle adherence as a *Core Measure*. The focus on adherence with these evidence-based bundled interventions is to foster early recognition and interventions in patients with sepsis so that patient outcomes improve. These assessments and interventions are collectively referred to as the *Sepsis Bundles* ([Chart 11-6](#)).

Rapid identification of the infectious source is a critical element in managing sepsis. Specimens of blood, sputum, urine, wound drainage, and tips of invasive catheters are collected for culture using aseptic technique. IV lines are removed and reinserted at alternate sites. If possible, urinary catheters are removed or changed. Any abscesses are drained, and necrotic areas are débrided. All cultures should be obtained prior to antibiotic administration. Current guidelines suggest that antibiotics should be initiated within the first hour of treatment of a patient with sepsis (Surviving Sepsis Campaign, 2019).

Fluid Replacement Therapy

Fluid replacement must be instituted to correct tissue hypoperfusion that results from the incompetent vasculature and the inflammatory response. Reestablishing tissue perfusion through aggressive fluid resuscitation is key to the management of sepsis and septic shock (Rhodes et al., 2017; Singer et al., 2016). An initial fluid challenge, which includes an IV infusion of at least 30 mL/kg of crystalloids over 30 minutes, may be required to aggressively treat sepsis-induced tissue hypoperfusion. In addition to monitoring BP, patient mentation, respiratory rate, fluid responsiveness after PLR, urine output, and serum lactate levels are monitored to assess effectiveness of fluid resuscitation.

Pharmacologic Therapy

If the infecting organism is unknown, broad-spectrum antibiotic agents are started until culture and sensitivity reports are received (Levy & Townsend, 2019; Rhodes et al., 2017), at which time the antibiotic agents may be changed to agents that are more specific to the infecting organism and less toxic to the patient.

If fluid therapy alone does not effectively improve tissue perfusion, vasopressor agents, specifically norepinephrine or dopamine, may be initiated to achieve a MAP of 65 mm Hg or higher. Inotropic agents may also be given to provide pharmacologic support to the myocardium. Packed red blood cells may be prescribed to support oxygen delivery and transport to the tissues. Neuromuscular blockade agents and sedation agents may be required to reduce metabolic demands and provide comfort to the patient. Deep vein thrombosis (DVT) prophylaxis with low-dose unfractionated heparin or low-molecular-weight heparin, in combination with mechanical prophylaxis (e.g., sequential compression devices) should be initiated, as well as medications for stress ulcer prophylaxis (e.g., H₂-blocking agents, proton pump inhibitors).

Chart 11-6

Surviving Sepsis Campaign Bundle and CMS Core Measure Monitoring Metrics

Complete within 1 h of patient presentation/symptoms

- Measure lactate level. Remeasure if initial lactate is >2 mmol/L
- Obtain blood cultures prior to administration of antibiotics
- Administer broad-spectrum antibiotics
- Begin rapid administration of 30 mL/kg crystalloid for hypotension or lactate ≥ 4 mmol/L (within 30 min)
- Administer vasopressors if patients is hypotensive during or after fluid resuscitation to maintain MAP ≥ 65 mm Hg

Reprinted with permission from Levy, M., Evans, L., & Rhodes, A. (2018). The Surviving Sepsis Campaign bundle: 2018 update. *Critical Care Medicine*, 46(6), 997–1000.

CMS Core Measure Metrics and Surviving Sepsis Bundles 2016:

Complete within 3 h of patient presentation/symptoms

- Obtain serum lactate level
- Obtain blood culture prior to administration of antibiotics
- Administer prescribed broad-spectrum antibiotics
- Initiate aggressive fluid resuscitation in patients with hypotension or elevated serum lactate (>4 mmol/L):
 - Minimum initial fluid bolus of 30 mL/kg using crystalloid solutions

Complete as soon as possible or within the first 6 h of patient presentation/symptoms

- Begin vasopressor agents if hypotension is not improved (MAP <65 mm Hg) after initial fluid resuscitation
- If hypotension persists after initial fluid administration (MAP <65 mm Hg) or initial lactate was ≥ 4 mmol/L, reassess intravascular volume status and tissue perfusion using two of the following assessment parameters, including vital signs, capillary refill, pulse, and skin findings, or two of the following:
 - Measurement of CVP and/or ScVO_2 (goal $>70\%$)
 - Bedside cardiovascular ultrasound
 - Dynamic assessment of fluid responsiveness with passive leg raise or fluid challenge

Additional interventions and targets for therapy in the early management of sepsis

- Support blood pressure to achieve a urine output of >0.5 mL/kg/h over a 6-h period
- Administer vasopressor agents if fluid resuscitation does not restore an effective BP and cardiac output:
 - Norepinephrine centrally given is the initial vasopressor of choice.

- Epinephrine, phenylephrine, or vasopressin should not be given as the initial vasoconstrictor in septic shock.
- Obtain blood, sputum, urine, and wound cultures and administer broad-spectrum antibiotics:
 - Cultures should be obtained prior to antibiotic administration.
 - Antibiotic administration should occur within 3 h of admission to the emergency department or within 1 h of inpatient admission.
- Support the respiratory system with supplemental oxygen and mechanical ventilation.
- Transfuse with packed red blood cells when hemoglobin is <7 g/dL to achieve a target hemoglobin of 7–9 g/dL in adults.
- Provide adequate IV sedation and analgesia; avoid the use of neuromuscular blockade agents when possible.
- Control serum glucose <180 mg/dL with IV insulin therapy.
- Implement interventions and medications to prevent deep vein thrombosis and stress ulcer prophylaxis.
- Discuss advance care planning with patients and families.

BP, blood pressure; CVP, central venous pressure; IV, intravenous; MAP, mean arterial pressure.

Adapted from Centers for Medicare & Medicaid Services (CMS). (2019). Hospital Toolkit for Adult Sepsis Surveillance. Retrieved on 9/10/19 at: www.cdc.gov/sepsis/clinicaltools/index.html; Levy, M., Evans, L., & Rhodes, A. (2018). The Surviving Sepsis Campaign bundle: 2018 update. *Critical Care Medicine*, 46(6), 997–1000; Levy, M. M., & Townsend, S. R. (2019). Early identification of sepsis on the hospital floors: Insights for implementation of the hour-1 bundle. *Society of Critical Care Medicine*. Retrieved on 9/10/19 at: www.survivingsepsis.org/SiteCollectionDocuments/Surviving-Sepsis-Early-Identify-Sepsis-Hospital-Floor.pdf

Nutritional Therapy

Aggressive nutritional supplementation should be initiated within 24 to 48 hours of ICU admission to address the hypermetabolic state present with septic shock (Reintam et al., 2017; Wang et al., 2019). Malnutrition further impairs the patient's resistance to infection. Enteral feedings are preferred to the parenteral route because of the increased risk of iatrogenic infection associated with IV catheters; however, enteral feedings may not be possible if decreased perfusion to the GI tract reduces peristalsis and impairs absorption.



Nursing Management

Nurses caring for patients in any setting must keep in mind the risks of sepsis and the high mortality rate associated with sepsis and septic shock. All invasive procedures must be carried out with aseptic technique after careful hand hygiene. In addition, IV lines, arterial and venous puncture sites, surgical incisions, traumatic wounds, and urinary catheters must be monitored for signs

of infection. Nursing interventions to prevent infection need to be implemented in the care of all patients. Nurses should identify patients who are at particular risk for sepsis and septic shock (i.e., older adults and immunosuppressed patients and those with extensive trauma, burns, or diabetes), keeping in mind that these high-risk patients may not develop typical or classic signs of infection and sepsis. However, confusion with or without agitation along with an increased respiratory rate may be the first sign of infection and sepsis in any adult patient (Ferguson, Coates, Osborn, et al., 2019).

Sepsis-3 recommends that patients in the ICU setting with infection be monitored for the development of sepsis by using the *Sepsis-Related Organ Failure Assessment Score* (also known as the *Sequential Organ Failure Assessment [SOFA]* score) (Singer et al., 2016; Vincent, Moreno, Takala, et al., 1996) ([Table 11-4](#)). The parameters that are monitored on the SOFA which include assessment of respiratory rate, platelets, bilirubin, MAP (and use of any vasopressors), serum creatinine, urine output, and Glasgow Coma Scale (GCS) score (see [Chapter 63](#)), may all be gathered and assessed by the nurse in the ICU setting. A drop of 2 points or more in a patient's SOFA score from baseline is suggestive of organ dysfunction. In a patient with infection, the presence of organ dysfunction suggests the development of sepsis (Singer et al., 2016).

For a patient not in the ICU setting who has an infection, *Sepsis-3* recommends that the Quick SOFA (qSOFA) scale be used to screen for the development of sepsis. The qSOFA is an easy measurement tool that nurses may readily use; the presence of any two of the three parameters on this scale suggests the development of sepsis. These parameters include a respiratory rate of 22 breaths/min or higher, a GCS score of less than 15 (any change in the patient's mentation), and a systolic BP of 100 mm Hg or less. In order to facilitate ease of use, therefore, *Sepsis-3* recommends that any change in the patient's mentation status be used when computing the qSOFA scale score (Singer et al., 2016). Another assessment tool that is frequently used in hospital-based settings that can identify patients with sepsis is the *Modified Early Warning System* (MEWS) (Institute for Healthcare Improvement [IHI], 2017). The nurse assesses the patient for changes in respiratory rate, heart rate, BP, consciousness, temperature, and urine output. Scores ranging from 0 to 3 are assigned to each variable, and a MEWS score greater than 4 is suggestive of the development of sepsis (IHI, 2017) ([Table 11-5](#)).

TABLE 11-4 The Sepsis-Related Organ Failure Assessment Score (Sequential Organ Failure Assessment [SOFA] Score)

SOFA Score	1	2	3	4
Respiration PaO ₂ /FiO ₂ , mm Hg	<400	<300	<200	<100 —with respiratory support—
Coagulation Platelets × 10 ³ /mm ³	<150	<100	<50	<20
Liver Bilirubin, mg/dL (μmol/L)	1.2–1.9 (20–32)	2.0–5.9 (33–101)	6.0–11.9 (102–204)	>12.0
Cardiovascular Hypotension	MAP <70 mm Hg	Dopamine ≤5 or dobutamine (any dose) ^a	Dopamine >5 or epinephrine ≤0.1 or norepinephrine ≤0.1	Dopamine >15 or epinephrine >0.1 or norepinephrine >0.1
Central Nervous System Glasgow Coma Scale	13–14	10–12	6–9	<6
Renal Creatinine, mg/dL (μmol/L) or urine output	1.2–1.9 (110–170)	2.0–3.4 (171–299)	3.5–4.9 (300–440) or <500 mL/day	>5.0

MAP, mean arterial pressure.

^aAdrenergic agents given for at least 1 h (doses given are in μg/kg/min).

Reprinted with permission from Vincent, J. L., Moreno, R., Takala, J., et al. (1996). The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. *Intensive Care Medicine*, 22(7), 707–710.

When caring for a patient with sepsis or septic shock, the nurse collaborates with other members of the health care team to identify the site and source of sepsis and the specific organisms involved. The nurse often obtains appropriate specimens for culture and sensitivity. Prescribed antibiotics are not given until these specimens are obtained. Hyperthermia (elevated body temperature) is common with sepsis and raises the patient's metabolic rate and oxygen consumption. Efforts may be made to reduce the temperature by administering acetaminophen or applying a hypothermia blanket. During these therapies, the nurse monitors the patient closely for shivering, which increases oxygen consumption. Efforts to increase comfort are important if the patient experiences fever, chills, or shivering.

The nurse administers prescribed IV fluids and medications, including antibiotic agents and vasoactive medications, to restore vascular volume. Because of decreased perfusion, serum concentrations of antibiotic agents that are normally cleared by the kidneys and liver may increase and produce toxic effects. Therefore, the nurse monitors blood levels (serum levels of antibiotic agents, procalcitonin, CRP, BUN, creatinine, WBC count, hemoglobin, hematocrit, platelet levels, coagulation studies) and reports changes to the primary provider. As with other types of shock, the nurse monitors the patient's hemodynamic status, fluid I&O, daily weight, and nutritional status. Close monitoring of serum albumin and prealbumin levels helps determine the patient's protein requirements.

TABLE 11-5 The MEWS (Modified Early Warning System)

MEWS (Modified Early Warning System)							
3	2	1	0	1	2	3	
Respiratory rate per minute	<8		9–14	15–20	21–29	>30	
Heart rate per minute	<40	40–50	51–100	101–110	111–129	>129	
Systolic blood pressure	<70	71–80	81–100	101–199		>200	
Conscious level (AVPU)	Unresponsive	Responds to Pain	Responds to Voice	Alert	New agitation; Confusion		
Temperature (°C)	<35.0	35.1–36	36.10–38	38.1–38.5		>38.6	
Hourly urine for 2 h	<10 mL/h	<30 mL/h	<45 mL/h				

Developed by Ysbyty Glan Clwyd, Conwy & Denbighshire National Health Service Trust, North Wales. Reprinted with permission from Institute for Healthcare Improvement (IHI). (2017). *Improvement stories: Early warning systems: Scorecards that save lives*. Retrieved on 11/6/19 at: <http://www.ihi.org/resources/Pages/ImprovementStories/EarlyWarningSystemsScorecardsThatSaveLives.aspx>

Neurogenic Shock

In **neurogenic shock**, vasodilation occurs as a result of a loss of balance between parasympathetic and sympathetic stimulation. Sympathetic stimulation causes vascular smooth muscle to constrict, and parasympathetic stimulation causes vascular smooth muscle to relax or dilate. The patient experiences a predominant parasympathetic stimulation that causes vasodilation lasting for an extended period, leading to a relative hypovolemic state. However, blood volume is adequate, because the vasculature is dilated; the blood volume is displaced, producing a hypotensive (low BP) state. The overriding parasympathetic stimulation that occurs with neurogenic shock causes a drastic decrease in the patient's systemic vascular resistance and bradycardia. Inadequate BP results in the insufficient perfusion of tissues and cells that is common to all shock states.

Neurogenic shock can be caused by spinal cord injury, spinal anesthesia, or other nervous system damage. It may also result from the depressant action of medications or from lack of glucose (e.g., insulin reaction) (see [Chart 11-5](#)). Neurogenic shock may have a prolonged course (spinal cord injury) or a short one (syncope or fainting). Normally, during states of stress, the sympathetic stimulation causes the BP and heart rate to increase. In neurogenic shock, the sympathetic system is not able to respond to body stressors. Therefore, the clinical characteristics of neurogenic shock are signs of parasympathetic stimulation. It is characterized by dry, warm skin rather than the cool, moist skin seen in hypovolemic shock. Another characteristic is hypotension with bradycardia, rather than the tachycardia that characterizes other forms of shock.



Medical Management

Treatment of neurogenic shock involves restoring sympathetic tone, either through the stabilization of a spinal cord injury or, in the instance of spinal

anesthesia, by positioning the patient properly. Specific treatment depends on the cause of the shock. (Further discussion of management of patients with a spinal cord injury is presented in [Chapter 63](#).)



Nursing Management

It is important to elevate and maintain the head of the bed at least 30 degrees to prevent neurogenic shock when a patient receives spinal or epidural anesthesia. Elevation of the head helps prevent the spread of the anesthetic agent up the spinal cord. In suspected spinal cord injury, neurogenic shock may be prevented by carefully immobilizing the patient to prevent further damage to the spinal cord (Kessler, Traini, Welk, et al., 2018). Nursing interventions are directed toward supporting cardiovascular and neurologic function until the usually transient episode of neurogenic shock resolves.

Patients with neurogenic shock have a higher risk for venous thromboembolism (VTE) formation because of increased pooling of blood from vascular dilation; this risk is greater in patients with neurogenic shock related to spinal cord injury. The nurse must assess the patient daily for any lower extremity pain, redness, tenderness, and warmth. If the patient complains of pain and objective assessment of the calf is suspicious, the patient should be evaluated for DVT. Passive range of motion of the immobile extremities helps promote circulation. Early interventions to prevent VTE include the application of pneumatic compression devices often combined with antithrombotic agents (e.g., low-molecular-weight heparin).

A patient who has experienced a spinal cord injury may not report pain caused by internal injuries. Therefore, in the immediate post-injury period, the nurse must monitor the patient closely for signs of internal bleeding that could lead to hypovolemic shock.

Anaphylactic Shock

Anaphylactic shock is caused by a severe allergic reaction when patients who have already produced antibodies to an antigen (foreign substance) develop a systemic antigen–antibody reaction; specifically, an immunoglobulin E (IgE)-mediated response. This antigen–antibody reaction provokes mast cells to release potent vasoactive substances, such as histamine or bradykinin, and activates inflammatory cytokines, causing widespread vasodilation and capillary permeability. The most common triggers are foods (especially peanuts), medications, and insect stings and bites (Pajno, Fernandez-Rivas, Arasi, et al., 2018) (see [Chart 11-5](#)). Anaphylaxis has three defining characteristics:

- Acute onset of symptoms

- Presence of two or more symptoms that include respiratory compromise, reduced BP, GI distress, and skin or mucosal tissue irritation
- Cardiovascular compromise from minutes to hours after exposure to the antigen

Signs and symptoms of anaphylaxis may present within 2 to 30 minutes of exposure to the antigen; however, occasionally some reactions may not develop for several hours (Chan & John, 2020). The patient may report headache, lightheadedness, nausea, vomiting, acute abdominal pain or discomfort, pruritus, and feeling of impending doom. Assessment may reveal diffuse erythema and generalized flushing, dyspnea (laryngeal edema), bronchospasm, cardiac arrhythmias, and hypotension. Characteristics of severe anaphylaxis usually include rapid onset of hypotension, neurologic compromise, respiratory distress, and cardiac arrest (Chan & John, 2020). Anaphylactoid reactions present similarly to anaphylaxis but are not mediated by IgE responses. Anaphylaxis and anaphylactoid reactions are often clinically indistinguishable (Pajno et al., 2018).



Medical Management

Treatment of anaphylactic shock requires removing the causative antigen (e.g., discontinuing an antibiotic agent), administering medications that restore vascular tone, and providing emergency support of basic life functions. Fluid management is critical, as massive fluid shifts can occur within minutes due to increased vascular permeability (Pajno et al., 2018). Intramuscular epinephrine is given for its vasoconstrictive action. Diphenhydramine is given intravenously to reverse the effects of histamine, thereby reducing capillary permeability. Nebulized medications, such as albuterol, may be given to reverse histamine-induced bronchospasm.

If cardiac arrest and respiratory arrest are imminent or have occurred, cardiopulmonary resuscitation (CPR) is performed. Endotracheal intubation may be necessary to establish an airway. IV lines are inserted to provide access for administering fluids and medications. See [Chapter 33](#) for further discussion of anaphylaxis and specific chemical mediators.



Nursing Management

The nurse has an important role in prevention and early recognition of anaphylactic shock. The nurse must assess all patients for allergies or previous reactions to antigens (e.g., medications, blood products, foods, contrast agents, latex) and communicate the existence of these allergies or reactions to others. In addition, the nurse assesses the patient's understanding of previous reactions

and steps taken by the patient and the family to prevent further exposure to antigens. When new allergies are identified, the nurse advises the patient to wear or carry identification that names the specific allergen or antigen.

When administering any new medication, the nurse observes all patients for allergic reactions. This is especially important with antibiotics, beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, aspirin, and nonsteroidal anti-inflammatory drugs (Chan & John, 2020). Previous adverse drug reactions increase the risk that the patient will develop a reaction to a new medication. If the patient reports an allergy to a medication, the nurse must be aware of the risks involved in the administration of similar medications.

At any diagnostic testing site, the nurse must identify patients who are at risk for anaphylactic reactions to contrast agents (radiopaque, dyelike substances that may contain iodine) used for diagnostic tests. The nurse must be knowledgeable about the clinical signs of anaphylaxis, must take immediate action if signs and symptoms occur, and must be prepared to begin CPR if cardiorespiratory arrest occurs.



Quality and Safety Nursing Alert

Patients with a known allergy to iodine or fish and those who have had previous allergic reactions to contrast agents are at high risk for anaphylactic reactions. The nurse should ask the patient about the allergy, signs and symptoms and severity, and if the patient has been tested for the allergy or carries an autoinjectable epinephrine device. This information must be communicated to the staff at the diagnostic testing site, including x-ray personnel.

Community health and home health nurses who administer medications, including antibiotic agents, in the patient's home or other settings, must be prepared to administer epinephrine intramuscularly in the event of an anaphylactic reaction.

After recovery from anaphylaxis, the patient and the family require an explanation of the event. Furthermore, the nurse provides education about avoiding future exposure to antigens and administering emergency medications to treat anaphylaxis (see [Chapter 33](#)).

Multiple Organ Dysfunction Syndrome

MODS is altered organ function in acutely ill patients that requires medical intervention to support continued organ function. It is another phase in the

progression of shock states. The actual incidence of MODS is difficult to determine, because it develops with acute illnesses that compromise tissue perfusion. Dysfunction of one organ system is associated with 20% mortality, and if more than four organs fail, the mortality is at least 60% (Sauaia, Moore, & Moore, 2017).

Pathophysiology

The precise mechanism by which MODS occurs remains unknown, but it is most commonly seen in patients with sepsis as a result of inadequate tissue perfusion. MODS frequently occurs toward the end of the continuum of septic shock when tissue perfusion cannot be effectively restored. It is not possible to predict which patients who experience shock will develop MODS, partly because much of the organ damage occurs at the cellular level and, therefore, cannot be directly observed or measured.

The clinical presentation of MODS is insidious; tissues become hypoperfused at both a microcellular and macrocellular level, eventually causing organ dysfunction that requires mechanical and pharmacologic intervention to support organ function. Organ failure usually begins in the lungs, and cardiovascular instability, as well as failure of the hepatic, GI, renal, immunologic, and central nervous systems, follows (Sauaia et al., 2017).

Clinical Manifestations

While it is not possible to predict MODS, clinical severity assessment tools may be used to anticipate patient risk of organ dysfunction and mortality. These clinical assessment tools include APACHE (Acute Physiology and Chronic Health Evaluation); SAPS (Simplified Acute Physiology Score); PIRO (Predisposing factors, the Infection, the host Response, and Organ dysfunction); and SOFA score (see [Table 11-4](#)) (Gustot, 2011; Kress & Hall, 2018).

In MODS, the sequence of organ dysfunction varies depending on the patient's primary illness and comorbidities before experiencing shock. Advanced age, malnutrition, and coexisting disease appear to increase the risk of MODS in acutely ill patients. For simplicity of presentation, the classic pattern is described. Typically, the lungs are the first organs to show signs of dysfunction. The patient experiences progressive dyspnea and respiratory failure that are manifested as ALI or ARDS, requiring intubation and mechanical ventilation (see [Chapters 19](#)). The patient usually remains hemodynamically stable but may require increasing amounts of IV fluids and vasoactive agents to support BP and cardiac output. Signs of a hypermetabolic state, characterized by hyperglycemia (elevated blood glucose level), hyperlactic acidemia (excess lactic acid in the blood), and increased BUN, are

present. The metabolic rate may be 1.5 to 2 times the basal metabolic rate. At this time, there is a severe loss of skeletal muscle mass (autocatabolism) to meet the high energy demands of the body.

After approximately 7 to 10 days, signs of hepatic dysfunction (e.g., elevated bilirubin and liver function tests) and renal dysfunction (e.g., elevated creatinine and anuria) are evident. As the lack of tissue perfusion continues, the hematologic system becomes dysfunctional, with worsening immunocompromise, increasing the risk of bleeding. The cardiovascular system becomes unstable and unresponsive to vasoactive agents, and the patient's neurologic response progresses to a state of unresponsiveness or coma.

The goal of all shock states is to reverse the tissue hypoperfusion and hypoxia. If effective tissue perfusion is restored before organs become dysfunctional, the patient's condition stabilizes. Along the septic shock continuum, the onset of organ dysfunction is an ominous prognostic sign; the more organs that fail, the worse the outcome.



Medical Management

Prevention remains the top priority in managing MODS. Older adult patients are at increased risk for MODS because of the lack of physiologic reserve and the natural degenerative process, especially immune compromise (Kress & Hall, 2018). Early detection and documentation of initial signs of infection are essential in managing MODS in older adult patients. Subtle changes in mentation and a gradual rise in temperature are early warning signs. Other patients at greater risk for MODS are those with chronic illness, malnutrition, immunosuppression, or surgical or traumatic wounds.

If preventive measures fail, treatment measures to reverse MODS are aimed at (1) controlling the initiating event, (2) promoting adequate organ perfusion, (3) providing nutritional support, and (4) maximizing patient comfort.



Nursing Management

The general plan of nursing care for patients with MODS is the same as that for patients with shock. Primary nursing interventions are aimed at supporting the patient and monitoring organ perfusion until primary organ insults are halted. Providing information and support to family members is a critical role of the nurse. The health care team must address end-of-life decisions to ensure that supportive therapies are congruent with the patient's wishes (see [Chapter 13](#)).

Promoting Communication

Nurses should encourage frequent and open communication about treatment modalities and options to ensure that the patient's wishes regarding medical management are met. Patients who survive MODS must be informed about the goals of rehabilitation and expectations for progress toward these goals, because massive loss of skeletal muscle mass makes rehabilitation a long, slow process. A strong nurse–patient relationship built on effective communication provides needed encouragement during this phase of recovery.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

Patients who experience and survive any type of shock or sepsis may have been unable to get out of bed for an extended period of time and are likely to have a slow, prolonged recovery. The patient and the family are educated about strategies to prevent further episodes of shock or sepsis by identifying the factors implicated in the initial episode (Ferguson et al., 2019). In addition, the patient and the family require education about assessments needed to identify the complications that may occur after the patient is discharged from the hospital. Depending on the type of shock and its management, the patient or the family may require education about treatment modalities such as emergency administration of medications, IV therapy, parenteral or enteral nutrition, skin care, exercise, and ambulation. The patient and the family are also educated about the need for gradual increases in ambulation and other activity. The need for adequate nutrition is another crucial aspect of education.

Continuing and Transitional Care

Because of the physical toll associated with recovery from shock and sepsis, patients may be cared for in a long-term care facility or rehabilitation setting after hospital discharge. Alternatively, a referral may be made for home, community-based, or transitional care. Patients may also experience PICS, a condition that is gaining increased recognition. PICS occurs after critically ill patients are discharged from the hospital. PICS involves physical, cognitive, and mental impairments that may adversely impact the patient's long-term recovery (Inoue, Hatakeyama, Kondo, et al., 2019). Nursing interventions to prevent PICS occur during the patient's critical illness and include early mobilization, assessment and management of delirium, promoting adequate

sleep, and assisting with mechanical ventilation liberation (e.g., weaning) as soon as possible. The nurse assesses the patient's physical status and monitors recovery. The adequacy of treatments continued at home and the ability of the patient and the family to cope with these treatments are also assessed. The patient is likely to require close medical supervision until complete recovery occurs. The nurse reinforces the importance of continuing medical care and helps the patient and the family identify and mobilize community resources.

CRITICAL THINKING EXERCISES

1 pq A 32-year-old patient is admitted after falling from a second-story balcony. The patient has a fractured femur, several fractured ribs, and possible head injury. The primary provider has prescribed lactated Ringer's solution to be infused at 250 mL/h to maintain a blood pressure >120 mm Hg and urine output >50 mL/h. Two large-bore IVs are started in each arm and IV fluids are initiated. The patient is alert to name only. The patient is typed and crossed for blood products, and a series of metabolic labs and a lactate level are drawn for analysis. The patient becomes unresponsive to name and begins moaning and grimacing. Vital signs are as follows: BP 92/72, HR 132 bpm, RR 32 bpm, SpO₂ 92% on 40% FiO₂ via a facemask with a urine output of 15 mL over the last hour. Identify 3–5 priority interventions that you should implement, the order in which they should be implemented, and explain how you made these decisions.

2 ebp A 76-year-old patient is scheduled for surgery for benign prostatic enlargement that is causing significant obstruction to his urinary function. The patient's past medical history includes chronic obstructive pulmonary disease, hypertension, and type 2 diabetes. Due to his medical history and surgical risks, he is not a candidate for general anesthesia and instead receives spinal anesthesia. Midway through the surgery his vital signs decrease abruptly to BP 88/32 mm Hg and HR 62 bpm, while his RR remains stable controlled by anesthesia with ventilatory support. Surgery is aborted for suspected neurogenic shock. The patient is transferred to the postanesthesia care unit (PACU) for monitoring. The surgeon explains the complication that has occurred to the patient's wife and tells her that a nurse will continue to provide updates to her by phone. The wife asks to see her husband, but the waiting room clerk says that family is not allowed in the PACU. An hour passes and the nurse does not call to update the wife. The clerk observes the wife quietly crying in a corner and calls the nurse caring for the patient to inform her of the wife's distress. As the nurse, what actions would you implement to facilitate patient- and family-centered care? What evidence supports these actions?

3 ipc An 88-year-old woman is admitted from an assisted living facility with acute decline in mental status. On admission vital signs are as follows: BP 100/76 mm Hg, HR 126 bpm, and RR 26 bpm. The patient is also incontinent of urine. As the nurse, you suspect a possible urinary tract infection and immediately assess the patient for sepsis. What other members of the interprofessional team will you need to collaborate with to facilitate early interventions to minimize the risk of complications? To implement a sepsis protocol, what additional information should you obtain prior to calling the provider? What should you anticipate from the

provider? How will you effectively communicate your assessment and recommendations to the provider and other members of the interprofessional team?

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*Asterisk indicates nursing research.

**Double asterisk indicates classic reference.

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Resources

American Association of Critical-Care Nurses Resources for Sepsis,
www.aacn.org/clinical-resources/sepsis

Institute for Healthcare Improvement, www.ihi.org

Sepsis Alliance, www.sepsis.org

Surviving Sepsis Campaign, www.survivingsepsis.org

12 Management of Patients with Oncologic Disorders

LEARNING OUTCOMES

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

1. Differentiate between characteristics of benign and malignant tumors.
2. Discuss the role of the nurse in the prevention and management of cancer.
3. Compare and contrast the goals of cancer care in prevention, diagnosis, cure, control, and palliation.
4. Describe the role of surgery, radiation therapy, chemotherapy, hematopoietic stem cell transplantation, immunotherapy, and targeted therapy in the treatment of cancer.
5. Use the nursing process as a framework for the care of the patient with cancer throughout the disease trajectory, from the time of diagnosis, to survivorship, and at the end of life.

NURSING CONCEPTS

Cellular Regulation
Managing Care
Tissue Integrity

GLOSSARY

- alopecia:** hair loss
- anaplasia:** pattern of growth in which cells lack normal characteristics and differ in shape and organization with respect to their cells of origin; usually, anaplastic cells are malignant
- angiogenesis:** growth of new blood vessels that allow cancer cells to grow
- apoptosis:** a normal cell mechanism of programmed cell death
- benign:** not cancerous; benign tumors may grow but are unable to spread to other organs or body parts
- brachytherapy:** delivery of radiation therapy through internal implants placed inside or adjacent to the tumor
- cancer:** a group of disorders characterized by abnormal cell proliferation, in which cells ignore growth-regulating signals in the surrounding environment
- carcinogenesis:** process of transforming normal cells into malignant cells
- carcinogens:** chemicals, physical factors, and other agents that cause cancer
- chemotherapy:** the use of medications to kill tumor cells by interfering with cellular functions and reproduction
- cytokines:** messenger substances that may be released by a cell to create an action at that site or may be carried by the bloodstream to a distant site before being activated; (*synonyms:* biochemical mediators, inflammatory mediators)
- extravasation:** leakage of intravenous medication from the veins into the subcutaneous tissues
- grading:** identification of the type of tissue from which the tumor originated and the degree to which the tumor cells retain the functional and structural characteristics of the tissue of origin
- graft-versus-host disease (GVHD):** an immune response initiated by T lymphocytes of donor tissue against the recipient's tissues (skin, gastrointestinal tract, liver); an undesirable response
- graft-versus-tumor effect:** the donor immune cell response against the malignancy; a desirable response
- immunotherapy:** the use of medications or other agents to stimulate or suppress components of the immune system to kill cancer cells
- malignant:** having cells or processes that are characteristic of cancer
- metastasis:** spread of cancer cells from the primary tumor to distant sites
- mucositis:** inflammation of the lining of the mouth, throat, and gastrointestinal tract often associated with cancer therapies
- myelosuppression:** suppression of the blood cell-producing function of the bone marrow

nadir: the lowest serum level of blood cells (i.e., white blood cells, red blood cells, and platelets) after therapy that has toxic effects on the bone marrow. Clinically, the nadir is most often used to describe the lowest absolute neutrophil count following chemotherapy

neoplasia: uncontrolled cell growth that follows no physiologic demand; cancer

neutropenia: abnormally low absolute neutrophil count

oncology: field or study of cancer

palliation: relief of symptoms and promotion of comfort and quality of life regardless of the disease stage

precision medicine: using advances in research, technology, and policies to develop individualized plans of care to prevent and treat disease

radiation therapy: the use of ionizing radiation to kill malignant cells

staging: process of determining the extent of disease, including tumor size and spread or metastasis to distant sites

stomatitis: inflammation of the oral tissues, often associated with some chemotherapeutic agents and radiation therapy to the head and neck region

targeted therapies: the use of medications or other agents to kill or prevent the spread of cancer cells by targeting specific part of the cell, with less negative effects on healthy cells

thrombocytopenia: decrease in the number of circulating platelets; associated with the potential for bleeding

toxicity: an unfavorable and unintended sign, symptom, or condition associated with cancer treatment

vesicant: substance that can cause inflammation, damage, and necrosis with extravasation from blood vessels and contact with tissues

Cancer is a large group of disorders with different causes, manifestations, treatments, and prognoses. Because cancer can involve any organ system and treatment approaches have the potential for multisystem effects, cancer nursing practice overlaps with numerous nursing specialties. Cancer nursing practice covers all age groups and is carried out in various settings, including acute care institutions, outpatient centers, physician offices, rehabilitation facilities, the home, and long-term care facilities. The scope, responsibilities, and goals of cancer nursing, also called **oncology** nursing, are as diverse and complex as those of any nursing specialty. Nursing management of the patient with oncologic disorders includes care of patients throughout the cancer trajectory from prevention through end-of-life care (see Fig. 12-1).

Precision medicine is possible because of the recent development of biologic databases (e.g., human genome sequencing), technologic advances that can identify unique characteristics of individual persons (e.g., genomics, cellular

assay tests), and computer-driven systems that can mine and analyze datasets (see Chapter 1). This is an exciting time for oncology as the overall goal of the precision medicine initiative is to focus on preventing and curing cancers (Ginsburg & Phillips, 2018).

Epidemiology

Cancer is a common health problem worldwide. In the United States, it was estimated in 2019 that more than 1,700,000 new cancer cases would be diagnosed, and that more than 600,000 Americans would die as a result of cancer (Siegel, Miller, & Jemal, 2019). Despite significant advances in science and technology, cancer is the second leading cause of death in the United States. The leading causes of cancer-related death in the United States in order of frequency and location are lung, prostate, and colorectal cancer in men and lung, breast, and colorectal cancer in women. Most cancer occurs in older adults; according to the American Cancer Society (ACS), 80% of all cancer diagnoses are in people 55 years of age or older (2019a). Overall, the incidence of cancer is slightly higher in women than in men (ACS, 2019a).

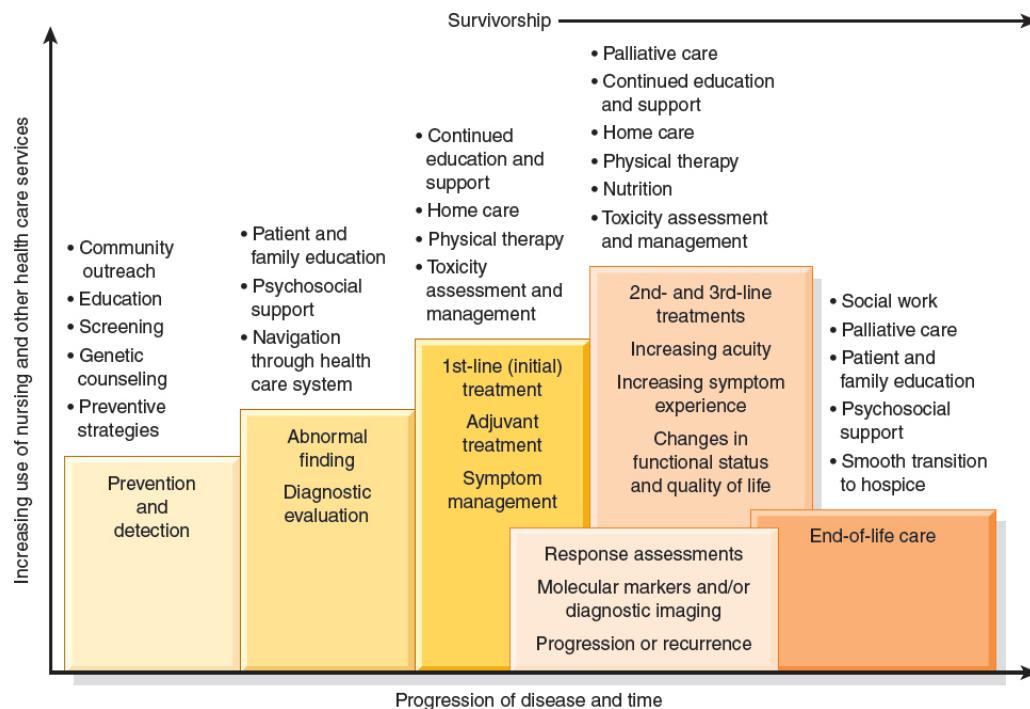


Figure 12-1 • Cancer care trajectory. The cancer care trajectory reflects the phases and care required during the continuum of the cancer experience from prevention and early detection through end-of-life care. Specialized nursing care is provided throughout the entire trajectory.

Cancer affects all groups of people; however, some groups are more adversely affected by cancer than others (National Cancer Institute [NCI], 2019a). Differences in cancer measures (e.g., incidence, screening rates, stage of diagnosis, morbidity, mortality) or cancer-related health conditions are termed *cancer health disparities*. These disparities often result from multiple, complex interrelated factors, including indicators of socioeconomic status (e.g., income, educational level, health care access), culture, diet, stress, the environment, and biology.

Cancer health disparities are most often observed among people of low-socioeconomic status, certain races/ethnicities, and those who reside in particular geographical locations (NCI, 2019a). For example, even though the overall rate of cancer deaths has declined, cancer death rates in non-Hispanic Black men and women remain substantially higher compared to all other racial and ethnic groups for most cancer types (DeSantis, Miller, Goding Sauer, et al., 2019). Hispanic/Latino Americans have a lower overall cancer incidence when compared to non-Hispanic White Americans; however, the incidence of infection-related cancers (e.g., liver cancer) is two times higher in those of Hispanic/Latino origin compared to non-Hispanic White Americans (Miller, Goding Sauer, Ortiz, et al., 2018).

Cancer incidence and death rates also vary by geography. In states where the prevalence of tobacco use is high (e.g., Kentucky), the incidence of lung cancer tends to be greater than in states where smoking is not as common (e.g., Utah) (Siegel et al., 2019). In locations where there is greater socioeconomic disparity, the incidence of cancer and overall cancer death rates are higher than in regions where there is not such a disparity (Siegel et al., 2019). For example, the incidence of cancers of the lung, colon and rectum, and cervix are substantially higher in Appalachian Ohio (southwestern region of the state) than in more affluent and more populated regions of the state (NCI, 2019a).

Pathophysiology of the Malignant Process

Cancer is a disease process that begins when a cell is transformed by genetic mutations of the cellular deoxyribonucleic acid (DNA). Genetic mutations may be inherited or acquired, leading to abnormal cell behavior (Norris, 2019). The initial genetically altered cell forms a clone and begins to proliferate abnormally, evading normal intracellular and extracellular growth-regulating processes or signals as well as the immune system defense mechanisms of the body. Genetic mutations may lead to abnormalities in cell signaling transduction processes (signals from outside and within cells that turn cell activities either on or off) that can in turn lead to cancer development. Ultimately cells acquire a variety of capabilities that allow them to invade surrounding tissues or gain access to lymph and blood vessels, which carry the cells to other areas of the body

resulting in **metastasis** or spread of the cancer (Pachmayr, Treese, & Stein, 2017).

Benign (noncancerous) and **malignant** (cancerous) cells differ in many cellular growth characteristics, including the method and rate of growth, ability to metastasize or spread, destruction of tissue, and ability to cause death. These differences are summarized in [Table 12-1](#). The degree of **anaplasia** (a pattern of growth in which cells lack normal characteristics and differ in shape and organization with respect to their cells of origin) is associated with increased malignant potential.

TABLE 12-1 Characteristics of Benign and Malignant Cells

Characteristics	Benign	Malignant
Cell	Well-differentiated cells resemble normal cells of the tissue from which the tumor originated.	Cells are undifferentiated and may bear little resemblance to the normal cells of the tissue from which they arose.
Mode of growth	Tumor grows by expansion and does not infiltrate the surrounding tissues; usually encapsulated.	Grows at the periphery and overcomes contact inhibition to invade and infiltrate surrounding tissues
Rate of growth	Rate of growth is usually slow.	Rate of growth is variable and depends on level of differentiation; the more anaplastic the tumor, the faster its growth.
Metastasis	Does not spread by metastasis.	Gains access to the blood and lymphatic channels and metastasizes to other areas of the body or grows across body cavities such as the peritoneum.
General effects	Usually a localized phenomenon that does not cause generalized effects unless its location interferes with vital functions.	Often causes generalized effects, such as anemia, weakness, systemic inflammation, weight loss, and CACS.
Tissue destruction	Does not usually cause tissue damage unless its location interferes with blood flow.	Often causes extensive tissue damage as the tumor outgrows its blood supply or encroaches on blood flow to the area; may also produce substances that cause cell damage.
Ability to cause death	Does not usually cause death unless its location interferes with vital functions.	Eventually causes death unless growth can be controlled.

CACS, cancer-related anorexia-cachexia syndrome.

Adapted from Norris, T. L. (2019). *Porth's pathophysiology: Concepts of altered health states* (10th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Carcinogenesis

Understanding the pathophysiology of cancer involves knowledge of the molecular process and proliferation patterns of cancers as well as the numerous etiologic factors that induce the malignant transformation of cells.

Molecular Process

Malignant transformation, or **carcinogenesis**, is thought to be at least a three-step cellular process, involving initiation, promotion, and progression (Norris, 2019). Agents that initiate or promote malignant transformation are referred to as **carcinogens**. A complete carcinogen is an agent that both initiates and promotes the development of cancer (e.g., cigarette smoking, asbestos).

During *initiation*, carcinogens (substances that can cause cancer), such as chemicals, physical factors, or biologic agents, cause mutations in the cellular DNA. Normally, these alterations are reversed by DNA repair mechanisms or the changes initiate **apoptosis** (programmed cellular death) or cell senescence. Cells can escape these protective mechanisms with permanent cellular mutations occurring, but these mutations usually are not significant to cells until the second step of carcinogenesis.

During *promotion*, repeated exposure to promoting agents (co-carcinogens) causes proliferation and expansion of initiated cells with increased expression or manifestations of abnormal genetic information, even after long latency periods. Promoting agents are not mutagenic and do not need to interact with the DNA. Promotion is reversible if the promoting substance is removed (a key focus in the prevention of cancer). Latency periods for the promotion of cellular mutations vary with the type of agent, the dosage of the promoter, and the innate characteristics and genetic stability of the target cell.

During *progression*, the altered cells exhibit increasingly malignant behavior. These cells acquire the ability to stimulate **angiogenesis** (growth of new blood vessels that allow cancer cells to grow), to invade adjacent tissues, and to metastasize. Cellular oncogenes are responsible for vital cell functions, including proliferation and differentiation. Cellular proto-oncogenes, such as those for the epidermal growth factor receptor (EGFR), transcription factors such as *c-Myc*, or cell signaling proteins such as *Kirsten ras (KRAS)*, act as “on switches” for cellular growth. Amplification of proto-oncogenes or overexpression of growth factors, such as epidermal growth factor (EGF), can lead to uncontrolled cell proliferation. Mutations that increase the activity of oncogenes also deregulate cell proliferation. Genetic alterations in the gene for *KRAS* have been associated with pancreatic, lung, and colorectal cancers (Mainardi, Mulero-Sánchez, Prahallad, et al., 2018). Just as proto-oncogenes “turn on” cellular growth, cancer suppressor genes “turn off,” or regulate, unneeded cellular proliferation. When suppressor genes are mutated, resulting in loss of function or expression, the cells begin to produce mutant cell populations that are different from their

original cellular ancestors. See [Chart 12-1](#) for further discussion of genetics concepts and cancer.

Proliferative Patterns

During the lifespan, various body tissues normally undergo periods of rapid or proliferative growth that must be distinguished from malignant growth activity. Several patterns of cellular adaptation include atrophy, hypertrophy, hyperplasia, metaplasia, and dysplasia (see [Chapter 5, Fig. 5-4](#)). Cancerous cells, described as malignant, demonstrate **neoplasia**, or uncontrolled cell growth that follows no physiologic demand. Although both benign and malignant growths are classified and named by tissue of origin, the *International Classification of Diseases for Oncology* (Fritz, Percy, Jack et al., 2013) is used by scientists and clinicians around the world as the nomenclature for malignant disease (see [Table 12-2](#)).

Etiology

Factors implicated or known to induce carcinogenesis include viruses and bacteria, physical agents, chemicals, genetic or familial factors, lifestyle factors, and hormones. Additional research is needed for a better understanding of the relationships among etiologic factors and cancer.

Viruses and Bacteria

It is estimated that 10% to 12% of all cancers worldwide are linked to viral infections (Lunn, Jahnke, & Rabkin, 2017). After infecting individuals, DNA viruses insert a part of their own DNA near the infected cell genes causing cell division. The newly formed cells that now carry viral DNA lack normal controls on growth. Examples of these viruses that are known to cause cancer include human papillomavirus (HPV) (cervical and head and neck cancers), hepatitis B virus (HBV) (liver cancer), and Epstein–Barr virus (EBV) (Burkitt lymphoma and nasopharyngeal cancer).

While there is little evidence to support a direct link between most bacteria and cancer, secondary responses to certain bacterial infections, such as the production of carcinogenic metabolites and inflammatory reactions, are suspected mechanisms of cancer development (van Elsland & Neefjes, 2018). Examples of bacteria that are associated with an increased risk of cancer include: *Helicobacter pylori* (stomach cancer), *Salmonella enteritidis* (colon cancer), and *Chlamydia trachomatis* (ovarian and cervical cancers).

Physical Agents

Physical factors associated with carcinogenesis include exposure to sunlight, radiation, chronic irritation or inflammation, tobacco carcinogens, industrial chemicals, and asbestos (ACS, 2019b).

Excessive exposure to the ultraviolet rays of the sun is associated with skin cancers in all individuals, although those with fair-skin are at highest risk.

Factors such as clothing styles (sleeveless shirts or shorts), the use of sunscreens, occupation, recreational habits, and environmental variables, including humidity, altitude, and latitude, all play a role in the amount of exposure to ultraviolet light (ACS, 2019c).

Chart 12-1



GENETICS IN NURSING PRACTICE

Genetics Concepts and Oncologic Disorders

Cancer is a genetic disease. Every phase of carcinogenesis is affected by multiple gene mutations. Some of these mutations are inherited (present in germ-line cells) and present a greater risk for a person to develop oncologic disorders; however, most (90%) are somatic mutations that are acquired mutations in specific cells. Examples of cancers influenced by genetics include:

Autosomal dominant:

- Breast and ovarian cancer
- Colorectal cancer
- Familial adenomatous polyposis
- Cowden syndrome
- Li–Fraumeni syndrome
- Lynch syndrome (hereditary nonpolyposis colon cancer)
- Multiple endocrine neoplasia types 1 and 2
- Neurofibromatosis types 1 and 2
- Prostate cancer
- Retinoblastoma
- Von Hippel–Lindau syndrome
- Wilms tumor

Autosomal recessive:

- Ataxia telangiectasis
- Endometrial cancer
- Gastrointestinal stromal tumor
- Familial melanoma syndrome
- Xeroderma pigmentosum

Nursing Assessments

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

Family History Assessment Specific to Oncologic Disorders

- Obtain information about both maternal and paternal sides of family for three generations.
- Obtain cancer history for at least three generations.
- Look for clustering of cancers that occur at young ages, multiple primary cancers in one individual, cancer in paired organs, and two or more close relatives with the same type of cancer suggestive of hereditary cancer syndromes.

Patient Assessment

- Assess for the following:
 - Physical findings that may predispose the patient to cancer, such as multiple colon polyps or the presence of more than one tumor. If a

tumor was previously diagnosed, inquire about the age of the patient when the first tumor was noted.

- Skin findings, such as atypical moles, that may be related to familial melanoma syndrome.
- Multiple *café-au-lait* spots, axillary freckling, and two or more neurofibromas associated with neurofibromatosis type 1.
- Facial trichilemmomas, mucosal papillomatosis, multinodular thyroid goiter or thyroid adenomas, macrocephaly, fibrocystic breasts, and other fibromas or lipomas related to Cowden syndrome.
- Assess for lifestyle risks (e.g., smoking, obesity, alcohol use).
- Determine potential occupational or environmental hazards that may generate exposure to inhaled chemicals, gases, or other irritants (e.g., toxic metals, asbestos, radon).

Management Issues Specific to Oncologic Disorders

- Assess patient's understanding of genetics factors related to their cancer.
- Offer appropriate genetics information and resources.
- Refer for cancer risk assessment when a hereditary cancer syndrome is suspected so that patient and family can discuss inheritance risk with other family members and availability of genetic testing.
- Provide support to patients and families with known genetic test results for hereditary cancer syndromes. Refer to support groups as appropriate.
- Participate in the management and coordination of risk reduction measures for those with known gene mutations.

Genetics Resources Specific to Oncologic Disorders

American Cancer Society: www.cancer.org

National Cancer Institute: www.cancer.gov

See also Chapter 6, Chart 6-7 for additional components of genetic counseling.

Exposure to ionizing radiation from repeated diagnostic x-ray procedures or with radiation therapy used to treat disease can cause cancer (ACS, 2019c). Improved x-ray equipment minimizes the risk of extensive radiation exposure. Radiation therapy used in cancer treatment and exposure to radioactive materials at nuclear weapon manufacturing sites or nuclear power plants in the past have been associated with a higher incidence of leukemia, multiple myeloma, and cancers of the breast, thyroid, and other tissues. Background radiation from the natural decay processes that produce radon has also been associated with lung cancer. Ventilation is advised in homes with high levels of trapped radon to allow the gas to disperse into the atmosphere.

Chemical Agents

Most cancers are thought to be related to environmental factors (ACS, 2019b). Most hazardous chemicals produce their toxic effects by altering DNA structure. This can occur in body sites distant from that of initial chemical exposure.

Tobacco use is thought to be the single most lethal chemical carcinogen; it accounts for about 30% of all cancer-related deaths (ACS, 2019a). Cigarette smoking is strongly associated with 12 different cancer types including: cancers of the oral cavity and pharynx, larynx, lung, esophagus, pancreas, uterine cervix, kidney, bladder, stomach, colorectal, liver, and myeloid leukemia.

Environmental tobacco smoke (ETS), otherwise known as secondhand smoke, has been linked to lung cancer—even in people who never smoked (ACS, 2019d). Nonsmokers who were exposed to ETS in the home or workplace have about a 20% to 30% greater risk of developing lung cancer (Centers for Disease Control and Prevention [CDC], 2019). There is also some evidence that ETS may be linked with cancers of the larynx, pharynx, nasal sinuses, brain, bladder, rectum, stomach, and breast (ACS, 2019d).

Other combustible forms of tobacco, such as cigars, pipes, roll-your-own products, and water pipes (or hookah), are also associated with increased cancer risk (ACS, 2019d).

TABLE 12-2 Classification of Cancer by Tissue of Origin

Classification	Tissue of Origin	Characteristics	Term	Examples
Carcinoma	Epithelial <ul style="list-style-type: none"> • Glandular epithelium 	Account for 80–90% of all cancers Organs or glands capable of secretion	Adenocarcinoma	Adenocarcinoma of the breast, lung, prostate
	• Squamous epithelium	Covers or lines all external and internal body surfaces	Squamous cell carcinoma	Squamous cell cancer of the skin, lung, esophagus
Sarcoma	Connective or Supportive <ul style="list-style-type: none"> • Bone • Cartilage • Adipose • Smooth muscle • Skeletal muscle • Fibrous tissue • Membranes lining body cavities • Blood vessels 	Most common form of cancer of the bone	Osteosarcoma	Osteosarcoma of the femur, humerus
	Rare, arises from within bones	Chondrosarcoma	Chondrosarcoma of the femur, pelvis	
	Arises from deep soft tissue	Liposarcoma	Liposarcoma of the retroperitoneum, thigh	
	Very rare	Leiomyosarcoma	Leiomyosarcoma of the uterus, intestines, stomach	
	Most common in young children	Rhabdosarcoma	Rhabdosarcoma of the head and neck, limbs	
	Often involves long or flat bones	Fibrosarcoma	Fibrosarcoma of the femur, tibia, mandible	
	Most often related to asbestos exposure	Mesothelial sarcoma or mesothelioma	Mesothelioma of the pleura or peritoneum	
	With liver involvement may be related to occupational exposure to vinyl chloride monomer	Angiosarcoma	Angiosarcoma of the liver, heart	
Myeloma	Plasma cells	Produced by B-cell lymphocytes; plasma cells produce antibodies	Not applicable (N/A)	N/A
Lymphoma	Lymphocytes	Two main classifications; may involve lymph nodes or body organs	Non-Hodgkin lymphoma Hodgkin lymphoma	B-cell lymphoma, T-cell lymphoma N/A
Leukemia	Hematopoietic cells in the bone marrow <ul style="list-style-type: none"> • White blood cells (WBCs) • Lymphocytes • Red blood cells (RBCs) 	May involve various cell lines produced in the bone marrow N/A N/A Involves overproduction of RBCs and is associated with increased levels of WBCs and platelets; also risk of additional bone marrow disease	Myelogenous Lymphocytic Erythremia	Acute myelogenous leukemia Acute lymphocytic leukemia Polycythemia vera

Adapted from Fritz, A., Percy, C., Jack, A., et al.; World Health Organization. (2013). International classification of diseases for oncology (ICD-O)-3rd edition, 1st revision. Retrieved on 7/22/2019 at: codes.iarc.fr/home

Electronic nicotine delivery systems (ENDS) including e-cigarettes, e-pens, e-pipes, e-hookah and e-cigars have gained increased popularity as an alternative to tobacco. While ENDS do not contain tobacco, most contain nicotine, which is highly addictive, and other potentially harmful substances, such as volatile organic compounds, formaldehyde, and flavoring chemicals (ACS, 2019d). Given that ENDS are relatively new to the market, the long-term health effects of these products remain unknown.

Smokeless tobacco products, such as chewing tobacco, snuff and snus, used most often by young adults aged 18 to 25 years, are associated with an increased risk of oral, pancreatic, and esophageal cancer (ACS, 2019d; Lipari & Van Horn, 2017).

Many chemical substances found in the workplace are carcinogens or co-carcinogens (ACS, 2019e). In the United States, carcinogens are classified by two federal agencies: the National Toxicology Program of the Department of Health and Human Services (HHS) and the Environmental Protection Agency's (EPA) Integrated Risk Information System (IRIS) (CDC, 2017). The CDC established the National Institute for Occupational Safety and Health (NIOSH) to provide occupational exposure limits and guidelines for protection of the workforce as regulated by the Occupational Safety and Health Act of 1970 (U.S. EPA, 2017). The extensive list of suspected chemical substances continues to grow and includes aromatic amines and aniline dyes; pesticides and formaldehydes; arsenic, soot, and tars; asbestos; benzene; cadmium; chromium compounds; nickel and zinc ores; wood dust; beryllium compounds; and polyvinyl chloride (ACS, 2019e). Betel quid, which are chewed as stimulants in some cultures, are also included (ACS, 2019e; Chen, Mahmood, Mariottini, et al., 2017).

Genetics and Familial Factors

Almost every cancer type has been shown to run in families. This may be due to a combination of genetic, environmental, and lifestyle factors. Genetic factors play a fundamental role in cancer cell development. Cancer has been associated with extra chromosomes, too few chromosomes, or translocated chromosomes (NCI, 2019b).

Approximately 5% to 10% of cancers in adults display a pattern of cancers suggestive of a familial predisposition (NCI, 2019b). Hereditary cancer syndromes represent a cluster of cancers identified by a specific genetic alteration that is inherited across generations. In these families, the associated genetic mutation is found in all cells in the body (germline mutation) and represents an inherited susceptibility to cancer for all family members who carry the mutation.

There are more than 50 hereditary syndromes identified by scientists that may predispose individuals to develop certain cancers (NCI, 2019b). The hallmarks of families with a hereditary cancer syndrome include cancer in two or more first-degree relatives (the parent, sibling, or child of an individual), onset of

cancer in family members younger than 50 years, the same type of cancer in several family members, individual family members with more than one type of cancer, and a rare cancer in one or more family members. There is also evidence of an autosomal dominant inheritance pattern of cancers affecting several generations of a family (NCI, 2019b).

There have been considerable advances in the recognition of inherited cancer susceptibility syndromes and in the ability to isolate and identify the inherited genetic mutations responsible. These advances have enabled the appropriate identification of families at risk for certain syndromes. Examples include hereditary breast and ovarian cancer syndrome (*BRCA1* and *BRCA2*) and multiple endocrine neoplasia syndrome (*MEN1* and *MEN2*) (see [Chart 12-1](#)). Other cancers associated with familial inheritance syndromes include nephroblastomas, pheochromocytomas, and colorectal, stomach, thyroid, renal, prostate, and lung cancers (NCI, 2019b).

Lifestyle Factors

Lifestyle factors (e.g., obesity, alcohol intake, poor diet, physical inactivity) were estimated to account for 16% of all cancer cases and 18% of all cancer deaths in 2017 (Islami, Goding Sauer, Miller, et al., 2018). These lifestyle factors were second only to cigarette smoking as a major modifiable risk factor associated with both cancer development and cancer mortality.

The risk of cancer increases with long-term ingestion of carcinogens or co-carcinogens or the absence of protective substances in the diet. Dietary substances that appear to increase the risk of cancer include fats, alcohol, salt-cured or smoked meats, nitrate- and nitrite-containing foods, and red and processed meats (World Cancer Research Fund [WCRF], 2018). Heavy alcohol use increases the risk of cancers of the mouth, pharynx, larynx, esophagus, liver, colon, rectum, and breast (WCRF, 2018).



Obesity has been linked to the development of cancers of the breast (in postmenopausal women), colon and rectum, endometrium, esophagus, kidney, and pancreas (WCRF, 2018). Obesity may be also associated with an increased risk for cancers of the gallbladder, liver, ovary, and cervix, and for multiple myeloma, Hodgkin lymphoma, and aggressive forms of prostate cancer. While there is a clear relationship between obesity and cancer, the etiology of cancer in the context of obesity remains poorly understood (NCI, 2017). Several possible mechanisms have been suggested, however, including that excess fat may cause chronic inflammation resulting in DNA damage, increased levels of certain hormones (e.g., estrogen, insulin, adipokines), and disruptions in levels of cell growth regulators (e.g., mammalian target of rapamycin and AMP-activated protein kinase)—all of which may increase the development of certain types of cancer. Multiple studies have long linked a sedentary lifestyle and lack of regular exercise to cancer development (Cannioto, Etter, Guterman, et al., 2017; Cannioto, Etter, LaMonte, et al., 2018).

Hormonal Agents

Tumor growth may be promoted by disturbances in hormonal balance, either by the body's own (endogenous) hormone production or by administration of exogenous hormones (Norris, 2019). Cancers of the breast, prostate, ovaries, and endometrium are thought to depend on endogenous hormonal levels for growth. Prenatal exposure to diethylstilbestrol (a synthetic form of the female hormone estrogen) has long been recognized as a risk factor for clear cell adenocarcinoma of the lower genital tract (Huo, Anderson, Palmer, et al., 2017).

Hormonal changes related to the female reproductive cycle are also associated with cancer incidence. Early onset of menses before age 12 and delayed onset of menopause after age 55, null parity (never giving birth), and delayed childbirth after age 30 are all associated with an increased risk of breast cancer (Chen, 2019). Increased numbers of pregnancies are associated with a decreased incidence of breast, endometrial, and ovarian cancers.

Women who take estrogen after menopause appear to have a decreased risk of breast cancer, but an increased risk of developing endometrial cancers (NCI, 2018). Thus, estrogen replacement alone is not used in women who have not had a hysterectomy. Combination estrogen and progesterone therapy is linked to a higher risk of breast cancer. The longer the combined therapy is used, the higher the risk of developing breast cancer. However, the risk substantially decreases when therapy is discontinued (NCI, 2018).

Role of the Immune System

In humans, transformed cells arise on a regular basis, but are recognized by surveillance cells of the immune system that destroy them before cell growth becomes uncontrolled (immune surveillance) (Norris, 2019). When the immune system fails to identify and stop the growth of transformed cells, a tumor can develop and progress.

Patients who are immunocompromised have an increased incidence of cancer. Transplant recipients who receive immunosuppressive therapy to prevent rejection of the transplanted organ have an increased incidence of cancer (Norris, 2019). Patients with acquired immune deficiency syndrome (AIDS) have an increased incidence of Kaposi sarcoma and other cancers. Patients who were previously treated for one cancer are at increased risk for secondary cancers (Norris, 2019).

Normal Immune Responses

Through the process of immune surveillance, an intact immune system usually has the ability to recognize and combat cancer cells through multiple, interacting cells and actions of the innate, humoral, and cellular components of the immune system (Jameson, Fauci, Kasper, et al., 2018; Norris, 2019). Tumor-associated antigens (TAAs; also called *tumor cell antigens*) are found on the membranes of many cancer cells. TAAs are processed by antigen-presenting cells (APCs) (e.g.,

macrophages and dendritic cells [very specialized cells of the immune system] that present antigens to both T and B lymphocytes) and are presented to T lymphocytes that recognize the antigen-bearing cells as foreign. Multiple TAAs have been identified—some are found in many types of cancer, some exist in the normal tissues of origin as well as the cancer cells, some exist in both normal and cancer cells but are overexpressed (exist in higher concentrations) in cancer cells, and others are very specific to certain cancer types (Norris, 2019).

In response to recognizing TAAs as foreign, T lymphocytes release several cytokines that elicit various immune system actions, including (1) proliferation of cytotoxic (cell-killing) T lymphocytes capable of direct destruction of cancer cells, (2) induction of cancer cell apoptosis, and (3) recruitment of additional immune system cells (B-cell lymphocytes that produce antibodies, natural killer cells, and macrophages) that contribute to the destruction and degradation of cancer cells (Jameson et al., 2018; Norris, 2019).

Immune System Evasion

Several theories postulate how malignant cells survive and proliferate, evading the elaborate immune system defense mechanisms (Jameson et al., 2018; Norris, 2019). If the body fails to recognize the TAAs on cancer cells or the function of the APCs is impaired, the immune response is not stimulated. Some cancer cells have been found to have altered cell membranes that interfere with APC binding and presentation to T lymphocytes. Tumors can also express molecules that induce T-lymphocyte anergy or tolerance such as PD-1 ligand. These molecules bind to PD-1 proteins on T lymphocytes and either block the killing of the tumor or induce cell death in the lymphocyte. In addition, cancer cells have been found to release cytokines that inhibit APCs as well as other cells of the immune system. When tumors do not possess TAAs that label them as foreign, the immune response is not alerted. This allows the tumor to grow too large to be managed by normal immune mechanisms.

The immunogenicity (immunologic appearance) of cancer cells can be altered through genetic mutations, allowing the cells to evade immune cell recognition (Norris, 2019). Conversely, mutations are the source for some TAAs. Tumor antigens may combine with the antibodies produced by the immune system and hide or disguise themselves from normal immune defense mechanisms. The tumor antigen–antibody complexes that evade recognition lead to a false message to decrease further production of antibodies as well as other immune system components.

Overexpression (abnormally high concentrations) of host suppressor T lymphocytes induced through the release of cytokines by malignant cells is thought to downregulate the immune response, thus permitting uncontrolled cell growth (Jameson et al., 2018). Suppressor T lymphocytes normally assist in regulating lymphocyte production and diminishing immune responses (e.g., antibody production) when they are no longer required. Low levels of antibodies and high levels of suppressor cells have been found in patients with multiple

myeloma, which is a cancer associated with hypogammaglobulinemia (low amounts of serum antibodies). Conversely, there is evidence that proliferation of helper T lymphocytes, which promote the immune response, is impaired by cytokines produced by cancer cells (Jameson et al., 2018). Without helper T lymphocytes, the immune system response is limited, and the cancer cells continue to proliferate. Understanding the role of the immune system and identification of the ways in which cancer evades the body's natural defenses provide the foundation for therapeutic approaches that seek to support and enhance the immune system's role in combating cancer (see [Chapter 31](#)).

Detection and Prevention of Cancer

Nurses in all settings play a key role in cancer detection and prevention. Primary, secondary, and tertiary prevention of cancer are all important.

Primary Prevention

Primary prevention is about reducing the risks of disease through health promotion and risk reduction strategies. Guidelines on nutrition and physical activity for cancer prevention can be found in [Chart 12-2](#).

An example of primary prevention is the use of immunization to reduce the risk of cancer through prevention of infections associated with cancer. The HPV vaccine is recommended to prevent cervical and head and neck cancers (Hashim, Genden, Posner, et al., 2019). The vaccine to prevent HBV infection is recommended by the CDC (2018) to reduce the risk of hepatitis and subsequent development of liver cancer.

Secondary Prevention

Secondary prevention involves screening and early detection activities that seek to identify precancerous lesions and early-stage cancer in individuals who lack signs and symptoms of cancer. ACS screening is advocated for many types of cancer (see [Table 12-3](#)) (ACS, 2018). Detection of cancer at an early stage may reduce costs, use of resources, and the morbidity associated with advanced stages of cancer and their associated complex treatment approaches. Many screening and detection programs target people who do not regularly practice health-promoting behaviors or lack access to health care. Nurses continue to develop community-based screening and detection programs that address barriers to health care or reflect the socioeconomic and cultural beliefs of the target population (Rees, Jones, Jones, et al., 2018; So, Kwong, Chen, et al., 2019).

The evolving understanding of the role of genetics in cancer cell development has contributed to prevention and screening efforts. Many centers offer cancer

risk evaluation programs that provide interdisciplinary in-depth assessment, screening, education, and counseling as well as follow-up monitoring for people at high risk for cancer (National Comprehensive Cancer Network [NCCN] 2019a,b). The NCI provides guidance for cancer risk assessment, counseling, education, and genetic testing (NCI, 2019b).

Chart 12-2  **HEALTH PROMOTION**

American Cancer Society Guidelines on Nutrition and Physical Activity for Cancer Prevention

Individual Choices

Achieve and Maintain a Healthy Weight Throughout Life

- Be as lean as possible throughout life without being underweight.
- Avoid excessive weight gain at all ages. For those who are currently overweight or have obesity, losing even a small amount of weight has health benefits and is a good place to start.
- Engage in regular physical activity and limit consumption of high-calorie foods and beverages as key strategies for maintaining a healthy weight.

Adopt a Physically Active Lifestyle

- Adults should engage in at least 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity physical activity each week, or an equivalent combination, preferably spread throughout the week.
- Children and adolescents should engage in at least 1 hour of moderate- or vigorous-intensity physical activity each day, with vigorous-intensity activity at least 3 days each week.
- Limit sedentary behavior such as sitting, lying down and watching television, and other forms of screen-based entertainment.
- Doing any intentional physical activity above usual activities, no matter what one's level of activity, can have many health benefits.

Consume a Healthy Diet, with an Emphasis on Plant Sources

- Choose foods and beverages in amounts that help achieve and maintain a healthy weight.
- Limit consumption of processed meat and red meats.
- Eat at least 2½ cups of vegetables and fruits each day.
- Choose whole grains in preference to processed (refined) grains.

If You Drink Alcoholic Beverages, Limit Consumption

- Drink no more than one drink per day for women or two per day for men.

Community Action

Public, private, and community organizations should work collaboratively at national, state, and local levels to implement policy environmental changes that:

- Increase access to affordable, healthy foods in communities, worksites, and schools, and decrease access to and marketing of foods and beverages of low nutritional value, particularly to youth.
- Provide safe, enjoyable, and accessible environments for physical activity in schools and worksites, and for transportation and recreation in communities.

Adapted from American Cancer Society. (2019i). ACS guidelines on nutrition and physical activity for cancer. Retrieved on 9/28/2018 at: Prevention www.cancer.org/healthy/eat-healthy-get-active/acs-guidelines-nutrition-physical-activity-cancer-prevention.html.

Unfolding Patient Stories: Doris Bowman • Part 1



Doris Bowman, a 39-year-old diagnosed with uterine fibroids, dysmenorrhea, and menorrhagia, is scheduled for a total abdominal hysterectomy with bilateral salpingoophorectomy. She has a family history of uterine and ovarian cancer. She asks the nurse what she can do to further reduce her risk for cancer and is also concerned for her family members.

What patient and family education should the nurse present on risk reduction strategies, health promotion, and cancer screening? (Doris Bowman's story continues in [Chapter 51](#).)

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

Tertiary Prevention

Improved screening, diagnosis, and treatment approaches have led to an estimated 16.9 million cancer survivors in the United States (ACS, 2019f). Tertiary prevention efforts focus on monitoring for and preventing recurrence of the primary cancer as well as screening for the development of second malignancies in cancer survivors. Survivors are assessed for the development of second malignancies such as lymphoma and leukemia, which have been associated with certain chemotherapy agents and the use of radiation therapy (ACS, 2019f). Survivors may also develop second malignancies not related to treatment, but rather genetic mutations related to inherited cancer syndromes, environmental exposures, and lifestyle factors.

Diagnosis of Cancer

A cancer diagnosis is based on assessment of physiologic and functional changes and results of the diagnostic evaluation. Patients with suspected cancer undergo extensive testing to (1) determine the presence and extent of cancer, (2) identify possible disease metastasis, (3) evaluate the function of involved and uninvolved body systems and organs, and (4) obtain tissue and cells for analysis, including evaluation of tumor stage and grade. The diagnostic evaluation includes a review

of systems; physical examination; imaging studies; laboratory tests of blood, urine, and other body fluids; procedures; and pathologic analysis. A selection of diagnostic tests is found in [Table 12-4](#).

Patients undergoing extensive testing may be fearful of the procedures and anxious about possible test results. Nurses help address the patient's fear and anxiety by explaining the tests to be performed, the sensations likely to be experienced, and the patient's role in the test procedures. The nurse encourages the patient and family to voice their fears about the test results, supports the patient and family throughout the diagnostic evaluation, and reinforces and clarifies information conveyed by the primary provider. The nurse also encourages the patient and family to communicate, share their concerns, and discuss their questions and concerns with one another.

Tumor Staging and Grading

A complete diagnostic evaluation includes identifying the stage and grade of the tumor. This is accomplished prior to treatment to provide baseline data for evaluating outcomes of therapy and to maintain a systematic and consistent approach to ongoing diagnosis and treatment. Cancer treatment options and prognosis are based on the cancer type; stage and grade of cancer; as well as the individual's health status and response to treatment (NCI, 2019c).

TABLE 12-3 American Cancer Society Screening Guidelines for the Early Detection of Cancer^a

Cancer Site	Population	Test or Procedure	Recommendation
Breast	Women, ages 40–54	Mammography	Women should undergo regular screening mammography starting at age 45 yrs. Women ages 45–54 should be screened annually. Women should have the opportunity to begin annual screening between the ages of 40 and 44.
	Women age 55 and over	Mammography	Transition to biennial screening or can continue annual screening. Continue screening if overall health is good and life expectancy is 10+ yrs.
Cervix	Women, ages 21–29	Papanicolaou (Pap) test	Screening should be done every 3 yrs with conventional or liquid-based Pap tests. HPV DNA testing only for abnormal Pap test.
	Women ages 30–65	Pap test and HPV DNA test	Screening should be done every 5 yrs with both the HPV test and the Pap test (preferred), or every 3 yrs with the Pap test alone (acceptable).
	Women ages 66+		Women ages 66+ who have regular cervical cancer screening with negative results should stop cervical cancer screening. Women who have had a total hysterectomy should stop cervical cancer screening.
Colorectal ^b	Men and women, ages 45–75. People aged 76–84, should discuss continued screening with their provider. People 85+ should no longer participate in colorectal cancer screenings.	Highly sensitive guaiac-based fecal occult blood test (gFOBT) or highly sensitive fecal immunochemical test (FIT)	Annual highly sensitive gFOBT or FIT testing.
		Multi-targeted	MT-sDNA testing every 3 yrs.

		stool DNA test (MT-sDNA) test	
	Flexible sigmoidoscopy (FSIG), or	Every 5 yrs.	
	Colonoscopy, or	Every 10 yrs.	
	CT colonography (virtual colonoscopy)	Every 5 yrs.	
Endometrial	Women, at menopause	At the time of menopause, women at average risk should be informed about risks and symptoms of endometrial cancer and encouraged to report any unexpected bleeding or spotting to their providers.	Not applicable (N/A)
Lung	Current or former smokers (quit within past 15 yrs) ages 55–74 in fairly good health with at least a 30 pack-year history.	Low-dose CT (LDCT)	<p>LDCT annually.</p> <p>Providers with access to high-volume, high-quality lung cancer screening and treatment centers should initiate a discussion about lung cancer screening with apparently healthy patients aged 55–74 who have at least a 30 pack-year smoking history, and who currently smoke or have quit within the past 15 yrs. Patient has been involved in the process of informed and shared decision making with a provider related to the potential benefits, limitations, and harms associated with screening for lung cancer with LDCT should occur before any decision is made to initiate lung cancer screening. Smoking cessation counseling remains a high priority for clinical attention in discussions with current smokers, who should be informed of their continuing risk of lung cancer. Screening should not be</p>

			viewed as an alternative to smoking cessation.
Prostate	Men, age 50+, African American Men, age 45+	Digital rectal examination (DRE) and prostate-specific antigen (PSA) test	Men who have at least a 10-yr life expectancy should have an opportunity to make an informed decision with their providers about whether to be screened for prostate cancer, after receiving information about the potential benefits, risks, and uncertainties associated with prostate cancer screening. Prostate cancer screening should not occur without an informed decision making process.
Cancer-related checkup	Men and women, age 20+	On the occasion of a periodic health examination, the cancer-related checkup should include examination for cancers of the thyroid, testicles, ovaries, lymph nodes, oral cavity, and skin, as well as health counseling about tobacco, sun exposure, diet and nutrition, risk factors, sexual practices, and environmental and occupational exposures.	N/A

CT, computed tomography; DNA, deoxyribonucleic acid; HPV, human papillomavirus.

^aAll individuals should become familiar with the potential benefits, limitations, and harms associated with cancer screening.

^bAll positive tests (other than colonoscopy) should be followed up with colonoscopy.

Adapted from American Cancer Society (ACS). (2018). American Cancer Society guidelines for the early detection of cancer. Retrieved on 7/5/2019 at: www.cancer.org/healthy/find-cancer-early/cancer-screening-guidelines/american-cancer-society-guidelines-for-the-early-detection-of-cancer.html

TABLE 12-4 Select Diagnostic Tests Used to Detect Cancer

Test	Description	Examples of Diagnostic Uses
Tumor marker identification	Analysis of biochemical mediators found in tumor tissue, blood, or other body fluids that are indicative of cancer cells or specific characteristics of cancer cells. These biochemical mediators may also be found in some normal body tissues.	Breast, colon, lung, ovarian, testicular, prostate cancers
Genetic tumor markers (also called prognostic indicators)	Analysis for the presence of mutations (alterations) in genes found in tumors or body tissues. Assists in diagnosis, selection of treatment, prediction of response to therapy, and risk of progression or recurrence.	Breast, lung, kidney, ovarian, brain cancers; leukemia; and lymphoma. Many uses of genetic profiling are considered investigational.
Mammography	Use of x-ray images of the breast.	Breast cancer
Magnetic resonance imaging (MRI)	Use of magnetic fields and radiofrequency signals to create sectioned images of various body structures.	Neurologic, pelvic, abdominal, thoracic, breast cancers
Computed tomography (CT) scan	Use of narrow-beam x-ray to scan successive layers of tissue for a cross-sectional view.	Neurologic, pelvic, skeletal, abdominal, thoracic cancers
Fluoroscopy	Use of x-rays that identify contrasts in body tissue densities; may involve the use of contrast agents.	Skeletal, lung, gastrointestinal cancers
Ultrasonography (ultrasound)	High-frequency sound waves echoing off body tissues are converted electronically into images; used to assess tissues deep within the body.	Abdominal and pelvic cancers
Endoscopy	Direct visualization of a body cavity or passageway by insertion of an endoscope into a body cavity or opening; allows tissue biopsy, fluid aspiration, and excision of small tumors. Used for diagnostic and therapeutic purposes.	Bronchial, gastrointestinal cancers
Nuclear medicine imaging	Uses IV injection or ingestion of radioisotopes followed by imaging of tissues that have concentrated the radioisotopes.	Bone, liver, kidney, spleen, brain, and thyroid cancers
Positron emission tomography (PET)	Through the use of a tracer, provides black-and-white or color-coded images of the biologic activity of a particular area, rather than its structure. Used in	Lung, colon, liver, pancreatic, head and neck cancers; Hodgkin and non-

	detection of cancer or its response to treatment.	Hodgkin lymphoma and melanoma
PET fusion	Use of a PET scanner and a CT scanner in one machine to provide an image combining anatomic detail, spatial resolution, and functional metabolic abnormalities.	See PET
Radioimmunoconjugates	Monoclonal antibodies are labeled with a radioisotope and injected IV into the patient; the antibodies that aggregate at the tumor site are visualized with scanners.	Colorectal, breast, ovarian, head and neck cancers; lymphoma and melanoma
Vascular imaging	Use of contrast agents that are injected into veins or arteries and monitored by fluoroscopy, CT, or MRI imaging in order to assess tumor vasculature. Used to assess tumor vascularity prior to surgical procedures.	Liver and brain cancers

Adapted from Fischbach, F. T., & Fischbach, M. A. (2018). *Fischbach's manual of laboratory and diagnostic tests* (10th ed.). Philadelphia, PA: Wolters Kluwer Health.

Chart 12-3

TNM Classification System

T The extent of the primary tumor

N The absence or presence and extent of regional lymph node metastasis

M The absence or presence of distant metastasis

The use of numerical subsets of the TNM components indicates the progressive extent of the malignant disease.

Primary Tumor (T)

Tx Primary tumor cannot be assessed

T0 No evidence of primary tumor

Tis Carcinoma in situ

T1, T2, T3, T4 Increasing size or local extent of the primary tumor

Regional Lymph Nodes (N)

Nx Regional lymph nodes cannot be assessed

N0 No regional lymph node metastasis

N1, N2, N3 Increasing involvement of regional lymph nodes

Distant Metastasis (M)

Mx Distant metastasis cannot be assessed

M0 No distant metastasis

M1 Distant metastasis

Adapted from National Comprehensive Cancer Network (NCCN). (2019c). Cancer staging guide. Retrieved on 7/20/2019 at: www.nccn.org/patients/resources/diagnosis/staging.aspx

Staging describes the size of the tumor, the existence of local invasion, lymph node involvement, and distant metastasis. Several systems exist for classifying the anatomic extent of disease. The tumor, nodes, and metastasis (TNM) system (see [Chart 12-3](#)) is the most common system used to describe the stage of many solid tumors (Amin, Greene, Edge, et al., 2017; NCCN, 2019c).

Staging provides a common language used by health care providers and scientists to accurately communicate about cancer across clinical settings and in research. These systems also provide a convenient shorthand notation that condenses lengthy descriptions into manageable terms for comparisons of treatments and prognoses.

Grading is the pathologic classification of tumor cells (NCI, 2019c). Grading systems seek to define the type of tissue from which the tumor originated and the degree to which the tumor cells retain the functional and histologic characteristics of the tissue of origin (differentiation). Samples of cells used to establish the tumor grade may be obtained from tissue scrapings, body fluids, secretions, washings, biopsy, or surgical excision. This information helps providers predict the behavior and prognosis of various tumors. The grade corresponds with a numeric value ranging from I to IV. Grade I tumors, also

known as well-differentiated tumors, closely resemble the tissue of origin in structure and function. Tumors that do not clearly resemble the tissue of origin in structure or function are described as poorly differentiated or undifferentiated and are assigned grade IV. These tumors tend to be more aggressive, less responsive to treatment, and associated with a poorer prognosis as compared to well-differentiated, grade I tumors. Various staging and grading systems are used to characterize cancers.

Anatomic Stage Group

Once the diagnosis, clinical stage, and histologic grade have been determined, the anatomic stage group, designated by I through IV (representing increasing severity of disease), is assigned to facilitate communication, treatment decisions, and estimation of prognosis. The anatomic stage group is also useful for comparing clinical outcomes.

Management of Cancer

Treatment options offered to patients with cancer are based on treatment goals for each specific type, stage, and grade of cancer. The range of possible treatment goals includes cure, a complete eradication of malignant disease; control, which includes prolonged survival and containment of cancer cell growth; or **palliation**, which involves relief of symptoms associated with the disease and improvement of quality of life. Treatment approaches are not initiated until the diagnosis of cancer has been confirmed and staging and grading have been completed.

The health care team and the patient and family must have a clear understanding of the treatment options and goals. Open communication and support are vital as those involved periodically reassess treatment plans and goals when complications of therapy develop or disease progresses.

Multiple modalities are commonly used in cancer treatment. Various approaches, including surgery, radiation therapy, chemotherapy, hematopoietic stem cell transplantation (HSCT), immunotherapy, and targeted therapy may be used together or at different times throughout treatment. Understanding the principles of each and how they interrelate is important in understanding the rationale and goals of treatment.

Surgery

Surgical removal of the entire cancer remains the ideal and most frequently used treatment method. However, the specific surgical approach may vary for several reasons. Diagnostic surgery is the definitive method for obtaining tissue to identify the cellular characteristics that influence all treatment decisions. Surgery

may be the primary method of treatment, or it may be prophylactic, palliative, or reconstructive.

Diagnostic Surgery

Diagnostic surgery, or biopsy, is performed to obtain a tissue sample for histologic analysis of cells suspected to be malignant. In most instances, the biopsy is taken from the actual tumor; however, in some situations, it is necessary to take a sample of lymph nodes near a suspicious tumor. Many cancers can metastasize from the primary site to other areas of the body through the lymphatic circulation. Knowing whether adjacent lymph nodes contain tumor cells helps the health care team plan the best therapeutic approach to combat cancer that has spread beyond the primary tumor site. The use of injectable dyes and nuclear medicine imaging can help identify the sentinel lymph node or the initial lymph node to which the primary tumor and surrounding tissue drain. Sentinel lymph node biopsy (SLNB), also known as sentinel lymph node mapping, is a minimally invasive surgical approach that in many instances has replaced lymphadenectomy (more invasive lymph node dissections) and the associated complications such as lymphedema and delayed healing. SLNB has been widely adopted for regional lymph node staging in selected cases of melanoma and breast cancer (NCCN, 2019d, 2019e).

Biopsy Types

Biopsy methods include excisional, incisional, and needle biopsy. The biopsy type is determined by the size and location of the tumor, the type of treatment anticipated if the cancer diagnosis is confirmed, and the need for surgery and general anesthesia. The biopsy method that allows for the least invasive approach while permitting the most representative tissue sample is chosen. Diagnostic imaging techniques can be used to assist in locating the suspicious lesion and to facilitate accurate tissue sampling. The patient and family are provided the opportunity and time to discuss the options before definitive plans are made.

Excisional biopsy is used for small, easily accessible tumors. In many cases, the surgeon can remove the entire tumor as well as the surrounding marginal tissues. The removal of normal tissue beyond the tumor area decreases the possibility that residual microscopic malignant cells may lead to a recurrence of the tumor. This approach not only provides the pathologist with the entire tissue specimen for the determination of stage and grade but also decreases the chance of seeding tumor cells (disseminating cancer cells throughout surrounding tissues).

Incisional biopsy is performed if the tumor mass is too large to be removed. In this case, a wedge of tissue from the tumor is removed for analysis. The cells of the tissue wedge must be representative of the tumor mass so that the pathologist can provide an accurate diagnosis. If the specimen does not contain

representative tissue and cells, negative biopsy results do not guarantee the absence of cancer.

Excisional and incisional approaches are often performed through endoscopy. However, a surgical procedure may be required to determine the anatomic extent or stage of the tumor. For example, a diagnostic or staging laparotomy (the surgical opening of the abdomen to assess malignant abdominal disease) may be necessary to assess malignancies such as gastric or colon cancer.

Needle biopsy is performed to sample suspicious masses that are easily and safely accessible, such as some masses in the breasts, thyroid, lung, liver, and kidney. Needle biopsies are most often performed on an outpatient basis. They are fast, relatively inexpensive, easy to perform, and may require only local anesthesia. In general, the patient experiences slight and temporary physical discomfort. In addition, the surrounding tissues are minimally disturbed, thus decreasing the likelihood of seeding cancer cells. Fine-needle aspiration (FNA) biopsy involves aspirating cells rather than intact tissue through a needle that is guided into a suspected diseased area. This type of specimen can only be analyzed by cytologic examination (viewing only cells, not tissue). Often, x-ray, computed tomography (CT) scanning, ultrasonography, or magnetic resonance imaging (MRI) is used to help locate the suspicious area and guide placement of the needle. FNA does not always yield enough material to permit accurate diagnosis, necessitating additional biopsy procedures. A core needle biopsy uses a specially designed needle to obtain a small core of tissue that permits histologic analysis. Most often, this specimen is sufficient to permit accurate diagnosis.

Surgery as Primary Treatment

When surgery is the primary approach in treating cancer, the goal is to remove the entire tumor or as much as is feasible (a procedure sometimes called *debulking*) as well as any involved surrounding tissue, including regional lymph nodes.

Two common surgical approaches used for treating primary tumors are local and wide excisions. Local excision, often performed on an outpatient basis, is warranted when the mass is small. It includes removal of the mass and a small margin of normal tissue that is easily accessible. Wide or radical excisions (*en bloc* dissections) include removal of the primary tumor, lymph nodes, adjacent involved structures, and surrounding tissues that may be at high risk for tumor spread. This surgical method may result in disfigurement and altered functioning, necessitating rehabilitation, reconstructive procedures, or both. However, wide excisions are considered if the tumor can be removed completely and the chances of cure or control are good.

Minimally invasive surgical techniques are increasingly replacing traditional surgery associated with large incisions for a variety of cancers (Yarbro, Wujcik, & Gobel, 2018). Advantages of minimally invasive approaches include less

surgical trauma, decreased blood loss, decreased incidence of wound infection and other complications associated with surgery, decreased surgical time and requirement for anesthesia, decreased postoperative pain and limited mobility, and shorter periods of recovery (Pache, Hübner, Jurt, et al., 2017).

Endoscopic surgery, an example of minimally invasive surgery, uses an endoscope with intense lighting and an attached multichip mini camera that is inserted into the body through a small incision. The surgical instruments are inserted into the surgical field through one or two additional small incisions, each about 1 to 2 cm in length. The camera transmits the image of the involved area to a monitor so that the surgeon can manipulate the instruments to perform the necessary procedure. Endoscopic surgery is used to treat many cancer-related conditions of the thorax (thoracoscopy) and abdomen (laparoscopy).

The use of robotics is another advancement in the surgical treatment of cancer (Yarbro, et al., 2018). The use of robotics during laparoscopic procedures permits the removal of tumors with more precision and dexterity than could be accomplished by laparoscopic surgery alone. Laparoscopic robotic-assisted surgery has been used to treat cancers of the colon, prostate and uterus; however, it remains unclear if long-term cancer-related outcomes are improved with robotic surgery when compared to more conventional surgical approaches (U.S. Food and Drug Administration [FDA], 2019).

Salvage surgery is an additional treatment option that uses an extensive surgical approach to treat the local recurrence of cancer after the use of a less extensive primary approach. Mastectomy to treat recurrent breast cancer after primary lumpectomy and radiation is an example of salvage surgery.

Surgery may completely excise limited areas of metastatic disease (referred to as oligometastatic disease) as well. An example would be colon cancer with one to three small areas of liver metastasis and no evidence of cancer elsewhere. In the past, patients with recurrent or metastatic disease were treated with palliation only as their disease was considered incurable. However, evidence now suggests that there is a possibility of a cure or prolonged survival for select subgroups of patients with certain cancer types (Jang, Kim, Jeong, et al., 2018; Ruiz, Sebagh, Wicherts, et al., 2018).

In addition to surgery that uses surgical blades or scalpels to excise the mass and surrounding tissues, several other types of techniques are available. **Table 12-5** provides examples of select techniques.

A multidisciplinary approach to patient care is essential for the patient undergoing cancer-related surgery. The effects of surgery on the patient's body image, self-esteem, and functional abilities are addressed. If necessary, a plan for postoperative rehabilitation is made before the surgery is performed. The growth and dissemination of cancer cells may have produced distant micrometastases by the time the patient seeks treatment. Therefore, attempting to remove wide margins of tissue in the hope of "getting all the cancer" may not be feasible. This reality substantiates the need for a coordinated multidisciplinary approach to cancer therapy.

Once the surgery has been completed, one or more additional (or adjuvant) modalities may be chosen to increase the likelihood of eradicating the remaining microscopic cancer cells that are undetectable by available diagnostic procedures. However, some cancers that are treated surgically in the very early stages (e.g., skin and testicular cancers) are curable without additional therapy.

TABLE 12-5 Select Techniques Used for Localized Destruction of Tumor Tissue

Type of Procedure	Description	Examples of Use
Chemosurgery	Use of chemicals or chemotherapy applied directly to tissue to cause destruction.	Intraperitoneal chemotherapy for ovarian cancer involving the abdomen and peritoneum.
Cryoablation	Use of liquid nitrogen or a very cold probe to freeze tissue and cause cell destruction.	Cervical, prostate, and rectal cancers.
Electrosurgery	Use of an electric current to destroy tumor cells.	Basal and squamous cell skin cancers.
Laser surgery	Use of light and energy aimed at an exact tissue location and depth to vaporize cancer cells (also referred to as photocoagulation or photoablation).	Dyspnea associated with endobronchial obstructions.
Photodynamic therapy	Intravenous (IV) administration of a light-sensitizing agent (hematoporphyrin derivative) that is taken up by cancer cells, followed by exposure to laser light within 24–48 h; causes cancer cell death.	Palliative treatment of dysphagia associated with esophageal and dyspnea associated with endobronchial obstructions.
Radiofrequency ablation (RFA)	Uses localized application of thermal energy that destroys cancer cells through heat: temperatures exceed 50°C (122°F).	Nonresectable liver tumors, pain control with bone metastasis.

Adapted from DeVita, V. T., Rosenberg S. A., & Lawrence, T. S., (Eds.). (2018). *Cancer: Principles & practice of oncology* (11th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

TABLE 12-6 Types of Palliative Surgery and Interventions

Procedure	Indications
Abdominal shunt placement	Ascites
Biliary stent placement	Biliary obstruction
Bone stabilization	Displaced bone fracture related to metastatic disease
Colostomy or ileostomy	Bowel obstruction
Cordotomy	Pain
Epidural catheter placement (for administering epidural analgesics)	Pain
Excision of solitary metastatic lesion	Metastatic lung, liver, or brain lesion
Gastrostomy, jejunostomy tube placement	Upper gastrointestinal tract obstruction
Hormone manipulation (removal of ovaries, testes, adrenals, pituitary)	Tumors that depend on hormones for growth
Nerve block	Pain
Percutaneous enteral gastrostomy (PEG) tube placement	Enteral nutrition
Pericardial drainage tube placement	Pericardial effusion
Peritoneal drainage tube placement	Ascites
Pleural drainage tube placement	Pleural effusion
Ureteral stent placement	Ureteral obstruction
Venous access device placement (for administering parenteral analgesics)	Pain

Prophylactic Surgery

Prophylactic or risk reduction surgery involves removing nonvital tissues or organs that are at increased risk of developing cancer. The following factors are considered when discussing possible prophylactic surgery:

- Family history and genetic predisposition
- Presence or absence of signs and symptoms
- Potential risks and benefits
- Ability to detect cancer at an early stage
- Alternative options for managing increased risk
- The patient's acceptance of the postoperative outcome

Colectomy, mastectomy, and oophorectomy are examples of prophylactic surgeries. Identification of genetic markers indicative of inherited cancer syndromes or a predisposition to develop some types of cancer plays a role in decisions concerning prophylactic surgeries. However, what is adequate justification for prophylactic surgery remains controversial. For example, several

factors are considered when deciding to proceed with a prophylactic mastectomy, including a family history of breast cancer; positive *BRCA1* or *BRCA2* findings; severity of overall breast cancer risk; a personal history of breast cancer; and individual factors (e.g., younger age, psychological well-being that may influence the patient's decision making process) (Chagpar, 2018; Schott, Vetter, Keller, et al., 2017). Prophylactic surgery is discussed with patients and families along with other approaches for managing increased risk of cancer development. Preoperative education and counseling, as well as long-term follow-up, are provided.

Palliative Surgery

The overall goal of palliative surgery in cancer care is to relieve symptoms and to improve the patient's quality of life (Fahy, 2019; Hanna, Blazer, & Mosca, 2012). Palliative surgery is often performed in an attempt to relieve symptoms such as ulceration, obstruction, hemorrhage, pain, and malignant effusions (see Table 12-6). When surgical cure is not possible, honest and informative communication with the patient and family about the goal of palliative surgery is essential to avoid false hope and disappointment. In certain cases, however, surgical intervention with palliative intent may also be performed as a supportive treatment to relieve symptoms along with other potentially curative cancer treatments (Fahy, 2019; Hanna et al., 2012). Thus, the role of palliative surgery today is no longer limited to end-of-life care.

Reconstructive Surgery

Reconstructive surgery may follow curative or extensive surgery in an attempt to improve function or obtain a more desirable cosmetic effect. It may be performed in one operation or in stages. The surgeon who will perform the surgery discusses possible reconstructive surgical options with the patient before the primary surgery is performed. Reconstructive surgery may be indicated for breast, head and neck, and skin cancers.

The nurse assesses the patient's needs and the impact that altered functioning and body image may have on quality of life. Nurses provide patients and families with opportunities to discuss these issues. The individual needs of the patient undergoing reconstructive surgery and their families must be accurately recognized and addressed.

Nursing Management

Patients undergoing surgery for cancer require general perioperative nursing care (see Chapters 14, 15, and 16). Surgical care is individualized according to age, organ impairment, specific deficits, comorbidities, cultural implications, and altered immunity. Combining other treatment methods, such as radiation and

chemotherapy, with surgery also contributes to postoperative complications, such as infection, impaired wound healing, altered pulmonary or renal function, and the development of venous thromboembolism (VTE). The nurse completes a thorough preoperative assessment for factors that may affect the patient undergoing the surgical procedure.

Preoperatively, the nurse provides the patient and family with verbal and written information about the surgical procedure as well as other interventions that may take place intraoperatively (e.g., radiation implants). Instructions concerning prophylactic antibiotic requirements, diet, and bowel preparation are also provided.

Patients who are undergoing surgery for the diagnosis or treatment of cancer may be anxious about the surgical procedure, possible findings, postoperative limitations, changes in normal body functions, and prognosis. The patient and family require time and assistance to process this information, possible changes, and expected outcomes resulting from the surgery.

The nurse serves as the patient advocate and liaison and encourages the patient and family to take an active role in decision making when possible. If the patient or family asks about the results of diagnostic testing and surgical procedures, the nurse's response is guided by the information that was conveyed previously. The nurse may be asked to explain and clarify information for patients and families that was provided initially but was not grasped because of intense anxiety. It is important that the nurse, as well as other members of the health care team, provide information that is consistent.

Postoperatively, the nurse assesses patient responses to surgery and monitors the patient for possible complications, such as infection, bleeding, thrombophlebitis, wound dehiscence, fluid and electrolyte imbalance, and organ dysfunction. The nurse also provides for the patient's comfort. Postoperative education addresses wound care, pain management, activity, nutrition, and medication information.

Plans for discharge, follow-up, home care, and subsequent treatment and rehabilitation are initiated as early as possible to ensure continuity of care from hospital to home or from a cancer referral center to the patient's local hospital and health care provider. Patients and families are encouraged to use community resources such as the ACS for support and information (see the Resources section at the end of this chapter).

Radiation Therapy

Approximately 60% of patients with cancer receive **radiation therapy** at some point during treatment (Halperin, Wazer, Perez, et al., 2019). Radiation may be used to cure cancer, as in thyroid carcinomas, localized cancers of the head and neck, and cancers of the cervix. Radiation therapy may also be used to control cancer when a tumor cannot be removed surgically or when local nodal metastasis is present. Neoadjuvant (prior to local definitive treatment) radiation

therapy, with or without chemotherapy, is used to reduce tumor size in order to facilitate surgical resection. Radiation therapy may be given prophylactically to prevent local recurrence or spread of microscopic cells from the primary tumor to a distant area (e.g., irradiating the breast and axilla following lumpectomy or mastectomy for breast cancer). Palliative radiation therapy is used to relieve the symptoms of locally advanced or metastatic disease, especially when the cancer has spread to the brain, bone, or soft tissue, or to treat oncologic emergencies, such as superior vena cava syndrome, bronchial airway obstruction, or spinal cord compression.

Two types of ionizing radiation—electromagnetic radiation (x-rays and gamma rays) and particulate radiation (electrons, beta particles, protons, neutrons, and alpha particles)—can be used to kill cells. The most lethal damage is the direct alteration of the DNA molecule within the cells of both malignant and normal tissues. Ionizing radiation can directly break the strands of the DNA helix, leading to cell death. It can also indirectly damage DNA through the formation of free radicals. If the DNA cannot be repaired, the cell may die immediately or may initiate apoptosis (Yarbro et al., 2018).

Replicating cells are most vulnerable to the disruptive effects of radiation (during DNA synthesis and mitosis, e.g., early S, G₂, and M phases of the cell cycle; see Fig. 12-2). Therefore, those body tissues that undergo frequent cell division are most sensitive to radiation therapy. These tissues include bone marrow, lymphatic tissue, epithelium of the gastrointestinal tract, hair follicles, and gonads. Slower-growing tissues and tissues at rest (e.g., muscle, cartilage, nervous system, connective tissues) are relatively radioresistant (less sensitive to the effects of radiation). However, it is important to remember that radiation therapy is localized treatment, and only the tissues that are within the treatment field are affected.

A radiosensitive tumor is one that can be destroyed by a dose of radiation that still allows for cell repair and regeneration in the surrounding normal tissue. If the radiation is delivered when most tumor cells are cycling through the cell cycle, the number of cancer cells destroyed (cell kill) is maximal. Radiation sensitivity is enhanced in tumors that are smaller in size and that contain cells that are rapidly dividing (highly proliferative) and poorly differentiated (no longer resembling the tissue of origin).

Radiation Dosage

The radiation dosage depends on the sensitivity of the target tissues to radiation, the size of the tumor, radiation tolerance of the surrounding normal tissues, and critical structures adjacent to the tumor target. The lethal tumor dose is defined as the dose that will eradicate 95% of the tumor yet preserve normal tissue. In external-beam radiation therapy (EBRT), the total radiation dose is delivered over several weeks in daily doses called *fractions*. This allows healthy tissue to repair and achieves greater cell kill by exposing more cells to the radiation as

they begin active cell division. Repeated radiation treatments over time (fractionated doses) also allow for the periphery of the tumor to be reoxygenated repeatedly, because tumors shrink from the outside inward. This increases the radiosensitivity of the tumor, thereby increasing tumor cell death (Morgan, Ten Haken, & Lawrence, 2018). Newer approaches take advantage of increased radiation beam conformality (better tumor targeting) to administer radiation in fewer doses with larger fractions sizes (hypo-fractionation and stereotactic body radiotherapy [SBRT]).

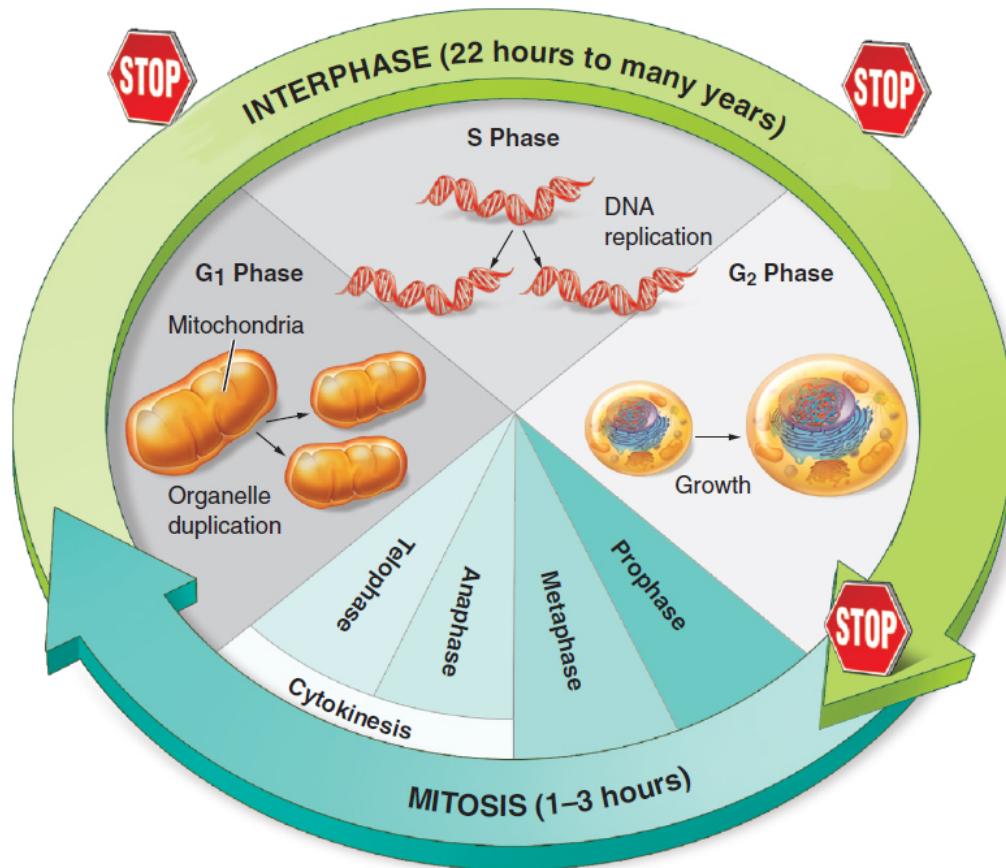


Figure 12-2 • Cell cycle. The cell cycle's four steps are illustrated beginning with G₁ and proceeding to M. The first growth phase (G₁), DNA synthesis phase (S), second growth phase (G₂), and mitosis (M) are illustrated. Reprinted with permission from Grossman, S. G., & Porth, C. M. (2014). *Porth's pathophysiology: Concepts of altered health states* (9th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Administration of Radiation

Radiation therapy can be given in various ways depending on the source of radiation used, the location of the tumor, and the type of cancer. The primary

radiotherapy modalities include EBRT, **brachytherapy** (a form of internal radiation), systemic (radioisotopes), and contact or surface molds.

External Radiation

EBRT is the most commonly used form of radiation therapy. The energy utilized in EBRT is generated either from a linear accelerator or from a unit that generates energy directly from a core source of radioactive material such as a GammaKnife™ unit. Through computerized software programs, both approaches can shape an invisible beam of highly charged photons or gamma rays to penetrate the body and target the tumor with pinpoint accuracy.

Advances in computer technology allow multiple imaging modalities (CT, MRI, and PET scans) to be used to provide three-dimensional images of the tumor, neighboring tissues at risk for microscopic spread, and surrounding normal tissues or organs at risk for radiation-induced injury. These images, referred to as volumetric images, allow the radiation oncologist to plan for multiple radiation beams directed from different angles and different planes so that the beams conform precisely around the tumor (referred to as conformal radiation). The dose of radiation that reaches the surrounding normal tissues is reduced, leading to much less tissue injury than in older forms of radiation therapy (Halperin et al., 2019). Treatment enhancements in EBRT include the ability to control different intensity or energy levels of radiation beams at different angles directed at the tumor, a process known as intensity-modulated radiation therapy (IMRT), which enables higher doses to be delivered to the tumor while sparing the important healthy structures surrounding the tumor (Halperin et al., 2019). IMRT can be given as standard daily fractions or as “hyperfractionated” twice-daily fractions, which shortens the duration of the patient’s treatment schedule. Image-guided radiation therapy (IGRT) uses continuous monitoring of the tumor with ultrasound, x-ray, or CT scans during the treatment to allow for automatic adjustment of the beams as the tumor changes shape or position in an effort to spare the healthy surrounding tissue and reduce side effects. Additional treatment enhancements include respiratory gating, where the treatment delivery is synchronized with the patient’s respiratory cycle, enabling the beam to be adjusted as the tumor or organ moves. These treatment advancements improve tumor destruction while reducing acute and long-term toxicities (Halperin et al., 2019).

Gamma rays generated from the spontaneous decay of naturally occurring solid source of radioactivity, such as cobalt-60, are one of the oldest forms of EBRT. With the advent of modern linear accelerators, the use of solid radioactive elements is confined primarily to the GammaKnife™ stereotactic radiosurgery unit, which is used as a one-time, high-dose delivery of EBRT for treatment of both benign and malignant intracranial lesions.

SBRT is another form of EBRT that uses higher doses of radiation to penetrate very deeply into the body to control deep-seated tumors that cannot be treated by other approaches such as surgery. SBRT is delivered with

considerably higher treatment fraction doses over a short span of time, usually 1 to 5 treatment days, in contrast to daily treatments for 5 days per week for 6 to 8 weeks for conventional EBRT. Specialized linear accelerators with the capability of robotically moving around the patient are used to deliver SBRT, such as the CyberKnife™, Trilogy™, and TomoTherapy™ delivery systems, which are now more commonly available.

Proton therapy is another approach to EBRT. Proton therapy utilizes high linear energy transfer (LET) in the form of charged protons generated by a large magnetic unit called a *cyclotron*. The advantage of proton therapy is that it is capable of delivering its high-energy dose to a deep-seated tumor, with decreased doses of radiation to the tissues in front of the tumor while virtually no energy exits through the patient's healthy tissue behind the tumor (Halperin et al., 2019). Proton therapy permits treatment of deep tumors near critical structures, such as the heart or major blood vessels.

Internal Radiation

Internal radiation includes localized implantation or systemic radionuclide administration. Brachytherapy delivers the dose of radiation to a localized area while systemic radiotherapy relies on strategies for getting the radionuclides closer to the tumor. The specific radioisotope used is selected based on its half-life, which is the time it takes for half of its radioactivity to decay, and the depth of penetration of the radiation.

Brachytherapy

Brachytherapy is the placement of radioactive sources within or immediately next to the cancer site in order to provide a highly targeted, intense dose of radiation beyond a dose that is usually provided by EBRT. In addition, this form of radiation delivery helps to spare exposure to normal surrounding tissue. The radiation source can be implanted by means of needles or rods, seeds, beads, ribbons, or catheters placed into body cavities (vagina, abdomen, pleura), lumens within organs, or interstitial tissue compartments (breast, prostate). Multiple imaging techniques such as ultrasound, CT, or MRI are used to guide placement of radiation sources. Patients may have many fears or concerns about internal radiation, and the nurse explains the various approaches and safety precautions that will be used to protect the patient, family, and health care staff.

Brachytherapy may be delivered as a temporary or a permanent implant. Temporary applications are delivered as high-dose radiation (HDR) for short periods of time, while low-dose radiation (LDR) is delivered over a more extended period. The primary advantage of HDR brachytherapy is that treatment time is shorter, there is reduced exposure to personnel, and the procedure can be performed on an outpatient basis over several days. HDR brachytherapy can be used for intraluminal, surface, interstitial, and intracavitary lesions. Intraluminal HDR brachytherapy involves the insertion of catheters or hollow tubes into the lumens of organs so that the radioisotope can be delivered as close to the tumor

bed as possible. Lesions in the bronchus, esophagus, rectum, or bile duct can be treated with this approach. Contact or surface application is used for treatment of tumors of the eye, such as retinoblastoma in children or ocular melanoma in adults.

Interstitial HDR implants, used in treating such malignancies as prostate, pancreatic, or breast cancer, may be temporary or permanent, depending on the site and radioisotope used. Based on the dose to be delivered (LDR or HDR), the implants may consist of seeds, needles, wires, strands, or small catheters positioned to provide a local radiation source. Prostate HDR therapy is one form of interstitial brachytherapy, in which radioactive strands or wires are placed, while the patient is under anesthesia, into hollow catheters that have been inserted in the perineum close to the prostate gland (Halperin et al., 2019).

Intracavitary radioisotopes are used to treat gynecologic cancers. In these malignancies, the radioisotopes are inserted into specially positioned applicators within the vagina. The applicator placement is verified by x-ray. Treatment can be achieved with either HDR or LDR brachytherapy sources, depending on the extent of disease. LDR therapy requires hospitalization because the patient is treated over several days. HDR intraoperative radiotherapy (IORT) has been used as a treatment approach for advanced gynecologic cancer that has spread to the paraaortic area or pelvic wall (Krengli, Pisani, Deantonio, et al., 2017).

Systemic radiotherapy (radiopharmaceutical therapy) involves oral or intravenous (IV) administration of a therapeutic radioactive isotope targeted to a specific tumor. Radioactive iodine (I-131) is a widely used form of systemic brachytherapy that is the primary treatment for thyroid cancer (Divgi, 2018). Radium-223 dichloride selectively targets prostate cancer bone metastases with high-energy, short-range alpha particles and is approved for the treatment of patients with symptomatic bone metastases and no known visceral metastatic disease (NCCN, 2019f). Radioisotopes are also used as a form of radioimmunotherapy for the treatment of refractory non-Hodgkin lymphoma (see [Chapter 30](#) for more information on lymphoma).

Toxicity

A **toxicity** is an unfavorable and unintended sign, symptom, or condition associated with cancer treatment. Toxicities associated with radiation therapy are most often localized in the region being irradiated and may be increased if concomitant chemotherapy is given (Yarbro et al., 2018). Acute or early toxicities most often begin within 2 weeks of the initiation of treatment and occur when normal cells within the treatment area are damaged and cellular death exceeds regeneration. Body tissues most affected are those that normally proliferate rapidly, such as the skin, the epithelial lining of the gastrointestinal tract, and the bone marrow.

Altered skin integrity is common and can include **alopecia** (hair loss) associated with whole brain radiation. Other skin reactions, referred to as

radiodermatitis, occur along a continuum ranging from erythema and dry desquamation (flaking of skin) to moist or wet desquamation (dermis exposed, skin oozing serous fluid) to, potentially, ulceration. Factors that contribute to the severity of radiodermatitis include the dose and form of radiation; use of concurrent chemotherapy, immunotherapy, or targeted therapy; inclusion of skin folds in the irradiated area; increased age, poor nutritional status, chronic sun exposure, current smoking status, and the presence of medical comorbidities, such as diabetes or kidney failure (Yarbro et al., 2018). Symptoms of radiodermatitis may necessitate treatment interruption, delays, or cessation of therapy. Re-epithelialization occurs after treatments have been completed. Hyperpigmentation, a less severe radiation-associated skin reaction, may develop about 2 to 4 weeks after the initiation of treatment.

Alterations in oral mucosa secondary to radiation therapy in the head and neck region include stomatitis (inflammation of the oral tissues), decreased salivation and xerostomia (dryness of the mouth), and change in or loss of taste. Depending on the targeted region, any portion of the gastrointestinal mucosa may be involved, causing **mucositis** (inflammation of the lining of the mouth, throat, and gastrointestinal tract). For example, patients receiving thoracic irradiation for lung cancer may experience acute esophageal irritation—associated chest pain and dysphagia. Anorexia, nausea, vomiting, and diarrhea may occur if the stomach or colon is in the radiation field. Symptoms subside and gastrointestinal re-epithelialization occurs after treatments have been completed. Bone marrow cells proliferate rapidly, and if sites containing bone marrow (e.g., the iliac crest or sternum) are included in the radiation field, anemia, leukopenia (decreased white blood cells [WBCs]), and thrombocytopenia (a decrease in platelets) may result. The patient is then at increased risk for infection and bleeding until blood cell counts return to normal.

Systemic side effects are commonly experienced by patients receiving radiation therapy. These include fatigue, malaise, and anorexia that may be secondary to biochemical mediators released when tumor cells are destroyed. Fatigue is commonly reported as one of the most distressing cancer-related symptoms. According to Kessels, Husson, and Van Der Feltz-Cornelis (2018), up to 99% of patients with cancer undergoing radiation therapy will experience fatigue. Additionally, patients often report increased severity of fatigue when radiation therapy is used along with other cancer treatments. These early effects tend to be temporary and most often subside within 6 months of the cessation of treatment.

Late effects (approximately 6 months to years after treatment) of radiation therapy may occur in body tissues that were in the field of radiation. These effects are chronic, usually a result of permanent damage to tissues, loss of elasticity, and changes secondary to a decreased vascular supply. Severe late effects include fibrosis, atrophy, ulceration, and necrosis and may affect the lungs, heart, central nervous system, and bladder. With advances in treatment planning and the accuracy of treatment delivery, the occurrence of late toxicities

has diminished. However, late or chronic symptoms, such as dysphagia, incontinence, cognitive impairment, and sexual dysfunction, may persist for several years with implications for survivors' overall health and quality of life (Kessels et al., 2018).

Nursing Management

Nurses anticipate, prevent, and work collaboratively with other providers to manage symptoms associated with radiation therapy in order to promote healing, patient comfort, and quality of life. Symptoms that are not appropriately managed may lead to poor outcomes as a result of interruptions, decreased doses, or early cessation of treatment (Lazarev, Gupta, Ghiassi-Nejad, et al., 2018; Wagner, Zhao, Goss, et al., 2018).

Ideally, nurses consider factors that may be predictive of radiation toxicities or radiosensitivity of tissues. In particular, advanced age, elevated radiation dose, and BMI have been associated with greater toxicity and symptoms (O'Gorman, Sasiadek, Denieffe, et al., 2015). The nature of the relationship between body mass index (BMI) and radiation toxicities is less clear. For example, a decreased BMI was found to be associated with an increased incidence of toxicities in women with cervical cancer (Rubinsak, Kang, Fields, et al., 2018); whereas, an increased BMI (obesity) was associated with increased incidence of late toxicities in men being treated for prostate cancer (Akthar, Liao, Eggner, et al., 2019). Consequently, the area of the body being irradiated must be used as the focus of nursing assessments of radiation toxicities.

In patients receiving EBRT, the nurse assesses the patient's skin, nutritional status, and general feelings of well-being throughout the course of treatment. Evidence-based protocols for nursing management of the toxicities associated with radiation therapy are used. If systemic symptoms such as fatigue occur the nurse explains that these symptoms are a result of the treatment and do not represent deterioration or progression of the disease. The nurse should recommend evidence-based interventions for the management of fatigue, which should include aerobic exercise, which is most effective when adherence is high (Kessels et al., 2018).

Protecting Caregivers

When the patient has a radioactive implant in place, the nurse and other health care providers need to protect themselves, as well as the patient, from the effects of radiation. Patients receiving internal radiation emit radiation while the implant is in place; therefore, contact with the health care team is guided by principles of time, distance, and shielding to minimize exposure of personnel to radiation. Specific instructions are provided by the radiation safety officer from the radiology department and specify the maximum time that can be spent safely in the patient's room, the shielding equipment to be used, and special precautions

and actions to be taken if the implant is dislodged (Halperin et al., 2019). Safety precautions used in caring for a patient receiving brachytherapy include assigning the patient to a private room, posting appropriate notices about radiation safety precautions, having staff members wear dosimeter badges, making sure that pregnant staff members are not assigned to the patient's care, prohibiting visits by children or pregnant visitors, limiting visits from others to 30 minutes daily, and seeing that visitors maintain a 6-foot distance from the radiation source.

Patients with seed implants typically return home; radiation exposure to others is minimal. Information about any precautions, if needed, is provided to the patient and family members to ensure safety. Depending on the dose and energy emitted by a systemic radionuclide, patients may or may not require special precautions or hospitalization (Halperin et al., 2019). The nurse should explain the rationale for these precautions to keep the patient from feeling unduly isolated.

Chemotherapy

Chemotherapy involves the use of antineoplastic drugs in an attempt to destroy cancer cells by interfering with cellular functions, including replication and DNA repair (Norris, 2019). Chemotherapy is used primarily to treat systemic disease rather than localized lesions that are amenable to surgery or radiation. Chemotherapy may be combined with surgery, radiation therapy, or both to reduce tumor size preoperatively (neoadjuvant), to destroy any remaining tumor cells postoperatively (adjuvant), or to treat some forms of leukemia or lymphoma (primary). The goals of chemotherapy (cure, control, or palliation) must be realistic because they will determine the medications that are used and the aggressiveness of the treatment plan.

Cell Kill and the Cell Cycle



Each time a tumor is exposed to chemotherapy, a percentage of the tumor cells (20% to 99%, depending on dosage and agent) are destroyed. Repeated doses of chemotherapy are necessary over a prolonged period to achieve regression of the tumor. Eradication of 100% of the tumor is almost impossible; the goal of treatment is eradication of enough of the tumor so that the remaining malignant cells can be destroyed by the body's immune system (Norris, 2019).

Actively proliferating cells within a tumor are the most sensitive to chemotherapy (the ratio of dividing cells to resting cells is referred to as the growth fraction). Nondividing cells capable of future proliferation are the least sensitive to antineoplastic medications and consequently are potentially dangerous. However, the nondividing cells must be destroyed to eradicate the disease. Repeated cycles of chemotherapy or sequencing of multiple

chemotherapeutic agents is used to achieve more tumor cell destruction by destroying the nondividing tumor cells as they begin active cell division.

Reproduction of both healthy and malignant cells follows the cell cycle pattern (see Fig. 12-2). All cells in the body (healthy and malignant) are affected by chemotherapy. The effect of chemotherapy on the body's cells is best described in relation to the cell cycle. The cell cycle time is the time required for one cell to divide and reproduce two identical daughter cells. The cell cycle of any cell has four distinct phases, each with a vital underlying function (Norris, 2019):

- G₁ phase—RNA and protein synthesis occurs
- S phase—DNA synthesis occurs
- G₂ phase—premitotic phase; DNA synthesis is complete, mitotic spindle forms
- Mitosis—duplicated chromosomes separate, and cell division occurs

Classification of Chemotherapeutic Agents

Chemotherapeutic agents may be classified by their mechanism of action in relation to the cell cycle. Agents that exert their maximal effect during specific phases of the cell cycle are termed *cell cycle-specific agents* (e.g., docetaxal, vinblastine, etoposide). These agents destroy cells that are actively reproducing by means of the cell cycle; most affect cells in the S phase by interfering with DNA and RNA synthesis. Other agents, such as plant alkaloids, are specific to the M phase, where they halt mitotic spindle formation. Chemotherapeutic agents that act independently of the cell cycle phases are termed *cell cycle-nonspecific agents* (e.g., busulfan, cisplatin, bleomycin). These agents usually have a prolonged effect on cells, leading to cellular damage or death. Many treatment plans combine agents that target different phases of the cell cycle to increase the number of vulnerable tumor cells killed during a treatment period (Dickens & Ahmed, 2018).

Chemotherapeutic agents are also classified by chemical group, each with a different mechanism of action. These include the alkylating agents, nitrosoureas, antimetabolites, antitumor antibiotics, topoisomerase inhibitors, plant alkaloids (also referred to as mitotic inhibitors), hormonal agents, and miscellaneous agents. The classification, mechanism of action, cell cycle specificity, and common side effects of select antineoplastic agents are listed in Table 12-7.

Chemotherapeutic agents from multiple categories may be used together to maximize cell destruction. Combination chemotherapy relies on agents with varying mechanisms, potential synergistic actions, and differing toxicities. The use of combination therapy also helps prevent the development of drug-resistant cells (Dickens & Ahmed, 2018).

Adjunct Chemotherapeutic Agents

In certain regimens, additional medications are given with chemotherapy agents to enhance activity or protect normal cells from injury. For example, leucovorin is often given with fluorouracil to treat colorectal cancer. Leucovorin, a compound similar to folic acid, helps fluorouracil bind with an enzyme inside of cancer cells and enhances the ability of fluorouracil to remain in the intracellular environment. Leucovorin also rescues normal cells from the toxic effects of high doses of methotrexate. When given at certain doses for the treatment of some forms of leukemia or lymphoma, methotrexate causes a folic acid deficiency in cells, resulting in cell death. Significant toxicity, including severe bone marrow suppression, mucositis, diarrhea and liver, and lung and kidney damage, can occur. Leucovorin helps to prevent or lessen these toxicities.

Administration of Chemotherapeutic Agents

Chemotherapy may be given in the hospital, outpatient center, or home setting by multiple routes. The route of administration depends on the type of agent; the required dose; and the type, location, and extent of malignant disease being treated. Standards for the safe administration of chemotherapy have been developed by the Oncology Nursing Society (ONS) and the American Society of Clinical Oncology (ASCO) (Neuss, Gilmore, Belderson, et al., 2017). Patient education is essential to maximize safety when chemotherapy is given in the home.

Dosage

The dosage of chemotherapeutic agents is based primarily on the patient's total body surface area, weight, previous exposure and response to chemotherapy or radiation therapy, and function of major organ systems. Dosages are determined to maximize cell kill while minimizing impact on healthy tissues and subsequent toxicities. The therapeutic effect may be compromised if modified and inadequate dosing is required due to toxicities. Modification of dosage is often required if critical laboratory values or the patient's symptoms indicate unacceptable or dangerous toxicities. Chemotherapy treatment regimens include standard-dose therapy, dose-dense regimens (giving chemotherapy more frequently than standard treatment regimens), and myeloablative therapy for HSCT. For certain chemotherapeutic agents, there is a maximum lifetime dose limit that must be adhered to because of the danger of long-term irreversible organ complications (e.g., because of the risk of cardiomyopathy, doxorubicin has a cumulative lifetime dose limit of 550 mg/m²).

Extravasation

Intravenously administered chemotherapy agents are additionally classified by their potential to damage tissue if they inadvertently leak from a vein into surrounding tissue; this leakage is called **extravasation**. The consequences of extravasation range from mild discomfort to severe tissue destruction, depending

on whether the agent is classified as an irritant or vesicant. Irritant agents induce a localized inflammatory reaction at the infusion or injection site, but usually do not cause permanent tissue damage (Olsen, LeFebvre, & Brassil, 2019).

Unlike irritants, **vesicants** are agents that cause inflammation, tissue damage, and possibly necrosis of tendons, muscles, nerves, and blood vessels if extravasation occurs (Olsen et al., 2019). Although the mechanism of vesicant actions varies with each drug, some agents bind to cell DNA and cause cell death that progresses to involve neighboring cells, whereas other agents are metabolized into cells and cause a localized, painful reaction that usually improves over time. Sloughing and ulceration of the tissue may progress to tissue necrosis that is so severe that skin grafting becomes necessary. The full extent of tissue damage may take several weeks to become apparent. Examples of commonly used agents classified as vesicants include dactinomycin, daunorubicin, doxorubicin, nitrogen mustard, mitomycin, vinblastine, and vincristine. Chemotherapy administration safety standards require the availability of defined extravasation management procedures, including antidote order sets and accessibility of antidotes in all settings where vesicant chemotherapy is given (Neuss et al., 2017; Olsen et al., 2019).

Chemotherapy is given only by those who have the knowledge and established competencies for vesicant and extravasation management (Neuss et al., 2017; Olsen et al., 2019). Prevention and management of extravasation are essential. Vesicant chemotherapy should never be given in peripheral veins involving the hand or wrist. Peripheral administration is permitted for short-duration infusions only, and placement of the venipuncture site should be on the forearm area using a soft, plastic catheter. For any frequent or prolonged administration of antineoplastic vesicants, right atrial silastic catheters, implanted venous access devices, or peripherally inserted central catheters (PICCs) should be inserted to promote safety during medication administration and reduce problems with access to the circulatory system ([Figs. 12-3](#) and [12-4](#)).

Hypersensitivity Reactions

Although hypersensitivity reactions (HSRs) can occur with any medication, many chemotherapy agents pose a high risk and have been associated with life-threatening outcomes (Olsen et al., 2019). HSRs are a subgroup of adverse drug reactions that are unexpected and associated with mild or progressively worsening signs and symptoms, such as rash, urticaria, fever, hypotension, cardiac instability, dyspnea, wheezing, throat tightness, and syncope. Immediate HSRs may appear within 5 minutes and up to 6 hours of an infusion. Delayed HSRs may occur after the completion of the infusion. Although patients may or may not react to the first infusion of a chemotherapy agent, repeated exposure increases the likelihood of a reaction.

TABLE 12-7



Select Antineoplastic Agents

Drug Class and Examples	Mechanism of Action	Cell Cycle Specificity	Common Side Effects
Alkylating Agents			
Busulfan, carboplatin, chlorambucil, cisplatin, cyclophosphamide, dacarbazine, altretamine ifosfamide, melphalan, nitrogen mustard, oxaliplatin, thiotepa	Bond with DNA, RNA, and protein molecules leading to impaired DNA replication, RNA transcription, and cell functioning; all resulting in cell death	Cell cycle—nonspecific	Bone marrow suppression, nausea, vomiting, cystitis (cyclophosphamide, ifosfamide), stomatitis, alopecia, gonadal suppression, renal toxicity (cisplatin), and development of secondary malignancies
Nitrosoureas			
Carmustine, lomustine or CCNU, semustine, streptozocin	Similar to alkylating agents; cross the blood-brain barrier	Cell cycle—nonspecific	Delayed and cumulative myelosuppression, especially thrombocytopenia; nausea, vomiting, pulmonary, hepatic, and renal damage
Topoisomerase I Inhibitors			
Irinotecan Topotecan	Induce breaks in the DNA strand by binding to enzyme topoisomerase, preventing cells from dividing	Cell cycle—specific (S phase)	Bone marrow suppression, diarrhea, nausea, vomiting, flulike symptoms (topotecan), rash (etoposide), hepatotoxicity (teniposide)
Topoisomerase II Inhibitors			
Etoposide Teniposide			
Antimetabolites			
5-Azacytidine, capecitabine, cytarabine, edatrexate, fludarabine, 5-fluorouracil (5-FU), gemcitabine, hydroxyurea, cladribine, 6-mercaptopurine, methotrexatepentostatin, 6-thioguanine	Interferes with the biosynthesis of metabolites or nucleic acids necessary for RNA and DNA synthesis; inhibits DNA replication and repair	Cell cycle—specific (S phase)	Nausea, vomiting, diarrhea, bone marrow suppression, stomatitis, renal toxicity (methotrexatepentostatin), hepatotoxicity (6-thioguanine), hand-foot syndrome (capecitibine)
Antitumor Antibiotics			
Bleomycin, dactinomycin, daunorubicin, doxorubicin, epirubicin, idarubicin, mitomycin,	Interfere with DNA synthesis by binding DNA;	Cell cycle—nonspecific	Bone marrow suppression, nausea, vomiting, alopecia, anorexia, cardiac toxicity

mitoxantrone, plicamycin	prevent RNA synthesis	(daunorubicin, doxorubicin), red urine (doxorubicin, idarubicin, epirubicin), pulmonary fibrosis (bleomycin)	
Mitotic Spindle Inhibitors			
<i>Plant alkaloids:</i> vinblastine, vincristine, vinorelbine	Arrest metaphase by inhibiting mitotic tubular formation (spindle); inhibit DNA and protein synthesis	Cell cycle— specific (M phase)	Bone marrow suppression (mild with vincristine), peripheral neuropathies, nausea and vomiting
<i>Taxanes:</i> paclitaxel, docetaxel	Arrest metaphase by inhibiting tubulin depolymerization	Cell cycle— specific (M phase)	Hypersensitivity reactions, bone marrow suppression, alopecia, peripheral neuropathies, mucositis
<i>Epothilones:</i> ixabepilone	Alters microtubules and inhibits mitosis	Cell cycle— specific (M phase)	Peripheral neuropathies, bone marrow suppression, hypersensitivity reactions, hepatic impairment
Hormonal Agents			
Androgens and antiandrogens, estrogens and antiestrogens, progestins and antiprogestins, aromatase inhibitors, luteinizing hormone- releasing hormone analogues, steroids	Bind to hormone receptor sites that alter cellular growth; block binding of estrogens to receptor sites (antiestrogens); inhibit RNA synthesis; suppress cytochrome P450 system	Cell cycle— nonspecific	Hypercalcemia, jaundice, increased appetite, masculinization, feminization, sodium and fluid retention, nausea, vomiting, hot flashes, vaginal estrogen dryness
Miscellaneous Agents			
Asparaginase, procarbazine	Inhibits protein, DNA, and RNA synthesis	Varies	Anorexia, nausea, vomiting, bone marrow suppression, hepatotoxicity, hypersensitivity reaction, pancreatitis
Arsenic trioxide	Causes fragmentation of DNA resulting in cell death; in acute promyelocytic leukemia, it		Nausea, vomiting, electrolyte imbalances, fever, headache, cough, dyspnea, electrocardiogram abnormalities

corrects protein changes and changes malignant T– cells into normal white blood cells.

DNA, deoxyribonucleic acid; RNA, ribonucleic acid.

Adapted from Comerford, K. C., & Durkin, M. T. (2020). *Nursing 2020 drug handbook*. Philadelphia, PA: Wolters Kluwer; Neuss, M. N., Gilmore, T. R., Belderson, K., et al. (2017). 2016 updated American Society of Clinical Oncology/Oncology Nursing Society chemotherapy administration safety standards, including standards for pediatric oncology. *Oncology Nursing Forum*, 44(1), 31–43.

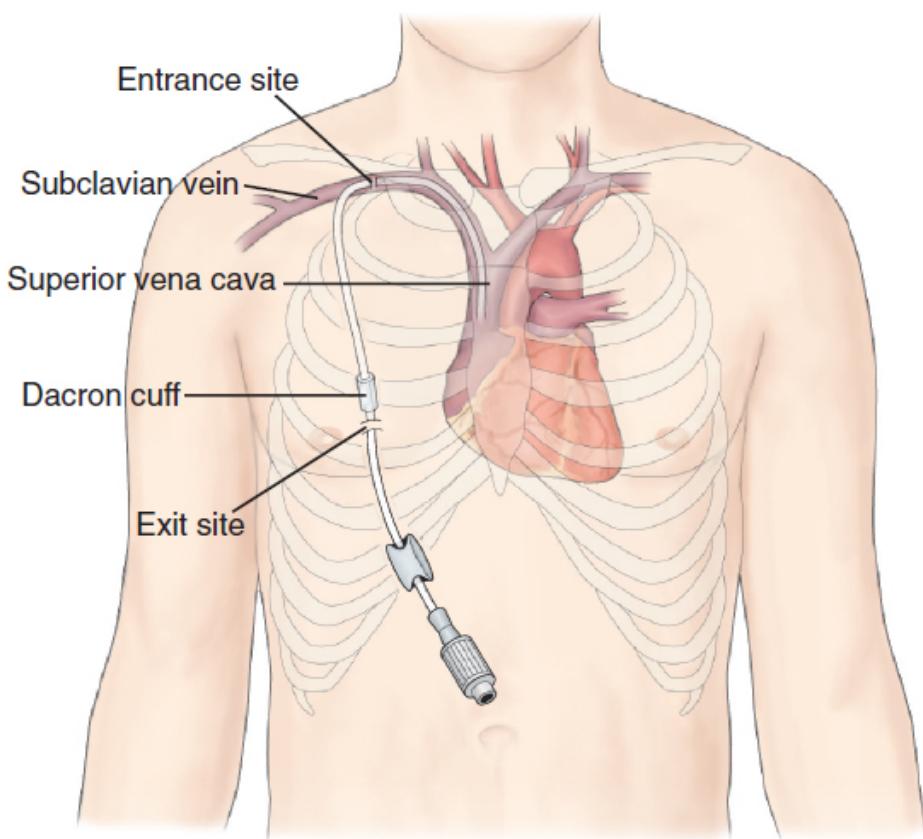


Figure 12-3 • Right atrial catheter. The right atrial catheter is inserted into the subclavian vein and advanced until its tip lies in the superior vena cava just above the right atrium. The proximal end is then tunneled from the entry site through the subcutaneous tissue of the chest wall and brought out through an exit site on the chest. The Dacron cuff anchors the catheter in place and serves as a barrier to infection.

Most immediate HSRs are immunoglobulin E (IgE)-mediated reactions—an allergic reaction (Olsen et al., 2019). Examples of agents that may cause an

allergic, IgE-mediated response include carboplatin, oxaliplatin, and L-asparaginase. However, some HSRs, such as anaphylactoid reactions, are non-IgE-mediated (nonallergic) and a result of cytokine release syndrome (CRS). Rituximab and cetuximab are examples of agents associated with non-IgE-mediated (nonallergic) HSRs. When signs and symptoms of HSR occur, the medication should be discontinued immediately, and emergency procedures initiated. Many institutions have developed specific protocols for responding to HSRs, including standing orders for administration of emergency medications (Roselló, Blasco, García Fabregat, et al., 2017). (See [Chapter 33](#) for further discussion of allergic reactions.)

For some chemotherapeutic agents, especially if they are essential in the treatment plan, desensitization procedures may be possible, and the patient is retreated with the agent at reduced dosages or slower infusion rates. Premedication regimens are used for certain chemotherapy agents to prevent or minimize reactions.

Toxicity

Toxicity associated with chemotherapy can be acute or chronic. Cells with rapid growth rates (e.g., epithelium, bone marrow, hair follicles, sperm) are very susceptible to damage, and the effects may manifest in virtually any body system.

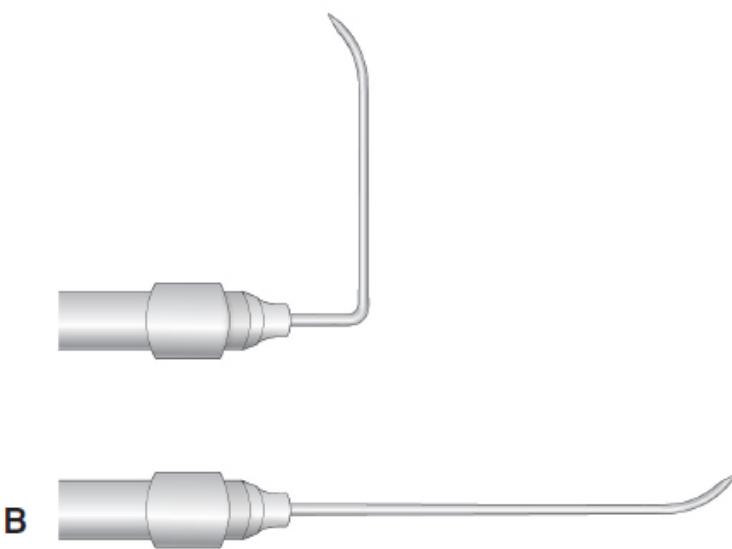
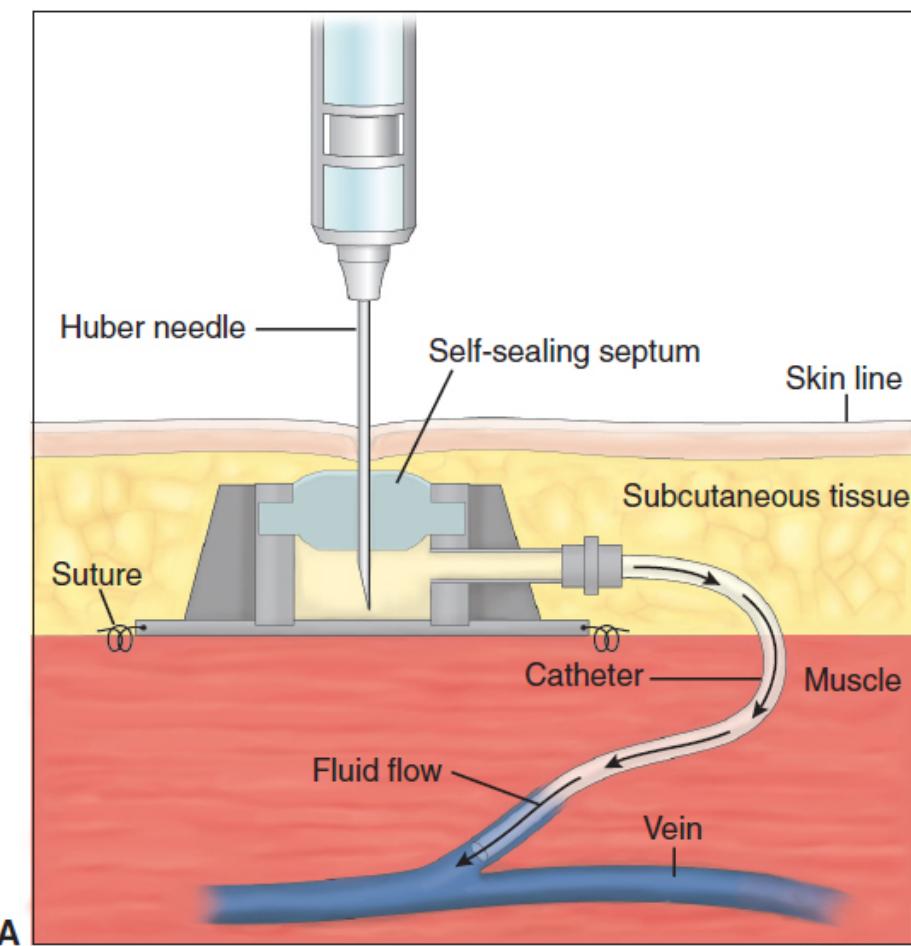


Figure 12-4 • Implanted vascular access device. **A.** A schematic diagram of an implanted vascular access device used for administration of medications, fluids, blood products, and nutrition. The self-sealing septum permits repeated puncture by Huber needles without damage or leakage. **B.** Huber needles used to enter the

implanted vascular port. The 90-degree needle is used for top-entry ports for continuous infusions.

Gastrointestinal System

The most common side effects of chemotherapy are nausea and vomiting, which may persist for 24 to 48 hours; delayed nausea and vomiting may occur up to 1 week after administration. The experience of chemotherapy-induced nausea and vomiting (CINV) may affect quality of life, psychological status, nutrition, fluid and electrolyte status, functional ability, compliance with treatment, and utilization of health care resources (Natale, 2018). Comorbidities, the underlying malignancy, other treatment modalities, and medications, as well as symptoms (e.g., pain), may contribute to CINV. Acute CINV is experienced in the first 24 hours after chemotherapy with a maximal intensity after 5 to 6 hours; delayed CINV occurs 24 hours posttreatment and may last as many as 7 days with a maximal intensity 48 to 72 hours after drug administration (anticipatory nausea and vomiting, occurring prior to administration of chemotherapy, may be a conditioned response triggered by a stimulus such as the smell of the infusion setting, the sight of the nurse, or the outpatient center waiting room) (Olsen et al., 2019).

Several mechanisms are responsible for the occurrence of nausea and vomiting, including activation of multiple receptors found in the vomiting center of the medulla, the chemoreceptor trigger zone, the gastrointestinal tract, the pharynx, and the cerebral cortex. Activation of neurotransmitter receptors in these areas is thought to induce CINV. Stimulation may originate through peripheral, autonomic, vestibular, or cognitive pathways. The primary neuroreceptors known to be implicated in CINV are 5-hydroxytryptamine (5-HT or serotonin) and dopamine receptors (Olsen et al., 2019).

The approach for managing CINV is based on the knowledge of the probability of emesis of the chemotherapy agents used. Algorithms are used to prevent and treat CINV based on national guidelines that consider this classification of chemotherapy agents (NCCN, 2019g).

Corticosteroids, phenothiazines, sedatives, and histamines are helpful, especially when used in combination with serotonin blockers to provide antiemetic protection (NCCN, 2019g). In order to manage delayed nausea and vomiting, antiemetic medications may be combined and are given for the first week at home after chemotherapy. Nonpharmacologic approaches such as relaxation techniques, imagery, acupressure, or acupuncture can help decrease stimuli contributing to symptoms and may be most helpful for patients with anticipatory nausea and vomiting. Small, frequent meals, bland foods, and comfort foods may reduce the frequency or severity of symptoms.

Stomatitis is commonly associated with some chemotherapy agents because of the rapid turnover of epithelium that lines the oral cavity. The entire gastrointestinal tract is susceptible to mucositis. Antimetabolites and antitumor

antibiotics are the major culprits in mucositis and other gastrointestinal symptoms, which can be severe in some patients.

Hematopoietic System

Many chemotherapy agents cause some degree of **myelosuppression** (depression of bone marrow function), resulting in leukopenia (decreased WBCs), **neutropenia** (decreased granulocytes), anemia (decreased red blood cells [RBCs]), thrombocytopenia (decreased platelets), and increased risk of infection and bleeding (Olsen et al., 2019). Depression of these cells is the usual reason for limiting the dose of the chemotherapy. Myelosuppression is predictable; for most agents, patients usually reach the point at which blood counts are lowest 7 to 14 days after chemotherapy has been given. During these 2 weeks, nurses anticipate associated toxicities, especially a fever associated with neutrophil count less than 1,500 cells/mm³. Frequent monitoring of blood cell counts is essential, and patients are educated about strategies to protect against infection, injury, and blood loss, particularly while counts are low.

Other agents—colony-stimulating factors (granulocyte colony-stimulating factor [G-CSF] and granulocyte-macrophage colony-stimulating factor [GM-CSF])—can be given after chemotherapy to stimulate the bone marrow to produce WBCs, especially neutrophils, at an accelerated rate, thus decreasing the duration of neutropenia. G-CSF and GM-CSF decrease the episodes of infection and the need for antibiotics and allow for more timely treatment cycles of chemotherapy with less need to reduce the dosage. Erythropoietin (EPO) stimulates RBC production, thus decreasing the symptoms of treatment-induced chronic anemia and reducing the need for blood transfusions. Interleukin 11 (IL-11) (oprelvekin) stimulates the production of megakaryocytes (precursors to platelets) and can be used to prevent and treat severe thrombocytopenia but has had limited use because of toxicities, such as HSR; capillary leak syndrome; pulmonary edema; atrial arrhythmias; and nausea, vomiting, and diarrhea (Olsen et al., 2019).

Renal System

Some chemotherapy agents can cause renal dysfunction by damaging the blood vessels or filtering structures of the kidneys (Olsen et al., 2019). Clinical manifestations of renal dysfunction from conventional chemotherapy can range from the asymptomatic elevation of serum electrolytes and creatinine levels to acute kidney injury (Merchan, Jhaveri, Berns, et al., 2017). Cisplatin, methotrexate, and mitomycin are particularly toxic to the kidneys (Olsen et al., 2019). Damage to the kidneys may also result in impaired water secretion, leading to syndrome of inappropriate secretion of antidiuretic hormone (SIADH). Rapid tumor cell lysis after chemotherapy results in increased urinary excretion of uric acid, which can cause renal damage. In addition, intracellular contents are released into circulation, resulting in hyperkalemia,

hyperphosphatemia, and hypocalcemia and obstructive nephropathy. (See later discussion of tumor lysis syndrome.)

The monitoring of laboratory values, including blood urea nitrogen (BUN), serum creatinine, creatinine clearance, and serum electrolytes is essential (Olsen et al., 2019). Adequate hydration and diuresis to prevent formation of uric acid crystals and administration of allopurinol may be used to prevent renal toxicity. Amifostine has demonstrated an ability to minimize renal toxicities associated with cisplatin, cyclophosphamide, and ifosfamide therapy.

Hemorrhagic cystitis is a bladder toxicity that can result from cyclophosphamide and ifosfamide, and alkylating agent (e.g., busulfan and thiotepa) therapy (Olsen et al., 2019). Hematuria can range from microscopic to frank bleeding with symptoms ranging from transient irritation during urination, dysuria, and suprapubic pain to life-threatening hemorrhage. Protection of the bladder focuses on aggressive IV hydration, frequent voiding, and diuresis.

Cardiopulmonary System

Several chemotherapy agents are associated with cardiac toxicity. Anthracyclines (e.g., daunorubicin, doxorubicin) are known to cause irreversible cumulative cardiac toxicities when total dosage reaches 400 mg/m² (Henriksen, 2018). If these agents are given in the presence of thoracic radiation therapy or other agents with cardiotoxicity potential, the cumulative dose limit is lower. Patients at increased risk for the development of cardiac toxicities include: extreme ages >65 years or <18 years, female gender, African American race, chest radiation, kidney failure, and preexisting cardiac disease (including hypertension) (Henriksen, 2018). Dexrazoxane has been used on a limited basis as a cardio-protectant when doxorubicin is needed in individuals who have already received a cumulative dose limit and continuation of therapy is deemed beneficial. Patients with known cardiac disease (e.g., heart failure) are treated with lower doses or agents not known to be associated with cardiac toxicity. Cardiac ejection fraction (volume of blood ejected from the heart with each beat) and other signs of heart failure must be monitored closely.

Bleomycin, carmustine, busulfan, mitomycin C, and paclitaxel/docetaxel, among other agents, have toxic effects on lung function, such as alveolar damage, bronchospasm, pneumonitis, and pulmonary fibrosis (Olsen et al., 2019). Therefore, patients are monitored closely for changes in pulmonary function, including pulmonary function test results. Patients with known lung disease are treated with alternative agents not known to cause pulmonary toxicity. When pulmonary toxicity occurs, the agent is discontinued and patients are treated with steroids and other supportive therapies.

Capillary leak syndrome with resultant pulmonary edema is an effect of cytarabine, mitomycin C, cyclophosphamide, and carmustine (Olsen et al., 2019). Subtle onset of dyspnea and cough may progress rapidly to acute respiratory distress and subsequent respiratory failure. Patients who are at significant risk for capillary leak syndrome are monitored closely.

Reproductive System

Testicular and ovarian function can be affected by chemotherapeutic agents, resulting in possible infertility (Olsen et al., 2019). Women may develop problems with ovulation or early menopause, whereas men may develop temporary or permanent azoospermia (absence of spermatozoa). Because treatment may damage reproductive cells, banking of sperm is often recommended for men before treatment is initiated (Oktay, Harvey, Partridge, et al., 2018). Options available for women prior to initiation of chemotherapy include cryopreservation (freezing) of oocytes, embryos, or ovarian tissue. Patients and their partners are informed about potential changes in reproductive function resulting from chemotherapy. In addition, many chemotherapy agents are known or thought to be teratogenic. Therefore, patients are advised to use reliable methods of birth control while receiving chemotherapy and not to assume that infertility has resulted (Olsen et al., 2019).

Neurologic System

Chemotherapy-induced neurotoxicity, a potentially dose-limiting toxicity, can affect the central, peripheral, and autonomic nervous systems (Olsen et al., 2019). Neurotoxicity characterized by metabolic encephalopathy can occur with ifosfamide, high-dose methotrexate, and cytarabine. With repeated doses, the taxanes and plant alkaloids, especially vincristine, can cause cumulative peripheral nervous system damage with sensory alterations in the feet and hands. These sensations can be described as tingling, pricking, or numbness of the extremities; burning or freezing pain; sharp, stabbing, or electric shock-like pain; and extreme sensitivity to touch. If unreported by patients or undetected, progressive motor axon damage can lead to loss of deep tendon reflexes, with muscle weakness, loss of balance and coordination, and paralytic ileus.

Some chemotherapy agents (e.g., paclitaxel and gemcitabine) can cause severe peripheral neuropathies that may lead to diminished quality of life and functional abilities and result in dose reductions, a change in chemotherapy regimen, or early cessation of treatment (Haryani, Fetzer, Wu, et al., 2017). Although often reversible, these side effects may take many months to resolve or persist indefinitely. Along with the usual paresthesias of the hands and feet, oxaliplatin has a unique and frightening neurotoxicity presentation that is often precipitated by exposure to cold and is characterized by pharyngolaryngeal dysesthesia consisting of lip paresthesia, discomfort or tightness in the back of the throat, inability to breathe, and jaw pain (Olsen et al., 2019).



Quality and Safety Nursing Alert

Patients receiving oxaliplatin must be instructed to avoid drinking cold fluids or going outside with hands and feet exposed to cold temperatures to avoid exacerbation of these symptoms for 3 to 4 days after therapy (Olsen et al., 2019). Cisplatin may cause peripheral neuropathies and hearing loss due to damage to the acoustic nerve.

Cognitive Impairment

Many patients with cancer have trouble with remembering dates, multitasking, managing numbers and finances, organization, face or object recognition, inability to follow directions, feeling easily distracted, and motor and behavioral changes. Although not completely understood, these are viewed as symptoms of cognitive impairment. Cognitive impairment is a multidimensional concept involving a decline in information-handling processes in several domains, including attention and concentration, executive function, information processing speed, language, motor function, visuospatial skill, learning, and memory (Jansen, 2017). Commonly referred to by patients as “chemo brain” or “chemo fog,” cognitive impairment has been associated with both cancer and cancer treatments, including surgery, radiation, chemotherapy, and targeted agents. The symptoms may be subtle or profound with potential negative effects on functional abilities, employment, independence, quality of life, and psychosocial status. Comorbidities, age, medications, pain, impaired nutrition, anemia, fatigue, fluid and electrolyte disturbances, organ dysfunction, infection, and hormonal imbalances are factors that may contribute to the experience of cognitive impairment and make it difficult to fully understand. Underlying mechanisms of cognitive impairment in patients with cancer being explored include neurotoxic effects, oxidative stress, hormonal changes, immune dysregulation, cytokine release, clotting, genetic predisposition, and accelerated aging processes (Jansen, 2017).

Fatigue

Cancer-related fatigue has been defined as an unusual, persistent, and subjective sense of tiredness that is not proportional to recent activity and interferes with usual functioning (NCCN, 2019f). Fatigue is a distressing side effect for most patients that greatly affects quality of life, during treatment and for months after treatment. The health care team works together to identify effective pharmacologic and nonpharmacologic approaches for fatigue management.

Nursing Management



Nurses play an important role in assessing and managing many of the problems experienced by patients receiving chemotherapy. Chemotherapy agents affect

both normal and malignant cells; therefore, their effects are often widespread, affecting many body systems.

Laboratory and physical assessments of metabolic indices and the dermatologic, hematologic, hepatic, renal, cardiovascular, neurologic, and pulmonary systems are critical in evaluating the body's response to chemotherapy. These assessments are performed prior to, during, and after a course of chemotherapy to determine optimal treatment options, evaluate the patient's response, and monitor toxicity. Patients are monitored for long-term effects of chemotherapy after active treatment has been completed during the period of survivorship (see [Chart 12-4](#)).

Assessing Fluid and Electrolyte Status

Anorexia, nausea, vomiting, altered taste, mucositis, and diarrhea put patients at risk for nutritional and fluid and electrolyte disturbances. Therefore, it is important for the nurse to assess the patient's nutritional and fluid and electrolyte status on an ongoing basis and to identify creative ways to encourage an adequate fluid and dietary intake.

Assessing Cognitive Status

Nurses should assess patients routinely for indications of cognitive impairment. Prior to the initiation of treatment, patients and families should be informed about the possibility of cognitive impairment. Nursing assessment plays an important role in determining the need for referral for neurocognitive evaluation and intervention (Jansen, 2017).

Chart 12-4

Potential Long-Term Complications of Cancer Chemotherapy

Abnormalities in senses of taste, smell, and touch
Abnormal balance, tremors, or weakness
Avascular necrosis
Cardiovascular toxicity (coronary artery disease, myocardial infarction, congestive heart failure, valvular heart disease, peripheral arterial disease)
Decreased libido
Dental caries
Dry mouth
Dysphagia
Dyspnea on exertion
Growth retardation in children
Herpes infections (zoster and varicella)
Hypothyroidism
Immune dysfunction
Infertility
Osteoporosis
Pericarditis (acute or chronic)
Pneumococcal sepsis
Pneumonitis (acute or chronic)
Secondary cancers:
 Acute myeloid leukemia
 Myelodysplastic syndromes
 Non-Hodgkin lymphomas
 Solid tumors (especially bone and soft tissue, lung, breast)
 Thyroid cancer
 Thymic hyperplasia

Adapted from Yarbro, C. H., Wujcik, D., & Gobel, B. H. (2018). *Cancer nursing: Principles and practice*. Burlington, MA: Jones & Bartlett Publishers.

Modifying Risks for Infection and Bleeding

Suppression of the bone marrow and immune system is expected and frequently serves as a guide in determining appropriate chemotherapy dosage but increases the risk of anemia, infection, and bleeding disorders. Nursing assessment and care address factors that would further increase the patient's risk. The nurse's role in decreasing the risk of infection and bleeding is discussed further in the Nursing Care of the Patient with Cancer section.

Administering Chemotherapy

Nurses must be aware of chemotherapy and other agents most associated with HSRs, strategies for prevention, signs and symptoms characteristic of HSRs, and the appropriate early and time-sensitive interventions for preventing progression to anaphylaxis. Nurses provide patient and family education that emphasizes two

key points: the importance of adhering to prescribed self-administered premedication before presenting to the infusion center and recognizing and reporting the signs and symptoms to the nurse once the infusion has started. Patients and families are also educated about signs and symptoms that may occur at home following discharge from the infusion area that may warrant medication administration or immediate transport to the emergency department for further assessment and treatment.

The local effects of the chemotherapeutic agent are also of concern. The patient is observed closely during administration of the agent because of the risk and consequences of extravasation. Prevention of extravasation is essential and relies on vigilant nursing care (Neuss et al., 2017). Selection of peripheral veins, skilled venipuncture, and careful administration of medications are essential. Peripheral administration is limited to short duration (less than 1 hour; IV push or bolus) infusions using only a soft, plastic catheter placed in the forearm area (Olsen et al., 2019). Continuous infusion of vesicants that takes longer than 1 hour or are given frequently are given only via a central line, such as a right atrial silastic catheter, implanted venous access device, or PICC. These long-term venous access devices promote safety during medication administration and reduce problems with repeated access to the circulatory system (see Figs. 12-3 and 12-4). Indwelling or subcutaneous venous access devices require consistent nursing care. Complications include infection and thrombosis (Voog, Campion, Du Rusquec, et al., 2018).

Indications of extravasation during administration of vesicant agents include the following:

- Absence of blood return from the IV catheter
- Resistance to flow of IV fluid
- Burning or pain, swelling, or redness at the site



Quality and Safety Nursing Alert

If extravasation is suspected, the medication administration is stopped immediately.

An extravasation kit should be readily available with emergency equipment and antidote medications, as well as a quick reference for how to properly manage an extravasation of the specific vesicant agent used (although evidence-based data regarding effective antidotes are limited) (Neuss et al., 2017; Olsen et al., 2019). Nurses should refer to their organization's policy and procedures for reporting, managing, and documenting extravasation. Safety standards require the availability of defined extravasation management procedures, including antidote order sets and accessibility of antidotes in all settings where vesicant chemotherapy is given (Neuss et al., 2017). Recommendations and guidelines for managing vesicant extravasation, which vary with each agent, have been issued

by individual medication manufacturers, pharmacies, and the ONS (Neuss et al., 2017; Olsen et al., 2019).

Difficulties or problems with administration of chemotherapeutic agents are brought to the attention of the primary provider promptly so that corrective measures can be taken to minimize local tissue damage.

The nurse evaluates the patient receiving neurotoxic chemotherapy, communicates findings with the medical oncologist, provides education to patients and families, and makes appropriate referrals for complete neurologic evaluation and occupational or rehabilitative therapies.

Preventing Nausea and Vomiting

Nurses are integral to the prevention and management of CINV. They collaborate with other members of the oncology care team to identify factors contributing to the experience of CINV and select effective antiemetic regimens that maximize currently available therapies. Nurses provide education for patients and families regarding antiemetic regimens and care for delayed CINV that may continue at home after the chemotherapy infusion has completed (NCCN, 2019g).

Managing Cognitive Changes

Although several approaches have been explored, no evidence-based guidelines for the prevention, treatment, or management of cognitive impairment have been established. Examples of nonpharmacologic approaches that nurses recommend to patients include exercise, natural restorative environmental intervention (walking in nature or gardening), and cognitive training programs (Jansen, 2017). Nurses should assist patients to address factors, such as fluid and electrolyte imbalances, nutrition deficits, fatigue, pain, and infection to minimize their contribution to cognitive impairment.

Managing Fatigue

Fatigue is a common side effect of chemotherapy. Nurses assist patients to explore the role that the underlying disease processes, combined treatments, other symptoms, and psychosocial distress play in the patient's experience of fatigue. In addition, nurses work with the patient and other team members to identify effective approaches for fatigue management (NCCN, 2019h).

Protecting Caregivers

Nurses involved in handling chemotherapeutic agents may be exposed to low doses of the agents by direct contact, inhalation, or ingestion (Menonna-Quinn, Polovich, & Marshall, 2019). Skin and eye irritation, nausea, vomiting, nasal mucosal ulcerations, infertility, low-birth-weight babies, congenital anomalies, spontaneous abortions, and mutagenic substances in urine have been reported in nurses preparing and handling chemotherapy agents. The Occupational and

Safety Health Administration (OSHA), the ONS, hospitals, and other health care agencies have developed specific precautions for health care providers involved in the preparation and administration of chemotherapy and for handling materials exposed to body fluids of those who have received these hazardous agents (see [Chart 12-5](#)) (Neuss et al., 2017; Olsen et al., 2019). Nurses must be familiar with their institutional policies and procedures regarding personal protective equipment, handling and disposal of chemotherapy agents and supplies, and management of accidental spills or exposures. Emergency spill kits should be readily available in any treatment area where chemotherapy is prepared or given. Precautions must also be taken when handling any bodily fluids or excreta from the patient, as many agents are excreted unaltered in urine and feces. Nurses in all treatment settings have a responsibility to educate patients, families, caregivers, assistive personnel, and housekeepers concerning precautions.

Hematopoietic Stem Cell Transplantation

HSCT has been used to treat several malignant and nonmalignant diseases for many years (Yarbro et al., 2018). In adults, HSCT is most commonly used to treat certain hematologic malignancies (e.g., malignant myeloma, acute leukemia, non-Hodgkin lymphoma), and less commonly some solid tumors (e.g., germ cell tumors, breast cancer, neuroblastomas).

The process of obtaining hematopoietic stem cells (HSCs) has evolved over the years (Yarbro et al., 2018). Historically, HSCs were obtained in the operating room by harvesting large amounts of bone marrow tissue from a donor under general anesthesia. However, peripheral blood stem cell collection using the process of apheresis now accounts for the vast majority of HSCT procedures (Yarbro et al., 2018). The cells collected are specially processed and reinfused into the patient. This method of collecting HSCs is a safe and a more cost-effective means of collection than the process of harvesting of marrow. Stem cells can also be collected from umbilical cord blood harvested from the placenta of newborns at birth that is cryopreserved and stored for later use (Yarbro et al., 2018).

Types of Hematopoietic Stem Cell Transplantation

Types of HSCT are based on the source of donor cells and the treatment (conditioning) regimen used to prepare the patient for stem cell infusion and eradicate malignant cells (Yarbro et al., 2018). These include:

- *Allogeneic HSCT* (AlloHSCT): From a donor other than the patient (may be a related donor such as a family member or a matched unrelated donor from the National Bone Marrow Registry or Cord Blood Registry)
- *Autologous*: From the patient

- *Syngeneic*: From an identical twin
- *Myeloablative*: Consists of giving patients high-dose chemotherapy and, occasionally, total-body irradiation
- *Nonmyeloablative*: Also called *mini-transplants*; does not completely destroy bone marrow cells

Chart 12-5

Safety in Handling Chemotherapy for Health Care Providers

- When preparing (compounding, reconstituting) chemotherapy for administration, use the following safety equipment to prevent exposure through inhalation, direct contact, and ingestion:
 - Class II or III biologic safety cabinet (BSC)
 - Closed-system transfer devices
 - Puncture and leak-proof containers, IV bags
 - Needleless systems (e.g., IV tubing and syringes)
- If BSC is not available when preparing chemotherapy for administration, use the following safety equipment to minimize exposure:
 - Surgical N-95 respirator to provide respiratory and splash protection
 - Eye and face protection (both face shield and goggles) working at or above eye level or cleaning a spill
- When preparing or administering chemotherapy or handling linens and other materials contaminated with chemotherapy or blood and body fluids of patients receiving chemotherapy, wear the following for personal protection:
 - Double layer of powder-free gloves specifically designated for chemotherapy handling (the inner glove is worn under the gown cuff and the outer glove is worn over the cuff)
 - Long sleeve, disposable gowns (without seams or closures that can allow drugs to pass through) made of polyethylene-coated polypropylene or other laminate materials
- Linens contaminated with chemotherapy or blood and body fluids of patients receiving chemotherapy should be placed in the following:
 - Closed-system, puncture- and leak-proof containers labeled “hazardous: chemotherapy contaminated linens”
 - Above referenced container maintained in the infusion center soiled utility room for outpatient settings
 - Above referenced container maintained in the patient room or soiled utility room for inpatient settings
- Chemotherapy preparation equipment (e.g., syringes, tubing, empty vials, etc.), gowns, and gloves should be disposed of in:
 - Closed-system, puncture- and leak-proof containers labeled “hazardous: chemotherapy contaminated waste”
- Wash hands with soap and water after removing gloves used to prepare or administer chemotherapy or clean contaminated linens and other materials
- “Spill kits” with the appropriate gowns, gloves, disposable absorbent materials for cleansing large areas, and hazard sign should be kept in all areas where chemotherapy is prepared and given
- Implement a quality improvement program addressing safe chemotherapy handling that includes the following:
 - Standard operating policies and procedures for:
 - Chemotherapy handling, preparation, and disposal

- Handling and disposal of chemotherapy spills
- Handling and disposal of blood and body fluids and contaminated materials of patients receiving chemotherapy
- Conduct competency-based education, training, and performance evaluations regarding chemotherapy safety procedures at orientation and at subsequent regular intervals
- Medical monitoring program to identify indicators of exposure
- Root cause analysis for all chemotherapy spills and exposure incidents

Adapted from National Institute for Occupational Safety and Health. (2008).

Personal protective equipment for health care workers who work with hazardous drugs. Retrieved on 7/11/2019 at: www.cdc.gov/niosh/docs/wp-solutions/2009-106/pdfs/2009-106.pdf?id=10.26616/NIOSH_PUB_2009_106;

Olsen, M. M., LeFebvre, K. B., & Brassil, K. (Eds.) (2019). *Chemotherapy and immunotherapy guidelines and recommendations for practice*. Pittsburgh, PA: Oncology Nursing Society.

AlloHSCTs are used primarily for diseases of the bone marrow and are dependent on the availability of a human leukocyte antigen-matched donor, which greatly limits the number of possible transplants. An advantage of AlloHSCT is that the transplanted cells should not be immunologically tolerant of a patient's malignancy and should cause a lethal **graft-versus-tumor effect** in which the donor cells recognize the malignant cells and act to eliminate them.

AlloHSCT may involve either myeloablative (high-dose) or nonmyeloablative (mini-transplant) chemotherapy (Yarbro et al., 2018). In ablative AlloHSCT, the recipient receives high doses of chemotherapy and possibly total-body irradiation to completely eradicate (ablate) the bone marrow and any malignant cells and help prevent rejection of the donor stem cells. The collected HSCs that are infused IV into the recipients travel to sites in the body where they produce bone marrow and establish themselves through the process of engraftment. Once engraftment is complete (8 to 10 days, sometimes longer), the new bone marrow becomes functional and begins producing RBCs, WBCs, and platelets. In nonablative AlloHSCT, the chemotherapy doses are lower and aimed at destroying malignant cells (without completely eradicating the bone marrow), thus suppressing the recipient's immune system to allow engraftment of donor stem cells. The lower doses of chemotherapy, associated with less organ toxicity and infection, can be used for older patients or those with underlying organ dysfunction for whom high-dose chemotherapy would be prohibitive (Yarbro et al., 2018). After engraftment, it is hoped that the donor cells will create a graft-versus-tumor effect. Before engraftment, patients are at high risk for infection, sepsis, and bleeding. Side effects of the high-dose chemotherapy and total-body irradiation can be acute and chronic (Negrin, 2018; Yarbro et al., 2018). Acute side effects include headache, alopecia, nausea, vomiting, mucositis, diarrhea, fluid and electrolyte imbalances, and acute kidney injury. Chronic side effects include infertility; pulmonary, cardiac, liver, and kidney

dysfunction; osteoporosis and avascular bone necrosis; diabetes; and secondary malignancies.

During the first 30 days after the conditioning regimen, AlloHSCT patients are at risk for developing hepatic sinusoidal obstructive syndrome (HSOS) (previously referred to as veno-occlusive disease) related to chemotherapy-induced inflammation of the sinusoidal epithelium (Negrin & Bonis, 2019). Inflammation causes embolization of RBCs, resulting in destruction, fibrosis, and occlusion of the sinusoids. Clinical manifestations of HSOS may include weight gain, hepatomegaly, increased bilirubin, and ascites. Although various approaches have been used to treat HSOS, evidence-based strategies have not emerged. The use of peripheral stem cells, specific chemotherapy dosing, and nonmyeloablative regimens have been associated with a decreased incidence (Negrin, 2018; Yarbro et al., 2018).

Graft-versus-host disease (GVHD), a major cause of morbidity and mortality in 30% to 50% of the allogeneic transplant population, occurs when the donor lymphocytes initiate an immune response against the recipient's tissues (e.g., skin, gastrointestinal tract, liver) during the beginning of engraftment (Yarbro et al., 2018). The donor cells view the recipient's tissues as foreign or immunologically different from what they recognize as "self" in the donor. To prevent GVHD, patients receive immunosuppressant drugs, such as cyclosporine, methotrexate, tacrolimus, or mycophenolate mofetil.

GVHD may be acute, occurring within the first 100 days, or chronic, occurring after 100 days (Yarbro et al., 2018). Clinical manifestations of acute GVHD include diffuse rash progressing to blistering and desquamation similar to second-degree burns; mucosal inflammation of the eyes and the entire gastrointestinal tract with subsequent diarrhea that may exceed 2 L per day; and biliary stasis with abdominal pain, hepatomegaly, and elevated liver enzymes progressing to obstructive jaundice. The first 100 days or so after AlloHSCT is crucial for patients; the immune system and blood-making capacity (hematopoiesis) must recover sufficiently to prevent infection and hemorrhage.

Autologous HSCT (AuHSCT) is considered for patients with disease of the bone marrow who do not have a suitable donor for AlloHSCT or for patients who have healthy bone marrow but require bone marrow-ablative doses of chemotherapy to cure an aggressive malignancy (Yarbro et al., 2018). The most common malignancies treated with AuHSCT include lymphoma and multiple myeloma. However, the use of AuHSCT has gained increasing acceptance in treating neuroblastoma, Ewing sarcoma, and germ cell tumors. Stem cells are collected from the patient and preserved for reinfusion; if necessary, they are treated to kill any malignant cells within the marrow, called *purging*. The patient is then treated with ablative chemotherapy and, possibly, total-body irradiation to eradicate any remaining tumor. Stem cells are then reinfused. Until engraftment occurs in the bone marrow sites of the body, there is a high risk of infection, sepsis, and bleeding. Acute and chronic toxicities from chemotherapy and radiation therapy may be severe. The risk of HSOS is also present after

autologous transplantation. No immunosuppressant medications are necessary after AuHSCT, because the patient does not receive foreign tissue. A disadvantage of AuHSCT is the risk that tumor cells may remain in the bone marrow despite high-dose chemotherapy (conditioning regimens).

Syngeneic transplants result in less incidence of GVHD and graft rejection; however, there is also less graft-versus-tumor effect to fight the malignancy. For this reason, even when an identical twin is available for marrow donation, another matched sibling or even an unrelated donor may be the most suitable donor to combat an aggressive malignancy (Yarbro et al., 2018).

Nursing Management

Nursing care of the patient undergoing HSCT is complex and demands a high level of skill. The success of HSCT is greatly influenced by nursing care throughout the transplantation process.

Implementing Care Before Treatment

All patients must undergo extensive evaluations before HSCT to assess the current clinical status of the disease. Nutritional assessments, extensive physical examinations, organ function tests, and psychological evaluations are conducted. Blood work includes assessing past infectious antigen exposure (e.g., hepatitis virus, cytomegalovirus, herpes simplex virus, human immunodeficiency virus, syphilis). The patient's social support systems and financial and insurance resources are also evaluated. Informed consent and patient education about the procedure and care before and after HSCT are vital.

Providing Care During Treatment

Skilled nursing care is required during the treatment phase of HSCT when high-dose chemotherapy (conditioning regimen) and total-body irradiation are given. The acute toxicities of nausea, diarrhea, mucositis, and hemorrhagic cystitis require close monitoring and symptom management by the nurse.

Nursing management during stem cell infusion consists of monitoring the patient's vital signs and blood oxygen saturation; assessing for adverse effects, such as fever, chills, shortness of breath, chest pain, cutaneous reactions, nausea, vomiting, hypotension or hypertension, tachycardia, anxiety, and taste changes; and providing strategies for symptom control, ongoing support, and patient education. During stem cell infusion, patients may experience adverse reactions to the cryoprotectant dimethylsulfoxide (DMSO) used to preserve the harvested stem cells. Less common toxicities include neurologic and renal impairment (Yarbro et al., 2018).



Quality and Safety Nursing Alert

Until engraftment of the new marrow occurs, the patient undergoing HSCT is at high risk for death from sepsis and bleeding.

A cluster of symptoms referred to as engraftment syndrome may occur during the neutrophil recovery phase in both allogeneic and autologous transplants. Clinical features of this syndrome vary widely but may include noninfectious fever associated with skin rash, weight gain, diarrhea, and pulmonary infiltrates, with improvement noted after the initiation of corticosteroid therapy rather than antibiotic therapy (Mutahar & Al-Anazi, 2017). Until engraftment is well established, the patient requires support with blood products and hematopoietic growth factors.

Potential infections may be bacterial, viral, fungal, or protozoan in origin. During the first 30 days following transplant, the patient is most at risk for developing reactivations of viral infections, including herpes simplex, EBV, cytomegalovirus, and varicella zoster. Mucosal denudement poses a risk for *Candida* (yeast) infection locally and systemically. Pulmonary toxicities offer the opportunity for fungal infections such as *Aspergillus*. Renal complications arise from the nephrotoxic chemotherapy agents used in the conditioning regimen or those used to treat infection (amphotericin B, aminoglycosides). A neutropenic diet is usually prescribed for patients to decrease the risk of exposure to foodborne infections from bacteria, yeast, molds, viruses, and parasites (Yarbro et al., 2018).

Tumor lysis syndrome and acute tubular necrosis are also potential complications after HSCT. Nursing assessment for signs of these complications is essential for early identification and treatment. GVHD requires skillful nursing assessment to detect early effects on the skin, liver, and gastrointestinal tract. HSOS resulting from the conditioning regimens used can result in fluid retention, jaundice, abdominal pain, ascites, tender and enlarged liver, and encephalopathy. Pulmonary complications, such as pulmonary edema, interstitial pneumonia, and other pneumonias, often complicate recovery.

Providing Care After Treatment

Nursing care following HSCT includes care of recipients and donors. These are discussed in the following sections.

Caring for Recipients

Ongoing nursing assessment during follow-up visits is essential to detect late effects of therapy after HSCT, which may occur 100 days or more after the procedure (Yarbro et al., 2018). Late effects include infections (e.g., varicella-zoster infection), restrictive pulmonary abnormalities, and recurrent pneumonias. Infertility often results due to total-body irradiation, chemotherapy, or both as components of the ablative regimen. Chronic GVHD can involve the skin, liver,

intestine, esophagus, eyes, lungs, joints, and vaginal mucosa. Cataracts may also develop after total-body irradiation.

There is a high potential of psychological distress following an HSCT, which has been associated with delayed recovery, mortality, and increased rates of complications, such as GVHD (Hermioni, Christina, Melanie, et al., 2019). Thus, psychosocial assessments by nursing staff must be ongoing and a priority. In addition to the multiple physical and psychological stressors affecting patients at each phase of the transplantation experience, the nature of the treatment and patient experience can place extreme emotional, social, financial, and physical demands on family, friends, and donors. Nurses assess the family and other caregivers' needs and provide education, support, and information about resources.

Caring for Donors

Like HSCT recipients, donors also require nursing care. They may experience mood alterations, decreased self-esteem, and guilt from feelings of failure if the transplantation fails. Family members must be educated and supported to reduce anxiety and promote coping during this difficult time. In addition, they must also be assisted to maintain realistic expectations of themselves as well as of the patient.

Immunotherapy and Targeted Therapy

Immunotherapy

Immunotherapy involves the use of medications or biochemical mediators to stimulate or suppress components of the immune system to kill cancer cells (ACS, 2019g). Over the past decade, advances in the understanding of the immune system and about its interaction with cancer have led to significant advances in immunotherapy. This has dramatically changed how patients with cancer are treated and has resulted in improvements in overall survival for many forms of cancer. There are several types of immunotherapy currently being used to treat cancer, including nonspecific immunotherapies, monoclonal antibodies, checkpoint inhibitors, cancer vaccines, and chimeric antigen receptor (CAR) T-cell therapy. Many immunotherapies are targeted therapies. Targeted therapies are discussed later in this chapter.

Nonspecific Immunotherapy

Nonspecific immunotherapy does not target the cancer cell directly, but rather boosts the immune system to enhance cancer cell destruction alone or along with other cancer treatments, such as chemotherapy or radiation therapy (ACS, 2019f). Common nonspecific immunotherapy agents include bacille Calmette-

Guérin (BCG) and cytokines (interferon, interleukins, and colony-stimulating factors).

BCG is one of the earliest immunotherapy agents used to treat cancer. BCG is a live attenuated strain of *Mycobacterium bovis*, which is closely related to the bacterium that causes tuberculosis in people. BCG is most commonly used to treat localized bladder cancer (ACS, 2019h). When instilled into the bladder, BCG serves as an antigen to stimulate an immune response. The intention is that the stimulated immune system will then eradicate malignant cells. Common toxicities associated with BCG therapy include mild flulike symptoms (e.g., fever, chills, malaise), bladder burning/discomfort, urinary frequency, and blood tinged urine, which typically last 2 to 3 days after treatment (ACS, 2019h).

The most commonly used nonspecific immunotherapy agents are cytokines. **Cytokines** are messenger substances that may be released by a cell to create an action at that site or may be carried by the bloodstream to a distant site before being activated; they are also called *biochemical or inflammatory mediators*. These substances are produced primarily by cells of the immune system to enhance or suppress the production and functioning of other components of the immune system and thus can be used to treat cancer or the adverse effects of some cancer treatments. Cytokines are grouped into families, such as interferons (IFNs), interleukins (ILs), and colony-stimulating factors. Colony-stimulating factors were described earlier in this chapter for their supportive role in myelosuppressive treatment modalities. (Refer to [Chapter 31](#) for more detailed discussion of the immune system.)

IFNs are cytokines with antiviral, antitumor, and immunomodulatory (inhibition or stimulation of the immune system) properties. Multiple antitumor effects of IFNs include antiangiogenesis, direct destruction of tumor cells, inhibition of growth factors, and disruption of the cell cycle (Yarbro et al., 2018). IFNs are used on a limited basis for the treatment of some solid and hematologic cancers. Similar to IFNs, ILs have immunomodulatory effects on other components of the immune response. IL-2, an interleukin made in a laboratory, has been approved as a treatment option for advanced kidney cancer and metastatic melanoma in adults.

Adverse effects such as fever, myalgia, nausea, and vomiting, as seen with IFN and IL-2 therapy, may not be life-threatening. However, other adverse effects with these therapies (e.g., capillary leak syndrome, pulmonary edema, hypotension) may become life-threatening. These severe toxicities have restricted the use of IFN and IL-2 clinically and have prompted clinicians to seek alternative anticancer therapies (Waldmann, 2018).

Monoclonal Antibodies (MoAbs)

MoAbs, another type of immunotherapy, have become available through technologic advances, enabling investigators to grow and produce targeted antibodies for specific malignant cells. Theoretically, this type of specificity allows MoAbs to destroy the cancer cells and spare normal cells. The specificity

of MoAbs is dependent on identifying key antigen proteins on the surface (outside) of tumors that are not present on normal tissues. When the MoAb attaches to the cell surface antigen, an important signal transduction pathway for communication between the malignant cells and the extracellular environment is blocked. The results may include an inability to initiate apoptosis, reproduce, or invade surrounding tissues.

The production of MoAbs involves injecting tumor cells that act as antigens into mice. B-cell lymphocytes in the spleen of the mouse produce immunoglobulin (antibodies) made in response to the injected antigens. Antibody-producing B-cells are combined with a cancer cell that can grow indefinitely in culture medium and continue producing more antibodies.

The combination of spleen cells and the cancer cells is referred to as a hybridoma. From hybridomas that continue to grow in the culture medium, the desired antibodies are harvested, purified, and prepared for diagnostic or therapeutic use (see Fig. 12-5). Advances in genetic engineering have led to the production of MoAbs with combinations of mouse and human components (*chimeric MoAbs*) or all-human components (*human MoAbs*). MoAbs made with human genes have greater immunologic properties and are less likely to cause allergic and infusion reactions (Norris, 2019).

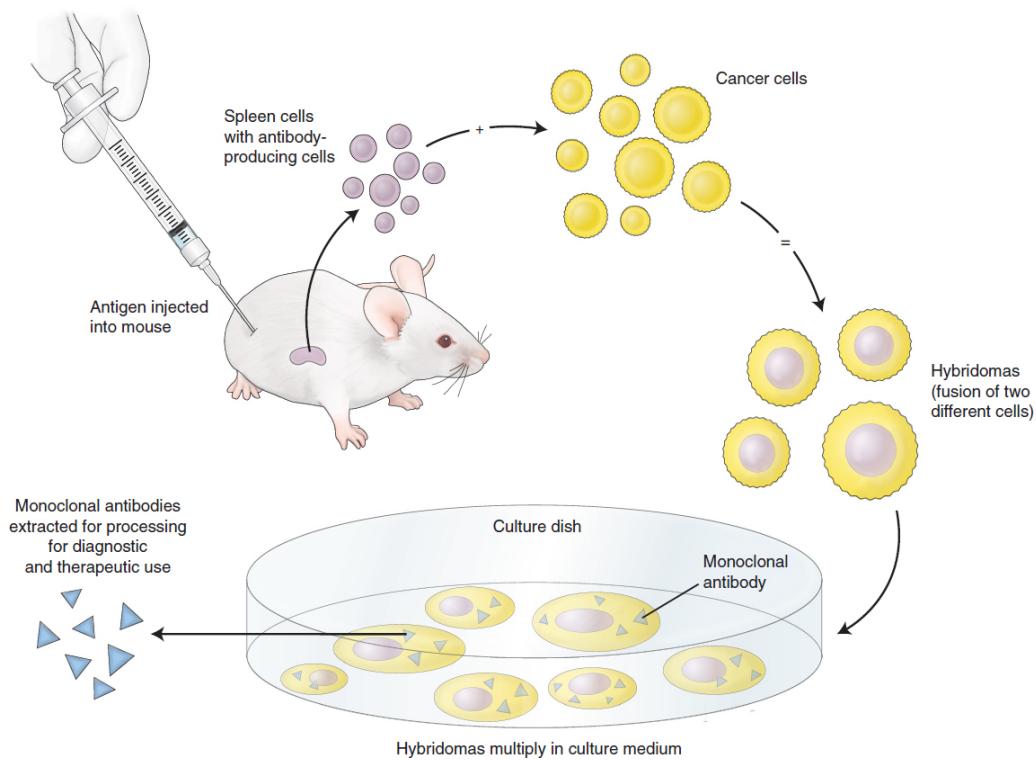


Figure 12-5 • Antibody-producing spleen cells are fused with cancer cells. This process produces cells called *hybridomas*. These cells, which can grow indefinitely in a culture medium, produce antibodies that are harvested, purified, and prepared for diagnostic or treatment purposes.

MoAbs (with names usually ending in -mab) are large particles that work on extracellular molecules, which are usually administered intravenously. Several MoAbs are used for the treatment in cancer using various extracellular (on the cell membrane) and intracellular targets. Trastuzumab targets the HER2 protein, an error of gene overexpression found in breast and other cancers (ACS, 2019g). Rituxumab is a MoAb that binds specifically with the CD20 antigen expressed by non-Hodgkin lymphoma and B-cell chronic lymphocytic leukemia.

Some MoAbs are used alone (termed naked MoAbs), whereas others are used in combination with agents that facilitate their antitumor actions (termed conjugated MoAbs). Ibritumomab tiuxetan, a MoAb conjugated with a radioactive isotope, is used in the treatment of certain types of lymphoma (ACS, 2019g). MoAbs are also used as aids in diagnostic evaluation of both primary and metastatic tumors through radiologic imaging and laboratory techniques (Olsen et al., 2019). For example, the process of immunohistochemistry uses a MoAb tagged with a stain that binds with the protein of interest, providing a visual stain for the presence or absence of the protein (Bishop, Cole, Zhang, et al., 2018). This type of test is used to identify the presence of estrogen and progesterone receptors on breast cancer cells to see if the cells will be responsive

to hormonal agents. Immunohistochemistry testing is also used to detect several proteins associated with hereditary nonpolyposis colon cancers. MoAbs are used to assist in the diagnosis of ovarian, colorectal, breast, and prostate cancers and some types of leukemia and lymphoma. MoAbs are also used in purging residual tumor cells from peripheral stem cell collections for patients who are undergoing HSCT after high-dose cytotoxic therapy. Researchers continue to explore the development and use of MoAbs, either alone or in combination with other substances such as radioactive materials, chemotherapy agents, toxins, and hormones.

MoAbs are associated with both common and unique toxicities (ACS, 2019g). Common toxicities associated with MoAbs include flulike symptoms (e.g., chills, weakness, malaise) headache, nausea and vomiting, diarrhea, rash, proteinuria, hypothyroidism, hypertension, and hepatotoxicity. Although mild to moderate allergic and infusion reactions are more commonly associated with chimeric MoAbs, severe HSRs have been seen with MoAb infusions. Other toxicities are specific to the substance attached to the MoAbs.

Checkpoint Inhibitors

The immune system uses inhibitory and acceleratory pathways and immune checkpoints to regulate the antitumor response (ACS, 2019g; Bayer, Amaya, Baniewicz, et al., 2017). Checkpoints are specific proteins on T-cells that need to be blocked (or inactivated) to initiate an immune response against cancer cells. Cancer cells use different mechanisms to avoid recognition by the immune system, thus allowing them to grow unchecked. Immune checkpoint inhibitors block proteins that diminish immune system function and prevent T lymphocytes from identifying and destroying cancer cells. When these mechanisms are blocked, the “brakes are off” and T-cells are released, allowing the immune system to identify and mount an immune response against cancer cells (this is termed an antitumor response).

Recently, special type of MoAbs were developed that block specific checkpoints on T-cells to enhance cancer cell surveillance and destruction by the immune system. These MoAbs are called check point inhibitors. Three classes of checkpoint inhibitors have been approved by the FDA for the treatment of cancer, including cytotoxic T-lymphocyte antigen-4 (CTLA-4), anti-programmed cell death protein 1 (PD-1) receptor, and anti-programmed cell death ligand 1(PD-L1) (Olsen et al., 2019). The CTLA-4 is an intracellular protein within resting T lymphocytes. CTLA receptors on certain T-cells prevent the overactivation of the immune response; thus, blocking this receptor allows for a persistent T-cell antitumor response (Bayer et al., 2017). An example of an immune check point inhibitor that blocks CTLA-4 is ipilimumab. Ipilimumab is currently approved for some forms of melanoma, colorectal cancer, and kidney cancer.

PD-1 is a checkpoint protein on activated T-cells that when bound to another protein called PD-L1 prevents the immune system from mounting a response

against normal cells (Olsen et al., 2019). PD-L1 is also located on some cancer cells, which disrupts immune surveillance and the antitumor response, thus allowing cancer cells to grow unchecked. Immune checkpoint inhibitors that block PD-1 (e.g., nivolumab and pembrolizumab) and PD-L1 (e.g., atezolizumab, avelumab, durvalumab) are used to treat a wide range of cancers, including: melanoma of the skin, non–small cell lung cancer, kidney cancer, bladder cancer, head and neck cancers, and Hodgkin lymphoma.

Immune checkpoint inhibitors exhibit a different toxicity profile from other anticancer therapies. Thus, the unique toxicities associated with immune checkpoint inhibitors are termed “immune-related adverse events” (irAes). These irAes are thought to be related to an inflammatory response to immune-related activity that can affect any organ or organ system; however, the exact etiology is not fully known (Olsen et al., 2019). IrAes often present with a delayed onset and the severity of toxicities can range from mild to potentially life-threatening. The most common irAes include dermatologic (e.g., rash and pruritus); gastrointestinal (e.g., diarrhea and colitis); pulmonary (e.g., pneumonitis); renal (e.g., nephritis); endocrine (e.g., hypophysitis [inflammation of the pituitary gland], thyroiditis, hyper- and hypothyroidism) toxicities. Prompt recognition of immune-related toxicities is essential to prevent treatment delays and to ensure prompt initiation of effective management strategies, which often include the use of anti-inflammatory medications, such as corticosteroids (Olsen et al., 2019).

Cancer Vaccines

Cancer vaccines mobilize the body’s immune response to prevent or treat cancer. These vaccines contain either portions of cancer cells alone or portions of cells in combination with other substances (adjuvants) that can augment or boost immune responses. *Autologous* vaccines are made from the patient’s own cancer cells, which are taken from tumor tissue obtained during biopsy or surgical intervention. The cancer cells are killed and prepared for injection back into the patient. *Allogeneic* vaccines are made from cancer cell lines that are immortalized cells that were originally obtained from other people who had a specific type of cancer. These cancer cells are grown in a laboratory and eventually killed and prepared for injection.

Prophylactic vaccines prevent disease. Three vaccines have been approved by the FDA for the prevention of HPV. HPV2 (Cervarix), recommended for use in females only, protects against HPV types 16 and 18 that are responsible for about 70% of all cervical cancers (ACS, 2019g). HPV4 (Gardasil) provides protection against four HPV types (6, 11, 16, and 18) and is recommended for use in both genders. HPV9 (Gardasil-9), recommended for both males and females, protects against nine HPV types associated with cervical, anal, vaginal, and vulvar cancers. HPV9 also protects against genital warts. All of the HPV vaccines are given as a series of three doses over 6 months. Although the role of HPV vaccines in prevention of oropharyngeal cancers related to HPV16 has not

been fully established, there are preliminary data to suggest effectiveness (Wang, Dickie, Sutavani, et al., 2018).

Vaccines are also used to treat existing disease. Therapeutic vaccines kill existing cancer cells and inhibit further cancer development. Sipuleucel-T (ACS, 2019g) is indicated for men with metastatic prostate cancer that is no longer responding to hormone therapy. It is the only therapeutic vaccine currently FDA approved. Therapeutic vaccines do not cure cancer but are associated with improved patient survival.

Chimeric Antigen Receptor (CAR) T–Cell Immunotherapy

A new addition to the treatment of cancer with immunotherapy is chimeric antigen receptor (CAR) T–cell therapy. CAR T–cell is a type of targeted immunotherapy that uses tumor-specific antigen recognition to target specific malignancies. CAR T–cell therapy involves the use of genetically modified T–cells to kill cancer cells (Lamprecht & Dansereau, 2019; Olsen et al., 2019). For example, in CART-19 therapy, cluster of differentiation (CD)–19 is the target antigen. This antigen is commonly overexpressed in acute lymphoblastic leukemia and certain types of non-Hodgkin lymphoma (Bayer et al., 2017). The only two CAR T–cell immunotherapy agents currently FDA-approved both target CD19 (tisagenlecleucel and axicabtagene ciloleucel). Tisagenlecleucel is approved to treat adults with relapsed or refractory non-Hodgkin lymphoma and young adults (up to 25 years old) with relapsed or refractory acute lymphoblastic leukemia. Axicabtagene and ciloleucel are approved to treat certain types of relapsed or refractory B-cell lymphoma (Olsen et al., 2019).

The process of making CAR genetically modified T–cells begins with collecting T–cells. T–cells may be collected from the patient (autologous) or collected from a healthy donor (allogeneic) using leukapheresis. The T–cells are sent to the laboratory where they are genetically altered by adding a specific chimeric antigen receptor. This process takes several weeks to make enough CAR T–cells needed for therapy. When there are enough CAR T–cells (based on the patient’s body weight), the cells are infused to the patient in a procedure similar to a blood transfusion (Lamprecht & Dansereau, 2019). A few days before a CAR T–cell infusion, the patient may be given chemotherapy to reduce the number of other immune cells to make room for the new CAR T–cells to expand and proliferate. Once infused, the CAR T–cells will continue to grow (up to a year or more postinfusion) while binding with tumor-specific antigen to kill cancer cells.

The two most common toxicities related to CAR T–cell therapy are CRS and neurologic toxicities (Anderson & Latchford, 2019). CRS, also referred to as cytokine storm, is the most common toxicity of CAR T–cell therapy and is experienced to some degree by most patients. During therapy, the infused CAR T–cells and other immune cells stimulate the release of inflammatory cytokines resulting in a systemic inflammatory response, which usually occurs within a few days of treatment. Clinical manifestations of CRS most commonly include

fever (hallmark sign), tachycardia, chills, myalgias, arthralgias, and fatigue. Although, if the CRS is severe, hypotension, dyspnea, hypoxia, respiratory distress, coagulopathies, and end-organ toxicities may occur (Anderson & Latchford, 2019). Neurologic toxicities can range from a headache and mild confusion to cerebral edema or intracranial hemorrhage. Although the exact etiology of neurologic toxicities related to CAR T-cell therapy is not yet known, it has been postulated that increased vascular permeability, increased cytokine levels, and the ability of the cells to cross the blood–brain barriers may be factors (Anderson & Latchford, 2019). Other toxicities seen with CAR T-cell therapy include tumor lysis syndrome, myelosuppression (neutropenia, anemia, and thrombocytopenia), and hypogammaglobulinemia.

Targeted Therapies

Normal cell growth is regulated by well-defined communication pathways between the environment surrounding the cell and the internal cell environment, the nucleus, and the intracellular cytoplasm. The cell membrane contains important protein receptors that respond to signals transmitted from the external cell environment and transmit that signal to the internal cell environment using enzymatic pathways called *signal transduction pathways*. Although normal cells have transduction pathways, scientific advances have led to the recognition that cancer, at the cellular level, is characterized by deregulated cell signaling transduction pathways (both intra- and extracellular pathways), as well as altered cell membrane receptors and proteins that play an important role in tumor initiation, growth, and spread (Norris, 2019). This improved understanding of cancer cell behavior has allowed scientists to develop molecular-based therapies, called targeted therapies.

Targeted therapies involve the use of agents to kill or prevent the spread of cancer cells by targeting a specific part of the cell, with less negative effects on healthy cells than conventional chemotherapy. These agents specifically target (like a lock and key mechanism) receptors, proteins, signal transduction pathways, and other processes to prevent the continued growth of cancer cells (NCI, 2019d). Targeted therapies allow for cancer treatment to be “personalized” to the unique molecular basis of the patient’s cancer. To determine if a patient would benefit from a targeted therapy, the patient’s cancer cells must be evaluated in the laboratory to determine if they have enough of the target molecule for the therapy to be effective. Thus, not all patients with the same type of cancer may benefit from the same targeted treatment. New unique molecular targets are regularly being identified, resulting in the discovery of many new targeted agents over the past decade (Olsen et al., 2019).

There are two main types of targeted therapies: monoclonal antibodies (previously discussed in the immunotherapy section) and small molecule drugs. Small molecule drugs (with names usually ending in -nib) target specific molecules on the inside of the cancer cells and are usually administered orally.

Targeted therapies are further classified in accordance with their mechanism of action (or the unique molecule targeted) (Olsen et al., 2019). There is a good amount of overlap in the mechanism of action of these agents because many targeted therapies work via complex pathways and have more than one target molecule. An overview of common categories of targeted therapies including tyrosine kinase inhibitors, EGFR inhibitors, vascular endothelial growth factor/receptor inhibitors, multikinase inhibitors, and proteasome inhibitors are discussed below using small molecule drugs as exemplars.

Tyrosine kinase inhibitors block a group of enzymes called tyrosine kinases, which regulate many cellular functions including signaling cellular growth and division (Olsen et al., 2019). Overexpression (too much) of tyrosine kinase is found in some cancer cells. Blocking these enzymes may prevent cancer cell growth. An example of a tyrosine kinase inhibitor is imatinib mesylate, which is used to treat cancers with an overexpression of BCR-ABL, such as chronic myeloid leukemias, acute lymphoblastic leukemias, and gastrointestinal stromal tumors (Comerford & Durkin, 2020).

Epidermal growth factor receptor (EGFR) inhibitors block a specific surface protein called epidermal growth factor (EGF), which is present on both normal and cancer cells. The overexpression of EGF in cancer cells promotes division and growth; thus, blocking EGFR can result in decreased cancer cell proliferation (Olsen et al., 2019). For example, erlotinib blocks the tyrosine kinase domain of the EGF protein to reduce cancer cell growth. Erlotinib is approved to treat advanced non-small cell lung cancer and advanced pancreatic cancer in combination with chemotherapy (Comerford & Durkin, 2020).

Vascular endothelial growth factor/receptor inhibitors (VEGFRIs) are agents that block vascular endothelial growth factor, which is produced by cells to stimulate **angiogenesis** (the growth of new blood vessels). As such, VEGFRIs inhibit angiogenesis, which prevents cancer cells from receiving adequate amounts of oxygen and nutrients needed for growth. VEGFRIs are used to treat various types of cancer, including soft tissue sarcomas, and thyroid, lung, kidney, and colorectal cancers. For example, axitinib is a VEGFRI approved to treat advanced renal cell carcinoma (kidney cancer) after failure of one prior systemic therapy (Comerford & Durkin, 2020; Olsen et al., 2019).

Multikinase inhibitors (MKIs) inhibit several intracellular and extracellular (surface) kinases that play a role in cellular growth and metastatic processes to reduce cancer cell growth and replication (Comerford & Durkin, 2020; Olsen et al., 2019). Sorafenib is an example of an MKI that inhibits several kinase receptor pathways (e.g., c-KIT, PDGFR, RAF, RET, VEGFR-1, VEGFR-2, VEGFR-3). Sorafenib is used to treat several different cancer types including liver, kidney, and thyroid cancers.

Proteasome inhibitors inhibit proteasome cellular complexes, that prevent certain enzymes from making proteins (Comerford & Durkin, 2020; Olsen et al., 2019). Proteasome helps maintain intracellular homeostasis by regulating proteins that facilitate cell division and prevent cell death. Thus, blocking

proteasome interferes with cell division and enhances cancer cell death. An example of a proteasome inhibitor is bortezomib, which is approved to treat multiple myeloma and certain types of lymphoma (e.g., mantle cell lymphoma).

Common toxicities that occur with targeted therapies include mucositis, nausea, vomiting, diarrhea, abdominal pain, electrolyte and fluid imbalances, skin rash, impaired wound healing, hypertension, and myelosuppression (decreased red blood cells, WBCs, and platelets) (Comerford & Durkin, 2020; Olsen et al., 2019). Peripheral neuropathy can also occur with some proteasome inhibitors. More serious complications of targeted therapy include cardiotoxicity (e.g., bortezomib) and hepatotoxicity (e.g., sorafenib).

Nursing Management

Patients receiving immunotherapy and targeted therapy have many of the same needs as patients undergoing other conventional cancer treatments. However, manipulation and stimulation of the immune system also creates unique challenges (Bayer et al., 2017; Olsen et al., 2019). Nurses must be aware of the adverse effects of these therapies and recognize the signs and symptoms of serious reactions to emergently institute appropriate interventions and supportive care. Nurses monitor patients for the impact of adverse effects on performance status and quality of life so that appropriate measures can be implemented to improve patient outcomes. The nurse must also assist in planning and evaluating patient care, and assess the need for education, support, and additional resources for both the patient and the family.

Patient education is important, as many of the toxicities are not only a source of physical discomfort, but they may also affect quality of life and patient adherence to treatment (Olsen et al., 2019; Yarbro et al., 2018). Key points of education related to immunotherapy and targeted therapy include providing information about (Bayer et al., 2017; Olsen et al., 2019; Yarbro et al., 2018):

- prescribed treatment(s) and associated toxicities to allow for prompt recognition of potentially treatment-limiting (e.g., peripheral neuropathy) and life-threatening (i.e., heart and liver failure) toxicities;
- the importance of reporting new symptoms or the exacerbation of existing symptoms promptly to the health care provider;
- how and when to contact the primary provider and seek the assistance of emergency medical services; and
- how to provide self-care during and after treatment.

Patient and family education should also include attention to general standards of care relevant to all anticancer treatment (such as principles of infection control, hand hygiene, nutrition and hydration, safe sexual practices, good skin care) and instructions to notify the health care provider about any newly prescribed or over-the-counter medications, including herbs, vitamins, and dietary supplements (Bayer et al., 2017).

Promoting Home, Community-Based, and Transitional Care

The nurse educates patients about self-care and assists in providing for continuing care. Some cancer immunotherapy and targeted therapies can be given subcutaneously (e.g., trastuzumab and denosumab) by the patient or family members at home. As needed, the home health nurse educates the patient and family how to administer these agents and monitors the use of appropriate technique as well as safe disposal of sharps and contaminated materials. The nurse also provides education about toxicities and helps the patient and family identify strategies to manage common side effects of immunotherapy and targeted therapy.

The use of oral medications to treat cancer has risen greatly in the past several years, especially with advances in targeted therapies, many of which are given by mouth. Since 2015, more than 50 oral anticancer medications were approved by FDA and it is estimated between 25% and 35% of all anticancer therapies being developed are orally administered (Dusetzina, Huskamp, Winn, et al., 2018; Kays, 2018). The rising use of oral therapies shifts the responsibility for delivery of treatment to patients and families in the home setting. Treatment approaches with newer mechanisms of action and associated toxicities, as well as the transition of responsibility to patients and families, increase the need for nurses to identify factors affecting adherence and to develop strategies to address adherence barriers (see [Table 12-8](#)).

TABLE 12-8 Strategies for Promoting Adherence to Oral Antineoplastic Agents

Assess for factors that may interfere with adherence to oral antineoplastic agents; develop plan of care that identifies and addresses specific assessment findings.

Barriers to Adherence	Strategies for Promoting Adherence
Sociodemographic Factors <ul style="list-style-type: none"> • Limited financial resources • Competing priorities for financial resources • Joblessness • Limited or no insurance • Racial or ethnic disparities • Lower level of education • Poor health literacy • Illiteracy • Non-English speaking • Lack of transportation • Lack of or limited social support • Rural residence 	<ul style="list-style-type: none"> • Refer to financial counseling through health care facility, local nonprofit health/oncology support/advocacy, or other nonprofit community advocacy organizations. • Refer to social worker for referrals as described above and/or for disability applications through employer or Social Security Administration, Medicaid, or Medicare applications. • Explore patient assistance programs for costs of health care, copays, medications, household costs, transportation services (costs or availability), and home care available through nonprofit organizations, oncology support/advocacy or other nonprofit community advocacy organizations, religious institutions, philanthropic organizations, pharmaceutical industry-sponsored programs, health care institution-specific programs; assist with financial documentation and application procedures as needed. • Explore assistance programs for other priorities competing for financial resources (e.g., for utility costs, gas, child care, food). • Assist patient to identify family, friends, or other available supports to assist with activities of daily living, household responsibilities, errands, shopping, meals, transportation or other responsibilities; assist with delegation of needs and schedules of availability if needed. • Assess preferred method of learning (e.g., verbal, visual, written materials); tailor instructional materials to patient needs, including language. • Include family, significant other, and friends in education whenever feasible. • Use return demonstration of behaviors and devices used to support adherence. • Provide contact information that spans 24 h for questions or problems. • Contact patient by phone or other means (e-mail, texting, video conference, telehealth) to assess for concerns in between follow-up visits to provider. • Encourage patient to use adherence reminders, such as pill boxes, medication calendars, checklists, medication diaries, alarms on cell phone or other devices/timers; explore availability of programmed telephone reminder services, text messages from family member, friend, or other caregiver; review diary at each visit. • Instruct patient to bring pill bottles to each follow-up visit; perform pill counts to monitor adherence. • Send postcard reminder to patient weekly (or less often) or 1 wk prior to due date for medication refill.

	<ul style="list-style-type: none"> Refer to home care for continued education and follow-up on adherence. Identify local pharmacies that supply oral medications for cancer therapies. Instruct patients to contact nurse if the pharmacy cannot fill the prescription within 24 h. Remind patients to anticipate need for adequate supply of medications prior to travel or vacation.
Age	<ul style="list-style-type: none"> Older adults; especially those >75 yrs Ensure that printed education materials and instructions are printed in black using at least 14-point sans serif font such as Arial or Calibri. Use illustrated education materials. Review and revise adherence strategies if patient status declines or changes. Explore availability of alarmed medication box.
Beliefs	<ul style="list-style-type: none"> Oral medications less effective or important than IV treatments Fatalism about disease outcomes Provide education regarding oral versus IV medications. Discuss goals of care and ongoing assessment of response to treatment.
Comorbidities	<ul style="list-style-type: none"> Preexisting chronic disease Vision or hearing impairments Communicate with primary care and other providers involved in care of patient regarding current cancer disease status and treatment; collaborate with other providers for ongoing management of nononcology issues or exacerbation of issues that may impact cancer treatment adherence. Collaborate with appropriate resources to assist with special needs and assistive devices for vision or hearing impairments.
Polypharmacy	<ul style="list-style-type: none"> Multiple medications for comorbidities or cancer treatment and symptom management Review all medications prescribed by oncology physicians and other providers involved in care of patient for preexisting chronic disease. Assess patient use of over-the-counter medications and other agents. Consult with pharmacist to identify medications and other agents that may be contraindicated or that may interfere with antineoplastic regimen. Collaborate with all providers prescribing medications in order to simplify or reduce number of required medications if possible.

	<ul style="list-style-type: none"> Provide patient, family, or other caregiver education regarding specific instructions when multiple medications are required. Provide written checklist for patient to utilize daily to check off each medication when taken. Check if patient prescriptions allow for refills; prescriptions should be for a finite period of time that concludes with next scheduled visit.
Psychiatric, Psychological, or Cognitive Concerns	<ul style="list-style-type: none"> Avoid initial education regarding oral medications at the same time of first provider visit; have patient and other learners return for another appointment to see nurse for education and follow-up with subsequent visit if deemed to be at high risk for adherence challenges. Discuss with provider the necessity for referral to psychiatrist to evaluate need for psychotropic medications. Refer patient for professional mental health counseling as needed. Identify additional supports for care and education as discussed earlier.
Disease Factors	<ul style="list-style-type: none"> Proactively assess and manage symptoms related to underlying disease or treatments. Provide patient, family, or other caregivers education about expected side effects and management strategies. Instruct patient to premedicate with antiemetic as prescribed 30 min prior to taking oral antineoplastic agent if needed for nausea or vomiting. Identify additional supports for care as discussed earlier. Assess need for referral to home physical or occupational therapy to address impaired mobility and need for assistive devices.
Communication Issues	<ul style="list-style-type: none"> Establish rapport and allow patients, families, and other caregivers time and opportunity to ask questions. Do not assume adherence to oral antineoplastic agents; emphasize the value and importance of adherence and assess potential barriers consistently throughout course of treatment at each follow-up visit. Communicate with primary provider regarding current cancer disease status, treatment and information regarding antineoplastic agents, such as drug–drug interactions, expected toxicities, and toxicities requiring prompt intervention.

IV, intravenous.

Adapted from Olsen, M. M., LeFebvre, K. B., & Brassil, K. (Eds.). (2019). *Chemotherapy and immunotherapy guidelines and recommendations for practice*. Pittsburgh, PA:

Oncology Nursing Society.

The nurse collaborates with physicians, social workers, third-party payers, and pharmaceutical companies to help the patient obtain reimbursement or support for the cost of oral cancer therapies and other required medications (Dusetzina et al., 2018). The nurse also reminds the patient about the importance of keeping follow-up appointments with the primary provider and assesses the patient's need for symptom management related to the underlying diseases or adverse effects of treatment. Home health nurses maintain communication with the primary provider regarding patient adherence and tolerance of treatment so that changes in care can be implemented in a timely fashion.

Complementary, Alternative, and Integrative Health Therapies

Integrative health care is viewed as a comprehensive, interdisciplinary approach to preventing and treating illness and promoting health that brings together complementary, alternative, and conventional therapies. The use of an integrative approach to health and wellness has grown within mainstream health care settings in the United States, particularly within oncology care (National Center for Complementary and Integrative Health [NCCIH], 2018).

Individuals use complementary approaches to prevent and treat cancer, although there are no data to support efficacy. Patients also use complementary approaches to manage symptoms related to cancer and associated treatments; some approaches are supported by clinical research while others are not. It is estimated that as many as 67% of people diagnosed with cancer use some form of complementary medicine (Wanchai, Armer, Smith, et al., 2017). However, many patients do not routinely communicate complementary practices to their health care providers because they usually are not asked about its use; they withhold the information, fearing that their provider would not approve, or they feel that the use of these approaches will not affect the conventional treatment they are receiving.

Although many complementary modalities can be a source of comfort and emotional support for patients, assessment of complementary therapy use is important for patient safety. Patients often perceive vitamins and dietary supplements as harmless, natural products that have no side effects or potential toxicities. In patients receiving any conventional therapies, the use of herbs or botanicals may interfere with drug metabolism, decrease or increase desired effects, or contain elements of uncertain pharmacologic capacities (NCCIH, 2018). Deep tissue massage and other manipulative therapies are contraindicated in patients with open wounds, radiodermatitis, thrombocytopenia, VTE, and coagulation disorders, and in those taking anticoagulants. See [Chapter 4](#) for more information on complementary, alternative, and integrative health therapies.



COVID-19 Considerations

The novel coronavirus disease 2019 (COVID-19) pandemic began in Wuhan, China, in late 2019. Since that time, several risks for both severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection and pathogenesis to COVID-19 have been posed (see [Chapter 66](#)). Epidemiologic findings from early data in China suggest that having cancer could be an important risk factor for becoming infected with SARS-CoV-2 as well as increasing the risk of mortality from COVID-19 (Deng, Yin, Chen, et al., 2020). Many cancer clinicians and researchers worldwide recognized during the early days of the pandemic that there was an urgent need to identify idiosyncratic risks and treatment issues that revolve around managing adults with both COVID-19 and cancer. As a result, the COVID-19 and Cancer Consortium (CCC19) was established (see Resources section for link to CCC19). An early cohort study sponsored by CCC19 of 928 patients from the United States, Canada, and Spain with either a history of cancer or active malignancy identified prognostic factors for mortality and severe COVID-19 (Kuderer, Choueiri, Shah, et al., 2020). Overall 13% of the patients died (Kuderer et al., 2020), a higher mortality rate than the 5.6% case fatality rate for all adult patients in the United States with COVID-19 (Johns Hopkins University & Medicine Coronavirus Resource Center, 2020). The researchers identified the following factors as associated with an increase in 30-day mortality: older age, male sex, a history of smoking, having two or more comorbidities, having an active cancer, and having received azithromycin plus hydroxychloroquine during treatment for COVID-19 (Kuderer et al., 2020). The four most common presenting symptoms of COVID-19 in these patients included fever, cough, fatigue or malaise, and dyspnea; the same as for all patients with COVID-19 (Kuderer et al., 2020). There was no association between having recently had surgery and either having severe COVID-19 or dying, suggesting that surgery indicated for patients with cancer should not be postponed due to pandemic concerns (Kuderer et al., 2020). CCC19 will continue to analyze registry data of patients with COVID-19 and cancer and publicize noteworthy findings as they become available, to facilitate optimal management of patients with COVID-19 and cancer (Kuderer et al., 2020).

Nursing Care of the Patient with Cancer

The outlook for patients with cancer has greatly improved because of scientific and technologic advances. However, as a result of the underlying disease or various treatment modalities, patients with cancer may experience a variety of secondary problems such as reduced WBC counts, infection, bleeding, skin and nutritional problems, pain, fatigue, and psychological stress. [Chart 12-6](#) provides a nursing care plan for the patient with cancer.

Maintaining Tissue Integrity

Some of the most frequently encountered disturbances of tissue integrity include stomatitis, skin and tissue reactions to radiation therapy, cutaneous toxicities associated with targeted therapy, alopecia, and metastatic skin lesions.

Stomatitis

Mucositis, a common side effect of radiation and some types of chemotherapy, refers to an inflammatory process involving the mucous membranes of the oral cavity and the gastrointestinal tract. **Stomatitis**, a form of mucositis, is an inflammatory process of the mouth, including the mucosa and tissues surrounding the teeth. Stomatitis is characterized by changes in sensation, erythema (mild redness), and edema or, if severe, by painful ulcerations, bleeding, and secondary infection. Stomatitis commonly develops within 3 to 14 days after patients receive certain chemotherapeutic agents (e.g., 5-fluorouracil and doxorubicin), immunotherapies (e.g., IL-2 and nivolumab); and targeted therapies (e.g., temsirolimus and everolimus). Stomatitis affects up to 100% of patients undergoing high-dose chemotherapy with HSCT, 90% of patients with malignancies of the head and neck receiving radiotherapy, and up to 40% of patients receiving standard-dose chemotherapy (Eilers, Asakura, Blecher, et al., 2017; Olsen et al., 2019). Stomatitis may be worse in patients with head and neck cancers who receive combined modality therapy of both radiation and chemotherapy. When severe, stomatitis can lead to interruptions, delays, and modifications in the course of treatment, all of which may contribute to less desirable patient outcomes. Severe oral pain can significantly affect swallowing, nutritional intake, speech, quality of life, coping abilities, and willingness to adhere to treatment regimens. In addition, stomatitis may lead to more frequent health care visits, hospitalizations, and increased health care costs (Berger, Schopohl, Bollig, et al., 2018). Stomatitis and mucositis are attributed to a cascade of molecular processes and submucosal endothelial cell destruction that begin almost immediately after the initiation of radiation and certain types of chemotherapy, prior to the development of signs and symptoms. Mucositis develops because of a sequence of related and interacting biologic events, culminating in injury and apoptosis of basal epithelial cells, leading to the loss of epithelial renewal, atrophy, and ulceration. Gram-positive and gram-negative organisms can invade the ulcerated tissue and result in infection.

Nursing assessment begins with an understanding of the patient's usual practices for oral hygiene and identification of individuals at risk for stomatitis. Oral cavity assessment is performed daily or at each patient visit (Olsen et al., 2019). Risk factors and comorbidities associated with stomatitis include poor oral hygiene, general debilitation, existing dental disease, prior irradiation to the head and neck region, impaired salivary gland function, the use of other medications that dry mucous membranes, myelosuppression, tobacco use, previous cancer treatment with a stomatotoxic agent or radiation therapy, diminished renal function, impaired nutritional status, and both older (>65 years) and younger (<20 years) ages (Olsen et al., 2019). The patient is also assessed for dehydration, infection, pain, and nutritional impairment resulting from mucositis.

Optimal evidence-based prevention and treatment approaches for stomatitis remain limited but continue to be studied across disciplines (Bowen, Gibson, Coller,

et al., 2019; Eilers et al., 2017). Most clinicians agree that maintenance of good oral hygiene, including brushing, flossing, rinsing, and dental care, is necessary to minimize the risk of oral complications associated with cancer therapies.

Palifermin, an IV-administered synthetic form of human keratinocyte growth factor, is beneficial in the prevention and management of stomatitis in patients with hematologic malignancies who are preparing for HSCT and in those undergoing chemotherapy for head and neck cancer (Bowen et al., 2019; Eilers et al., 2017). Palifermin promotes epithelial cell repair and accelerated replacement of cells in the mouth and gastrointestinal tract. Careful timing of administration and monitoring are essential for effectiveness and to detect adverse effects. Other approaches recommended for practice include cryotherapy (topical application of oral ice during infusions), consistent oral hygiene, low-level laser therapy, and sodium bicarbonate mouth rinses (Bowen et al., 2019; Eilers et al., 2017; Olsen et al., 2019). Additional aspects of care are discussed in [Chart 12-6](#): nursing care plan for patients with cancer.

Chart 12-6



PLAN OF NURSING CARE

The Patient with Cancer

NURSING DIAGNOSIS: Risk for infection associated with inadequate defenses related to myelosuppression secondary to radiation or antineoplastic agents

GOAL: Prevention of infection

Nursing Interventions	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess patient for evidence of infection. <ol style="list-style-type: none"> a. Check vital signs every 4 hours. b. Monitor white blood cell (WBC) count and differential each day. c. Inspect all sites that may serve as entry ports for pathogens (IV sites, wounds, skin folds, bony prominences, perineum, and oral cavity). 2. Report fever ($\geq 38.3^{\circ}\text{C}$ [101°F] or $\geq 38^{\circ}\text{C}$ [100.4°F] for >1 hour) (see Table 12-10), chills, diaphoresis, swelling, heat, pain, erythema, exudate on any body surfaces. Also report change in respiratory or mental status, urinary frequency or burning, malaise, myalgias, arthralgias, rash, or diarrhea. 3. Obtain cultures and sensitivities as indicated before initiation of antimicrobial treatment (wound exudate, sputum, urine, stool, blood). 4. Initiate measures to minimize infection. 	<ol style="list-style-type: none"> 1. Signs and symptoms of infection may be diminished in the immunocompromised host. Prompt recognition of infection and subsequent initiation of therapy will reduce morbidity and mortality associated with infection. 2. Early detection of infection facilitates early intervention. 3. Tests identify the organism and indicate the most appropriate antimicrobial therapy. The use of inappropriate antibiotics enhances proliferation of additional flora and encourages growth of antibiotic-resistant organisms. 4. Exposure to infection is reduced. <ol style="list-style-type: none"> a. Preventing contact with pathogens helps prevent infection. b. Hands are significant source of contamination. c. Incidence of rectal and perianal abscesses and subsequent 	<ul style="list-style-type: none"> • Demonstrates normal temperature and vital signs • Exhibits absence of signs of inflammation: local edema, erythema, pain, and warmth • Exhibits normal breath sounds on auscultation • Takes deep breaths and coughs every 2 hours to prevent respiratory dysfunction and infection. • Exhibits absence of pathogens on cultures • Avoids contact with others with infections • Avoids crowds • All personnel carry out hand hygiene after each voiding and bowel movement. • Excoriation and trauma of

<ul style="list-style-type: none"> a. Discuss with patient and family: <ul style="list-style-type: none"> 1. Placing patient in private room if absolute WBC count <1000/mm³. 2. Importance of patient avoiding contact with people who have known or recent infection or recent vaccination. b. Instruct all personnel in careful hand hygiene before and after entering room. c. Avoid rectal or vaginal procedures (rectal temperatures, examinations, suppositories; vaginal tampons). d. Use stool softeners to prevent constipation and straining. e. Assist patient in practice of meticulous personal hygiene. f. Instruct patient to use electric razor. g. Encourage patient to ambulate in room unless contraindicated. h. Provide patient and family education on food hygiene and safe food handling. i. Each day change water pitcher, 	<p>systemic infection is high. Manipulation may cause disruption of membrane integrity and enhance progression of infection.</p> <ul style="list-style-type: none"> d. Minimizes trauma to tissues e. Prevents skin irritation f. Minimizes skin trauma g. Minimizes chance of skin breakdown and stasis of pulmonary secretions h. No evidence supports dietary restrictions of avoiding raw or fresh fruit and vegetables for patients who are neutropenic. General precautions regarding food handling and storage are recommended. i. Stagnant water is a source of infection. <p>5. Hospital-acquired sepsis is closely associated with IV catheters.</p> <ul style="list-style-type: none"> a. Incidence of infection is increased when catheter is in place >72 hours. b. Chlorhexidine is effective against many gram-positive 	<p>skin are avoided.</p> <ul style="list-style-type: none"> • Trauma to mucous membranes is avoided (avoidance of rectal thermometers, suppositories, vaginal tampons, perianal trauma). • Uses evidence-based procedures and techniques if participating in management of invasive lines or catheters • Uses electric razor • Is free of skin breakdown and stasis of secretions • Adheres to dietary and environmental precautions • Exhibits no signs of sepsis or septic shock • Exhibits normal vital signs, cardiac output, and arterial pressures when monitored
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<p>denture cleaning fluids, and respiratory equipment containing water.</p> <p>5. Assess IV sites every day for evidence of infection.</p> <ul style="list-style-type: none"> a. Change peripheral short-term IV sites every other day. b. Cleanse skin with chlorhexidine before arterial puncture or venipuncture. c. Change central venous catheter dressings every 48 hours. d. Change all solutions and infusion sets every 72–96 hours. e. Follow Infusion Nursing Society guidelines for care of peripheral and central venous access devices. <p>6. Avoid intramuscular injections.</p> <p>7. Avoid insertion of urinary catheters; if catheters are necessary, use aseptic technique.</p> <p>8. Educate patient or family member to administer granulocyte (or granulocyte-macrophage) colony-stimulating factor when prescribed.</p> <p>9. Advise patient to avoid exposure to animal excreta, discuss dental procedures with primary provider, avoid</p>	<p>and gram-negative pathogens.</p> <p>c. Allows observation of site and removes source of contamination.</p> <p>d. Once introduced into the system, microorganisms can grow in infusion sets despite replacement of container and high flow rates.</p> <p>e. Infusion Nursing Society collaborates with other nursing subspecialties in determining guidelines for IV access care.</p> <p>6. Reduces risk for skin abscesses.</p> <p>7. Rates of infection greatly increase after urinary catheterization.</p> <p>8. Granulocyte colony-stimulating factor decreases the duration of neutropenia and the potential for infection.</p> <p>9. Minimizes exposure to potential sources of infection and disruption of skin integrity</p>	<ul style="list-style-type: none"> • Demonstrates ability to administer colony-stimulating factor • Has bowel movements at regular intervals without constipation or straining • Patient hygiene is maintained. • Absence of IV catheter-related infection • Absence of skin abscesses • Absence of urinary catheter-related infection
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vaginal douche, and avoid vaginal or rectal manipulation during sexual contact during the period of neutropenia.

NURSING DIAGNOSIS: Risk for impaired skin integrity: erythematous and wet desquamation reactions to radiation therapy

GOAL: Maintenance of skin integrity

Nursing Intervention	Rationale	Expected Outcomes
<p>1. In erythematous areas:</p> <ul style="list-style-type: none"> a. Avoid the use of soaps, cosmetics, perfumes, powders, lotions, and ointments; non-aluminum-based deodorant may be used on intact skin. b. Use only lukewarm water to bathe the area. c. Avoid rubbing or scratching the area. d. Avoid shaving the area with a straight-edged razor. e. Avoid applying hot-water bottles, heating pads, ice, and adhesive tape to the area. f. Avoid exposing the area to sunlight or cold weather. g. Avoid tight clothing in the area. Use cotton clothing. h. Topical agents such as Aquaphor, 	<p>1. Care to the affected areas must focus on preventing further skin irritation, drying, and damage.</p> <ul style="list-style-type: none"> a. These substances may cause pain and additional skin irritation and damage. b. Avoiding water of extreme temperatures and soap minimizes additional skin damage, irritation, and pain. c. Rubbing, scratching, or both will lead to additional skin irritation, damage, and increased risk of infection. d. The use of razors may lead to additional irritation and disruption of skin integrity and increased risk of infection. e. Avoiding extreme temperatures minimizes 	<ul style="list-style-type: none"> • Avoids use of soaps, powders, and other cosmetics on site of radiation therapy • States rationale for special care of skin • Exhibits minimal change in skin • Avoids trauma to affected skin region (avoids shaving, constricting and irritating clothing, extremes of temperature, and the use of adhesive tape) • Reports change in skin promptly • Demonstrates proper care of blistered or open areas

- radiacare gel, aloe vera, or biafine may be used, and low- or medium-potency corticosteroid cream may be given if pruritus is present.
2. If wet desquamation occurs:
 - a. Do not disrupt any blisters that have formed.
 - b. Avoid frequent washing of the area.
 - c. Report any blistering.
 - d. Use prescribed creams or ointments; topical antibacterial creams may help to dry a wet wound (e.g., Silvadene cream).
 - e. If area weeps, apply a nonadhesive absorbent dressing.
 - f. If the area is without drainage, moisture and vapor-permeable dressings, such as hydrocolloids and hydrogels on noninfected areas, have been used in many settings.
 - g. Consult with wound-ostomy-continence nurse (WOCN) and primary provider if eschar forms.
- additional skin damage, irritation, burns, and pain.
- f. Sun exposure or extreme cold weather may lead to additional skin damage and pain.
 - g. Allows air circulation to affected area
 - h. May aid healing; however, evidence supporting the benefits of topical agents is lacking.
2. Open weeping areas are susceptible to bacterial infection. Care must be taken to prevent introduction of pathogens.
 - a. Disruption of skin blisters disrupts skin integrity and may lead to increased risk of infection.
 - b. Frequent washing may lead to increased irritation and skin damage, with increased risk of infection.
 - c. Blistering of skin represents progression of skin damage.
 - d. Anecdotally believed to decrease irritation and inflammation of the area and promote healing; although a variety of products are used in many settings, there are
- Exhibits absence of infection of blistered and opened areas.
 - Wound is free of development of eschar

few randomized controlled trials with evidence to support one product or intervention over another.

- e. Easier to remove and associated with less pain and trauma when drainage dries and adheres to dressing.
- f. May promote healing; however, randomized controlled clinical trial support is lacking in the setting of moist desquamation. Hydrocolloid dressings may enhance comfort.
- g. Eschar must be removed to promote healing and prevent infection. WOCNs have expertise in the care of wounds.

NURSING DIAGNOSIS: Impaired oral mucous membrane integrity: stomatitis**GOAL:** Maintenance of intact oral mucous membranes

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Assess oral cavity daily using the same assessment criteria or rating scale.2. Identify individuals at increased risk for stomatitis and related complications.3. Instruct patient to report oral burning, pain, areas of redness, open lesions on oropharyngeal mucosa and lips, pain associated with swallowing, or decreased tolerance to temperature extremes of food.4. Encourage and assist as needed in oral hygiene.	<ol style="list-style-type: none">1. Provides baseline for later evaluation; maintains consistency in assessment findings2. Patient and treatment variables are associated with the incidence and severity of stomatitis as well as related complications such as delayed healing and infection.3. Identification of initial stages of stomatitis will facilitate prompt interventions, including modification of treatment as prescribed by primary provider.4. Patients who are having discomfort or pain, or other symptoms related to the disease and treatment, may require encouragement and assistance in performing oral hygiene. Oral hygiene is maintained to prevent complications of	<ul style="list-style-type: none">• States rationale for frequent oral assessment and hygiene• Factors associated with the incidence, severity, and complications are identified prior to initiation of cancer treatment• Oral mucosal assessment is conducted at baseline and on an ongoing basis• Oral hygiene practices are initiated prior to development of stomatitis.• Identifies signs and symptoms of stomatitis to report to nurse or primary provider• Participates in recommended oral hygiene regimen• Avoids mouthwashes with alcohol• Brushes teeth and mouth with soft toothbrush• Uses lubricant to keep lips soft and nonirritated• Avoids hard-to-chew, spicy, hot foods or other irritating foods

Nursing Intervention	Rationale	Expected Outcomes
Preventive		
<p>1. Advise patient to avoid irritants such as commercial mouthwashes, alcoholic beverages, and tobacco.</p> <p>2. Brush with soft toothbrush using nonabrasive toothpaste for 90 seconds after meals and at bedtime; allow toothbrush to air dry before storing; floss at least once daily or as advised by the clinician; patients who have not previously flossed regularly should not initiate flossing during stomatotoxic treatment; rinse mouth four times a day with a bland rinse (normal saline, sodium bicarbonate, or saline and sodium bicarbonate); avoid irritating foods (acidic, hot, rough, and spicy); use water-based moisturizers to protect lips.</p> <p>3. Consider use of oral ice chips during stomatotoxic chemotherapy infusions.</p> <p>4. Consider use of low-level laser therapy.</p>	<p>stomatitis, such as infection.</p> <p>1. Alcohol content of mouthwashes and tobacco smoke will dry oral tissues and potentiate breakdown.</p> <p>2. Limits trauma and removes debris. Patients who have not previously flossed regularly should not initiate flossing during stomatotoxic treatment due to potential for injury to the oral mucosa and increased susceptibility to infection.</p> <p>3. Oral cryotherapy has demonstrated reduced oral mucositis incidence, severity, and pain; improved quality of life; and minimizes chances of complications of oral mucositis</p> <p>4. Low-energy level laser therapy has demonstrated decreased severity, duration, and pain associated with stomatitis.</p>	<p>Maintains adequate hydration</p> <ul style="list-style-type: none"> • Exhibits clean, intact oral mucosa • Exhibits no ulcerations or infections of oral cavity • Exhibits no evidence of bleeding • Reports absent or decreased oral pain • Reports no difficulty swallowing • Exhibits healing (reepithelialization) of oral mucosa within 5–7 days (mild stomatitis)

5. Consider administration of palifermin as prescribed for patients receiving high-dose chemotherapy.
6. Maintain adequate hydration.
7. Provide written instruction and education to patients on the above items.
5. Palifermin, a recombinant keratinocyte growth factor (KGF) that stimulates the growth of cells lining the mouth and intestinal tract, has been shown to decrease the severity and duration of stomatitis.
6. Maintenance of hydration prevents mucosal drying and breakdown.
7. Written information reinforces patient education and provides the patient and family with a source.

Mild stomatitis

(generalized erythema, limited ulcerations, small white patches: *Candida*)

1. Use normal saline mouth rinses every 1–4 hours.
2. Use soft toothbrush or toothette.
3. Remove dentures except for meals; be certain that dentures fit well.
4. Apply water-soluble lip lubricant.
5. Avoid foods that are spicy or hard to chew and those with extremes of temperature.
1. Assists in removing debris, thick secretions, and bacteria
2. Minimizes trauma
3. Minimizes friction and discomfort
4. Promotes comfort
5. Prevents local trauma
- Exhibits healing of oral tissues within 10–14 days (severe stomatitis)
- Exhibits no bleeding or oral ulceration
- Consumes adequate fluid and food
- Exhibits absence of dehydration and weight loss
- Exhibits no evidence of infection

Severe stomatitis

(confluent ulcerations with bleeding and white patches covering >25% of oral mucosa)

1. Obtain tissue samples for culture and sensitivity tests of areas of infection.
 2. Assess ability to chew and swallow; assess gag reflex.
 3. Use oral rinses (may combine in solution saline, anti-*Candida* agent, such as mycostatin, and topical anesthetic agent [described later]) as prescribed, or place patient on side and irrigate mouth; have suction available.
 4. Remove dentures.
 5. Use toothette or gauze soaked with solution for cleansing.
 6. Use water-soluble lip lubricant.
 7. Provide liquid or pureed diet.
 8. Monitor for dehydration.
 9. Minimize discomfort.
 - a. Consult primary provider for use of topical anesthetic, such as dyclonine and diphenhydramine, or viscous lidocaine.
 - b. Administer systemic analgesics as prescribed.
1. Assists in identifying need for antimicrobial therapy
 2. Patient may be in danger of aspiration
 3. Facilitates cleansing and provides for safety and comfort
 4. Prevents trauma from ill-fitting dentures
 5. Limits trauma and promotes comfort
 6. Promotes comfort and minimizes loss of skin integrity
 7. Ensures intake of easily digestible foods without chewing
 8. Decreased oral intake and ulcerations potentiate fluid deficits
 9. Promotes healing
 - a. Alleviates pain and increases sense of well-being; promotes participation in oral hygiene and nutritional intake
 - b. Adequate management of pain related to

- c. Perform mouth care as described.
- severe stomatitis can facilitate improved quality of life, participation in other aspects of activities of daily living, oral intake, and verbal communication.
- c. Promotes removal of debris, healing, and comfort

NURSING DIAGNOSIS: Impaired skin integrity associated with rash

GOALS: Maintenance of skin integrity

Nursing Intervention	Rationale	Expected Outcomes
Prevention		
<p>1. Instruct patients to avoid sunlight through use of protective clothing, use of sun screen with SPF of 30 with physical blockers (zinc oxide, titanium dioxide), or avoidance of direct sun exposure.</p> <p>2. Maintain adequate oral hydration.</p> <p>3. Avoid long hot showers or baths, harsh soaps and laundry detergents, perfumes, and nonhypoallergenic cosmetics.</p> <p>4. Apply emollients; apply hydrocortisone 1% cream with moisturizer at least twice daily; administer doxycycline 100 mg</p>	<p>1. Many agents are associated with photosensitivity; sunburn would intensify inflammation associated with rash and potentiate loss of skin integrity</p> <p>2. Prevents skin dryness related to dehydration</p> <p>3. Prevents skin irritation, dryness, flaking, and inflammation</p> <p>4. Minimizes dryness, flaking, and disruption of skin integrity</p>	<ul style="list-style-type: none"> • Sun exposure will be limited; no development of sun burn • Absence of dehydration • Participates in skin care regimen as instructed • Absence of dryness, flaking

twice per day or
minocycline, as
prescribed

Treatment

1. Apply topical treatment as prescribed: clindamycin 1%, fluocinonide 0.05% cream twice a day, or alclometasone 0.05% cream twice a day
 2. For severe papulopustular rash: Administer systemic treatment as prescribed: doxycycline 100 mg twice per day; minocycline 100 mg daily; or isotretinoin at low doses of 20–30 mg per day
 3. Assess for development of infection: obtain cultures of pustules and administer appropriate antibiotics as prescribed by the physician
1. Recommended as treatment to minimize skin disruption and prevent infection by Multinational Association of Supportive Care in Cancer (MASCC)
 2. Recommended as treatment to minimize skin disruption and prevent infection by Multinational Association of Supportive Care in Cancer (MASCC)
 3. Prompt recognition and treatment of infection are necessary to prevent bacteremia, sepsis, and further patient compromise
- Rash severity does not interfere with level of comfort and adherence to targeted therapy as prescribed; absence of local or systemic infection
 - Rash severity does not interfere with level of comfort and adherence to targeted therapy as prescribed; absence of local or systemic infection
 - Local infection is controlled; absence of sepsis

NURSING DIAGNOSIS: Impaired tissue integrity: alopecia**GOAL:** Maintenance of tissue integrity; coping with hair loss

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Discuss potential hair loss and regrowth with patient and family; advise that hair loss may occur on body parts other than the head.2. Explore potential impact of hair loss on self-image, interpersonal relationships, and sexuality.3. Prevent or minimize hair loss through the following:<ol style="list-style-type: none">a. Use scalp cooling (hypothermia), if appropriate.b. Cut long hair before treatment.c. Use mild shampoo and conditioner, gently pat dry, and avoid excessive shampooing.d. Avoid electric curlers, curling irons, dryers, clips, barrettes, hair sprays, hair dyes, hair extensions, weaves, braids, dreadlocks, hair straightening products, and permanent waves.e. Avoid excessive combing or brushing; use wide-toothed comb.4. Prevent trauma to scalp.<ol style="list-style-type: none">a. Use sunscreen or wear hat when in the	<ol style="list-style-type: none">1. Provides information so that patient and family can begin to prepare cognitively and emotionally for loss2. Facilitates coping and maintenance of interpersonal relationships3. Retains hair as long as possible.<ol style="list-style-type: none">a. Decreases hair follicle uptake of chemotherapy (not used for patients with leukemia or lymphoma because tumor cells may be present in blood vessels or scalp tissue)b. Minimizes hair loss due to the weight and manipulation of hair4. Preserves tissue integrity<ol style="list-style-type: none">a. Assists in maintaining skin integrityb. Prevents ultraviolet light exposure5. Minimizes change in appearance<ol style="list-style-type: none">a. Wig that closely resembles hair color and style is	<ul style="list-style-type: none">• Identifies alopecia as potential side effect of treatment• Identifies positive and negative feelings and threats to self-image• Verbalizes meaning that hair and possible hair loss have for them• States rationale for modifications in hair care and treatment• Uses mild shampoo and conditioner, and shampoos hair only when necessary• Avoids hair dryer, curlers, sprays, and other stresses on hair and scalp• Wears hat or scarf over

<p>sun.</p> <p>5. Suggest ways to assist in coping with hair loss.</p> <ul style="list-style-type: none"> a. Purchase wig or hairpiece before hair loss. b. If hair loss has occurred, take photograph to wig shop to assist in selection. c. Begin to wear wig before hair loss. d. Contact the American Cancer Society for donated wigs or a store that specializes in this product. e. Wear hat, scarf, or turban. <p>6. Encourage patient to wear own clothes and retain social contacts.</p> <p>7. Explain that hair growth usually begins again once therapy is completed.</p>	<p>more easily selected if hair loss has not begun.</p> <p>b. Facilitates adjustment</p> <p>c. Enables patient to be prepared for loss and facilitates adjustment</p> <p>d. Provides options to patient and assists with financial burden if necessary</p> <p>e. Conceals loss and protects scalp</p> <p>6. Assists in maintaining personal identity</p> <p>7. Reassures patient that hair loss is usually temporary</p>	<p>hair when exposed to sun</p> <ul style="list-style-type: none"> • Takes steps to deal with possible hair loss before it occurs; purchases wig or hairpiece if desired • Maintains hygiene and grooming • Interacts and socializes with others
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NURSING DIAGNOSIS: Impaired nutritional status associated with nausea and vomiting

GOAL: Patient experiences less nausea and vomiting associated with therapies; weight loss is minimized

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess the patient's previous experiences and expectations of nausea and vomiting, including causes and interventions used. 2. Adjust diet before and after drug administration according to patient preference and tolerance. 3. Prevent unpleasant sights, odors, and sounds in the environment. 4. Use distraction, music therapy, biofeedback, self-hypnosis, relaxation techniques, and guided imagery before, during, and after chemotherapy. 5. Administer prescribed antiemetics, sedatives, and corticosteroids before chemotherapy and afterward as needed. 6. Ensure adequate fluid hydration before, during, and after drug administration; assess intake and output. 7. Encourage frequent oral hygiene. 	<ol style="list-style-type: none"> 1. Identifies patient concerns, misinformation, and potential strategies for intervention; also gives patient sense of empowerment and control. 2. Each patient responds differently to food after chemotherapy. A diet containing foods that relieve or prevent nausea or vomiting is most helpful. 3. Unpleasant sensations can stimulate the nausea and vomiting center. 4. Decreases anxiety, which can contribute to nausea and vomiting. Psychological conditioning may also be decreased. 5. Administration of antiemetic regimen before onset of nausea and vomiting limits the adverse experience and facilitates control. Combination drug therapy reduces nausea and vomiting through various triggering mechanisms. 6. Adequate fluid volume dilutes drug levels, decreasing stimulation of vomiting receptors. 	<ul style="list-style-type: none"> • Identifies previous triggers of nausea and vomiting • Exhibits decreased apprehension and anxiety • Identifies previously used successful interventions for nausea and vomiting • Reports decrease in nausea • Reports decrease in incidence of vomiting • Consumes adequate fluid and food when nausea subsides • Demonstrates use of distraction, relaxation, and imagery when indicated • Exhibits normal skin turgor and

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| 8. Provide pain-relief measures, if necessary. | 7. Reduces unpleasant taste sensations | moist mucous membranes |
| 9. Consult with dietitian as needed. | 8. Increased comfort increases physical tolerance of symptoms. | • No additional weight loss |
| 10. Assess and address other contributing factors to nausea and vomiting, such as other symptoms, constipation, gastrointestinal irritation, electrolyte imbalance, radiation therapy, medications, and central nervous system metastasis. | 9. Interdisciplinary collaboration is essential in addressing complex patient needs. | |
| | 10. Multiple factors may contribute to nausea and vomiting. | |

NURSING DIAGNOSIS: Impaired nutritional status associated with anorexia, cachexia, or malabsorption

GOAL: Maintenance of nutritional status and of weight within 10% of pretreatment weight

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess and address factors that interfere with oral intake or are associated with increased risk of decreased nutritional status. 2. Initiate appropriate referrals for interdisciplinary collaboration to manage factors that interfere with oral intake. 3. Educate patient to avoid unpleasant sights, odors, and sounds in the environment during mealtime. 4. Suggest foods that are preferred and well tolerated by the patient, preferably high-calorie and high-protein foods. Respect ethnic and cultural food preferences. 5. Encourage adequate fluid intake, but limit fluids at mealtime. 	<ol style="list-style-type: none"> 1. Multiple patient or treatment-related factors are associated with increased risk of impaired nutritional intake, such as radiation to the head, neck, and thorax; stomatoxic or emetogenic chemotherapy; prior oral, head, and neck surgery; mucositis; impaired swallowing or dysphagia; poor dentition; cough or dyspnea. 2. Other disciplines may be more appropriate for assessment and management of issues such as swallowing impairments (speech therapy), fatigue and decreased physical ability (physical and occupational therapy), nutritional assessment and determination of patient needs (nutritionist), cough and dyspnea (respiratory therapy), poor dentition (dental medicine), depression/anxiety (social worker, psychologist, or psychiatrist). 3. Anorexia can be stimulated or increased with noxious stimuli. 	<ul style="list-style-type: none"> • Factors associated with increased risk for impaired nutritional intake are identified • Factors associated with increased risk of impaired nutritional intake are identified and addressed, whenever possible, through interdisciplinary collaboration • Patient and family identify minimal nutritional requirements • Maintains or increases weight and body cell mass as per goals identified by nutritionist • Reports decreasing anorexia and increased interest in eating • Demonstrates normal skin turgor

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| 6. Suggest smaller, more frequent meals. | 4. Foods preferred, well tolerated, and high in calories and protein maintain nutritional status during periods of increased metabolic demand. | • Identifies rationale for dietary modifications; patient and family verbalize strategies to minimize nutritional deficits |
| 7. Promote relaxed, quiet environment during mealtime with increased social interaction as desired. | 5. Fluids are necessary to eliminate wastes and prevent dehydration. Increased fluids with meals can lead to early satiety. | • Participates in calorie counts and diet histories |
| 8. If patient desires, serve alcoholic beverages at mealtime with foods. | 6. Smaller, more frequent meals are better tolerated because early satiety is less likely to occur. | • Uses relaxation techniques and guided imagery before meals |
| 9. Consider cold foods, if desired. | 7. A quiet environment promotes relaxation. Social interaction at mealtime may foster appetite, divert focus on food, and promote enjoyment of eating. | • Exhibits laboratory and clinical findings indicative of adequate nutritional intake: normal serum levels of protein, albumin, transferrin, iron, blood urea nitrogen (BUN), creatinine, vitamin D, electrolytes, hemoglobin, hematocrit, and lymphocytes; normal urinary creatinine levels |
| 10. Encourage nutritional supplements and high-protein foods between meals. | 8. Alcoholic beverages may stimulate appetite and add calories. | • Consumes diet containing required nutrients |
| 11. Encourage frequent oral hygiene, particularly prior to meals. | 9. Cold, high-protein foods are often more tolerable and less odorous than hot foods. | • Carries out oral hygiene before meals |
| 12. Address pain and other symptom management needs. | 10. Supplements and snacks add protein and calories to meet nutritional requirements. | |
| 13. Increase activity level as tolerated. | 11. Oral hygiene may stimulate appetite and increase saliva production. | |
| 14. Decrease anxiety by encouraging verbalization of fears and concerns; use relaxation techniques and guided imagery at mealtime. | 12. Pain and other symptoms impair appetite and nutritional intake. | |
| 15. Instruct patient and family about body alignment and proper positioning at mealtime. | 13. Increased activity promotes appetite. | |
| 16. Collaborate with dietician to provide nutritional | 14. Relief of anxiety may increase appetite. | |

- counseling; instruct patient and family regarding enteral tube feedings of commercial liquid diets, elemental diets, or other foods as prescribed.
17. Collaborate with dietician or nutrition support team to instruct patient and family regarding home parenteral nutrition with lipid supplements as prescribed.
18. Administer appetite stimulants as prescribed by primary provider.
19. Encourage family and friends not to nag or cajole patient about eating.
20. Assess and address other contributing factors to nausea, vomiting, and anorexia such as electrolyte imbalance, radiation therapy, medications, and central nervous system metastasis.
15. Proper body position and alignment are necessary to aid chewing and swallowing.
16. Nutritional counseling may improve outcomes. Tube feedings may be necessary in the severely debilitated patient who has a functioning gastrointestinal system but is unable to maintain adequate oral intake.
17. Parenteral nutrition with supplemental fats supplies needed calories and proteins to meet nutritional demands, especially in the nonfunctional gastrointestinal system.
18. Although the mechanism is unclear, medications such as megestrol acetate have been noted to improve appetite in patients with cancer and human immunodeficiency virus infection.
19. Pressuring patient to eat may cause conflict and unnecessary stress.
20. Multiple factors contribute to anorexia and nausea.
- Reports decreased pain or other symptoms; symptoms do not interfere with oral intake
 - Reports decreasing episodes of nausea and vomiting
 - Participates in increasing levels of activity as measured by assessment of performance status
 - Family and friends do not focus efforts on encouraging food intake
 - States rationale for use of tube feedings or parenteral nutrition
 - Demonstrates ability to manage enteral feedings or parenteral nutrition, if prescribed
 - Maintains body position and alignment needed to facilitate chewing and swallowing

NURSING DIAGNOSIS: Fatigue**GOAL:** Decreased fatigue level

Nursing Intervention	Rationale	Expected Outcomes
1. Assess patient and treatment factors that are associated with or increase fatigue (e.g., anemia, fluid and electrolyte imbalances, pain, anxiety, etc.) 2. Institute interventions to address factors contributing to fatigue (e.g., correct electrolyte imbalance, manage pain, collaborative management of anemia, administer prescribed antidepressants, anxiolytics, hypnotics, or psychostimulants, as indicated) 3. Encourage balance of rest and exercise; avoiding extended periods of inactivity. At minimum, promote patient's normal sleep habits. 4. During active treatment, rearrange daily schedule and organize activities to conserve energy expenditure; encourage patient to ask for others' assistance with	1. Multiple factors are associated with or contribute to cancer-related fatigue. Although fatigue is common in patients receiving chemotherapy or radiation therapy, there are several factors that can be modified or addressed, such as dehydration, electrolyte abnormalities, organ impairment, anemia, impaired nutrition, pain and other symptoms, depression, anxiety, impaired mobility, and dyspnea 2. Addressing factors contributing to fatigue assists in managing fatigue (e.g., lowered hemoglobin and hematocrit predispose patient to fatigue due to decreased oxygen availability especially in a setting of impaired mobility that requires increased energy expenditure). 3. Sleep helps to restore energy levels. Prolonged napping during the day may interfere with sleep habits. 4. Reorganization of activities can reduce energy losses and stressors.	<ul style="list-style-type: none">• Factors contributing to fatigue are assessed and managed whenever possible• Exhibits acceptable serum value levels for nutritional indices (see Imbalanced Nutrition)• Reports decreased pain or other symptoms• Consumes diet with recommended nutritional intake• Achieves or maintains appropriate weight and body mass• Maintains adequate hydration• Reports decreasing levels of fatigue• Adopts healthy lifestyle practices• Rests when fatigued

- necessary chores, such as housework, child care, shopping, and cooking. During periods of profound fatigue, consider reduced job workload, if necessary and possible, by reducing number of hours worked per week.
5. Encourage protein, fat, and calorie intake at least equal to that recommended for the general public.
 6. Encourage the use of relaxation techniques and guided imagery.
 7. Encourage participation in planned exercise programs involving aerobic, resistance, and flexibility training based on individual limitations and safety measures.
 - a. Minimum exercise for survivors depending on individual capabilities ranges from 10 minutes of light exercise, yoga, or stretching daily to 30 minutes of moderate to vigorous activity
 5. Protein and calorie depletion decreases activity tolerance; preventing malnutrition, achieving and maintaining recommended weight and body mass assist in management of fatigue.
 6. Promotion of relaxation and psychological rest limits contribution to physical fatigue.
 7. Various approaches to exercise programs have demonstrated increases in endurance and stamina and lower fatigue.
 8. Many providers fail to discuss the role of exercise and healthy lifestyle practices for patients during and after cancer treatment. Patients may be more likely to utilize the benefits of exercise in addressing fatigue if they receive a formal prescription.
 9. Creates community partnerships, a nonclinical environment of support, fosters increased awareness of survivorship needs, and provides referral sources that can reach more survivors.
 10. A CET designs and administers fitness assessments and exercise programs specific to an
- Reports adequate sleep
 - Requests assistance with activities appropriately
 - Uses relaxation exercises and imagery to decrease anxiety and promote rest
 - Reports no breathlessness during activities
 - Reports improved ability to relax and rest
 - Exhibits improved mobility and decreased fatigue
 - Fatigue does not interfere with ability to participate in activities of daily living or pleasure

8. Collaborate with other cancer providers to encourage them to give patients a prescription to exercise and explain role of exercise in cancer treatment.
individual's cancer diagnosis, treatment, current recovery status; possesses basic understanding of cancer diagnoses, treatments, and potential adverse effects.
9. Partner with community organizations (e.g., YMCA) to develop and offer cancer survivor specific rehab/exercise programs.
10. Collaborate with physical and occupational therapy or refer to American College of Sports Medicine (ACSM) Certified Cancer Exercise Trainer (CET) to identify safe and appropriate activities.

NURSING DIAGNOSIS: Chronic pain**GOAL:** Relief of pain and discomfort

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none">1. Use pain scale to assess pain and discomfort characteristics: location, quality, frequency, duration, etc., at baseline and on an ongoing basis. Assure patient that2. you know the pain is real and will assist them in reducing it.3. Assess prior pain experiences and previous management strategies the patient found successful.4. Assess other factors contributing to patient's pain: fear, fatigue, other symptoms, psychosocial distress, etc.5. Provide education to patient and family about prescribed analgesic regimen.6. Address myths or misconceptions and lack of knowledge about the use of opioid analgesics.	<ol style="list-style-type: none">1. Provides baseline for assessing changes in pain level and evaluation of interventions2. Fear that pain will not be considered real increases anxiety and reduces pain tolerance.3. Helps to individualize pain management approaches and identify potential challenges or approaches that should not be utilized because of safety or other issues4. Provides data about factors that decrease the patient's ability to tolerate pain and increase pain level5. Analgesics tend to be more effective when given early in pain cycle, around the clock at regular intervals, or when given in long-acting forms; breaks the pain cycle; premedication with analgesics is used for activities that cause increased pain or breakthrough pain.6. Barriers to adequate pain management involve patients' fear of side effects, fatalism about the possibility of achieving pain control,	<ul style="list-style-type: none">• Reports decreased level of pain and discomfort on pain scale• Reports less disruption in activity and quality of life from pain and discomfort• Reports decrease in other symptoms and psychosocial distress• Adheres to analgesic regimen as prescribed• Barriers to adequately addressing pain do not interfere with strategies for managing pain.• Takes an active role in administration of analgesia• Identifies additional effective pain-relief strategies• Uses previously employed successful pain-relief strategies appropriately• Identifies or utilizes nonpharmacologic pain-relief strategies and

- | | | |
|--|---|--|
| <p>7. Collaborate with patient, primary provider, and other health care team members when changes in pain management are necessary.</p> <p>8. Consult with palliative care providers or team throughout the cancer continuum.</p> <p>9. Explore nonpharmacologic and complementary strategies to relieve pain and discomfort: distraction, imagery, relaxation, cutaneous stimulation, acupuncture, etc.</p> | <p>fear of distracting providers from treating the cancer, belief that pain is indicative of progressive disease, and fears about addiction. Professional health providers also have demonstrated limited knowledge about evidence-based approaches to pain.</p> <p>7. New methods of administering analgesia must be acceptable to patient, primary provider, and health care team to be effective; patient's participation decreases the sense of powerlessness.</p> <p>8. Palliative care specialists provide expertise and contribute to symptom management regardless of stage of disease or treatment within the cancer continuum, not only during end-stage disease. Palliative care can improve quality of life, length of survival, symptom burden, mood, and efficient utilization of health services.</p> <p>9. Increases the number of options and strategies available to patient that serve as adjuncts to pharmacologic interventions.</p> | <ul style="list-style-type: none"> • Reports successful decrease in pain • Reports that decreased level of pain permits participation in other activities and events and quality of life |
|--|---|--|

NURSING DIAGNOSIS: Grief associated with loss; altered role functioning

GOAL: Appropriate progression through grieving process

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Encourage verbalization of fears, concerns, and questions regarding disease, treatment, and future implications. 2. Explore previous successful coping strategies. 3. Encourage active participation of patient or family in care and treatment decisions. 4. Visit family and friends to establish and maintain relationships and physical closeness. 5. Encourage ventilation of negative feelings, including projected anger and hostility, within acceptable limits. 6. Allow for periods of crying and expression of sadness. 7. Involve spiritual advisor as desired by the patient and family. 8. Refer patient and family to professional counseling as indicated to alleviate 	<ol style="list-style-type: none"> 1. An increased and accurate knowledge base decreases anxiety and dispels misconceptions. 2. Provides frame of reference and examples of coping. 3. Active participation maintains patient independence and control. 4. Frequent contacts promote trust and security and reduce feelings of fear and isolation. 5. This allows for emotional expression without loss of self-esteem. 6. These feelings are necessary for separation and detachment to occur. 7. This facilitates the grief process and spiritual care. 8. Goal is to facilitate the grief process or adaptive methods of coping. 9. Grief work is variable. Not every person uses every phase of the grief process, and the time spent in dealing with each phase varies with every person. To complete grief work, this variability must be allowed. 	<p>The patient and family:</p> <ul style="list-style-type: none"> • Progress through the phases of grief as evidenced by increased verbalization and expression of grief. • Identify resources available to aid coping strategies during grieving. • Use resources and supports appropriately. • Discuss the future openly with each other. • Discuss concerns and feelings openly with each other. • Use nonverbal expressions of concern for each other. • Develop positive or adaptive coping mechanisms for processing of grief.

pathologic or
nonadaptive
grieving.

9. Allow for progression through the grieving process at the individual pace of the patient and family.

NURSING DIAGNOSIS: Disturbed body image and situational low self-esteem associated with changes in appearance, function, and roles

GOAL: Improved body image and self-esteem

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Assess patient's feelings about body image and level of self-esteem. 2. Identify potential threats to patient's self-esteem (e.g., altered appearance, decreased sexual function, hair loss, decreased energy, role changes). Validate concerns with patient. 3. Encourage continued participation in activities and decision making. 4. Encourage patient to verbalize concerns. 5. Individualize care for the patient. 6. Assist patient in self-care when fatigue, lethargy, nausea, vomiting, and other symptoms prevent independence. 7. Assist patient in selecting and using cosmetics, scarves, hair pieces, hats, and clothing that increase their sense of attractiveness. 8. Encourage patient and partner to share 	<ol style="list-style-type: none"> 1. Provides baseline assessment for evaluating changes and assessing effectiveness of interventions. 2. Anticipates changes and permits patient to identify importance of these areas to them. 3. Encourages and permits continued control of events and self. 4. Identifying concerns is an important step in coping with them. 5. Prevents or reduces depersonalization and emphasizes patient's self-worth. 6. Physical well-being improves self-esteem. 7. Promotes positive body image. 8. Provides opportunity for expressing concern, intimacy, affection, and acceptance. 	<ul style="list-style-type: none"> • Identifies concerns of importance • Takes active role in activities • Maintains participation in decision making • Verbalizes feelings and reactions to losses or threatened losses • Participates in self-care activities • Permits others to assist in care when they are unable to be independent • Exhibits interest in appearance, maintains grooming, and uses aids (cosmetics, scarves, etc.) appropriately if desired • Participates with others in conversations and social events and activities • Verbalizes concern about sexual partner or significant others • Explores alternative ways of expressing concern and affection • The patient and significant other can maintain level of intimacy and express affection and acceptance

- concerns about altered sexuality and sexual function and to explore alternatives to their usual sexual expression.
9. Refer to collaborating specialists as needed.

COLLABORATIVE PROBLEM: Potential complication: risk for bleeding problems

GOAL: Prevention of bleeding

Nursing Intervention	Rationale	Expected Outcomes
<ol style="list-style-type: none"> 1. Monitor for factors increasing risk of bleeding (thrombocytopenia, elevated INR/PT/PTT, decreased fibrinogen or other clotting factors, use of medications affecting platelets or other clotting indices) 2. Assess for and educate patient/family about signs and symptoms of bleeding: <ol style="list-style-type: none"> a. Petechiae or ecchymosis (bruising) b. Decrease in hemoglobin or hematocrit c. Prolonged bleeding from invasive procedures, venipunctures, 	<ol style="list-style-type: none"> 1. The underlying cancer, antineoplastic agents or other medications may interfere with normal mechanisms of clotting. 2. Early detection promotes early intervention. <ol style="list-style-type: none"> a. Petechiae and ecchymosis indicate injury to microcirculation and larger vessels. b. Decreased hemoglobin or hematocrit may indicate blood loss. c. Prolonged bleeding may indicate 	<ul style="list-style-type: none"> • Signs and symptoms of bleeding are identified • Exhibits no blood in feces, urine, or emesis • Exhibits no bleeding of gums or injection/venipuncture sites • Exhibits no ecchymosis (bruising) or petechiae • Patient and family identify ways to prevent bleeding • Uses recommended measures to reduce risk of bleeding (uses soft toothbrush, shaves with electric razor only) • Exhibits normal vital signs • Reports that environmental hazards have been reduced or removed • Maintains hydration

	minor cuts or scratches	abnormal clotting indices.	<ul style="list-style-type: none"> Reports absence of constipation Avoids substances interfering with clotting Absence of tissue destruction Exhibits normal mental status and absence of signs of intracranial bleeding Avoids medications that interfere with clotting (e.g., aspirin) Absence of epistaxis and cerebral bleeding
d.	Frank or occult blood in any body fluids	d. Occult blood in body fluids indicates bleeding.	
e.	Bleeding from any body orifice	e. Indicates blood loss	
f.	Altered mental status	f. Altered mental status may indicate decreased cerebral tissue oxygenation or bleeding.	
g.	Hypotension; tachycardia	g. Hypotension or tachycardia may indicate blood loss.	
3.	Instruct patient and family about ways to minimize risk of bleeding.	3. Patient can participate in self-protection.	
a.	Use soft toothbrush for mouth care.	a. Prevents trauma to oral tissues	
b.	Avoid commercial mouthwashes.	b. Contains high alcohol content that will dry oral tissues	
c.	Use electric razor for shaving.	c. Prevents trauma to skin	
d.	Use emery board for nail care.	d. Reduces risk of trauma to nail beds	
e.	Avoid foods that are difficult to chew.	e. Prevents oral tissue trauma	
f.	Keep lips moisturized with water-based lubricant	f. Prevents skin from drying	
g.	Maintain fluid intake of at least 3 L per 24 hours unless contraindicated	g. Prevents skin and oral tissue membranes from drying	
h.	Use stool softeners or increase bulk in diet.	h. Prevents trauma to rectal	
i.	Recommend use of water-based lubricant before		

- sexual intercourse.
4. Initiate measures to minimize bleeding. Draw all blood for lab work with one daily venipuncture for hospitalized patients.
- Avoid taking temperature rectally or administering suppositories and enemas.
 - Avoid intramuscular injections; use smallest needle possible.
 - Apply direct pressure to injection and venipuncture sites for at least 5 minutes.
 - Avoid bladder catheterizations; use smallest catheter if catheterization is necessary.
 - Avoid medications that will interfere with clotting (e.g., aspirin).
 - Recommend use of water-based lubricant before sexual intercourse.
 - Platelet transfusions as prescribed; administer prescribed diphenhydramine
- mucosa from straining
- Prevents friction and tissue trauma
 - Measures are taken to minimize bleeding
 - Minimizes blood loss
 - Bleeding may occur from intramuscular injection sites, particularly if large bore needles are used
 - Bleeding may occur if direct pressure is not applied for a long enough time period
 - Prevents trauma to urethra
 - Minimizes risk of bleeding
 - Helps prevent bleeding from small skin tears
 - Platelet count <20,000/mm³ ($0.02 \times 1012/L$) is associated with increased risk of spontaneous bleeding.
- Allergic reactions to blood products are associated with antigen-

- hydrochloride or hydrocortisone sodium succinate to prevent reaction to platelet transfusion.
- h. Supervise activity when out of bed.
 - i. Caution against forceful nose blowing.
- antibody reaction that causes platelet destruction
- h. Reduces risk of falls
 - i. Prevents trauma to nasal mucosa and increased intracranial pressure

Adapted from Corbitt, N., Harrington, J., Kendall, T. (2017). Putting evidence into practice: Prevention of bleeding. Retrieved on 7/23/2019 at: www.ons.org/pep/bleeding; Eilers, J. G., Asakura, Y., Blecher, C. S., et al. (2017). Putting evidence into practice: Mucositis. Retrieved on 7/7/2019 at: www.ons.org/pep/mucositis; Gosselin, T., Beamer, L., Ciccolini, K., et al. (2017). Putting evidence into practice: Radiodermatitis. Retrieved on 7/29/2019 at: www.ons.org/pep/radiodermatitis; Miaskowski, C. A., Brant, J. M., Caldwell, P., et al. (2017). Putting evidence into practice: Chronic pain. Retrieved on 7/19/2019 at: www.ons.org/pep/chronic-pain; Mitchell, S. A., Albrecht, T. A., Omar Alkaiyat, M., et al. (2017). Putting evidence into practice: Fatigue. Retrieved on 7/2/2019 at: www.ons.org/pep/fatigue; Thorpe, D. M., Conley, S. B., Drapek, L., et al. (2017). Putting evidence into practice: Anorexia. Retrieved on 7/28/2019 at: www.ons.org/pep/anorexia; Williams, L., Ciccolini, K., Johnson, L. A., et al. (2017). Putting evidence into practice: Skin reactions. Retrieved on 7/23/2019 at: www.ons.org/pep/skin-reactions; and Wilson, B. J., Ahmed, F., Crannell, C. E., et al. (2017). Putting evidence into practice: Preventing infection – general. Retrieved on 7/5/2019 at: www.ons.org/pep/prevention-infection-general

Radiation-Associated Impairment of Skin Integrity

Although advances in radiation therapy have resulted in decreased incidence and severity of skin impairments, patients may still develop radiodermatitis, formerly called radiation dermatitis, associated with pain, irritation, pruritus, burning, skin sloughing without drainage (dry desquamation) or with drainage (wet desquamation), and diminished quality of life (Gosselin, Beamer, Ciccolini, et al., 2017; Olsen et al., 2019). Nursing care for patients with radiodermatitis includes maintenance of skin integrity, cleansing, promotion of comfort, pain reduction, prevention of additional trauma, prevention and management of infection, and promotion of a moist wound-healing environment (Olsen et al., 2019). In order to prevent impaired skin integrity, patients are advised to use moisturizer on the skin, avoid sun exposure to the area of treatment, and avoid tape or bandages and other sources of irritation or trauma. Although a variety of methods and products are used in clinical practice for patients with radiation-induced skin impairment, there is

limited evidence to support their value (Gosselin et al., 2017). Patients with skin and tissue reactions to radiation therapy require careful skin care to prevent further skin irritation, drying, and damage, as discussed in the nursing care plan (see [Chart 12-6](#), Risk for impaired skin integrity: erythematous and wet desquamation reactions to radiation therapy).

Alopecia

The temporary or permanent thinning or complete loss of hair is a potential adverse effect of whole brain radiation therapy, various chemotherapies and targeted agents. Alopecia usually begins 1 to 3 weeks after the initiation of chemotherapy and radiation therapy; regrowth most often begins within 8 weeks after the last treatment (Olsen et al., 2019). Some patients who undergo radiation to the head may sustain permanent hair loss. The onset of gradually progressing alopecia and body hair loss associated with targeted therapies generally occurs 1 to 3 months after the start of treatment and may be patchy appearing as temporal or frontal hair loss (Barton-Burke, Ciccolini, Mekas, et al., 2017). This type of hair loss is usually reversible after the end of therapy and in some cases beginning sooner. Several targeted agents are associated with changes in hair growth rate, curliness, texture, and pigmentation. Although health care providers may view hair loss as a minor issue, for many patients it is a major assault on body image, challenging to self-esteem, and resulting in psychosocial distress and depression. Despite the significant psychosocial impact of alopecia, few studies have addressed methods to prevent or minimize the impact of alopecia. The use of cryotherapy to the head (scalp cooling) has been shown to be effective in reducing alopecia during chemotherapy administration (Ross & Fischer-Cartlidge, 2017; Rugo, Melin, & Voigt, 2017). However, due to reports of scalp metastasis and a lack of safety data, cryotherapy is not recommended for use in patients with hematologic malignancies (Rugo et al., 2017). Nurses provide information about hair loss and support the patient and family in coping with changes in body image. Patients are assisted to identify proactive choices that may empower them to improve responses to cancer and perceived lack of control as discussed in the nursing care plan (see [Chart 12-6](#), Impaired tissue integrity: alopecia).

Malignant Skin Lesions

Skin lesions may occur with local extension or metastasis of the tumor into the epithelium and its surrounding lymph and blood vessels. Either locally invasive or metastatic cancer to the skin may result in erythema, discolored nodules, or progression to wounds involving edema, exudates, and tissue necrosis. The most extensive lesions involve ulceration (referred to as fungating lesions) with an overgrowth of malodorous microorganisms. These lesions are a source of considerable pain, discomfort, and embarrassment. Although skin lesions occur in various malignancies, they are most commonly associated with breast cancer.

Ulcerating skin lesions usually indicate advanced or disseminated disease that is unlikely to be eradicated but may be controlled or palliated through systemic treatment (chemotherapy and targeted therapy) or radiation therapy. Local care of

these lesions is a nursing priority. Nurses carefully assess malignant skin lesions for the size, appearance, condition of surrounding tissue, odor, bleeding, drainage, and associated pain or other symptoms, including evidence of infection. The potential for serious complications such as hemorrhage, vessel compression/obstruction, or airway obstruction, especially in head and neck cancer, should be noted so that the caregiver can be instructed in palliative measures to maintain patient comfort.

Nursing care (see [Chart 12-6](#)) also includes wound cleansing, reduction of superficial bacteria, control of bleeding, odor reduction, protection from further skin trauma, and pain management. The patient and family require emotional support, assistance, and guidance in providing wound care and addressing comfort measures at home.

Promoting Nutrition

Most patients with cancer experience some weight loss during their illness. Thus nurses need to promote good nutrition throughout treatment.

Nutritional Impairment

Anorexia, malabsorption, and cancer-related anorexia-cachexia syndrome (CACS) are some common nutritional problems. Impaired nutritional status may contribute to both physical and psychosocial consequences (see [Chart 12-7](#)). Nutritional concerns include decreased protein and caloric intake, metabolic or mechanical effects of the cancer, systemic disease, side effects of the treatment, or the patient's emotional status.

Chart 12-7

Potential Consequences of Impaired Nutrition in Patients with Cancer

- Anemia
- Decreased survival
- Immune incompetence and increased incidence of infection
- Delayed tissue and wound healing
- Fatigue
- Diminished functional ability
- Decreased capacity to continue antineoplastic therapy
- Increased hospital admissions
- Increased length of hospital stay
- Impaired psychosocial functioning

Anorexia

Among the many causes of anorexia in patients with cancer are alterations in taste, manifested by increased salty, sour, and metallic taste sensations, and altered responses to sweet and bitter flavors. Taste changes contribute to decreased appetite and nutritional intake and subsequently protein-calorie malnutrition. Taste

alterations may result from mineral (e.g., zinc) deficiencies, increases in circulating amino acids and cellular metabolites, or the administration of chemotherapeutic agents. Patients undergoing radiation therapy to the head and neck may experience “mouth blindness,” which is a severe impairment of taste.

Anorexia may occur because patients develop early satiety after eating only a small amount of food. This sense of fullness occurs secondary to a decrease in digestive enzymes, abnormalities in the metabolism of glucose and triglycerides, and prolonged stimulation of gastric volume receptors, which convey the feeling of being full. Psychological distress (e.g., fear, pain, depression, isolation) throughout illness may also have a negative impact on appetite. Patients may develop an aversion to food because of nausea and vomiting associated with treatment.

Malabsorption

Some patients with cancer are unable to absorb nutrients from the gastrointestinal system as a result of tumor activity, cancer treatments, or both. Malignancy can affect gastrointestinal activity in several ways (e.g., impaired enzyme production, interference with both protein and fat digestion) that can lead to increased gastrointestinal irritation, peptic ulcer disease, and fistula formation.

Chemotherapy and radiation associated with mucositis cause damage to mucosal cells of the bowel, resulting in impaired nutrient absorption. Abdominal irradiation has been associated with sclerosis of intestinal blood vessels and fibrotic changes in the gastrointestinal tissue, both impacting nutrient absorption. Surgical intervention may change peristaltic patterns, alter gastrointestinal secretions, and reduce the absorptive surfaces of gastrointestinal mucosa, all of which contribute to malabsorption.

Cancer-Related Anorexia-Cachexia Syndrome

CACS is a complex biologic process that results from a combination of increased energy expenditure and decreased intake (Mattox, 2017; Olsen et al., 2019). This syndrome can occur in both the curative and palliative stages of treatment and care. Combined immunologic, neuroendocrine, and metabolic processes give rise to anorexia, unintentional weight loss, and increased metabolic demand with impaired metabolism of glucose and lipids. As this syndrome continues, altered metabolic processes and tumor responses lead to cytokine release, causing generalized systemic inflammation. The patient experiences continued weight loss and malnutrition characterized by loss of adipose tissue, visceral protein, and skeletal muscle mass. Patients with CACS complain of loss of appetite, early satiety, and fatigue. It is estimated that 50% of patients with cancer experience anorexia and cachexia; this percentage increases to as high as 86% in patients with advanced cancer at the end of life (Olsen et al., 2019). Protein losses are associated with the development of anemia, peripheral edema, and progressive debilitation. The signs and symptoms of cancer cachexia and progressive debilitation result in decreased quality of life, psychological distress, and anxiety for both patient and family as they respond to actual and perceived impending losses, fear, lack of control, and helplessness.

Nursing care is integral to an interdisciplinary approach that addresses the multiple factors contributing to impaired nutritional status in patients with cancer (see [Chart 12-6](#)).

General Nutrition Considerations

Assessment of the patient's nutritional status is conducted at diagnosis and monitored throughout the course of treatment and follow-up. Early identification of patients at risk for problems with intake, absorption, and cachexia, particularly during the early stages of disease, can facilitate timely implementation of specifically targeted interventions that attempt to improve quality of life, treatment outcomes, and survival (Krishnasamy, Yoong, Chan, et al., 2017). Current weight, weight loss, diet and medication history, patterns of anorexia, nausea and vomiting, diarrhea, and situations and foods that aggravate or relieve symptoms are assessed and addressed.

The type of cancer, stage, and treatment approaches are considered so that proactive measures to support nutrition can be identified. For example, patients with head and neck cancers who are treated with radiation therapy, or some combination of surgery, radiation, chemotherapy or targeted agents, are at high risk for inadequate oral intake and nutritional deficits. In many centers, these patients have a percutaneous endoscopic gastrostomy (PEG) tube placed for enteral nutrition prior to initiation of treatment and the onset of mucositis, weight loss, and other consequences of impaired oral intake. A speech therapy consult may be helpful for patients with oropharyngeal or laryngeal tumors or surgical interventions that are anticipated to effect swallowing, secretion management, speech, or respiratory function.

Whenever possible, every effort is made to maintain adequate nutrition through the oral route. Prokinetic agents such as metoclopramide are used to increase gastric emptying in patients with early satiety and delayed gastric emptying. Other pharmacologic interventions such as megestrol acetate or corticosteroids (on a short-term basis) may be used to improve appetite. Oral nutritional supplements are encouraged to meet nutritional needs and to maintain or improve weight gain and physical functioning. Approaches incorporate nutritional counseling, exercise, pharmacologic interventions to combat anorexia, and symptom management when feasible (NCCN, 2019g). If adequate nutrition cannot be maintained by oral intake, nutritional support via the enteral route may be necessary as discussed previously. When needed, the patient and family are taught to administer enteral nutrition in the home. Home health nurses assist with patient education and monitor the patient's symptoms and response to enteral nutrition.

When malabsorption is a problem, enzyme and vitamin replacement may be instituted. Additional strategies include changing the feeding schedule, using simple diets, and relieving diarrhea. If malabsorption is severe, or the cancer involves the upper gastrointestinal tract, parenteral nutrition may be necessary. However, patients receiving parenteral nutrition are at increased risk for complications, including catheter-related and systemic infection. The use of parenteral nutrition in patients with advanced or end-stage cancer is seldom used and controversial (Jatoi & Kelly, 2019). Parenteral nutrition can be given in several ways: by a long-term

venous access device such as a right atrial catheter (see Fig. 12-3), implanted venous port (see Fig. 12-4), or PICC (see Fig. 12-6). The nurse educates the patient and family to care for the venous access device and to administer parenteral nutrition. Home health nurses provide education and assist with or supervise parenteral nutrition administration in the home.

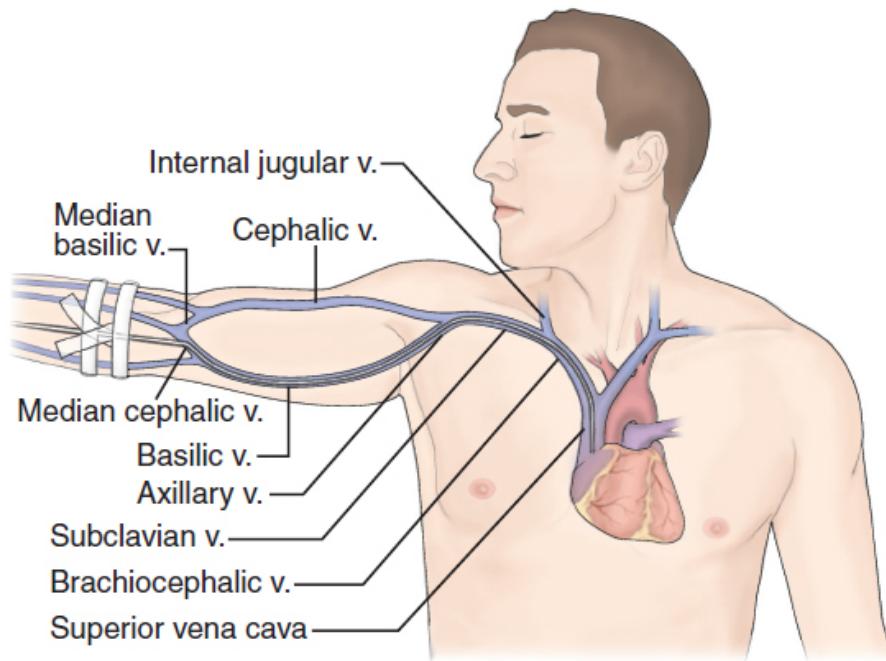


Figure 12-6 • A peripherally inserted central catheter is advanced through the cephalic or basilic vein to the axillary, subclavian, or brachiocephalic vein or the superior vena cava.

Relieving Pain

More than half of patients with cancer experience pain throughout the cancer trajectory. Moderate to severe pain is reported by approximately 28% of patients with cancer during treatment and as many as 52% of those with advanced disease (van den Beuken-van Everdingen, Hochstenbach, Joosten, et al., 2016). Although cancer pain may be acute, it is more frequently characterized as chronic. (See Chapter 9 for more information on pain.) As in other situations involving pain, the experience of cancer pain is influenced by physical, psychosocial, cultural, and spiritual factors.

Cancer can cause pain in various ways (see Table 12-9). Initially, pain is most often related to the underlying cancer process. Pain is also associated with various cancer treatments. Acute pain is linked with trauma from surgery. Occasionally, chronic pain syndromes, such as postsurgical neuropathies (pain related to nerve tissue injury), occur. Some chemotherapeutic agents cause tissue necrosis, peripheral neuropathies, and stomatitis—all potential sources of pain—whereas radiation therapy can cause pain secondary to skin, nervous tissue, or organ

inflammation. Patients with cancer may have other sources of pain, such as arthritis or migraine headaches, that are unrelated to the underlying cancer or its treatment.

The nurse assesses the patient for the source and site of pain as well as those factors that influence the patient's perception and experience of pain, such as fear and apprehension, fatigue, anger, and social isolation. Pain assessment scales are useful for assessing the patient's pain before and after pain-relieving interventions are instituted to assess the effectiveness of interventions. Other symptoms that contribute to the pain experience, such as nausea and fatigue, are assessed and addressed as well.

TABLE 12-9 Examples of Sources of Cancer Pain

Source	Descriptions	Underlying Cancer
Bone metastasis	Throbbing, aching	Breast, prostate, myeloma
Ischemia	Sharp, throbbing	Kaposi sarcoma
Lymphatic or venous obstruction	Dull, aching, tightness	Lymphoma, breast, Kaposi sarcoma
Nerve compression, infiltration	Burning, sharp, tingling	Breast, prostate, lymphoma
Organ obstruction	Dull, crampy, gnawing	Colon, gastric
Organ infiltration	Distention, crampy	Liver, pancreatic
Skin inflammation, ulceration, infection, necrosis	Burning, sharp	Breast, head and neck, Kaposi sarcoma

Adapted from van den Beuken-van Everdingen, M. H., Hochstenbach, L. M., Joosten, E. A., et al. (2016). Update on prevalence of pain in patients with cancer: Systematic review and meta-analysis. *Journal of Pain and Symptom Management*, 51(6), 1070–1090; Yarbro, C. H., Wujcik, D., & Gobel, B. H. (2018). *Cancer nursing: Principles and practice*. Burlington, MA: Jones & Bartlett Publishers.

Today, most people expect pain to disappear or resolve quickly. Although it is often controllable, advanced cancer pain is commonly irreversible and not quickly resolved. For many patients, pain is often seen as a signal that cancer is advancing, and that death is approaching. As patients anticipate pain and anxiety increases, pain perception heightens, producing fear and further pain. Thus, chronic cancer pain can lead to a cycle progressing from pain to anxiety to fear and back to pain, especially when the pain is not adequately managed. Inadequate pain management is most often the result of misconceptions and insufficient knowledge about pain assessment and management on the part of patients, families, and health care providers (Scarborough & Smith, 2018). Chapter 9 provides information regarding factors contributing to the pain experience, pain perception, and tolerance as well as pharmacologic and nonpharmacologic nursing interventions addressing pain. The nursing care plan (see Chart 12-6) also provides strategies for nursing assessment and management of chronic pain. Analgesics are administered based on the patient's reported level of pain. A cancer pain algorithm, developed as a set of analgesic guiding principles, is given in Figure 12-7.

Pharmacologic and nonpharmacologic approaches, even those that may be invasive, are considered in managing cancer-related pain regardless of the patient's status along the cancer trajectory. The nurse assists the patient and family to take an active role in managing pain. The nurse provides education and support to correct fears and misconceptions about opioid use. Inadequate pain management leads to a diminished quality of life characterized by distress, suffering, anxiety, fear, immobility, isolation, and depression.

Decreasing Fatigue

Fatigue is one of the most frequent and distressing symptoms experienced by patients receiving cancer therapy. Patients report that fatigue persists and interferes with activities of daily living for months to years after the completion of treatment (Wang, Yang, Miao, et al., 2018). Fatigue rarely exists in isolation; patients typically experience other symptoms concurrently, such as pain, dyspnea, anemia, sleep disturbances, or depression. The relationship between sleep disturbances, fatigue, and depression (as well as other clinical factors) in older adults with cancer is an active area of research.

In assessing fatigue, the nurse distinguishes between *acute fatigue*, which occurs after an energy-demanding experience, and *cancer-related fatigue*, which is defined as “a distressing persistent, subjective sense of physical, emotional or cognitive tiredness or exhaustion related to cancer or cancer treatment that is not proportional to recent activity and interferes with usual functioning” (NCCN, 2019h). Acute fatigue serves a protective function, whereas cancer-related fatigue does not. The exact mechanisms of fatigue are not well understood and are multifactorial in nature.

Despite the commonly occurring experience of fatigue in patients with cancer, a reliable assessment tool has not been identified. The experience of fatigue is highly subjective, with patient descriptors varying greatly (NCCN, 2019h). The nurse assesses physiologic and psychological factors that can contribute to fatigue (see [Chart 12-8](#)).

Exercise is an effective approach to the management of cancer-related fatigue (Mitchell, Albrecht, Omar Alkaiyat, et al., 2017). Psychoeducational approaches, cognitive-behavioral therapy to address sleep, progressive muscle relaxation, yoga, and mindfulness meditation may be effective measures to facilitate fatigue management (Mitchell et al., 2017). Nurses assist patients with strategies to minimize fatigue or help the patient cope with fatigue as described in the nursing care plan (see [Chart 12-6](#), Fatigue). Occasionally, pharmacologic interventions are utilized, including antidepressants for patients with depression, anxiolytics for those with anxiety, hypnotics for patients with sleep disturbances, and psychostimulants for some patients with advanced cancer or fatigue that does not respond to other interventions (NCCN, 2019h; Mitchell et al., 2017). See the Nursing Research profile in [Chart 12-9](#).

Chart 12-8

Sources of Fatigue in Patients with Cancer

- Anxiety associated with fear, diagnosis, role changes, uncertainty of future
- Disturbed sleep pattern related to cancer therapies, anxiety, and pain
- Electrolyte imbalances due to vomiting, diarrhea
- Risk for impaired nutritional intake due to nausea, vomiting, cancer-related anorexia-cachexia syndrome
- Impaired physical mobility due to neurologic impairments, surgery, bone metastasis, pain, and analgesic use
- Impaired tissue integrity due to stomatitis, mucositis
- Ineffective breathing associated with cough and dyspnea
- Ineffective protection secondary to neutropenia, thrombocytopenia, anemia
- Pain, pruritus
- Uncertainty and education needs related to disease process, treatment

Adapted from the National Comprehensive Cancer Network (NCCN). (2019h).

NCCN guidelines for supportive care: Cancer related fatigue – version 1.2019.

Retrieved on 7/20/2019 at:

www.nccn.org/professionals/physician_gls/pdf/fatigue.pdf

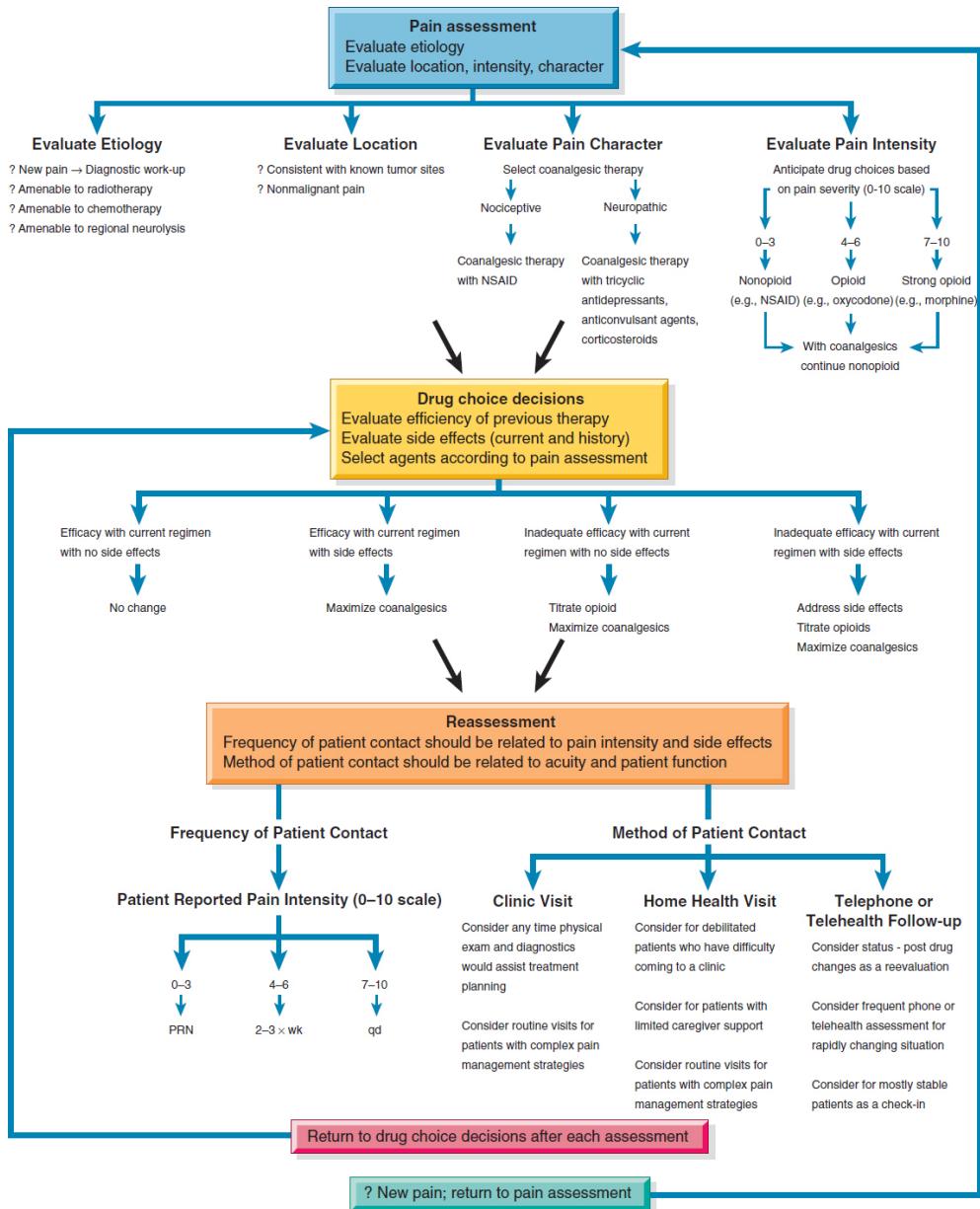


Figure 12-7 • The cancer pain algorithm (highest-level view) is a decision-tree model for pain treatment. NSAID; nonsteroidal anti-inflammatory drug; PRN, as needed; qd, once a day; wk, week. Adapted from DuPen, A. R., DuPen, S., Hansberry, J., et al. (2000). An educational implementation of a cancer pain algorithm for ambulatory care. *Pain Management Nursing*, 1(4), 118.

Chart 12-9 NURSING RESEARCH PROFILE

Poor Sleep and Fatigue in Women with Breast Cancer

Overcash, J., Tan, A., & Noonan, A. (2018). Factors associated with poor sleep in older women diagnosed with breast cancer. *Oncology Nursing Forum*, 45(3), 359–371.

Purpose

Breast cancer is the most common form of cancer in women. The risk for developing breast cancer increases with age, as does the incidence of poor quality of sleep. The purpose of this study was to determine the relationship among poor sleep and gait, grip (hand) strength, cognitive status, and symptoms (depression, pain, fatigue).

Design

Women with breast cancer (any stage) aged 69 years or older were recruited to participate in this descriptive, cross-sectional study for an outpatient cancer clinic in the midwestern region of the United States. Instruments used to collect data in this study included: the Timed Up and Go Test (gait), Jamar Hydraulic Hand Dynamometer (grip strength), the Mini-Cog (cognitive status), the Geriatric Depression Scale (depression), Numeric Pain Scale (pain), the Brief Fatigue Inventory (fatigue), and the Pittsburgh Sleep Quality Index (sleep).

Findings

Sixty women with breast cancer, with a mean age of 77.6 (range 69.0 to 93.0) years, participated in this study. Strong to moderate relationships were found between pain and fatigue ($r = 0.58, p = 0.001$); pain and depression ($r = 0.40, p = 0.002$); and depression and fatigue ($r = 0.66, p = 0.001$). Patients with greater severity of fatigue ($OR = 1.57, 95\% CI = 1.32 – 2.16$]), depression ($OR = 1.46, 95\% CI = 1.07 – 1.99$]), and pain ($OR = 1.41, 95\% CI = 1.04 – 1.92$]) were found to be at significantly higher risk of having poor sleep. Fatigue was found to have the strongest association with poor sleep. Poor sleep was not significantly related to gait, grip strength, or cognitive status in this study (all p -values > 0.05).

Nursing Implications

Nurses need to be aware of the symptoms that can affect sleep in older women with breast cancer, both during and following treatment. In this study, fatigue was found to strongly relate to poor sleep, which suggests that nurses must assess for and develop strategies to address both symptom concerns. Nurses should encourage older women with breast cancer to engage in physical activity (exercise) as part of their daily routine, to decrease fatigue and enhance sleep. Given that fatigue, depression, pain, and poor sleep may contribute to an increased risk for falls in older adults, nurses must pay particular attention to safety when caring for women with breast cancer and their families.

Improving Body Image and Self-Esteem

The nurse identifies potential threats to the patient's body image and assesses the patient's ability to cope with the many bodily changes that may be experienced throughout the course of disease and treatment. Entry into the health care system

may be accompanied by depersonalization. Threats to self-concept occur as the patient faces the realization of illness, disfigurement, possible disability, and death. To accommodate treatments or because of the disease, many patients with cancer are forced to alter their lifestyles. Priorities and values change when body image is threatened. Disfiguring surgery, hair loss, cachexia, skin changes, altered communication patterns, and sexual dysfunction can threaten the patient's self-esteem and body image.

A creative and positive approach is essential when caring for patients with altered body image. Nursing strategies for addressing issues related to body image and self-esteem are included in the nursing care plan (see [Chart 12-6](#)). The nurse serves as an active listener and counselor to both the patient and the family. Possible influences of the patient's culture and age are considered when discussing concerns and potential interventions.

Addressing Sexuality

The physiologic processes associated with cancer; potential short- and long-term effects of cancer treatments; and psychosocial, emotional, and spiritual responses to the entire experience may lead patients to confront a variety of sexuality-based issues. Patients at the greatest risk of sexual dysfunction are those with tumors that involve the sexual or pelvic organs and those whose treatment affects the hormonal systems mediating sexual function (Olsen et al., 2019). Although sexuality is an important component of overall health, many nurses and other health professionals are hesitant to include sexuality in their discussions with patients (Almont, Farsi, Krakowski, et al., 2019). In offering a holistic approach to the care of patients with cancer, nurses should initiate discussions about sexuality and assess sexual health.

Infertility, a common consequence of cancer and cancer treatments, can be of concern to patients and their partners. The potential for impaired fertility is discussed and options reviewed for fertility preservation (Olsen et al., 2019). Fertility plans are discussed and determined prior to the initiation of any therapy that may compromise reproductive abilities.

Referrals are made as needed for specialized evaluation and interventions that are beyond the scope of nursing intervention.

Assisting in the Grieving Process

A cancer diagnosis need not indicate a fatal outcome. Many forms of cancer are curable, and others may be controlled for long periods of time similar to the course of other chronic diseases. Despite the tremendous advances in cancer treatment, many patients and their families still view cancer as a fatal disease that is inevitably accompanied by pain, suffering, debilitation, and emaciation. Grieving is a normal response to these fears and to actual or potential losses: loss of health, normal sensations, body image, social interaction, intimacy, independence, and usual social roles. Patients, families, and friends may grieve for the loss of quality time to spend with others, the loss of future and unfulfilled plans, and the loss of control over the patient's body and emotional reactions. Nurses continue to assess the patient and

family for positive or maladaptive coping behaviors, interpersonal communication, and evidence of the need for additional psychosocial support or interventions such as referral for professional counseling.

If the patient enters the terminal phase of disease, the nurse may assess that the patient and family members are at different stages of grief. In such cases, the nurse assists the patient and family to acknowledge and cope with their reactions and feelings. The nurse also empowers the patient and family to explore preferences for issues related to end-of-life care, such as withdrawal of active disease treatment, desire for the use of life-support measures, and symptom management approaches. Oncology nurses respectfully support the patient's spiritual or religious views and facilitate contact with their preferred clergy member, if desired. In addition, nurses consider the patient's cultural beliefs and practices when addressing issues related to grief. After the death of a patient with cancer, home health or hospice nurses follow up with surviving family members for bereavement counseling to facilitate expression and coping with feelings of loss and grief. (See [Chapter 13](#) for further discussion of end-of-life issues.)

Management of Psychosocial Distress

Despite advances in cancer care, patients experience varying levels of psychosocial distress related to actual or potential losses, fear of the unknown, symptoms due to cancer or cancer treatments, changes in usual family and social roles, financial concerns, and a sense of loss of control. Psychosocial distress is defined as a multifactorial unpleasant emotional experience of a psychological (cognitive, behavioral, emotional), social, or spiritual nature that may interfere with the ability to cope effectively with cancer, its physical symptoms and its treatment (NCCN, 2019i). Distress occurs along a continuum, ranging from common normal feelings of vulnerability, sadness, and fears to problems that can become disabling, such as depression, anxiety, panic, social isolation, and existential and spiritual crisis. Distress that is not addressed can have a significant impact on the overall well-being, interpersonal relationships, cognitive abilities, and adherence to treatment regimens, leading to poor outcomes.

Nurses need to screen patients for psychosocial distress during the cancer experience. Patients are supported in managing various sources and levels of distress. Referral to mental health providers may be helpful to address specific concerns (NCCN, 2019i).

Monitoring and Managing Potential Complications

Nurses need to pay particular attention to monitoring for and helping manage complications such as infection, septic shock, bleeding, and thrombocytopenia.

Infection

For patients in all stages of cancer, the nurse assesses factors associated with the development of infection. Although infection-associated morbidity and mortality have greatly decreased, prevention and prompt treatment of infection are essential

in patients with cancer (Olsen et al., 2019). Often, more than one predisposing factor is present in patients with cancer. The nurse monitors laboratory studies to detect early changes in WBC counts. Common sites of infection, such as the pharynx, skin, perianal area, urinary, and respiratory tracts, are assessed on a regular basis. However, the typical signs of infection (swelling, redness, drainage, and pain) may not occur in myelosuppressed patients because of decreased circulating WBCs and a diminished local inflammatory response. Fever may be the only sign of infection (NCCN, 2019j). The nurse monitors the patient for sepsis, particularly if invasive catheters or long-term IV catheters are in place.

WBC function is often impaired in patients with cancer. Among the five types of WBCs (neutrophils [granulocytes], lymphocytes, monocytes, basophils, and eosinophils), neutrophils serve as the body's primary initial defense against invading organisms. Comprising 60% to 70% of the body's WBCs, neutrophils act by engulfing and destroying infective organisms through phagocytosis. Both the total WBC count and the concentration of neutrophils are important in determining the patient's ability to fight infection. A decrease in circulating WBCs is referred to as leukopenia. The terms granulocytopenia and neutropenia refer to a decrease in neutrophils.

A differential WBC count identifies the relative numbers of WBCs and permits tabulation of polymorphonuclear neutrophils (PMNs) or segmented neutrophils (mature neutrophils, reported as “polys,” PMNs, or “segs”) and immature forms of neutrophils (reported as bands, metamyelocytes, and “stabs”). The absolute neutrophil count (ANC) is calculated by the following formula:

$$\text{ANC} = (\text{segmented neutrophils [%]} + \text{band [%]}) \\ \times \text{WBC count (cell/mm}^3\text{)}$$

Example: (25% seg + 25% bands)
 $\times 6000 \text{ WBC cell/mm}^3 = 3000 \text{ ANC}$

Neutropenia, an abnormally low ANC, is associated with an increased risk of infection. The risk of infection rises as the ANC decreases. As the ANC declines below 1500 cells/mm³, the risk of infection rises. An ANC less than 500 cells/mm³ reflects a severe risk of infection (NCCN, 2019j). The **nadir** is the lowest ANC following chemotherapy, targeted therapy, or radiation therapy that suppresses bone marrow function. Severe neutropenia may necessitate delays in administration of myelosuppressive therapies or treatment dose adjustments, although the use of the hematopoietic growth factors (e.g., colony-stimulating factors; see previous discussion) has reduced the severity and duration of treatment-associated neutropenia as well as infection-related morbidity and early death (NCCN, 2019j). The administration of these growth factors assists in maintaining treatment schedules, drug dosages, treatment effectiveness, and quality of life.

Patients with febrile neutropenia are assessed for factors that increase the risk of infection and for sources of infection through cultures of blood, sputum, urine, stool, IV and urinary or other catheters, and wounds, if appropriate (see [Table 12-10](#)). In addition, a chest x-ray is usually obtained to assess for pulmonary infection.

Defense against infection is compromised in many ways. The integrity of the skin and mucous membranes is challenged by multiple invasive diagnostic procedures, by the adverse effects of all cancer treatment modalities, and by the detrimental effects of immobility. Impaired nutrition as a result of CACS, nausea, vomiting, diarrhea, and the underlying disease alters the body's ability to combat invading organisms. Medications such as antibiotics disturb the balance of normal flora, allowing the overgrowth of normal flora and pathogenic organisms. Other medications can also alter the immune response (see [Chapter 31](#)). Cancer itself may lead to defects in cellular and humoral immunity. Advanced cancer may cause obstruction of hollow viscera (e.g., intestines), blood, and lymphatic vessels, creating a favorable environment for proliferation of pathogenic organisms. In some patients, tumor cells infiltrate bone marrow and prevent normal production of WBCs.

TABLE 12-10 Assessment of Neutropenic Fever in Patients with Cancer

Fever Criteria	Neutropenia Criteria		
<ul style="list-style-type: none"> Any one-time temperature of 38.3°C (101°F) or Any temperature of ≥38°C (100.4°F) or ≥1 h 	<ul style="list-style-type: none"> <500 neutrophils/mcL or <1000 neutrophils/mcL and predicted to decline to ≤500 neutrophils/mcL over the next 48 h 		
Assessment Targets for Neutropenic Fever Evaluation			
Infection Risk Factors	Physical Assessment	Diagnostic Procedures	Microbiologic Cultures
<ul style="list-style-type: none"> Chronic comorbid illnesses Underlying hematologic malignancies Immunosuppression related to factors other than neutropenia (e.g., immunosuppressive therapy following allogeneic HSCT or hypogammaglobulinemia [decreased gammaglobulin production]) Solid tumors causing obstructions of the bronchial tree, ureters, colon, or biliary ducts Advanced or refractory underlying malignancy Prolonged duration of neutropenia Age ≥65 yrs Limited mobility or debilitation Medications (e.g., corticosteroids such as prednisone) Antibiotic therapy or prophylaxis Recent surgery for diagnosis or treatment Chemotherapy or targeted therapy received within the last 7–10 days Prior treatment with multiple types of chemotherapy regimens Recent radiation therapy; especially to areas 	<ul style="list-style-type: none"> Skin, pressure points, wounds Surgical or biopsy incision sites IV access or reservoir sites Drainage catheter sites Tracheostomy site Lungs and sinuses Perivaginal and perirectal area Alimentary canal, abdomen Neurologic assessment Vital signs 	<ul style="list-style-type: none"> Diagnostic imaging as appropriate to identify abscesses, fistulas, pneumonia, obstruction, etc. Complete blood count/differential Serum chemistries, liver function tests Renal function tests Pulse oximetry or arterial blood gases Lumbar puncture for CSF analysis 	<ul style="list-style-type: none"> Blood (peripheral and central venous access if applicable) Urine (especially with indwelling catheter) Skin wounds, lesions, incision sites, catheter exit sites Catheter tips when feasible Drainage from catheters Stool, diarrhea Sputum CSF

- associated with bone marrow reserves
- Prior documented infections
- Impaired skin integrity
- Invasive drainage or urinary catheters
- Peripheral or central venous access devices
- Exposures (travel, others with infection, blood administration, pets)
- Diarrhea
- Poor nutritional status
- Recent lumbar puncture or indwelling Ommaya reservoir™ (long-term intraventricular catheter for administration of chemotherapy into CSF and ventricles)

CSF, cerebrospinal fluid; HSCT, hematopoietic stem cell transplantation; IV, intravenous.

Adapted from National Comprehensive Cancer Network (NCCN). (2019j). NCCN clinical practice guidelines in oncology: Prevention and treatment of cancer-related infection—version 1.2019. Retrieved on 7/14/2019 at:

www.nccn.org/professionals/physician_gls/pdf/infections.pdf; Palmore, T. N., Parta, M., Cuellar-Rodriguez, J., et al. (2018). Infections in the cancer patient. In DeVita, V. T., Lawrence, T. S., Rosenberg, S. A. (Eds.). *Cancer: Principles & practice of oncology* (11th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Nurses are in a key position to assist in preventing and identifying symptoms of infection, as discussed in the nursing care plan (see [Chart 12-6](#)). Clinical practice guidelines developed by the ONS, the Infusion Nurses Society (INS), the NCCN, and the ASCO are used to guide prevention and management of infection. Interventions to prevent infection and alternative patient education formats for infection-related instruction are high nursing research priorities.

Gram-positive bacteria (*Streptococcus*, enterococci, and *Staphylococcus* species) and gram-negative organisms (*Escherichia coli*, *Klebsiella pneumoniae*, *Enterobacter*, and *Pseudomonas aeruginosa*) are the most frequently isolated causes of infection. Fungal organisms, such as *Candida albicans*, also contribute to the incidence of serious infection. Viral infections in immunocompromised patients are caused most often by herpes simplex, respiratory syncytial, parainfluenza, and influenza A and B viruses.

Fever is reported promptly as it is an important sign of infection in patients when associated with neutropenia (Olsen et al., 2019). Patients with neutropenic fever (see [Table 12-10](#)) are assessed for infection and reported promptly (NCCN, 2019j). Antibiotics may be prescribed after cultures of wound drainage, exudates, sputum, urine, stool, or blood are obtained. Careful consideration is given to the underlying malignancy, prior antineoplastic treatment, ANC, comorbidities, and other patient-

related factors prior to the identification of the most appropriate antibiotic therapy. Evidence-based guidelines are available for prevention and treatment of cancer-related infections (NCCN, 2019j). Patients with neutropenia are treated with broad-spectrum antibiotics before the infecting organism is identified because of the increased risk of mortality associated with untreated infection. Broad-spectrum antibiotic therapy targets the most likely major pathogenic organisms. It is important for these medications to be given and taken promptly as scheduled to achieve adequate blood levels. Once the offending organism is identified, more specific antimicrobial therapy is prescribed as appropriate. Nurses provide education for patients and families regarding prevention of infection, signs and symptoms to report, and the importance of adherence to prescribed antimicrobial therapy.



Septic Shock

The nurse assesses the patient frequently for signs and symptoms of infection and inflammation throughout the trajectory of cancer care. Sepsis and septic shock are life-threatening complications that must be prevented or detected and treated promptly. Although all patients with cancer are at risk, patients who are neutropenic or who have hematologic malignancies are at the greatest risk. Patients with signs and symptoms of impending sepsis and septic shock require immediate hospitalization and aggressive treatment in the intensive care setting. See [Chapter 11](#) for discussion of sepsis and septic shock.

Bleeding and Thrombocytopenia

Platelets are essential for normal blood clotting and coagulation (hemostasis). **Thrombocytopenia**, a decrease in the circulating platelet count, is the most common cause of bleeding in patients with cancer and is usually defined as a platelet count less than $100,000/\text{mm}^3$ ($0.1 \times 10^{12}/\text{L}$). The risk of bleeding increases when the platelet count decreases below $50,000/\text{mm}^3$ ($0.05 \times 10^{12}/\text{L}$). At platelet counts lower than $10,000/\text{mm}^3$ ($0.02 \times 10^{12}/\text{L}$), the risk for spontaneous bleeding is increased (Olsen et al., 2019).

Thrombocytopenia often results from bone marrow depression after certain types of chemotherapy and radiation therapy and with tumor infiltration of the bone marrow. In some cases, platelet destruction is associated with hypersplenism (enlarged spleen) and abnormal antibody function, which occur with leukemia and lymphoma. Bacterial and viral infections may lead to early platelet destruction or impaired bone marrow production of platelets and subsequent thrombocytopenia. Some medications (e.g., heparin, vancomycin) may cause bone marrow toxicity leading to decreases in circulating platelets. Less commonly, posttransfusion complications may lead to antibody destruction of platelets causing profound thrombocytopenia (Mones & Soff, 2019). The nursing care plan addresses nursing assessment parameters and interventions for patients at risk for bleeding (see [Chart 12-6](#)).



Quality and Safety Nursing Alert

Although laboratory test results confirm the diagnosis of thrombocytopenia, the patient who is developing thrombocytopenia may display early signs and symptoms. Thus, nurses need to observe keenly for petechiae and ecchymoses, which are early indicators of decreasing platelet levels. Early detection promotes early intervention.

Additional medications may be prescribed to address bleeding or thrombocytopenia based on the underlying etiology. See [Chapter 29](#) for further discussion of assessment and treatment of thrombocytopenia and coagulopathies.

VTE, a common problem for patients with cancer, includes deep venous thrombosis (DVT), pulmonary embolism (PE), superficial venous thrombosis (SVT), and thrombosis in other abdominal or thoracic venous tributaries such as the mesenteric veins or the superior vena cava. The incidence of VTE has been linked to an increased likelihood of death in patients with cancer (NCCN, 2019k). Factors associated with risk for VTEs in cancer patients include preexisting underlying or cancer-related coagulopathies, medications including chemotherapy, hospitalizations, surgical procedures, increased age, debilitation, immobility, infection, and peripheral and central venous catheters (NCCN, 2019k). Patients with cancer are monitored for associated risk factors and are assessed on an ongoing basis for VTE. Nurses provide patient and family education regarding symptoms of VTE to report to the provider. Recommendations for prophylaxis are based on national guidelines and patient risk factors. Nursing assessment findings associated with VTE are reported promptly so that VTE evaluation and treatment can be initiated expeditiously.

Promoting Home, Community-Based, and Transitional Care



Educating Patients About Self-Care

Most commonly, patients with cancer are diagnosed and treated in the outpatient setting. Nurses in outpatient settings often have the responsibility for patient education and helping coordinate care in the home (see [Chart 12-10](#)). The shift of care from acute care facilities to the home or outpatient settings as well as the increasing use of oral antineoplastic agents places significant responsibility for care on the patient and family. In order to maintain optimal patient outcomes and quality of life, patients and families require support and information that will prepare them to engage in self-care. Education initially focuses on the most immediate care needs likely to be encountered at home.

Approaches to preparing patients for self-care responsibilities are chosen with consideration of the patient's preferred learning strategies, level of education, and health literacy (Howell, Harth, Brown, et al., 2017). Nurses are in a prime role to

promote self-care by ensuring that the educational needs of patients and families are met across time points of the cancer continuum (Eller, Lev, Yuan, et al., 2018; White, Cohen, Berger, et al., 2017). Easily understood, concrete, objective information that assists patients to understand what to expect and includes sensory and temporal components is important. Symptoms, side effects of treatment, and changes in the patient's status that should be reported are discussed and reinforced with printed materials that can be referred to in the home. Nurses help to empower patients by sharing strategies to manage side effects or other symptoms. Additional learning needs are based on the priorities conveyed by the patient and family as well as on the complexity of care required (Howell et al., 2017).

Chart 12-10



HOME CARE CHECKLIST

The Patient Receiving Care for an Oncologic Disorder

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

- State the impact of cancer treatment on physiologic functioning, ADLs, IADLs, roles, relationships, and spirituality.
- State changes in lifestyle (e.g., diet, activity) necessary to maintain health.
- State the name, dose, side effects, frequency, and schedule for all medications.
- Demonstrate how to administer the chemotherapy agent in the home:
 - Describe safe storage and handling of oral chemotherapy/immunotherapy/targeted therapy agents in the home.
 - Demonstrate safe disposal of needles, syringes, IV supplies, or unused chemotherapy medications.
 - List possible side effects of chemotherapy/immunotherapy/targeted therapy agents and suggested management approaches.
- List possible side effects of radiation therapy and suggested management approaches.
- List complications of medications/therapeutic regimen necessitating a call to the nurse or primary provider.
- List complications of medications/therapeutic regimen necessitating a visit to the emergency department.
- Locate list of names and contact details of resource personnel involved in care (e.g., home health nurse, infusion services, IV vendor, equipment company, radiation therapy department).
- Explain treatment plan and importance of upcoming visits to the primary provider.
- State how to obtain medical supplies after discharge.
- Identify durable medical equipment needs, proper usage, and maintenance necessary for safe utilization.
- Demonstrate usage of adaptive equipment for ADLs.
- Identify community resources for peer and caregiver/family support:
 - Identify sources of support (e.g., friends, relatives, faith community)
 - Identify phone numbers of support groups for people with cancer and their caregivers/families
 - State meeting locations and times
- Identify the need for health promotion, disease prevention, and screening activities

ADL, activities of daily living; IADL, independent activities of daily living.

Technologic advances allow home administration of IV chemotherapy, blood products, and antibiotics; enteral or parenteral nutrition; and parenteral analgesics. Patients and families are taught to care for vascular access devices, infusion pumps, various types of drainage catheters, and on occasion complex wounds. The importance of patient safety and infection control is included in patient and family education. Although nurses are often available to provide some assistance with

cancer care in the home, patients and families need to acquire the requisite knowledge and skills that will enable them to develop a strong sense of self-efficacy to not only foster self-care, but also to promote a more positive health status and better quality of life (Eller et al., 2018; White et al., 2017).

Continuing and Transitional Care

Referral for home or transitional care is often indicated for patients with cancer. The responsibilities of the nurse include assessing the home environment and suggesting modifications in the home or in care to help address the patient's physical and safety needs. Home health nurses also assess the psychosocial impact of cancer on the patient and family so that appropriate interventions can be identified or referrals for support services are instituted.

Ongoing nursing visits or phone contact from the home or transitional care nurse assist in prevention, early identification, prompt reporting, and management of patient problems. Timely modifications in therapy to manage symptoms and adverse effects of treatment may decrease patient suffering and decrease emergency department visits or hospital admissions. Continued contact facilitates evaluation of the patient's progress, responses to treatment, and assessment of the ongoing needs of the patient and family. It is necessary to assess the patient's and family's understanding of the treatment plan and management strategies and to reinforce previous education. The nurse facilitates coordination of patient care by maintaining close communication with all involved health care providers. The nurse may make referrals and coordinate available community resources (e.g., local office of the ACS, home aides, church groups, faith community nurses, support groups) to assist patients and caregivers.



Gerontologic Considerations

Approximately 55% of cancer incidence and 70% of cancer deaths occur in persons aged 65 years or older (ACS, 2019a). The rising number of individuals over age 65 with cancer has led to the emergence of geriatric oncology, a multidimensional and multidisciplinary approach to treating growing numbers of older adults with cancer.

Nurses working with older adults must understand the normal physiologic changes that occur with aging and the implications for the patient with cancer (see [Table 12-11](#)). These changes that affect all body systems may ultimately influence older patients' responses to cancer treatment. In addition, many older patients have other chronic diseases requiring multiple medications. The existence of comorbidities and multiple medications may contribute to drug interactions and toxicities in older patients.

The understanding of the effects and tolerance of chemotherapy, immunotherapy, targeted therapies, and radiation in the older adult is limited, as older adults have been underrepresented in oncology clinical trials. Potential chemotherapy-related toxicities, such as renal impairment, myelosuppression, fatigue, and cardiomyopathy, may increase as a result of declining organ function and diminished physiologic reserves. The recovery of normal tissues after radiation

therapy may be delayed, and older adult patients may experience more severe adverse effects, such as mucositis, nausea and vomiting, and myelosuppression. Because of impaired healing and declining pulmonary and cardiovascular functioning, older patients are slower to recover from surgery. Older patients are also at increased risk for complications, such as atelectasis, pneumonia, and wound infections.

TABLE 12-11  Age-Related Changes and Their Effects on Patients with Cancer

Age-Related Changes	Implications
Impaired immune system	Use special precautions to avoid infection; monitor for atypical signs and symptoms of infection.
Altered drug absorption, distribution, metabolism, and elimination	Mandates careful calculation of chemotherapy and frequent assessment for drug response and side effects; dose adjustments may be necessary.
Increased prevalence of other chronic diseases	Monitor for effect of cancer or its treatment on patient's other chronic diseases; monitor patient's tolerance for cancer treatment; monitor for interactions with medications used to treat chronic diseases.
Diminished renal, respiratory, and cardiac reserve	Be proactive in prevention of decreased renal function, atelectasis, pneumonia, and cardiovascular compromise; monitor for side effects of cancer treatment.
Decreased skin and tissue integrity; reduction in body mass; delayed healing	Prevent pressure ulcers secondary to immobility; monitor skin and mucous membranes for changes related to radiation or chemotherapy; monitor nutritional status.
Decreased musculoskeletal strength	Prevent falls; assess support for performing activities of daily living in home setting; encourage safe use of assistive mobility devices.
Decreased neurosensory functioning: loss of vision, hearing, and distal extremity tactile senses	Provide instruction modified for patient's hearing and vision changes; provide instruction concerning safety and skin care for distal extremities; assess home for safety.
Altered social and economic resources	Assess for financial concerns, living conditions, and resources for support.
Potential changes in cognitive and emotional capacity	Provide education and support modified for patient's level of functioning and safety.

Adapted from Eliopoulos, C. (2018). *Gerontological nursing* (10th ed.). Philadelphia, PA: Wolters Kluwer.

Cancer Survivorship

In the United States, there are currently an estimated 16.9 million adult cancer survivors; by 2030, that number is estimated to be 22.1 million (ACS, 2019f). Advances in cancer screening, treatment, and management of complications have contributed to a lengthened survival period for many, with long-term survival

becoming possible for many patients. *Cancer survivorship* has been defined as the period from cancer diagnosis through the remaining years of life and focuses on the health and life of a person beyond diagnostic and treatment phases. Although individuals vary and many types of cancers and treatments exist, the acute, long-term, and late effects of cancer and its treatment may have multiple long-term physical, cognitive, psychological, social, and financial consequences that can impact activities of daily living, ultimately affecting quality of life (ACS, 2019f).

Survivorship care is often based on expert opinion and experience rather than on evidence-based practices. Knowledge regarding survivorship concerns continues to evolve. The Institute of Medicine has identified four components of survivorship care, the period that follows primary treatment for cancer and lasts until end of life. In addition to a summary of prior diagnosis and treatment, survivorship care includes monitoring and treatment for late effects related to disease and prior treatments, physical and vocational rehabilitation, psychosocial support and counseling as necessary, surveillance and screening for new and recurrent cancer, and coordination between specialists and primary care providers to ensure that all of the survivor's needs are met (Hewitt, Greenfield, & Stovall, 2006) (see [Table 12-12](#)). Advocacy organizations across the country have recommended that a survivorship care plan be provided to all patients with cancer and their primary provider at the completion of treatment. The survivorship care plan includes a summary of cancer diagnosis, treatment, recommendations for follow-up care, including approaches to treat symptoms, rehabilitative needs, monitoring for late effects, and surveillance and screening for new and recurrent cancer. Referrals for specific services such as lymphedema therapy, chronic pain management, and genetic counseling are also provided. Nurses assist in the development of the survivorship care plan and provide education and care for cancer survivors. Nurses, other health care providers, public health professionals, and patient advocates design and conduct research to identify needs of cancer survivors and evidence-based approaches to care.

TABLE 12-12 Components of Survivorship Care

Component	Examples of Care
Prevention and detection of new and recurrent cancer	Mammography (per ACS guidelines) Papanicolaou (Pap) smears (per ACS guidelines) Smoking cessation programs Nutrition counseling
Surveillance for cancer spread, recurrence, or second cancers	Colonoscopy post colon cancer Mammography post breast cancer Liver function tests post colon cancer Prostate-specific antigen post prostate cancer
Intervention for consequences of cancer and its treatments	Lymphedema therapy Pain management Enterostomal therapy Fertility care Psychosocial support or counseling Reconstructive surgery
Coordination between specialists and primary providers to meet health needs	Care for comorbidities (e.g., diabetes) Influenza vaccination Bone densitometry Monitoring for chemotherapy induced cardiotoxicity

ACS, American Cancer Society.

Adapted from Hewitt, M., Greenfield, S., & Stovall, E. (Eds.). (2006). *From cancer patient to cancer survivor: Lost in translation*. Washington, DC: Institute of Medicine and National Research Council, National Academies Press. Components of survivorship care provided by the Institute of Medicine report on cancer survivorship.

Providing Care to the Patient with Advanced Cancer

The patient with advanced cancer needs to be monitored for oncologic emergencies and have appropriate treatment should emergencies occur. These patients may have end-of-life and palliative care needs that must be addressed.

Providing Care in Oncologic Emergencies

Table 12-13 discusses select nursing and medical care of oncologic emergencies.

As a result of advances in all aspects of cancer care, it is more common that individuals are living with cancer that has advanced beyond the original site of development to regional or distant sites. Patients with advanced cancer are likely to experience many of the problems described previously, although more often and to a greater degree. Symptoms of pain, anorexia, weight loss, CACS, fatigue, and impaired functional status and mobility make patients more susceptible to depressive symptoms, skin breakdown, fluid and electrolyte imbalances, and infection.

Treatment for the patient with advanced cancer is likely to be palliative rather than curative, with an emphasis on prevention and appropriate management of pain.

The use of long-acting analgesic agents at set intervals, rather than on an “as needed” basis, is recommended in addressing pain management. Working with the patient and family, as well as with other health care providers, to manage pain is essential to increase the patient’s comfort and offer some sense of control. Other medications (e.g., sedatives, tranquilizers, muscle relaxants, antiemetics) are added to assist in palliating additional symptoms and promoting quality of life.

TABLE 12-13 Oncologic Emergencies: Manifestations and Management

Oncologic Emergency	Clinical Manifestations and Diagnostic Findings	Medical and Nursing Management
<p>Superior Vena Cava Syndrome (SVCS)</p> <p>Compression or invasion of the superior vena cava by tumor; enlarged lymph nodes; intraluminal thrombus that obstructs venous circulation; or drainage of the head, neck, arms, and thorax. Most often associated with lung cancer, SVCS is also associated with lymphoma, thymoma, and testicular cancers and mediastinal metastases from breast cancer. If untreated, SVCS may lead to cerebral anoxia (because not enough oxygen reaches the brain), laryngeal edema, bronchial obstruction, and death.</p>	<p>Clinical Manifestations:</p> <p>Gradually or suddenly impaired venous drainage giving rise to:</p> <ul style="list-style-type: none"> progressive dyspnea (shortness of breath), cough, hoarseness, chest pain, and facial swelling Edema of the neck, arms, hands, and thorax and reported sensation of skin tightness, difficulty swallowing, and stridor. Possibly engorged and distended jugular, temporal, and arm veins. Dilated thoracic vessels causing prominent venous patterns on the chest wall. Increased intracranial pressure, associated visual disturbances, headache, and altered mental status <p>Diagnostic Findings:</p> <p>Diagnosis confirmed by:</p> <ul style="list-style-type: none"> Clinical findings Chest x-ray Thoracic CT scan Thoracic MRI 	<p>Medical Management:</p> <p>Radiation therapy to shrink tumor or enlarged lymph nodes and relieve symptoms.</p> <p>Chemotherapy for sensitive cancers (e.g., lymphoma, small cell lung cancer) or when the mediastinum has been irradiated to maximum tolerance.</p> <p>Anticoagulant or thrombolytic therapy for central venous catheter-related intraluminal thrombosis.</p> <p>Percutaneously placed intravascular stents may be priority consideration rather than surgery unless symptoms are rapidly progressing.</p> <p>Supportive measures such as oxygen therapy, corticosteroids, and diuretics (in cases of fluid overload).</p> <p>Nursing Management:</p> <p>Identify patients at risk for SVCS.</p> <p>Provide patient and family education regarding signs and symptoms to report.</p> <p>Monitor and report clinical manifestations of SVCS.</p> <p>Monitor cardiopulmonary and neurologic status.</p>

	Venogram if intraluminal thrombosis is suspected	Avoid upper extremity venipuncture and blood pressure measurement; instruct patient to avoid tight or restrictive clothing and jewelry on fingers, wrist, and neck. Facilitate breathing and drainage from upper portion of body by instructing patient to maintain some elevation of head and upper body with semi-Fowler position; avoid completely supine or prone position (this helps to promote comfort and reduce anxiety associated with dependent and progressive edema). Promote energy conservation to minimize dyspnea. Monitor the patient's fluid volume status; administer fluids cautiously to minimize edema. Assess for thoracic radiation-related problems such as mucositis with resultant dysphagia (difficulty swallowing) and esophagitis. Monitor for chemotherapy-related problems, such as myelosuppression. Provide postoperative care as appropriate.
Spinal Cord Compression	Clinical Manifestations: Local inflammation, edema, venous stasis, and impaired blood supply to nerve tissues. Local or radicular back or neck pain along the dermatomal areas innervated by the affected nerve root (e.g., thoracic radicular pain extends in a band around the chest or abdomen). Pain exacerbated by movement, supine recumbent position, coughing, sneezing, or the	Medical Management: Radiation therapy to reduce tumor size and halt progression; corticosteroid therapy to decrease inflammation and swelling at the compression site. Surgery to debulk tumor and stabilize the spine if symptoms progress despite radiation therapy or if vertebral fracture or bone fragments lead to additional nerve damage; surgery is also an option when the tumor is not radiosensitive or is located in an area that was previously irradiated. Minimally invasive surgical procedures, referred to as vertebral augmentation, may be used for patients with vertebral fractures to attain stability of the bone, prevent nerve compression, and decrease pain. Procedures include: Vertebroplasty: involves percutaneous injection of polymethyl methacrylate (PMMA), a bone cement filler, into the vertebral body. Kyphoplasty: a balloon is inserted into the damaged vertebral body and then inflated to create a cavity within the bone that can be filled with bone cement. The balloon
Most commonly caused by compression of the cord and its nerve roots by a metastatic paravertebral tumor that extends into the epidural space; vertebral bone metastasis leading to bone collapse and displacement impinging on the spinal cord or nerve roots; and less commonly, primary malignancy of the cord. May potentially lead to significant and permanent neurologic impairment associated with multiple physical, psychosocial consequences.		
Most often associated with cancers that metastasize to the bone such as breast, lung, and prostate cancers and lymphoma. Also seen in nasopharyngeal cancer and multiple myeloma.		

<p>About 60% of spinal cord compressions occur at the thoracic level, 30% in the lumbosacral level, and 10% in the cervical and sacral regions. The prognosis depends on the severity and rapidity of onset.</p>	<p>Valsalva maneuver. Neurologic dysfunction and related motor and sensory deficits (numbness, tingling, feelings of coldness in the affected area, inability to detect vibration, loss of positional sense). Motor loss ranging from subtle weakness to flaccid paralysis. Bladder or bowel dysfunction depending on level of compression (above S2, overflow incontinence; from S3 to S5, flaccid sphincter tone and bowel incontinence).</p>	<p>helps to compress the fracture fragments together as the cavity is created.</p>
<p>Diagnostic Findings: Percussion tenderness at the level of compression; abnormal reflexes; and sensory and motor abnormalities. MRI is a preferred diagnostic tool; may also utilize x-rays, bone scans, and CT scan.</p>	<p>Radiofrequency vertebral augmentation: similar to kyphoplasty; instead of the balloon, a small navigational cannula is inserted into the vertebra to create small pathways for cement. The cement is heated with radiofrequency to create an ultra-high viscosity that is thought to promote bone stability. Chemotherapy as adjuvant to other local therapies for patients with chemosensitive cancers, such as lymphoma or small cell lung cancer.</p>	<p><i>Note:</i> Despite treatment, patients with poor neurologic function before treatment are less likely to regain complete motor and sensory function; patients who develop complete paralysis usually do not regain all neurologic function.</p>
	<p>Nursing Management: Perform ongoing assessment of neurologic function to identify existing and progressing dysfunction. Control pain with pharmacologic and nonpharmacologic measures.</p>	

		<p>Prevent complications of immobility resulting from pain and decreased function (e.g., skin breakdown, urinary stasis, thrombophlebitis, decreased clearance of pulmonary secretions).</p> <p>Maintain muscle tone by assisting with range-of-motion exercises in collaboration with physical and occupational therapists; patients with unstable vertebral fractures do not initiate physical therapy until spine stabilization procedures have been completed.</p> <p>Institute intermittent urinary catheterization and bowel training programs for patients with bladder or bowel dysfunction.</p> <p>Provide encouragement and support to the patient and family coping with pain and altered functioning, lifestyle, roles, and independence.</p> <p>Institute appropriate referrals for home care and physical and occupational therapy.</p> <p>Provide patient and family education about pharmacologic and nonpharmacologic interventions</p>
Hypercalcemia	<p>Clinical Manifestations:</p> <p>Fatigue, weakness, confusion, decreased level of responsiveness, hyporeflexia, nausea, vomiting, constipation, ileus, polyuria (excessive urination), polydipsia (excessive thirst), dehydration, and arrhythmias.</p> <p>Diagnostic Findings:</p> <p>Total serum calcium level >10.4 mg/dL (2.6 mmol/L)</p> <p>Ionized serum calcium >1.29 mmol/L</p>	<p>Medical Management:</p> <p>Identify patients at risk for hypercalcemia and assess for signs and symptoms of hypercalcemia. Treatment of the underlying malignancy (e.g., chemotherapy, radiation therapy, hormone therapy, immunotherapy or targeted therapy). Reduce serum calcium levels: Oral hydration (3–4 L of fluid daily unless contraindicated by existing renal or cardiac disease) or IV hydration followed by diuretics (forced diuresis). Avoid dietary supplements and medications that can increase serum calcium levels (e.g., thiazide diuretics, nonsteroidal anti-inflammatory drugs; and vitamins A and D, and calcium supplements). Bisphosphonate therapy may be indicated for the long-term management. Maintenance of nutritional intake without restricting normal calcium intake. Dietary and pharmacologic interventions such as stool softeners and laxatives for constipation. Antiemetic therapy for nausea and vomiting.</p> <p>Nursing Management:</p> <p>Monitor closely for changes in mental status; fluid and electrolyte imbalances;</p>

			and renal, gastrointestinal, cardiac dysfunction. Monitor treatment effectiveness and for the presence of side effects. Ensure proper hydration and monitor fluid and electrolyte balance closely. Educate patients who are at-risk and their families to recognize and report signs and symptoms of hypercalcemia. Encourage mobilization / weight-bearing activities, as tolerated, to limit bone resorption. Maintain patient safety and ensure comfort. Educate patient and families about prescribed management strategies.
Tumor Lysis Syndrome (TLS)	<p>Clinical Manifestations:</p> <p>Potentially fatal complication that occurs spontaneously or more commonly following radiation, immunotherapy, targeted therapy, or chemotherapy-induced cell destruction of large or rapidly growing cancers such as leukemia, lymphoma, and small cell lung cancer. The release of tumor intracellular contents (nuclei acids, electrolytes, and debris) leads to rapidly induced electrolyte imbalances—hyperkalemia, hyperphosphatemia (leading to hypocalcemia), and hyperuricemia—that can have life-threatening end-organ effects on the myocardium, kidneys, and central nervous system.</p> <p>Nervous System: Clinical manifestations depend on the extent of metabolic abnormalities.</p> <p>Neurologic: Fatigue, weakness, memory loss, altered mental status, muscle cramps, tetany, paresthesias (numbness and tingling), and seizures.</p> <p>Cardiac: Elevated blood pressure, shortened QT complexes, widened QRS waves, altered T waves, arrhythmias, and cardiac arrest.</p> <p>Gastrointestinal: Anorexia, nausea, vomiting, abdominal cramps, diarrhea, and increased bowel sounds.</p> <p>Renal: Flank pain, oliguria, anuria, kidney injury, and acidic urine pH.</p>	<p>Medical Management:</p> <p>To prevent kidney injury and restore electrolyte balance. Aggressive fluid hydration is initiated 24–48 h before and after the initiation of cytotoxic therapy to increase urine volume and eliminate uric acid and electrolytes.</p> <p>Diuresis with a loop diuretic or osmotic diuretic, if urine output is inadequate.</p> <p>Allopurinol therapy to inhibit the conversion of nucleic acids to uric acid (oral or IV). Rasburicase may be used to convert already formed uric acid to allantoin, which is highly water soluble and eliminated in urine.</p> <p>Administration of a cation-exchange resin, such as sodium polystyrene sulfonate to treat hyperkalemia by binding and eliminating potassium through the bowel.</p> <p>Administration of IV sodium bicarbonate, hypertonic dextrose, and regular insulin temporarily shifts potassium into cells and lowers serum potassium levels if a rapid decrease in potassium is necessary.</p> <p>Administration of phosphate-binding gels, such as aluminum hydroxide, to treat hyperphosphatemia by promoting phosphate excretion in the feces.</p> <p>Hemodialysis when patients are unresponsive to the standard approaches for managing uric acid and electrolyte abnormalities.</p> <p>Identify at-risk patients.</p> <p>Institute essential preventive measures (e.g., fluid hydration, medications) as prescribed.</p> <p>Assess patient for signs and symptoms of electrolyte imbalances.</p>	

<p><i>Other:</i> Gout, malaise, and pruritis.</p> <p>Diagnostic Findings:</p> <p>Electrolyte imbalances identified by serum electrolyte measurement and urinalysis (see Chapter 10); electrocardiogram to detect cardiac arrhythmias.</p> <p>Clinical TLS is diagnosed when ≥ 1 of 3 conditions arise either 3 days prior to or 7 days after cytotoxic cancer therapy:</p> <ul style="list-style-type: none"> acute kidney injury (defined as a rise in creatinine to ≥ 1.5 times the upper limit of normal that is not attributable to medications, arrhythmias, and seizures). 	<p>Nursing Management:</p> <p>Monitor for signs and symptoms of TLS in patients who are at-risk (e.g., those with hematologic malignancies).</p> <p>Monitor vital signs, laboratory values (i.e., electrolyte), fluid balance closely (i.e., daily weights, intakes and outputs), and cardiac status closely.</p> <p>Educate patients and families about risk factors for TLS; teach about current treatments (e.g., hydration, dietary restrictions [e.g., potassium and phosphorous], and pharmacologic therapy); signs and symptoms of complications; and when to report symptoms indicating electrolyte disturbances to the provider.</p>
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CT, computed tomography; IV, intravenous; MRI, magnetic resonance imaging.

Adapted from Kaplan, M. (Ed.). (2018). *Understanding and managing oncologic emergencies: A resource for nurses*. Pittsburgh, PA: Oncology Nursing Society; Rimmer, A., & Yahalom, J. (2018). Superior vena cava syndrome. In DeVita, V. T., Lawrence, T. S., Rosenberg, S. A. (Eds.). *Cancer: Principles & practice of oncology* (11th ed.). Philadelphia, PA: Lippincott Williams & Wilkins; Stein, S., & Deshpande, H. A. (2018). Metabolic emergencies. In DeVita, V. T., Lawrence, T. S., Rosenberg, S. A. (Eds.). *Cancer: Principles & practice of oncology* (11th ed.). Philadelphia, PA: Lippincott Williams & Wilkins; and Szerlip, N., Beeler, W. H., & Spratt, D. E. (2018). Spinal cord compression. In DeVita, V.T., Lawrence, T. S., Rosenberg, S. A. (Eds.). *Cancer: Principles & practice of oncology* (11th ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

If the patient is a candidate for radiation therapy or surgical interventions to relieve pain or other symptoms, the potential benefits and risks of these procedures (e.g., percutaneous nerve block, cordotomy) are explained to the patient and family. Measures are taken to prevent complications that result from altered sensation, immobility, and changes in bowel and bladder function.

Weakness, altered mobility, fatigue, and inactivity typically increase with advanced cancer as a result of the disease, treatment, inadequate nutritional intake,

or dyspnea. The nurse works with the patient and family to identify realistic goals and promote comfort. Measures include use of energy conservation methods to accomplish tasks and activities that the patient values most.

Efforts are made to provide the patient with as much control and independence as desired but with assurance that support and assistance are available when needed. In addition, health care teams work with the patient and family to ascertain and comply with the patient's wishes about treatment methods and care as the terminal phase of illness and death approach.

Hospice

The needs of patients with end-stage illness are best met by a comprehensive interdisciplinary specialty program that focuses on quality of life; palliation of symptoms; and provision of physical, psychosocial, and spiritual support for patients and families when cure and control of the disease are no longer possible. The concept of hospice best addresses these needs.

Hospice care is often delivered through coordination of specialty services provided by hospitals, home care programs, and the community. Patients need to be referred to hospice services in a timely fashion so that complex patient and family needs can be addressed. See [Chapter 13](#) for detailed discussion of end-of-life care.

CRITICAL THINKING EXERCISES

1  A 38-year-old man with early-onset colorectal cancer completed his first cycle of chemotherapy 2 days ago. He is now being admitted to the inpatient oncology unit with febrile neutropenia. What interventions and education should be considered for febrile neutropenia? What is the evidence for these interventions and education that you identified for febrile neutropenia? How strong is that evidence, and what criteria will you use to assess the strength of that evidence?

2  A 76-year-old woman, with a past medical history that is significant for cataracts, arthritis, and hypertension, was recently diagnosed with stage II breast cancer. She is being discharged today from the hospital following a bilateral mastectomy with several surgical drains in place. What are the priorities for the assessment of this patient to ensure her readiness for discharge to home? What will be your priorities when educating this patient for self-care?

3  You are caring for a 53-year-old man with diffuse large B-cell lymphoma who received CAR T-cell therapy 2 days ago. How does CAR T-cell therapy differ from conventional chemotherapy? What type of referrals might be appropriate for this patient? What members of the interprofessional health care team do you anticipate as being integral to the care of this patient?

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*Asterisk indicates nursing research.

**Double asterisk indicates classic reference.

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Resources

- American Association of Cancer Research (AACR), www.aacr.org
- American Cancer Society (ACS), www.cancer.org
- American College of Surgeons Commission on Cancer (CoC), www.facs.org/quality-programs/cancer/coc
- American Pain Society (APS), www.ampainsoc.org
- American Society of Clinical Oncology (ASCO), www.asco.org
- American Society for Radiation Oncology (ASTRO), www.astro.org
- Association of Oncology Social Work (AOSW), www.aosw.org
- Cancer Care, www.cancercare.org/
- Centers for Disease Control and Prevention (CDC): Cancer Control and Prevention, www.cdc.gov/cancer/
- Hospice and Palliative Nurses Association (HPNA), www.hpna.org
- LIVESTRONG Survivorship Centers of Excellence, www.livestrong.org
- National Cancer Institute (NCI), www.cancer.gov
- National Coalition for Cancer Survivorship, www.canceradvocacy.org
- National Comprehensive Cancer Network (NCCN), www.nccn.org
- National Hospice and Palliative Care Organization, www.nhpco.org
- National Institutes of Health, National Center for Complementary and Integrative Health (NCCIH), nccih.nih.gov
- OncoLink (cancer resources), www.oncolink.org
- Oncology Nursing Society (ONS), www.ons.org
- Quackwatch, www.quackwatch.org

The Bone Marrow Foundation, bonemarrow.org/
The Cancer Support Community, www.cancersupportcommunity.org
The COVID-19 and Cancer Consortium, ccc19.org
The Leukemia and Lymphoma Society, www.lls.org

13 Palliative and End-of-Life Care

LEARNING OUTCOMES

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

1. Compare and contrast the settings where palliative care and end-of-life care are provided.
2. Describe the principles and components of hospice care in the United States, including the Medicare hospice benefit.
3. Apply skills for communicating with patients who are seriously ill and their families in order to provide culturally and spiritually sensitive care.
4. Identify components of uncomplicated grief and mourning and implement nursing measures to support patients and families.

NURSING CONCEPTS

Assessment
Comfort
End-of-Life
Ethics
Grief and Loss
Palliative Care

GLOSSARY

autonomy: self-determination; in the health care context, the right of the person to make choices about the use and discontinuation of medical interventions

bereavement: period during which mourning for a loss takes place

grief: personal feelings that accompany an anticipated or actual loss

hospice: a coordinated program of interdisciplinary care and services for terminally ill patients and their families

interdisciplinary collaboration: communication and cooperation among members of diverse health care disciplines jointly to plan, implement, and evaluate care

Medicare Hospice Benefit: a Medicare Part A entitlement that provides for comprehensive, interdisciplinary palliative care and services for eligible beneficiaries who have a terminal illness and a life expectancy of less than 6 months

mourning: individual, family, group, and cultural expressions of grief and associated behaviors

palliative care: a patient/person and family-centered approach to care that attends to the physical, functional, psychological, spiritual, and existential aspects of a serious illness

palliative sedation: controlled and monitored use of certain sedatives and nonopioid medications with the intention of reducing the patient's level of consciousness for the relief of refractory pain and symptoms that have not responded to other management measures; the purpose is not to hasten the patient's death but to relieve intractable symptoms

prognosis: the expected course of an illness and the chance for recovery

spirituality: personal belief systems that focus on a search for meaning and purpose in life, intangible elements that impart meaning and vitality to life, and a connectedness to a higher or transcendent dimension

terminal illness: progressive, irreversible illness that despite cure-focused medical treatment will result in the patient's death

Nurses have a significant and lasting effect on the way patients live until they die, the manner in which death occurs, and the enduring memories of that death for patients' families. The contemporary definition of nursing includes "...the diagnosis and treatment of human responses, and advocacy in the care of individuals, families, groups, communities, and populations" (American Nurses Association [ANA], 2015, p. 1). There may be no group more important than seriously ill and dying patients.

Knowledge about palliative and end-of-life principles of care and the ability to recognize the unique response of each patient and family to a given illness

are essential components required to support the unique values, preferences, and goals of a person's care. Nurses have an opportunity to bring research, education, and practice together to impact the culture of dying, allowing for much-needed improvement to care that is relevant across practice settings, age groups, cultural backgrounds, and illnesses. Nurses in all settings are likely to encounter patients who are seriously ill and families who can benefit from palliative care during advanced illness and at end-of-life. This chapter presents concepts about death and dying in the United States, settings for end-of-life care of the dying, and ways that nurses can address the health issues of patients who are terminally ill.

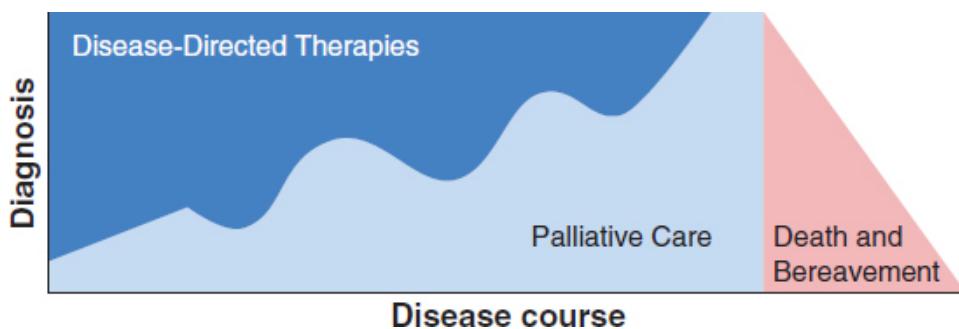


Figure 13-1 • This model shows the intersection of disease-directed therapies and palliative care beginning at diagnosis and continuing to death and bereavement. Adapted from Ferrell, B. R., Twaddle, M. L., Melnick., A., et al. (2018). National consensus project clinical practice guidelines for quality palliative care guidelines, 4th edition. *Journal of Palliative Medicine*, 21(12), 1684–1689.

Palliative Care and End-of-Life Care

Palliative care uses an interdisciplinary model of care, focusing on symptom management and psychosocial/spiritual support for those with serious, life-limiting illnesses. Palliative care aims to improve quality of life for people and families through early integration into the plan of care strategies for managing pain and symptoms and for reducing burdensome care transitions through interdisciplinary teamwork, care coordination, clinician-patient communication, and decisional support. It is appropriate for patients at any age and at any stage in a serious illness, even while pursuing disease-directed or curative therapies, and extending into bereavement for families (Fig. 13-1). Palliative care can be viewed as both an approach to care and as a structured system for care delivery that aims to optimize quality of life by anticipating, preventing, and treating suffering (National Consensus Project for Quality Palliative Care [NCP], 2018). **Interdisciplinary collaboration** is an essential

component which is rooted in communication and cooperation among the various disciplines, with each member of the team contributing to a single integrated care plan that addresses the needs of the patient and the family. In contrast, multidisciplinary care refers to participation of clinicians with varied backgrounds and skill sets but without coordination and integration of care into a unified plan.

Hospice is a type of palliative care, focusing on comfort at the end-of-life. When patients enroll in hospice, they have made the decision to forego disease-directed therapies and focus solely on the relief of symptoms associated with their illness and the dying process. Both palliative care and hospice clinicians are skilled in the delivery of end-of-life care (which may include the months or weeks prior to death), expert pain and symptom management, life review and bereavement support. Focus on care of the dying is motivated by the aging of the population, the prevalence of and publicity surrounding life-threatening illnesses, and the increasing likelihood of a prolonged period of chronic illness prior to death. [Figure 13-2](#) depicts four illness trajectories: sudden death, terminal illness, organ failure, and frailty. Sudden death is unexpected and accounts for the minority of deaths in this day and age. The other trajectories depict a common course in three types of chronic life-limiting illness: (1) terminal illness (e.g., cancer) is generally diagnosed when one is highly functioning, with a short steady decline before death; (2) organ failure (e.g., heart failure, end-stage kidney disease, or chronic lung disease) usually follows a slow decline after diagnosis, with episodic illnesses, perhaps exacerbation or hospitalization, where one has difficulty returning back to functional baseline; and (3) frailty (e.g., dementia) is typically diagnosed when one is already frail, with a slow decline over years. With the exception of sudden death, functional status can be a good prognostic indication; one such scale, the Palliative Performance Scale, is based on ambulation, activity, outward evidence of disease, self-care, intake, and level of consciousness (10-point increments, with 100% being fully functional and independent) (Hui, Park, Liu, et al., 2016).

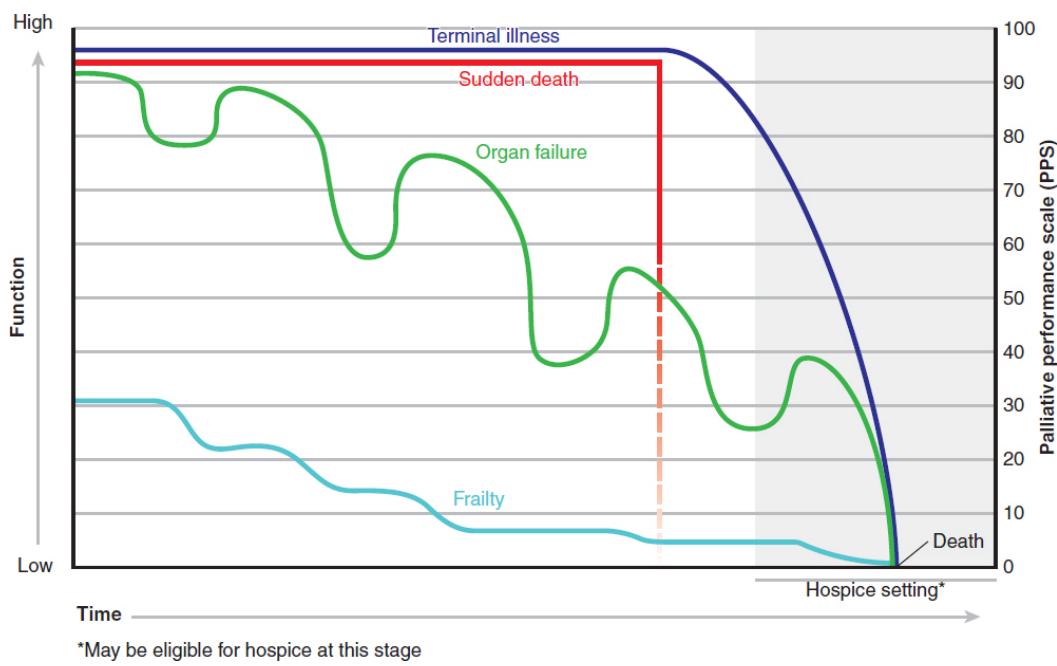


Figure 13-2 • The four trajectories of illness.

The Evolution of End-of-Life Care

In the 20th century, chronic, degenerative diseases replaced communicable diseases as the major causes of death. In the earlier part of the 20th century, most deaths occurred at home and most families had direct experience with death, through providing care to family members at the end-of-life and then mourning their losses together as a family unit. As the place of death shifted from home to hospitals, families became increasingly distanced from the death experience, placing the care of the patient into the health care provider's hands.

In the latter half of the 20th century, a technologic imperative practice pattern among health care professionals emerged, along with an expectation among patients and families that every available means to extend life must be tried. In the 21st century, technologic intervention at the end-of-life continues to have profound implications, affecting how clinicians care for the dying, how family and friends participate in care, how patients and families understand and choose among end-of-life care options, how families prepare for **terminal illness** (a progressive, irreversible illness that despite medical treatment will result in the patient's death) and death, and how they heal after the death of a loved one. Palliative and end-of-life care continues to evolve with the technology and medical interventions that are used to care for the seriously ill.

Between 2000 and 2050, the number of people older than 85 years is expected to quadruple (West, Cole, Goodkind, et al., 2014). More than half of the aged population has one or more chronic, serious illness (West et al.,

2014). The incidence of life-limiting illness is expected to continue to rise. Numerous initiatives aimed at improving end-of-life care have been launched in recent years, spurred by a widespread call for substantive change in the way Americans deal with death. In 2014, the Institute of Medicine (IOM) released a consensus report titled *Dying in America: Improving Quality and Honoring Individual Preferences Near the End of Life*. The report presented a summary of findings from a review of the evidence and provided recommendations for improving palliative and end-of-life care. The authors noted that patients experienced many transitions in settings, frequent and potentially avoidable hospital readmission, and inconsistent referral to palliative care. Recommendations include widespread and timely access to, and comprehensive coverage for palliative care services, improved clinician–patient communication, greater emphasis on advance care planning, professional education and development, and stronger public education and engagement (IOM, 2014).

The NCP identified eight key domains that underline a more comprehensive and humane approach to the care of the seriously ill patient at any stage, age, setting or prognosis, with the most recent update released in 2018 and outlined in [Chart 13-1](#) (NCP, 2018). These practice guidelines have been endorsed by more than 80 national organizations, such as the Institute for Healthcare Improvement (IHI), American Cancer Society (ACS), Hospice and Palliative Nurses Association (HPNA), and the National Hospice and Palliative Care Organization (NHPCO), to structure and evaluate quality palliative and end-of-life programs for all those with serious illness at any stage, age, setting or prognosis.

In 2017, the ANA in collaboration with HPNA developed a *Call for Action—Nurses Lead and Transform Palliative Care*, which outlined the five areas of palliative nursing care: clinical practice, education, policy, research, and administration. In the document, the ANA and HPNA outline ways that nurses can transform palliative care, both as primary and specialty palliative care clinicians (ANA, 2017a). Primary palliative care is defined as the fundamental palliative care skills that all health care providers should have, including basic symptom assessment and management and the ability to explore goals of care through therapeutic communication. In contrast, the palliative care specialist addresses complex symptoms and navigates difficult goals of care conversations and family conflict. As a measure to better incorporate primary palliative care into nursing education, the American Association of Colleges of Nursing (AACN) recommended a new curriculum for palliative education for undergraduate nurses, including developing an online version of the End-of-Life Nursing Education Consortium (ELNEC) (Ferrell, Malloy, Mazanec, et al., 2016). See Resources section at end of chapter.

Settings for Palliative Care and End-of-Life Care

As the landscape of care at the end-of-life moved from the home to a more institutionalized death, the models of palliative care delivery changed as well. At hospice's inception, end-of-life care was home based or at a free-standing hospice. Palliative care was largely hospital based; it was born out of academic medical centers, where some health care providers felt that everyone deserves to receive treatment based on the tenets of hospice care throughout the progression of serious illness (Morrison, 2013). Current models for palliative and end-of-life care delivery will be described in greater detail in the following sections and include (NCP, 2018):

- Institution-based palliative care programs (e.g., programs within hospitals or long-term care facilities)
- Outpatient-based palliative care programs (e.g., outpatient clinics, ambulatory settings)
- Community-based palliative care program (consultative teams collaborate with hospice or home health agencies, to support patients not yet receiving hospice care in their place of residence)
- Hospice care (under Medicare Part A)

As our population ages and is expected to develop multiple comorbidities, the need for a palliative care workforce also grows. Yet, experts project a workforce shortage of all palliative clinicians that may not recover for nearly three decades. Proposed policy changes include (Kamal, Wolf, Troy, et al., 2019):

Chart 13-1

Overview of National Consensus Project Guidelines, Fourth Edition

Domain 1: Structure and Processes of Care

- **Overview:** Palliative care is based on a comprehensive interdisciplinary assessment of the patient and the family, by all clinicians with assistance from palliative care specialists as needed.
- **Clinical Implications:** Palliative care may improve quality of life for patients and families when well-trained interdisciplinary clinicians create care plans based on patient-stated values and preferences.

Domain 2: Physical Aspects of Care

- **Overview:** Pain, other symptoms, and side effects are managed based on the best available evidence, with attention to disease-specific pain and symptoms, which is skillfully and systematically applied.
- **Clinical Implications:** Interdisciplinary teams treat physical symptoms through recognition that physical symptoms impact a patient's emotional and spiritual well-being. Clinicians also consider goals, cultural, and developmental needs when choosing pharmacologic and nonpharmacologic treatments.

Domain 3: Psychological and Psychiatric Aspects of Care

- **Overview:** Psychological status, including need for assistance with distress, coping, family conflict, grief support and resources is assessed and managed based on the best available evidence, which is skillfully and systematically applied. When necessary, psychiatric issues are addressed and treated.
- **Clinical Implications:** Initial assessment is usually performed by social workers with assistance from psychologists and psychiatrists as needed.

Domain 4: Social Aspects of Care

- **Overview:** Comprehensive interdisciplinary assessment identifies the environmental and social factors that may impact the patient's and family members' quality of life, and a plan of care is developed to respond to these needs as effectively as possible.
- **Clinical Implications:** Coordination of care to address social and environmental vulnerabilities.

Domain 5: Spiritual, Religious, and Existential Aspects of Care

- **Overview:** Spiritual and existential dimensions are assessed and responded to based on the best available evidence, which is skillfully and systematically applied.
- **Clinical Implications:** The interdisciplinary team assesses each patient's sources of meaning and purpose, while respecting patient-specific beliefs, values, and traditions.

Domain 6: Cultural Aspects of Care

- **Overview:** Palliative care team members assess and attempt to meet the needs of the patient, family, and community in a culturally sensitive manner.
- **Clinical Implications:** Health care providers must examine their own biases to work to avoid judgment and provide culturally sensitive care.

Domain 7: Care of the Patient Nearing the End-of-Life

- **Overview:** Care is provided by interdisciplinary team members skilled in end-of-life care domains including expert symptom management, skilled communication, and grief/bereavement support.
- **Clinical Implications:** Signs and symptoms of impending death are recognized and communicated in developmentally appropriate language for the patient, family, and children, if applicable, with respect to information preferences.

Domain 8: Ethical and Legal Aspects of Care

- **Overview:** The patient's goals, preferences, and choices are respected within the limits of applicable state and federal law, within current accepted standards of medical care, and form the basis for the plan of care.
- **Clinical Implications:** Knowledge of ethical principles (beneficence, nonmaleficence, justice, self-determination) as well as pertinent health care laws is required. Clinicians must determine moral agency and understand when to engage surrogate decision makers to employ substituted judgment.

Adapted from Ferrell, B. R., Twaddle, M. L., Melnick., A., et al. (2018). National consensus project clinical practice guidelines for quality palliative care guidelines, 4th edition. *Journal of Palliative Medicine*, 21(12), 1684–1689.

- The adoption of the Palliative Care and Hospice Education and Training Act (PCHETA), which if passed would increase the field by over 9000 practitioners.
- Policy to financially support palliative fellowship training across disciplines.
- Further research defining nonphysician palliative care specialist clinicians (nurses, social workers, chaplaincy).
- Creative reimbursement models that support the full interdisciplinary team.
- Policy to promote resiliency and, in turn, prevent burnout. (For more on burnout see later section entitled “Professional Caregiver Issues.”)

Institution-Based Palliative Care

The landmark Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments (SUPPORT) documented troubling deficiencies in the care of the dying in hospital settings (SUPPORT Principal Investigators, 1995). Subsequently, the IOM's *Dying in America* (2014), the NCP *Clinical Practice Guidelines* (2018), and the ANA-HPNA *Call for Action* (2017a) have been shaping palliative and end-of-life care through developing and encouraging adherence to standards of care for patients with advanced illness and at end-of-life that will apply regardless of where the patients are receiving care (ANA, 2017a; IOM, 2014; NCP, 2018). There has been a steady increase in American hospital-based palliative care, with reportedly 90% of hospitals with 300 beds or more offering palliative care, compared to 56% of hospitals with fewer than 300 beds (Dumanovsky, Augustin, Rogers, et al., 2016).

In the hospital, the delivery of palliative care is typically through an interdisciplinary consultation service where primary teams consult specialists for one or more of the following reasons:

- Pain management
- Symptom management
- Goals of care discussions
- End-of-life issues
- Psychosocial distress
- Spiritual or existential distress

The core interdisciplinary team usually consists of physicians, nurses, advanced practice nurses, social workers, and chaplains (National Palliative Care Registry, 2017). Additional team members may include pharmacists, nutritionists, music or art therapists, ethicists, and psychologists. Occasionally, institutions have palliative care units where palliative care clinicians oversee a patient's care when palliative care needs supersede other hospital conditions. The most common reason for palliative care consultation in the hospital setting is goals of care discussion; those referred for care planning are most often older with a serious illness other than cancer and typically have a full code status (Bischoff, O'Riordan, Marks, et al., 2017).

In 2011, The Joint Commission launched an advanced certification program for palliative care to recognize hospitals that provide exceptional patient and family-centered care (The Joint Commission, 2018). The Center to Advance Palliative Care (CAPC) has a plethora of institutional resources for developing hospital–hospice partnerships to provide high-quality palliative care for hospitalized patients and for addressing the palliative care needs of other specialized populations. See Resources section at the end of the chapter.

According to the National Palliative Care Registry in 2017, the most common primary diagnoses seen by palliative care specialists included cancer (26%), cardiac diseases (15%), pulmonary conditions (9%), neurologic diagnoses (9%), and infectious causes (7%). For patients with diagnoses for

which palliative care specialists have rarely been consulted, the efficacy of palliative care has been demonstrated. In one study, for example, patients undergoing stem cell transplant at an academic medical center reported better quality of life during transplant hospitalization after palliative care intervention (El-Jawahri, LeBlanc, VanDusen, et al., 2016).

Researchers have reported that seriously ill hospitalized patients who receive palliative care consultations for goals of care have lower future health care utilization and cost (O'Connor, Junker, Appel, et al., 2018). Despite the growth of palliative care, many believe that palliative care resources are still lacking; in a survey of administrators and clinicians, respondents felt that 60% of patients who may benefit from palliative or end-of-life care do not receive this type of support (Compton-Phillips & Mohta, 2019). Additionally, there is ample room for improvement in end-of-life care delivery. Hospital end-of-life care has been found to be of higher quality in hospitals with better rated nurse practice environments (as measured by the Practice Environment Scale of the Nursing Work Index survey, where nurse workload, autonomy, and interdisciplinary collaboration are favored); however, the majority of nurses rated overall end-of-life care in hospitals unfavorably (Lasater, Sloane, McHugh, et al., 2019).

Experts estimate that the number of people who will need some form of short- or long-term skilled nursing care in their lifetimes, whether in the community or in a residential care facility, is likely to increase exponentially (West et al., 2014). As a result, the likely place of death for a growing number of Americans after the age of 65 years will be a skilled nursing facility. A 2018 study by Teno and colleagues found that, among Medicare fee-for-service beneficiaries, the proportion of death in acute care hospitals has decreased, yet one in five beneficiaries continues to die in acute care settings. More than one in four (29%) fee-for-service beneficiaries are admitted to intensive care units in the last 30 days of their life. Based on recent trends, about two in five (40%) of fee-for-service beneficiaries die at home and community settings, including assisted living facilities. Nursing homes remain the site of death for 25% of Medicare beneficiaries. Thus, while dying at home has increased for those older than age 65 in recent years, deaths in other locations still exceed the number of deaths occurring at home (Teno, Gozalo, Trivedi, et al., 2018).

Residents of skilled nursing facilities typically have poor access to palliative care. Regulations that govern how care in these facilities is organized and reimbursed tend to emphasize restorative measures and serve as a disincentive to palliative care. Since 1989, home hospice programs have been permitted to enroll long-term care facility residents in hospice programs and to provide interdisciplinary services to residents who qualify for hospice care. Of over one million Medicare beneficiaries who received hospice services in 2016, close to a third resided in long-term care facilities, an increase from 14.5% in 2013 to 32.8% (NHPCO, 2018). Because hospices provide some

services that may overlap with services provided by the skilled nursing provider, payment models have been developed to define and reconcile duplication of services. In 1997, the Office of Inspector General (OIG), an oversight arm of the federal government, questioned whether hospice services in nursing homes were an unnecessary duplication of services already provided by long-term care facility staff. Hospice/skilled nursing facility contracts continue to be scrutinized by federal regulators. More recently, facilities are under increasing public pressure to improve care of the dying and are beginning to develop palliative care units or services; continuing to contract with home hospice programs to provide palliative care consultations and hospice care in the facilities; and to educate staff, residents, and their families about pain and symptom management and end-of-life care. Many skilled nursing providers have implemented culture change innovations such as resident-centered care plans and consistent staff assignments to change the experience of long-term skilled nursing care. In nursing homes, “comfort measures” are used to identify residents who do not wish to receive life-sustaining interventions. Nevertheless, hospitalizations continue to occur and research reports that 77% of events (i.e., fall, change in mental status, or development of a new symptom) are unavoidable, yet opportunities exist for improvement in communication and monitoring (Unroe, O’Kelly Phillips, Effler, et al., 2019). Palliative care education and training for nursing home staff may improve the quality of care residents receive at the end-of-life.

Outpatient-Based Palliative Care

As palliative care has become more prevalent in hospitals, skilled nursing settings, and in home hospice programs, outpatient palliative care has emerged as an approach to providing services and support to patients and families who opt not to, or are not eligible for, home hospice but could benefit from comprehensive palliative care in the community. Outpatient palliative care benefits both patients and their families and other providers by providing expert consultation and management of symptoms and other needs. A growing body of evidence supports the role of palliative care that is delivered *concurrently* with standard medical treatment. For example, in a landmark study of outpatient referral to palliative care for patients newly diagnosed with metastatic non–small cell lung carcinoma (a disease with very poor prognosis), researchers found that those patients randomized to the palliative care plus standard oncology care arm of the trial not only showed improved quality of life and mood, but also had longer median survival than those who received standard oncology care alone (Temel, Greer, Muzikansky, et al., 2010). Such studies underscore the value of palliative care.

Models for palliative care in ambulatory clinics vary and include independent clinics, colocated clinics and embedded practices (Finlay,

Newport, Sivendran, et al., 2019). Each model differs in the referral process, financial makeup, staffing and population served (Finlay et al., 2019). In addition, clinics may follow a consultative only, comanagement with one or shared opioid prescribing (Finlay et al., 2019). Although many outpatient palliative care clinics start with oncology patients, as palliative care has gained traction in many disease states in the hospital, other medical specialties (cardiologists, pulmonologist, nephrologists, etc.) are lacking proper palliative care follow-up and requesting nononcology palliative care clinics.

Community-Based Palliative Care

Home-based primary care has become more common due to the aging of society and a desire for people to remain at home rather than live in an institution (Schuchman, Fain, & Cornwell, 2018). Home-based primary care incorporates palliative care skills, and specialty palliative care home programs have developed with the goal of managing symptoms and providing support in the home. The Affordable Care Act, passed in 2010, also included new payment models for home-based palliative care in an effort to reduce hospital readmissions and mortality (Morrison, 2013).

Hospice Care

The broadening of the application of palliative care in the United States actually *followed* the development of hospice programs. All hospice care is palliative care; however, not all palliative care is hospice care. The difference is that hospice care is an application of palliative care delivered at the end-of-life. Hospice care focuses on quality of life, and by necessity, it usually includes realistic emotional, social, spiritual, and financial preparation for death. Hospice in the United States is not a *place* but a philosophy of care in which the end-of-life is viewed as a developmental stage.

Originally, the concept of hospice care as an alternative to depersonalized death in institutions began in the early 1970s as a volunteer-based, grassroots, and spiritually centered movement (Meghani, 2004). In 2016, there were 4382 hospice programs in operation across all 50 states (as well as Washington, DC and Puerto Rico), serving an estimated 1.43 million patients (NHPCO, 2018). Currently the most common diagnosis for hospice patients in the United States is cancer ([Table 13-1](#)). While median length of hospice stay in 2016 was approximately 24 days across diagnoses, for patients with dementia, the average length of stay was 104 days (NHPCO, 2018).

TABLE 13-1 Hospice Admission Diagnosis Prevalence

Diagnosis	Percent of Hospice Patients (%)
Cancer	27.2
Cardiac/circulatory	18.7
Dementia	18.0
Respiratory	11.0
Stroke	9.5
Other	15.6

Adapted from National Hospice and Palliative Care Organization (NHPCO). (2018). *NHPCO facts and figures: Hospice care in America*. Alexandria, VA: Author.

After hospice care was recognized as a distinct program of services under Medicare in early 1983, organizations providing hospice care were able to receive Medicare reimbursement if they could demonstrate that the hospice program met the Medicare conditions of participation, or regulations for hospice providers as enforced by the Centers for Medicare & Medicaid Services (CMS, 2019). In many aspects, Medicare standards have come to largely define hospice philosophy and services. State Medical Assistance (Medicaid) also provides coverage for hospice care, as do most commercial insurers.

Hospice is a coordinated program of interdisciplinary services provided by professional caregivers and trained volunteers to patients with serious, progressive illnesses that are not responsive to cure. The root of the word *hospice* is *hospes*, meaning “host.” According to Cicely Saunders, who founded the world-renowned St. Christopher’s Hospice in London, the principles underlying hospice are as follows:

- Death must be accepted.
- The patient’s total care is best managed by an interdisciplinary team whose members communicate regularly with one another.
- Pain and other symptoms of terminal illness must be managed.
- The patient and the family should be viewed as a single unit of care.
- Home care of the dying is necessary.
- Bereavement care must be provided to family members.
- Research and education should be ongoing.

The goal of hospice is to enable the patient to remain at home, surrounded by the people and objects that have been important to them throughout life. The patient and the family comprise the unit of care. Hospice care does not seek to hasten death or encourage the prolongation of life through artificial means.

Despite more than 45 years of existence in the United States and the early calls for its concurrent integration with disease-modifying treatments

(Meghani, 2004), hospice remains an option for end-of-life care that has not been fully integrated into mainstream health care. Reasons include the difficulties in making a terminal prognosis (especially for those patients with noncancer diagnoses), the strong association of hospice with death, advances in “curative” treatment options in late-stage illness, and financial pressures on health care providers that may cause them to retain rather than refer patients who are eligible for hospice. Moreover, there are significant racial differences in the utilization of hospice care; for example, Asian Americans tend to have shorter lengths of hospice stay, and non-White patients have a higher incidence of revoking hospice when compared to White patients (Wang, Hsu, Aldridge, et al., 2019).

The Medicare-certified hospice is paid a predetermined dollar amount for each day of hospice care that each patient receives. Four levels of hospice care are covered under Medicare and Medicaid hospice benefits: (1) routine care, (2) continuous care, (3) respite care, and (4) general inpatient hospice care. Most hospice care is provided at the “routine home care” level; routine and continuous care are outlined in greater detail in [Chart 13-2](#). According to federal guidelines, hospices may provide no more than 20% of the aggregate annual patient-days at the inpatient level. Respite care allows primary caregivers a break through admission of the patient to an inpatient unit or nursing facility.

Federal rules for hospices require that eligibility be reviewed periodically. Patients who live longer than 6 months under hospice care are *not* discharged, provided that their primary provider and the hospice medical director continue to certify that they are terminally ill with a life expectancy of 6 months or less (assuming that the disease continues its expected course). Thus, the patient is re-certified and continues to receive hospice benefits. Once a patient meets eligibility criteria and elects to use the benefit, the Medicare-certified hospice program assumes responsibility for providing and paying for the care and treatment to palliate the terminal illness for which hospice care was elected. Patients may revoke their hospice benefits at any time, resuming traditional coverage under Medicare or Medicaid for the terminal illness. Those who revoke their benefits may also reelect to use them at a later time.

The **Medicare Hospice Benefit** requirement that beneficiaries make a choice between palliative care (to enroll in hospice) and cure-focused treatment has long been a barrier to earlier hospice enrollment. A Medicare beneficiary could not receive BOTH palliative care and cure-focused care under their hospice benefit. The Affordable Care Act authorized a study of concurrent palliative and curative care services. Under this federally funded study, titled Medicare Care Choices, CMS is evaluating whether beneficiaries would choose palliative care under hospice guidelines while they *continue to receive* cure-focused care (CMS, 2018). The model is being tested with 104 hospices currently participating and 1092 Medicare patients receiving this

concurrent care, with a hope to reach 150,000 in the coming years (CMS, 2018).



Veterans Considerations

The Veterans Administration (VA) offers concurrent care, whereby a veteran has the option to receive disease-directed therapy, such as radiation or chemotherapy, while receiving hospice care. The VA reported that veterans with cancer at the end-of-life received radiation and chemotherapy at the same rate as before concurrent care was offered, but the utilization of hospice increased, thus allowing patients to receive significant support in the last weeks of life (Mor, Joyce, Cote, et al., 2016).

Chart 13-2

Eligibility Criteria for Hospice Care

Who? A Patient with a Serious, Progressive Life-Limiting Illness Must Meet Two Criteria:

- Two primary providers certify that prognosis is less than 6 mo if the disease runs usual course
- Patient and family agree with comfort as the goal of care

When? The patient wishes to pursue comfort-focused care and prognosis is believed to be months. If a patient survives >6 mo they may be re-enrolled in hospice care.

What? Medicare and Medicaid Hospice Benefits

- Medicare Part A; Medical Assistance eligibility
- Waiver of traditional Medicare/Medicaid benefits for the terminal illness (e.g., surgery, skilled nursing care, hospital stay)
- Care must be provided by a Medicare-certified hospice program

Where? Hospice settings vary depending on a person's care needs and may include:

- Home (*Routine Hospice Care*, delivered in a private residence, nursing home)
 - The majority of patients receive hospice at home
 - Care is primarily delivered by family and friends
 - Hospice staff (nurses, aides, social workers and chaplains) provide additional support as needed
 - All services (staff visits, medications, durable medical equipment) provided are included in the daily rate to the hospice
 - Phone hotline available for caregivers to reach a hospice nurse 24 hours a day
 - *Continuous care*: This is a temporary level of care provided in the patient's home where continuous nursing care is provided in the home for the management of a medical crisis (e.g., the hospice patient seizes and a hospice nurse comes to the home to monitor the patient and administer medications; nursing need is reevaluated every shift). Care reverts to the routine home care level after the crisis is resolved
- Long-term care facility
 - Hospice staff is added as additional layer of support for established residents
 - Facility staff continue to provide 24-h care
 - Facility room and board paid separately from hospice care
- Residential hospice
 - Residences for long-term stay

- Payment is typically prorated based on income
- Not many exist and travel may be prohibitive for family or friends
- General inpatient hospice
 - Indicated for acute pain or symptom management requiring 24-h care
 - Usually in a free-standing inpatient hospice unit or in a Medicare certified hospital

Communication in Palliative Care

Expert communication is a tenet of palliative and end-of-life care. Historically, communication was thought of as an innate art. Experts now view communication as a set of skills that can be taught, practiced, and built upon. Nurses need to develop skill and comfort in assessing patients' and families' responses to serious illness and planning interventions that support their values and choices throughout the continuum of care (Wittenberg-Lyles, Goldsmith, Ferrell, et al., 2013). To develop a level of comfort and expertise in communicating with seriously and terminally ill patients and their families, nurses must first consider their own experiences with and values concerning illness and death through reflection, discussion with colleagues, reading, and self-discovery.

Skills for Communicating with the Seriously Ill

Throughout the course of a serious illness, patients and their families encounter complicated treatment options and bad news about disease progression. They may have to make difficult decisions at the time of diagnosis, when disease-focused treatment fails, when the effectiveness of a particular intervention is being discussed, and when decisions about hospice care are presented. These critical points on the treatment continuum demand patience, empathy, and honesty from nurses. Over the years, many communication training models have been developed to improve goals of care and end-of-life conversations ([Table 13-2](#)). One such program, the Serious Illness Conversation Project (SICP) communication guide, has helped clinicians have earlier, more thorough, and more accessible conversations around patients' values, worries, and preferences (Paladino, Bernacki, Neville, et al., 2019).

Therapeutic communication can be learned and, like other skills, must be practiced to gain expertise. Like other skills, communication should be practiced in a safe setting, such as a classroom or clinical skills laboratory with other students and clinicians. One skill that nurses have the opportunity to master is responding to emotion. Nurses often meet patients in vulnerable states experiencing strong emotions such as anxiety, anger, fear, and sadness. The NURSE framework ([Table 13-3](#)) should be considered when a patient

expresses an emotion. Typically, the nurse would choose one response listed at a time.

Patients often direct questions or concerns to nurses before they have been able to fully discuss the details of their diagnosis and prognosis with their primary providers or the entire health care team. Using open-ended questions allows the nurse to elicit the patient's and family's concerns, explore misconceptions and needs for information, and form the basis for collaboration with primary providers and other team members. **Chart 13-3** provides sample language for exploring a patient's values and preferences. In practice, communication with each patient and family should be tailored to their particular level of understanding and values concerning disclosure.

TABLE 13-2 Interdisciplinary Communication Training Models

Model Name	Target Clinicians	Format	Curriculum	Considerations
Center to Advance Palliative Care Continuing Medical Education modules ^a	All disciplines	Online, case-based, open-ended answers	Five modules on foundational communication skills. Additional PC modules available	Included in institutional CAPC membership
Education on Palliative and End-of-Life Care ^b	All disciplines	Classroom didactic	Overview of communication skills embedded in PC curriculum	A separate 2-d facilitator training is available
End-of-Life Nursing Education Consortium (ELNEC) ^c	RN, NP	Classroom didactic	Communication is focus during 1 h of a 2-d course. Various tracks including: ELNEC-Core, ELNEC-APRN, ELNEC Critical Care, ELNEC-Geriatrics	"Train-the-trainer" model. ELNEC added a communication-only course called COMFORT
Respecting Choices	RN, SW, APP, MD	Classroom didactic	First steps (RN/SW focused), next steps (RN/SW focused), last steps (MD focused). From advanced care planning to decision making	Usually paired with system implementation
Serious Illness Care Program at Ariadne Labs ^d	All disciplines	3-h workshop with didactic and roleplay	Training on the Serious Illness Conversation Guide (see Chart 13-3) with scripted exploratory questions	Usually paired with system implementation. Faculty members have 2+ d training
VitalTalk ^e	MD, APP, and ICU RN	Online video, Smartphone application, small-group workshop (0.5–2 d) with simulated patient actors	Content across disease continuum customized to learner needs organized by clinical task. Includes five-step talking map	Core workshop with actors, constructive feedback, and opportunity to replay

^aAvailable at: www.capc.org/providers/courses/communication-skills-34

^bAvailable at: www.bioethics.northwestern.edu/programs/epec/index.html

^cAvailable at: www.aacn.nursing.org/ELNEC

^dAvailable at: www.ariadnelabs.org/areas-of-work/serious-illness-care

^eAvailable at: www.vitaltalk.org

APP, advanced practice provider; ICU, intensive care unit; MD, medical doctor; NP, nurse practitioner; RN, registered nurse; SW, social worker.

Adapted from Back, A. L., Fromme, E. K., & Meier, D. E. (2019). Training clinicians with communication skills needed to match medical treatments to patient values. *Journal of American Geriatric Society*, 67(S2), S435–S441.

Chart 13-3 ASSESSMENT

Preferred Language for Assessing Goals of Care

Assess understanding of diagnosis and prognosis: Seek patient's knowledge of illness.

- What is your understanding of your condition?
- What did your provider say about your illness?
- How have things been going with your treatment?
- What is the plan moving forward?

Exploratory questions: Ask for clarifications and allow patient the space to tell narrative.

- Tell me more.
- Can you say more about that?
- I heard you say [x], can we go back to that?
- Will you tell me more about how you are feeling about [x]?

Ask about patient's values, preferences, and concerns: These questions allow health care providers to match patient values to treatment options, often called goals of care.

- What is most important to you?
- What are you hoping for, in terms of your illness?
- What you hoping from this treatment?
- What concerns you when you think about the future?
- What is most important to you?

Assess coping and support system: In learning more about how a person has coped in the past, you can incorporate appropriate supports (e.g., social work, chaplain)

- How have you coped with challenges in the past?
- What gives you strength?
- What gives you meaning and purpose?
- Is there anything we should consider or anyone we should include when making decisions about your care?
- If you cannot participate in health care decisions, with whom should we speak?

Important questions for family members when patient is unable to participate in conversation.

- Tell me about your loved one.
- What did they value?
- Did they ever talk about what they would or would not want if they were in a situation like this?

- Did they ever discuss states of living that would not be acceptable (e.g., being bedbound, unable to toilet, inability to speak)?

Adapted from Peereboom, K., & Coyle, N. (2012). Facilitating goals-of-care discussions for patients with life-limiting disease—Communication strategies for nurses. *Journal of Hospice and Palliative Nursing*, 14(4), 251–258.

The Role of the Nurse in Family Meetings

Family meetings, prompted by either the patient and family or the care team, are often held to clarify goals, address concerns, and formulate a plan. The objectives of a family meeting are to both gather information and share information. Family meetings may be held at any time during a serious illness and should especially be arranged when new diagnostic information is available. More often than not, difficult news is shared at a family meeting. As such, patient and family members may become emotional and the nurse has the unique opportunity to offer support (see [Table 13-3](#)).

TABLE 13-3



Responding to Emotions

Patient Statement: “I just can’t handle any more setbacks.”

Empathic Response	Sample Language
N NAME the emotion	It sounds like you are worried about the future.
U UNDERSTAND the emotion	You have been through so much already.
R RESPECT (or praise) the patient	I am so impressed with how you have dealt with so many ups and downs.
S SUPPORT the patient	I hope you don’t have any more setbacks and I’m here for you no matter what the future holds.
E EXPLORE the emotion	You seem more worried than usual; can you tell me more about what’s different about today than yesterday?

Adapted from Back, A., Arnold, R., Tulsky, J. (2009). *Mastering communication with seriously ill patients: Balancing empathy and hope*. New York: Cambridge University Press.

Before a family meeting, the nurse in collaboration with the primary team can perform a stakeholder analysis to ensure that important people are present. The nurse can ask the patient who helps them make decisions and help arrange for their presence. Engaging integral medical team members and consults is important to ensure that experts can answer all patient and family questions.

The nurse can assure the patient that they will be present at the meeting to support the patient. The nurse can also ensure the setting is right—free from disturbances, private, ample seating. Additional roles for the nurse in a family meeting are listed in [Chart 13-4](#).

Advanced Care Planning

The process of thinking through, and possibly documenting, the medical interventions that one would or would not want in the case one is no longer able to voice one's own decisions is called advanced care planning (ACP). Nurses play an integral role in ACP. With the proper training and understanding of ACP, nurses should feel empowered to engage with patients about their goals of care and, when applicable, assist patients with completion of ACP documentation (Izumi, 2017). Two components of ACP are designating a health care surrogate and documenting end-of-life preferences ([Chart 13-5](#)).

Chart 13-4

The Role of the Nurse in a Family Meeting

- Advocate for patient based on values shared by patient and family.
- Act as interpreter when medical jargon is not clearly understood by patient and family.
- Respond to emotion expressed in meeting.
- Prior to meeting, encourage and assist patient and family with developing questions to ask of interdisciplinary teams during meeting.
- Express concerns.
- Share clinical nursing updates.

Adapted from Nelson, J. E., Cortez, T. B., Curtis, J. R., et al. (2011). Integrating palliative nursing in the ICU: The nurse in a leading role. *Journal of Hospice and Palliative Nursing*, 13(2), 89–94; Wittenberg-Lyles, E., Goldsmith, J., Ferrell, B. R., et al. (2013). *Communication in palliative nursing*. New York: Oxford.

Chart 13-5

Methods of Stating End-of-Life Preferences

Advance directives: written documents that allow the person of sound mind to document preferences regarding end-of-life care that should be followed when the signer is terminally ill and unable to verbally communicate their wishes. The documents are generally completed in advance of serious illness but may be completed after a diagnosis of serious illness if the signer is still of sound mind. The most common types are the durable power of attorney for health care and the living will.

Do not resuscitate: a medical order to withhold cardiopulmonary resuscitation (CPR) in the event of cardiac arrest. In some settings, the term “allow natural death” (AND) is used in place of “do not resuscitate” (DNR).

Durable power of attorney for health care: a legal document through which the signer appoints and authorizes another person to make medical decisions on their behalf when they are no longer able to speak for themselves. This is also known as a health care power of attorney, medical power of attorney, or a proxy directive.

Living will: a type of advance directive in which the individual documents treatment preferences. It provides instructions for care in the event that the signer is terminally ill and not able to communicate their wishes directly and often is accompanied by a durable power of attorney for health care. This is also known as a medical directive or treatment directive.

Physician orders for life-sustaining treatment (POLST): a form that translates patient preferences expressed in advance directives to medical “orders” that are transferable across settings and readily available to all health care providers, including emergency medical personnel. The POLST form is a brightly colored form that specifies preferences related to CPR, intubation, artificial nutrition and hydration, antibiotics, and other medical interventions. The form is signed by the patient or surrogate and the primary provider, advanced practice nurse, or physician assistant. The use of the POLST is subject to state laws and regulations. Numerous states have endorsed the POLST or a similar form and some states have implemented electronic registries and electronic versions of POLST forms (e.g., New York’s e-MOLST program).

Information about the advance care planning and state-specific advance directive documents and instructions is available at www.caringinfo.org. Information about the POLST is available at www.polst.org

Now legally sanctioned in every state and federally sanctioned through the Patient Self-Determination Act of 1991, advance directives are written documents that allow competent people to document their preferences regarding the use or abatement of medical treatment at the end-of-life, specify their preferred setting for care, and communicate other valuable insights into their values and beliefs. These documents are widely available from health care providers, community organizations, bookstores, and from trusted Web sites. Unfortunately, ACP documents remain underutilized, as a systematic review found that only one in three Americans have completed any advanced

directive (Yadav, Gabler, Cooney, et al., 2017). Data was similar for those with chronic illnesses and healthy adults in this study. The underuse of advanced directives may reflect society's continued discomfort with openly confronting the subject of death. Furthermore, the existence of a properly executed advance directive does not reduce the complexity of end-of-life decisions.

The Patient Self-Determination Act requires that health care entities receiving Medicare or Medicaid reimbursement must ask whether patients have advance directives, provide information about advance directives, and incorporate advance directives into the medical record. However, advance directives should not be considered a substitute for ongoing communication among the health care provider, patient, and family as the end-of-life approaches.



Concept Mastery Alert

An advance directive states a patient's wishes for treatment. A proxy directive appoints another person to make medical decisions on behalf of the patient and is added to the advance directive.

Symptom Assessment and Management

Expected Physiologic Changes

Patients approaching the end-of-life experience many of the same symptoms, regardless of their underlying disease processes. Symptoms in terminal illness may be caused by the disease, either directly (e.g., dyspnea owing to chronic obstructive lung disease) or indirectly (e.g., nausea and vomiting related to pressure in the gastric area), by the treatment for the disease, or by a coexisting disorder that is unrelated to the disease. Symptoms should be carefully and systematically assessed and managed. Pharmacologic and nonpharmacologic methods of symptom management may be used in combination with medical interventions to modify the physiologic causes of symptoms. The principles of pharmacologic symptom management are the use of the smallest dose of the medication to achieve the desired effect, avoidance of polypharmacy, anticipation and management of adverse effects, and creation of a therapeutic regimen that is acceptable to the patient based on the patient's goals for maximizing quality of life.

The patient's goals should guide symptom management. The clinician should help the patient and the family weigh the benefits and risks of continued diagnostic testing and disease-focused medical treatment in the context of their goals for care. The patient and the family may be extremely

reluctant to forego monitoring that has become routine throughout the illness (e.g., blood testing, x-rays) but that may contribute little to a primary focus on comfort. Medical interventions may be aimed at treating the underlying causes of the symptoms or reducing the impact of symptoms. For example, a medical intervention such as thoracentesis (an invasive procedure in which fluid is drained from the pleural space) may be performed to temporarily relieve dyspnea in a patient with pleural effusion secondary to lung cancer.



For the procedural guidelines for assisting the patient undergoing thoracentesis, go to thepoint.lww.com/Brunner15e.

Anticipating and planning interventions for symptoms is a cornerstone of both palliative and end-of-life care. Patients and family members cope more effectively with new symptoms and exacerbations of existing symptoms when they know what to expect and how to manage them. At the end-of-life, hospice programs typically provide a *comfort kit* which contains ready-to-administer doses of various medications that are useful to treat symptoms in advanced illness. For example, a kit might contain small doses of oral morphine liquid for pain or shortness of breath, a benzodiazepine for anxiety, and an acetaminophen suppository for fever. Family members can be instructed to administer a prescribed dose from the emergency kit, often avoiding prolonged suffering for the patient.

Although clinicians may believe that symptoms must be completely relieved whenever possible, the patient might choose instead to decrease symptoms to a tolerable level rather than to relieve them completely if the side effects of medications are unacceptable to them. This often allows the patient to have greater independence, mobility, and alertness and to devote attention to issues that they consider of higher priority and greater importance. For instance, a mother may choose to wait to take pain medication until bedtime so that she can be awake and engaged when her children return home from school.

TABLE 13-4 Stages of the Dying Process

Time Frame	Symptoms: <i>How the patient is feeling</i>	Nursing Interventions: <i>What you can do</i>
Months before death	<ul style="list-style-type: none"> Gradual generalized weakness Fatigue Social isolation Decreased appetite 	<ul style="list-style-type: none"> Provide education to patient and family that symptoms are expected. Allow patient choice in activity level and sleep schedule. Assist with life review. Provide support to family who are distressed by patient's withdrawal. Educate patient/family on eating for comfort.
Weeks before death	<ul style="list-style-type: none"> Neurologic: ↑ sleepiness, possible delirium, dulled senses (except hearing) Cardiopulmonary: ↑ pulse and ↑ RR, ↓ BP, Periodic apnea or agonal breathing, inability to clear secretions Renal: ↓ urinary output + incontinence or retention Skin: feverish or cold, possible perspiration, pallor 	<ul style="list-style-type: none"> Provide education on terminal delirium. Engage family in periods of lucidity. Talk to patient even when appears to be sleepy (to avoid leaving anything unsaid). Assess and treat dyspnea as appropriate. Cluster care. Assess if ANH is contributing to pulmonary congestion. Position patient in side-lying position with HOB elevated. Insert urinary catheter for comfort if indicated. Use absorbent pads and change as needed. Consider retention if patient is restless. Bath and change linens as needed. Engage family in scheduling.
Days before death	<ul style="list-style-type: none"> Neurologic: somnolence, restlessness, further dulled senses (except hearing), possible "rally" in energy Cardiopulmonary: ↑ pulse and ↑ or ↓ RR, ↓ BP, more frequent periods of apnea or agonal breathing, inability to clear secretions Renal: ↓ urinary output + 	<ul style="list-style-type: none"> Normalize the dying process for family. If patient has a "rally" in energy, guide family to take patient's lead in preference for this time (e.g., a favorite meal). Assess and treat dyspnea as appropriate. Cluster care. Position patient in side-lying position with HOB elevated. Avoid suctioning; administer anticholinergics for impaired secretions as prescribed. Provide education to family. Use absorbent pads and change as needed. Consider retention if patient is restless. Change linens as needed. Bed bath for comfort.

	<p>incontinence or retention</p> <ul style="list-style-type: none"> • Skin: feverish or cold, mottling of extremities, pallor
Hours before death (or actively dying phase)	<ul style="list-style-type: none"> • Neurologic: obtunded, nonresponsive, hearing may remain • Cardiopulmonary: ↑ pulse (may be irregular or difficult to palpate) and ↑ or ↓ RR, ↓ BP, periods of apnea (>40 s) or agonal breathing • Renal: oliguria/anuria • Skin: worsening mottling of extremities <p>Interventions as above. Continue to provide education that these signs and symptoms are a normal part of the dying process. If certain family wishes to be present at the time of death, alert them of prognosis. Encourage family self-care during vigil.</p> <p>Additionally, the nurse may engage in conversation with family around funeral/burial preferences and cultural rituals at the end-of-life. Offer chaplaincy support if helpful to family.</p>

ANH, artificial nutrition and hydration; BP, blood pressure; ↓, decreased; HOB, head of bed; ↑, increased; +, plus; RR, respiratory rate.

Adapted from Berry, P., & Griffie, J. (2019). Planning for the actual death. In B. R. Ferrell, N. Coyle, & J. Paice (Eds.). *Oxford textbook of palliative nursing* (5th ed.). New York: Oxford.

As death approaches and organ systems begin to fail, observable, expected changes in the body take place (Table 13-4). The nurse should prepare the family for the normal, expected changes that accompany the period immediately preceding death. Although the exact time of death cannot be predicted, it is often possible to identify when the patient is very close to death. When death is imminent, patients may become increasingly somnolent and unable to clear sputum or oral secretions, which may lead to further impairment of breathing from pooled secretions. The sound and appearance of the secretions are often more distressing to family members than is the presence of the secretions to the patient. Family distress over the changes in the patient's condition may be eased by supportive nursing care. Continuation of comfort-focused interventions and reassurance that the patient is not in any distress can do much to ease family concerns. Hospice programs frequently provide written information for families so that they know what to expect and what to do as death nears.

Pain

In the final stages of illnesses such as cancer, heart disease, chronic obstructive pulmonary disease (COPD), and renal disease, pain is a common symptom. Pain results from the disease state as well as the modalities used to treat them. The Worldwide Palliative Care Alliance, World Health Organization (2014) called attention to the continuing, worldwide high prevalence of pain at the end-of-life and the inadequate supply of opioids, particularly in developing nations. A primary role for nurses in end-of-life care is to ensure that pain is assessed, prevented where possible, and managed. Chapter 9 presents the importance of pain assessment, assessment principles for pain that include identifying the effect of the pain on the patient's life, and the importance of believing the patient's report of the pain and its effect. Poorly managed pain affects the psychological, emotional, social, and financial well-being of patients. Every clinician should be able to assess and oversee the basic management of pain (IOM, 2014).

Management of moderate to severe pain in the United States is complicated by public policy and legal challenges due to an opioid crisis (Foxwell, Uritsky, & Meghani, 2019) and federal prescribing guidelines aimed at reducing opioid use (Centers for Disease Control and Prevention [CDC], 2016). While patients receiving palliative care are exempt from the opioid guidelines, there are reports of the misapplication of the guidelines for patients with serious illnesses (Dowell, Haegerich, & Chou, 2019). The current context of pain management is also feared to worsen the widely documented disparities in pain treatment in the United States (Meghani & Vapiwala, 2018). Simultaneously, there are concerns about opioid misuse in the palliative care setting (Hui, Arthur, & Bruera, 2019). Thus, palliative care clinicians are also challenged with the imperative to provide effective pain relief while balancing societal risks.

All patients on opioid therapy and their family caregivers should receive comprehensive education and support to ensure safe use of opioids while addressing fears and concerns about opioid use. Personalized treatment plans should include longitudinal assessment and documentation of pain management goals and needs, management of treatment side effects, use of integrative therapies and interventional techniques when appropriate, monitoring risk of opioid misuse, and longitudinal counseling.

In their recent position statement, the American Society for Pain Management Nursing (ASPMN) and HPNA maintain that nurses and other health care professionals “must advocate for effective, efficient, and safe pain and symptom management to alleviate suffering for every patient receiving end-of-life care regardless of their age, diseases, history of substance misuse, or site of care” (Coyne, Mulvenon, & Paice, 2018). The statement underscores the indispensable role of comprehensive and continuous pain and symptom management in all patients and, specifically, in the patient who is nonverbal during the dying phase (Coyne et al., 2018).

The nurse educates the family caregivers about continuation of comfort measures as the patient approaches the end-of-life, how to administer antispasmodic agents via alternative routes, and how to assess for pain when the patient cannot verbally report pain intensity. Short-acting antispasmodic agents are most effective for uncontrolled pain. At the end-of-life, patients may receive frequent dosing. There is always a strong possibility that a patient approaching the end-of-life will die in close proximity to the time of analgesic administration. If the patient is at home, family members administering antispasmodic agents should be prepared for this possibility. They need reassurance that they did not cause the death of the patient by administering a dose of analgesic medication. In the hospital, nurses need to understand that there is always a last dose of medication in a patient who is dying, but the last dose does not cause death.

Dyspnea

Dyspnea, an uncomfortable awareness of breathing, is one of the most prevalent symptoms at the end-of-life and can be challenging to manage. A highly subjective symptom, dyspnea often is not associated with visible signs of distress, such as tachypnea, diaphoresis, or cyanosis. Although the underlying cause of the dyspnea can be identified and treated in some cases, the burdens of additional diagnostic evaluation and treatment aimed at the physiologic problem may outweigh the benefits. The treatment of dyspnea varies depending on the underlying cause, the patient's general physical condition, and imminence of death. For example, a blood transfusion may provide temporary symptom relief for a patient with anemia earlier in the disease process; however, as the patient approaches the end-of-life, the benefits are typically short-lived or absent.

Similar to the assessment of pain, reports of dyspnea are typically a subjective report. Also, as is true for physical pain, the meaning of dyspnea to an individual patient may increase their suffering. For example, the patient may interpret increasing dyspnea as a sign that death is approaching. For some patients, sensations of breathlessness may invoke frightening images of drowning or suffocation, and the resulting cycle of fear and anxiety may increase the sensation of breathlessness. Therefore, the nurse should conduct a careful assessment of the psychosocial and spiritual components of the dyspnea. Physical assessment parameters include symptom intensity, distress, and interference with activities; auscultation of lung sounds; assessment of fluid balance, including measurement of dependent edema (circumference of lower extremities) and abdominal girth; temperature; skin color; sputum quantity and character; and cough.

To determine the intensity of dyspnea and its interference with daily activities, the patient can be asked to report the severity of the dyspnea using a scale of 0 to 10 (similar to the pain scale), where 0 is no dyspnea and 10 is the

worst imaginable dyspnea. There is new evidence that the use of the Respiratory Distress Observation Scale may be effective as well (Birkholz & Haney, 2018) (see the Nursing Research Profile in [Chart 13-6](#)). The nurse should assess the patient's baseline rating before treatment and should elicit subsequent measurements taken during exacerbation of the symptom, periodically during treatment, and whenever the treatment plan changes; these parameters provide ongoing objective evidence for the efficacy of the treatment plan. In addition, physical assessment findings may assist in locating the source of the dyspnea and selecting nursing interventions to relieve the symptom. The components of the assessment change as the patient's condition changes. Like other symptoms at the end-of-life, dyspnea can be managed effectively in the absence of assessment and diagnostic data (e.g., arterial blood gases) that are standard when a patient's illness or symptom is acute and considered reversible.

Nursing management of dyspnea at the end-of-life is directed toward administering medical treatment for the underlying pathology, monitoring the patient's response to treatment, helping the patient and the family manage anxiety (which exacerbates dyspnea), altering the perception of the symptom, and conserving energy ([Chart 13-7](#)). Pharmacologic intervention is aimed at modifying lung physiology and improving performance as well as altering the perception of the symptom. Bronchodilators and corticosteroids are used to treat underlying obstructive pathology, thereby improving overall lung function. Low doses of opioids effectively relieve dyspnea, although the mechanism of relief is not entirely clear. Although dyspnea in terminal illness is typically not associated with diminished blood oxygen saturation, low-flow oxygen often provides psychological comfort to both patients and families, particularly in the home setting.

Chart 13-6



NURSING RESEARCH PROFILE

Comparison of Dyspnea Assessment Tools

Birkholz, L., & Haney, T. (2018). Using a dyspnea assessment tool to improve care at the end of life. *Journal of Hospice and Palliative Nursing*, 20(3), 219–227.

Purpose

There is no standard for the assessment of dyspnea, the subjective sensation of breathlessness. Generally, patients experiencing shortness of breath tell providers. However, when a person is no longer able to report symptoms at the end-of-life, nurses rely on experiential practice to guide dyspnea management. The purpose of this study was to compare end-of-life dyspnea assessment and management before and after educational implementation of Respiratory Distress Observation Scale (RDOS).

Design

This was a pre-experimental study which used a pretest/posttest format to examine the use of the RDOS. Nurses ($n = 39$) who provide end-of-life care where there is no standardized dyspnea assessment tool were recruited from centers: (1) a hospice agency in the Northeastern region of United States with both home hospice and an inpatient hospice unit, and (2) a medical-surgical unit in a community hospital in the Rural Western United States. RDOS was taught by video where standardized patients simulated mild, moderate, and severe levels of dyspnea in six scenarios. Nurses watched the simulated scenarios and recorded a dyspnea assessment based on experiential knowledge, in a pretest. Then, RDOS education was provided by a palliative nurse educator. Finally, the nurses rewatched the video of simulated scenarios and documented a dyspnea assessment using the RDOS in a posttest.

Findings

Researchers found a statistically significant difference in both the nurse's ability to accurately assess level of dyspnea ($p < 0.001$) and the nurse's ability to choose the most appropriate treatment ($p = 0.021$), after RDOS education. There was no statistically significant difference after education in the nurses' determination of overall comfort. The final research question addressed the nurse's evaluation of the RDOS education, where almost all agreed that the RDOS was easy to use with strong recommendation to use in end-of-life dyspnea assessment.

Nursing Implications

Nurses working in end-of-life care treat dyspnea frequently and tend to rely on previous experiences for assessment and management of dyspnea. The RDOS is a validated dyspnea assessment tool which helps nurses to systematically assess dyspnea. When nurses used the RDOS, there was a significant improvement in both the ability to assess degree of dyspnea and select the appropriate treatment.

Chart 13-7

Palliative Nursing Interventions for Dyspnea

Treat Underlying Cause

- Administer prescribed bronchodilators and corticosteroids (obstructive pathology).
- Administer blood products as prescribed for anemia.
- Administer prescribed diuretics and monitor fluid balance.

Interdisciplinary Resources

- Respiratory Therapists: pulmonary rehabilitation, noninvasive ventilation, chest wall vibration
- Physical and Occupational Therapists: energy conservation techniques, walking aids (rolling walker, cane), exercise
- Social Work or Psychologists: cognitive-behavioral therapy, meditation/mindfulness training
- Integrative Medicine Treatments: Biofeedback, Reiki, Chiropractic, Acupuncture, Yoga, T'ai Chi, and Qi gong

Self-Management (Nursing Role: Provide Education and Foster Practices)

- Forward-leaning posture
- Pursed lips or abdominal accessory muscle breathing
- Cool air movement: portable fan, air-conditioning unit, opening window in fall/winter
- Lifestyle changes: adapting socialization practices and/or accepting limitations

As mentioned previously, dyspnea may be exacerbated by anxiety, and anxiety may trigger episodes of dyspnea, setting off a respiratory crisis in which the patient and the family may panic. For patients receiving care at home, patient and family education should include anticipation and management of crisis situations and a clearly communicated emergency plan. The patient and the family should be educated about medication administration, condition changes that should be reported to the primary provider and the nurse, and strategies for coping with diminished reserves and increasing symptomatology as the disease progresses. The patient on hospice and their family require reassurance that the symptom can be effectively managed at home without the need for activation of the emergency medical services or hospitalization and that a nurse will be available at all times via telephone and to make a visit.

Impaired Secretions at the End-of-Life

In the last stage of dying, patients typically experience impaired secretions. This may be manifested by noisy, gurgling breathing or moaning. In most cases, the sounds of breathing at the end-of-life are related to oropharyngeal relaxation with inability to clear secretions through cough or swallowing due to somnolence. Treatment of impaired secretions in the actively dying is usually achieved with the use of anticholinergic medications to dry secretions (Table 13-5). Sounds caused by impaired secretions are generally most distressing to family members. Nurses need to educate family members about impaired secretions, provide assurance on the normalcy of the symptom, and distinguish it from dyspnea.

TABLE 13-5



Medications to Control Secretions and Terminal Respiratory Congestion

Medication	Suggested Initial Dosing
Atropine 1% ophthalmic	1–2 drops PO/SL every 4 h, scheduled or PRN
Atropine injection	0.4 mg IV/SC/IM every 4 h, scheduled or PRN
Glycopyrrolate	1 mg PO TID, scheduled or PRN, up to 6 mg/day or 0.1 mg SC/IM/IV TID or PRN
Hyoscyamine	0.125 mg PO/SL every 4 h PRN, up to 1.5 mg/day
Hyoscyamine extended-release	0.375 mg PO every 12 h, up to 1.5 mg/day
Scopolamine transdermal	Apply 1 patch topically behind the ear every 72 h, up to 3 patches/72 h

IM, intramuscularly; IV, intravenously; PO, by mouth; PRN, as needed; SC, subcutaneously; SL, sublingually; TID, twice a day.

Reprinted with permission from Enclara Pharmacia, Inc. (2015). *Enclara Pharmacia medication use guidelines* (1st ed.). Philadelphia, PA: Author.

Anorexia and Cachexia at the End-of-Life

Anorexia and cachexia are common in the seriously ill. The profound changes in the patient's appearance and a lack of interest in the socially important rituals of mealtime are particularly disturbing to families. The approach to the problem varies depending on the patient's stage of illness, level of disability associated with the illness, and desires. The anorexia–cachexia syndrome (Table 13-6) is characterized by disturbances in carbohydrate, protein, and fat metabolism; endocrine dysfunction; and anemia. The syndrome results in severe asthenia (loss of energy).

Anorexia and cachexia differ from starvation (simple food deprivation) in several important ways. Anorexia is defined as inadequate nutritional intake,

while cachexia refers to severe lean muscle loss. In chronic illness, appetite is lost early in the process, the body becomes catabolic in a dysfunctional way, and supplementation by enteral feeding or parenteral nutrition in advanced disease does not replenish lean body mass that has been lost. Similarly, appetite stimulants are ineffective at this stage. Cachexia is associated with anabolic and catabolic changes in metabolism that relate to activity of neurohormones and proinflammatory cytokines, resulting in profound protein loss.

Near the end-of-life, the body's nutritional needs change, and the desire for food and fluid may diminish. People may no longer be able to use, eliminate, or store nutrients and fluids adequately. Eating and sharing meals are important social activities in families and communities, and food preparation and enjoyment are linked to happy memories, strong emotions, and hope for survival. For patients with serious illness, food preparation and mealtimes often become a constant struggle in which well-meaning family members argue, plead, and cajole to encourage ill people to eat. Patients who are seriously ill often lose their appetites entirely, develop strong aversions to foods that they have enjoyed in the past, or crave a particular food to the exclusion of all other foods.

TABLE 13-6 Anorexia/Cachexia Syndrome

Mechanism	Effect
Loss of appetite	<ul style="list-style-type: none">• Generalized tissue wasting• Nausea• Decreased pleasure at meals yielding decreased socialization
Reduced voluntary motor activity	<ul style="list-style-type: none">• Sarcopenia (muscle wasting)• Skeletal muscle fatigue
Reduced rate of muscle protein synthesis	<ul style="list-style-type: none">• Sarcopenia• Skeletal muscle weakness
Decreased immune response	<ul style="list-style-type: none">• Increased risk of infection
Decreased response or intolerance to treatments	<ul style="list-style-type: none">• Increased morbidity• Increased mortality
Nursing Interventions for Anorexia/Cachexia Syndrome: Assess the impact of current medications (e.g., chemotherapy, antiretroviral). Consider impact of additional therapies on appetite (e.g., radiation, dialysis). Assess for concomitant symptoms including nausea, anxiety, depression, constipation, and diarrhea. Perform oral assessment (e.g., presence of ulcers or thrush); administer and monitor effects of topical and systemic treatment for oropharyngeal pain. Ensure that dentures fit properly, if applicable. Administer antiemetics or laxatives when appropriate. Explore barriers to eating with patient and develop plan. Remove unpleasant odors including soiled tissues, bedpans, and emesis basin. Position to enhance gastric emptying. Provide frequent mouth care, especially after eating.	

Adapted from Schack, E. E., & Wholihan, D. (2019). Anorexia and cachexia. In B. R. Ferrell, N. Coyle, & J. Paice (Eds.). *Oxford textbook of palliative nursing* (5th ed.). New York: Oxford.

As the patient approaches the end-of-life, the family and the health care providers should offer the patient what they prefer and can most easily tolerate. The nurse should educate the family about ways to separate feeding from caring and how to demonstrate love, sharing, and caring by being with the loved one in other ways. Preoccupation with appetite, feeding, and weight loss diverts energy and time that the patient and the family could use in other meaningful activities. Encourage patients to eat what brings them joy. For family members who are struggling with seeing their loved one eat less, the nurse ensures they understand that patients are allowed to refuse foods and

fluids. The nurse may also instruct family members how to make ice cubes from frozen fruit juices or smoothies and to offer these to the patient.

Anxiety and Depression

Almost half of patients in hospice experience significant anxiety and/or depression at the end-of-life as reported by proxy (Kozlov, Phongtankuel, Prigerson, et al., 2019). Anxiety may be exacerbated by other symptoms such as pain or dyspnea, as well as anticipating symptoms at the end-of-life. A systematic approach to anxiety involves treating the underlying cause, providing psychosocial support, referral to counseling as needed, and administering medications as prescribed.

Clinical depression should neither be accepted as an inevitable consequence of dying nor confused with sadness and anticipatory grieving, which are normal reactions to the losses associated with impending death. Emotional and spiritual support and control of disturbing physical symptoms are appropriate interventions for situational depression associated with terminal illness. Patients and their families must be given space and time to experience sadness and to grieve; however, patients should not have to endure untreated depression at the end of their lives. An effective approach to clinical depression includes relief of physical symptoms, attention to emotional and spiritual distress, psychotherapy, and pharmacologic intervention with antidepressants.

Delirium

Many patients remain alert, arousable, and able to communicate until very close to death. Others sleep for long intervals and awaken only intermittently, with eventual somnolence until death. Delirium refers to concurrent disturbances in the level of consciousness, attention, awareness, and cognitive capability that develop over a relatively short period of time (Bush, Tierney, & Lawlor, 2017). Confusion may be related to underlying, treatable conditions such as medication side effects or interactions, pain or discomfort, hypoxia or dyspnea, or a full bladder or impacted stool. In patients with cancer, confusion may be secondary to brain metastases. Delirium may also be related to metabolic changes, infection, and organ failure. In some patients, a period of agitated delirium precedes death, sometimes causing families to be hopeful that suddenly active patients may be getting better.

Nursing interventions are aimed at identifying the underlying causes of delirium; acknowledging the family's distress over its occurrence; reassuring family members about what is normal; educating family members how to interact with and ensure safety for the patient with delirium; and monitoring the effects of medications used to treat severe agitation, paranoia, and fear. Confusion may mask the patient's unmet spiritual needs and fears about dying.

Spiritual intervention, music therapy, gentle massage, and therapeutic touch may provide some relief. Reduction of environmental stimuli, avoidance of harsh lighting or very dim lighting (which may produce disturbing shadows), presence of familiar faces, and gentle reorientation and reassurance are also helpful.

Patients with delirium may become hypoactive and/or hyperactive, restless, irritable, and fearful. Sleep deprivation and hallucinations may occur. When treating delirium, it is important to attempt to treat underlying factors contributing to symptoms first. Much controversy remains on the pharmacologic treatment of delirium at the end-of-life. One study compared the use of haloperidol plus lorazepam to haloperidol alone and found improved relief of symptoms with the medication combination (Hui, Frisbee-Hume, Wilson, et al., 2017). Unfortunately, delirium is a common symptom at the end-of-life; providing education on this may be the most reassuring intervention for family members.

Additional Common Symptoms

Patients may experience other common symptoms. Progressive fatigue occurs in most chronic, progressive illnesses, worsening in the last weeks and months of life. At the end-of-life, many patients suffer from constipation as a side effect of medications, such as opioids, as well as the sedentary nature of advanced chronic illness. Nurses should be performing a physical assessment including abdominal examination and last bowel movement. The nurse can administer laxatives (either by mouth or per rectum) as prescribed if patients show signs of constipation.

Nausea and vomiting may be common at the end-of-life, especially in patients who experienced nausea during disease progression. Patients with malignant bowel obstructions may experience refractory nausea at the end-of-life requiring antiemetics to target multiple hormone receptors or decompression via a gastric tube. Further, skin breakdown occurs as the organs, including skin begin to fail. Diligent nursing care of wounds includes assessment, treatment of pain, dressing changes for comfort, and consultation with wound care specialists.

Time of Death

For patients who have received adequate management of symptoms and for families who have received adequate preparation and support, the actual time of death is commonly peaceful and occurs without struggle. Nurses may or may not be present at the time of a patient's death. In many states, nurses are authorized to make the pronouncement of death and sign the death certificate when death is expected. The determination of death is made through a physical examination that includes auscultation for the absence of breathing and heart

sounds. Home care or hospice programs in which nurses make the time-of-death visit and pronounce death have policies and procedures to guide the nurse's actions during this visit. Immediately on cessation of vital functions, the body begins to change. It becomes dusky or bluish, waxen appearing, and cool; blood darkens and pools in dependent areas of the body (e.g., the back and the sacrum if the body is in a supine position); and urine and stool may be evacuated.

Immediately after death, family members should be allowed and encouraged to spend time with the deceased. Normal responses of family members at the time of death vary widely and range from quiet expressions of grief to overt expressions that include wailing and prostration. The family members' desire for privacy during their time with the deceased should be honored. Family members may wish to independently manage or assist with care of the body after death. In the home, after-death care of the body frequently includes culturally specific rituals such as bathing the body. Home care agencies and hospices vary in the policies surrounding removal of tubes. In the absence of specific guidance from the organization, the nurse should shut off infusions of any kind (intravenous or tube feeding) and leave intravenous access devices, feeding tubes, catheters, and wound dressings in place. When an expected death occurs in the home setting, the family generally will have received assistance with funeral arrangements in advance of the death. The funeral home should be called and the funeral director will transport the body directly to the funeral home. In the hospital or long-term care facility, nurses follow the respective facility's procedure for preparation of the body and transportation to the facility's morgue. However, the needs of families to remain with the deceased, to wait until other family members arrive before the body is moved, and to perform after-death rituals should be honored.

Some patients may have elected to donate tissue such as corneas, bone, veins, or heart valves for transplant. Typically, this information would be known to the care team before the patient dies. In some cases, such as late referral or an unexpectedly rapid progression, the patient's wishes with respect to donation might be unknown. If the patient died at home or in a nursing home and the deceased's wishes are known, a team member should contact the organ procurement organization through which the deceased had arranged to donate tissue. That organization will ordinarily transport the body for tissue procurement and to the funeral home.

Providing Psychosocial and Spiritual Support

Many patients suffer unnecessarily when they do not receive adequate attention for the symptoms accompanying serious illness. Careful evaluation of

the patient should include not only the physical problems but also the psychosocial and spiritual dimensions of the patient's and family's experience of serious illness. This approach contributes to a more comprehensive understanding of how the patient's and family's life has been affected by illness and leads to nursing care that addresses their needs in every dimension.

Hope and Meaning in Illness

Clinicians and researchers have observed that although specific hopes may change over time, hope generally persists in some form across every stage of illness. In terminal illness, hope represents the patient's imagined future, forming the basis of a positive, accepting attitude and providing the patient's life with meaning, direction, and optimism. When hope is viewed in this way, it is not limited to cure of the disease; instead, it focuses on what is achievable in the time remaining. Many patients find hope in working on important relationships and creating legacies. Patients who are terminally ill can be extremely resilient, reconceptualizing hope repeatedly as they approach the end-of-life.

Numerous nurse researchers have studied the concept of hope, and they have related its presence to spirituality, quality of life, and transcendence (Tarbi & Meghani, 2019). Hope is a multidimensional construct that provides comfort as a person endures life threats and personal challenges. The following are hope-fostering and hope-hindering activities among patients who are terminally ill and in hospice with various diagnoses:

- *Hope-fostering categories:* Love of family and friends, spirituality/faith, setting goals and maintaining independence, positive relationships with clinicians, humor, personal characteristics, and uplifting memories
- *Hope-hindering categories:* Abandonment and isolation, uncontrollable pain/discomfort, and devaluation of personhood

Nurses can support hope for the patient and the family by using effective listening and communication skills, thus encouraging realistic hope that is specific to their needs for information, expectations for the future, and values and preferences concerning the end-of-life. Nurses must engage in self-reflection and identify their own biases and fears concerning illness, life, and death. As nurses become more skilled in working with patients who are seriously ill, they can become less determined to fix and more willing to listen; more comfortable with silence, grief, anger, and sadness; and more fully present with patients and their families.

Nursing interventions for enabling and supporting hope include the following:

- Answering questions about illness in terms the patient understands

- Listening attentively
- Encouraging sharing of feelings
- Providing accurate information
- Encouraging and supporting patients' control over their circumstances, choices, and environment whenever possible
- Assisting patients to explore ways for finding meaning in their lives
- Facilitating effective communication within families
- Making referrals for psychosocial and spiritual counseling
- Assisting with the development of supports in the home or community when none exist

Providing Culturally Sensitive Care at the End-of-Life

Nurses are responsible for educating patients and their caregivers and for supporting them as they adapt to life with the illness. Nurses can assist patients and families with life review, values clarification, treatment decision making, and end-of-life goals. The only way to do this effectively is to try to appreciate and understand the illness from the patient's perspective.

The nurse's role is to assess the values, preferences, and practices of every patient, regardless of ethnicity, gender identity, socioeconomic status, or background. The nurse can share knowledge about a patient's and family's cultural beliefs and practices with the health care team and facilitate the adaptation of the care plan to accommodate these practices.

Although death, grief, and mourning are universally accepted aspects of living, the values, expectations, and practices during serious illness as death approaches and after death are culturally bound and expressed. Health care providers may share similar values concerning end-of-life care and may find that they are inadequately prepared to assess for and implement care plans that support culturally diverse perspectives. Historical mistrust of the health care system and unequal access to even basic medical care may underlie the beliefs and attitudes among ethnically diverse populations. In addition, lack of education or knowledge about end-of-life care treatment options and language barriers may influence decisions among many socioeconomically disadvantaged groups.

Nurses should be both culturally aware and sensitive in their approaches to communication with patients and families about death. To provide effective patient- and family-centered care at the end-of-life, nurses must be willing to set aside their own assumptions and attitudes so that they can discover what type and amount of disclosure is most meaningful to each patient and family within their unique belief systems. For example, a nurse may find that a female patient prefers to have her eldest son make all of her care decisions. Institutional practices and laws governing informed consent are also rooted in

the Western notion of autonomous decision making and informed consent. If a patient wishes to defer decisions to her son, the nurse can work with the team to negotiate informed consent, respecting the patient's right not to participate in decision making and honoring her family's cultural practice.

Every person has unique needs at the end-of-life and deserves to be respected as a person. Those who identify as lesbian, gay, bisexual, transgender, and queer may need special attention to psychological stressors of serious illness and ACP to designate a preferred surrogate, as this may be outside of the biologic family. An additional concern for some transgender patients is loss of identity after death; for instance, not being buried in clothes of their preferred gender or honored with the name they chose (Higgins & Hynes, 2019).

The nurse should assess and document the patient's and family's specific beliefs, preferences, and practices regarding end-of-life care, preparation for death, and after-death rituals. [Chart 13-8](#) identifies suggested language for cultural and spiritual assessment. The discomfort of novice nurses with asking questions and discussing this type of sensitive content can be reduced by prior practice in a classroom or clinical skills laboratory, observation of interviews conducted by experienced nurses, and partnering with experienced nurses during the first few assessments.

Spiritual Care

Attention to the spiritual component of the illness experienced by the patient and the family is not new within the context of nursing care, yet many nurses lack the comfort or skills to assess and intervene in this dimension. Spirituality contains features of religiosity; however, the two concepts are not interchangeable. **Spirituality** includes domains such as how a person derives meaning and purpose from life, one's beliefs and faith, sources of hope, and attitudes toward death (Puchalski, 2015).

The spiritual assessment is a key component of comprehensive nursing assessment for patients who are terminally ill and their families. While the nursing assessment should include religious affiliation, the nurse keeps in mind that spirituality is a broader concept than just religion (see [Chart 13-8](#)). In addition to the assessment of the role of religious faith and practices, important religious rituals, and connection to a religious community, the nurse should further explore:

- The harmony or discord between the patient's and the family's beliefs
- Other sources of meaning, hope, and comfort
- The presence or absence of a sense of peace of mind and purpose in life

- Spiritual or religious beliefs about illness, medical treatment, and care of the sick

A four-step spiritual assessment process using the acronym FICA involves asking the following questions (Puchalski & Romer, 2000):

- Faith and belief: Do you consider yourself to be a spiritual or religious person? What is your *faith* or belief? What gives your life meaning?

Chart 13-8



ASSESSMENT

Cultural and Spiritual Issues at the End-of-Life

Cultural Assessment

- Tell me more about yourself and your family.
- How are decisions made in your family?
- In order to provide you with the best care, are there any customs or practices important to you that should be included in your care plan?

Spiritual Assessment

- How is your spirit?
- Are you at peace?
- What gives you meaning and purpose?
- What are you most hoping for?
- Is anything worrying you?
- Is there anything you have not done that you wish/need to do?

Assessing Cultural and Spiritual Practices Surrounding Death

- What do you believe happens after death?
- Would you like your family to be involved in the care of your body?
- Are there certain people you would like to care for your body after death (e.g., women only or spiritual care provider)?
- Immediately after death are there any rituals, practices, or ceremonies that should be performed? Are there ways that we can help facilitate these?

Culturally Competent Communication Skills

- Respect uniqueness of each patient.
- Listen attentively to patient's narratives.
- Assess values and preferences for care.
- Address concerns.
- Anticipate when communication may be difficult and engage interdisciplinary team to provide support.

Adapted from Cormack, C., Mazanec, P., & Panke, J. T. (2019). Cultural considerations in palliative care. In B. R. Ferrell, N. Coyle, & J. Paice (Eds.). *Oxford textbook of palliative nursing* (5th ed.). New York: Oxford; Taylor, E. J. (2019). Spiritual screening, history, and assessment. In B. R. Ferrell, N. Coyle, & J. Paice (Eds.). *Oxford textbook of palliative nursing* (5th ed.). New York: Oxford.

- *Importance and Influence:* What *importance* does faith have in your life? Have your beliefs *influenced* the way you take care of yourself

and your illness? What role do your beliefs play in regaining your health?

- Community: Are you a part of a spiritual or religious *community*? Is this of support to you and how? Is there a group of people you really love or who are important to you?
- Address in care: How would you like me to *address* these issues in your health care?

For most people, contemplating one's own death raises many issues, such as the meaning of existence, the purpose of suffering, and the existence of an afterlife. When faced with a grave prognosis, many patients experience spiritual or existential distress where a sense of meaning or purpose is lost. Other manifestations of spiritual distress include questioning or blaming their god. Nurses may identify distress when patients ask questions like, "Why me?" or "Why is God punishing me?" When hearing such questions, nurses should engage a spiritual care provider. Near the end-of-life, many draw strength from religion and spirituality and may turn to spiritual care providers for support; however, one study showed that 75% of clergy want more education surrounding end-of-life issues (Sanders, Chow, Enzinger, et al., 2017). Nurses may be perfectly poised to deliver point-of-care education to spiritual care providers. Further, the interdisciplinary team can organize a debriefing session to enrich education for all disciplines.

Another phenomenon associated with spirituality at the end-of-life is spiritual pain, defined as a pain deep in one's soul not manifested as physical symptoms, which may be experienced in the seriously ill. Although prevalence is not known in all illnesses, in those with advanced cancer as many as 67% of patients may experience spiritual pain, which is correlated with lower quality of life (Perez-Cruz, Langer, Carrasco, et al., 2019).

Grief, Mourning, and Bereavement

A wide range of feelings and behaviors are normal, adaptive, and healthy reactions to the loss of a loved one. **Grief** refers to the personal feelings that accompany an anticipated or actual loss; see [Table 13-7](#) for types of grief. **Mourning** refers to individual, family, group, and cultural expressions of grief and associated behaviors. **Bereavement** refers to the period of time during which mourning for a loss takes place. Both grief reactions and mourning behaviors change over time as people learn to live with the loss. Although the pain of the loss may be tempered by the passage of time, loss is an ongoing developmental process, and time does not heal the bereaved person completely. That is, the bereaved do not get over a loss entirely, nor do they return to who they were before the loss. Rather, they develop a new sense of who they are and where they fit in a world that has changed dramatically and permanently.

TABLE 13-7 Characteristics of Types of Grief and Nursing Interventions

Type of Grief	Characteristics
Anticipatory	Unconsciously preparing for what might happen <i>Examples:</i> Grief at diagnosis at loss of “normal” life; preparing for the loss of a limb for amputation
Uncomplicated	Range of emotions experienced after a loss moving toward adjustment; brief periods of relapse common <i>Examples:</i> Missing a deceased grandparent during holidays
Complicated or Prolonged	Intense response after loss where profound emotions persist usually >1 y <i>Example:</i> Widow who stops caring for herself after the death of her husband and sobs at any mention of his name a year later
Disenfranchised	Grieving person feels that society does not acknowledge or support person’s right to grieve <i>Examples:</i> Mistress, homosexual partner, colleagues
Unresolved	Traumatic or unexpected losses <i>Examples:</i> Death of a child; suicide; disaster-related death
Nursing Interventions: Assessment of self-care and social supports. Employ assessment tools, such as the Inventory of Complicated Grief. Referral to professional counseling services.	

Adapted from Corless, I. B., & Meisenh, J. B. (2019). Bereavement. In B. R. Ferrell, N. Coyle, & J. Paice (Eds.). *Oxford textbook of palliative nursing* (5th ed.). New York: Oxford; Limbo, R., Kobler, K., & Davies, B. (2019). Grief and bereavement in perinatal and pediatric palliative care. In B. R. Ferrell, N. Coyle, & J. Paice (Eds.). *Oxford textbook of palliative nursing* (5th ed.). New York: Oxford.

Anticipatory Grief and Mourning

People experience grief in different ways. Kübler-Ross (1969) originally described five stages of grief including (1) denial, (2) anger, (3) bargaining, (4) depression, and (5) acceptance. Newer models of grief acknowledge that grieving is not a linear process and many more emotions may be present than originally identified by Kübler-Ross. Additionally, people may feel conflicting emotions, such as hope and guilt when thinking about the future, at the same time. The Dual Process Model ([Fig. 13-3](#)) of coping with bereavement allows oscillation between the loss-oriented process and the restoration-oriented process (Stroebe & Schut, 1999). The Dual Process Model assumes fluidity in bereavement and normalizes the individual experience by offering that people will experience competing and complicated emotions after the death of a loved one.

Individual and family coping with the anticipation of death is complicated by the varied and conflicting trajectories that grief and mourning may assume in families. For example, the patient may be experiencing sadness while contemplating role changes that have been brought about by the illness, and the patient’s spouse or partner may be expressing or suppressing feelings of anger about the current changes in role and impending loss of the relationship.

Others in the family may cope with fear using withdrawal. Each person copes in their own way as there is no *right* way to cope.

The nurse should assess the characteristics of the family system and intervene in a manner that supports and enhances the cohesion of the family unit. The nurse can suggest that family members talk about their feelings and understand them in the broader context of anticipatory grief and mourning. Acknowledging and expressing feelings, continuing to interact with the patient in meaningful ways, and planning for the time of death and bereavement are adaptive family behaviors. Professional support provided by grief counselors, whether in the community, at a local hospital, in the long-term care facility, or associated with a hospice program, can help both the patient and the family sort out and acknowledge feelings and make the end-of-life as meaningful as possible.

Grief and Mourning After Death

When a loved one dies, family members enter a new phase of grief and mourning as they begin to accept the loss, feel the pain of permanent separation, and prepare to live a life without the deceased. Even if the loved one dies after a long illness, preparatory grief experienced during the terminal illness does not preclude the grief and mourning that follow the death. With a death after a long or difficult illness, family members may experience conflicting feelings of relief that the loved one's suffering has ended, compounded by guilt and grief related to unresolved issues or the circumstances of death. Grief work may be especially difficult if a patient's death was painful, prolonged, accompanied by unwanted interventions, or unattended. Families that had no preparation or support during the period of imminent death may have a more difficult time finding a place for the painful memories.

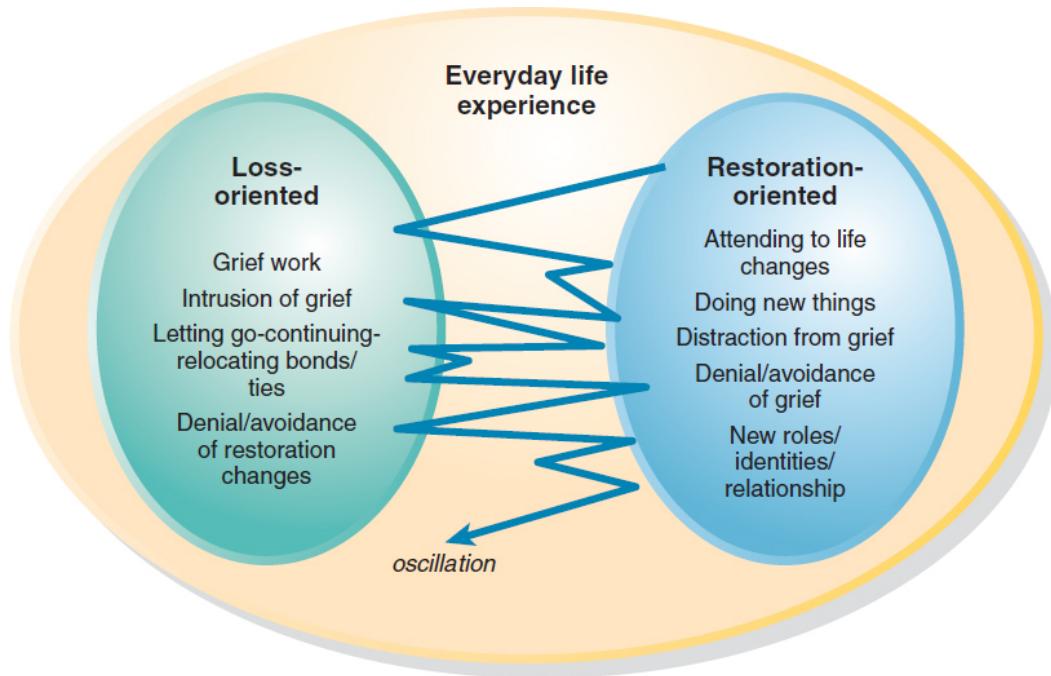


Figure 13-3 • The Dual Process Model of coping with bereavement. Adapted with permission from Stroebe, M. S., & Schut, H. (1999). The Dual Process Model of coping with bereavement: Rationale and description. *Death Studies*, 23, 197–224, by permission of Taylor & Francis Ltd (<http://www.tandfonline.com>) and Stroebe MS & Schut H. doi: 10.1080/074811899201046.

Feelings of grief are often profound; however, bereaved people eventually reconcile the loss and find a way to reengage with their lives. Grief and mourning are affected by several factors, including individual characteristics, coping skills, and experiences with illness and death; the nature of the relationship to the deceased; factors surrounding the illness and the death; family dynamics; social support; and cultural expectations and norms.

After-death rituals, including preparation of the body, funeral practices, and burial rituals, are socially and culturally significant ways in which family members begin to accept the reality and finality of death. Preplanning of funerals is common, and hospice professionals, in particular, help the family make plans for death, often involving the patient, who may wish to play an active role. Preplanning of the funeral relieves the family of the burden of making decisions in the intensely emotional period after a death.

In general, the period of mourning is an adaptive response to loss during which mourners come to accept the loss as real and permanent, acknowledge and experience the painful emotions that accompany the loss, experience life without the deceased, overcome impediments to adjustment, and find a new way of living in a world without the loved one. Particularly immediately after

the death, mourners begin to recognize the reality and permanence of the loss by talking about the deceased in past terms and telling and retelling the story of the illness and death. Societal norms in the United States are frequently at odds with the normal grieving processes of people; time excused from work obligations is typically measured in days, and mourners are often expected to get over the loss quickly and get on with life.

In reality, the work of grief and mourning takes time, and avoiding grief work after the death often leads to long-term adjustment difficulties. According to Rando (2000), mourning for a loss involves the “undoing” of psychosocial ties that bind mourners to the deceased, personal adaptation to the loss, and learning to live in the world without the deceased. Six key processes of mourning allow people to accommodate to the loss in a healthy way (Rando, 2000):

- Recognition of the loss
- Reaction to the separation and experiencing and expressing the pain of the loss
- Recollection and reexperiencing the deceased, the relationship, and the associated feelings
- Relinquishing old attachments to the deceased
- Readjustment to adapt to the new world without forgetting the old
- Reinvestment

Although many people complete the work of mourning with the informal support of families and friends, many find that talking with others who have had a similar experience, such as in formal support groups, normalizes the feelings and experiences and provides a framework for learning new skills to cope with the loss and create a new life. Hospitals, hospices, religious organizations, and other community organizations often sponsor bereavement support groups. When a person dies while enrolled in hospice, families receive proactive bereavement from the hospice agency for an average of 13 months after the death. Hospice bereavement programs vary, yet bereavement support usually starts with scheduled calls to deceased primary contact. Bereavement may then be set up as individual counseling or group sessions, such as widow or parents support groups. Hospices may offer specialized child bereavement support services. Bereavement staff may consist of specially trained nurses, child psychologists, counselors, child life specialists, and/or music or art therapists. If a person dies and is not enrolled in hospice, some palliative care teams offer bereavement or help to connect families with a hospice for bereavement resources. When one cancer center implemented a bereavement program, surveyed bereaved family members felt that acknowledgment of their loved one through a formal condolence letter had a positive impact (Morris & Block, 2015).

Complicated Grief and Mourning

Complicated grief and mourning are characterized by prolonged feelings of sadness and feelings of general worthlessness or hopelessness that persist long after the death, prolonged symptoms (depression, anxiety, insomnia, fatigue) that interfere with activities of daily living, or self-destructive behaviors such as alcohol or substance abuse and suicidal ideation or attempts (Mason & Duffy, 2019). Certain risk factors that increase the likelihood of complicated grief include death of a child or spouse, multiple losses, and history of trauma (Toftaglen, Kip, Witt, et al., 2017). Complicated grief and mourning require professional assessment and can be treated with psychological interventions and, in some cases, with medications.

Special Issues for the Nurse in End-of-Life Care

Nursing care often intersects with moral and ethical dilemmas (see [Chapter 1](#) for further discussion of ethics). Nurses are encouraged to personally examine their respective moral compasses in order to provide the best, unbiased care for patients and families. In caring for patients at the end-of-life, questions of *right* and *wrong* may arise in relation to treatment options. The ANA's *Code of Ethics for Nurses* provides a framework for the nurse to support patients, with guiding principles being the patient's right to self-determination and the nurse's adherence to professional nursing standards (ANA, 2015). When approaching an ethical dilemma, the nurse should take the following steps: define the problem, clarify facts and assumptions, compile a list of all options, evaluate options with interdisciplinary team input, and choose the most appropriate option and implement the plan (Prince-Paul & Daly, 2019). The most common ethical dilemmas a nurse will encounter are determining decisional capacity, withholding or withdrawing life-prolonging measures—including, but not limited to, ventilator support, dialysis, artificial nutrition and hydration—requests for hastening death, and concerns related to proxy decision making. Select ethical issues are discussed in greater detail in the following sections.

Medically Administered Nutrition and Hydration

Although nutritional supplementation may be an important part of the treatment plan in early or chronic illness, unintended weight loss and dehydration are expected characteristics of progressive illness. As illness progresses, patients, families, and clinicians may believe that without artificial nutrition and hydration, patients who are terminally ill will *starve*, causing profound suffering and hastened death. The use of artificial nutrition and hydration (tube and intravenous fluids and feeding) carries considerable risks

and generally does not contribute to comfort at the end-of-life (Casarett, Kapo, & Kaplan, 2005; Marcolini, Putnam, & Aydin, 2018). Similarly, survival is not increased when patients who are terminally ill with advanced dementia receive enteral feeding, and no data support an association between tube feeding and improved quality of life in these patients (De & Thomas, 2019). Furthermore, in patients who are close to death, symptoms associated with dehydration such as dry mouth, confusion, and diminished alertness are common and typically do not respond to artificial nutrition and hydration (Danis, Arnold, & Savarese, 2018). Dry mouth can generally be managed through nursing measures such as mouth care, and environmental changes with medications can help to diminish confusion.

Palliative Sedation at the End-of-Life

Effective control of symptoms can be achieved under most conditions; however, some patients may experience distressing, intractable symptoms. Although **palliative sedation** remains controversial, it is offered in some settings to patients who are close to death or who have symptoms that do not respond to conventional pharmacologic and nonpharmacologic approaches, resulting in unrelieved suffering. Palliative sedation is distinguished from euthanasia and physician-assisted suicide (PAS) in that the intent of palliative sedation is to relieve symptoms, not to hasten death (see [Chart 13-9](#)). Proportionate palliative sedation uses the minimum drug necessary to relieve the symptom while preserving consciousness, whereas palliative sedation induces unconsciousness, which is more controversial (Quill, Lo, Brock, et al., 2009). Palliative sedation is most commonly used when the patient exhibits intractable pain, dyspnea, seizures, or delirium, and it is generally considered appropriate in only the most difficult situations. Palliative sedation is accomplished through an infusion of one or more pharmacologic agents in doses adequate to eliminate signs of discomfort. Before implementing palliative sedation, the health care team should assess for the presence of underlying and treatable causes of suffering, such as depression or spiritual distress. Finally, the patient and the family should be fully informed about the use of this treatment and alternatives. A retrospective study of Italian hospice patients reported that palliative sedation was documented as discussed in only half of the cases where it was implemented (Ingravallo, de Nooijer, Pucci, et al., 2018).

Published guidelines for pharmacologic management of palliative sedation vary widely internationally (Abarshi, Rietjens, Robijn, et al., 2017) but most mention the use of midazolam, a short-acting benzodiazepine (Lux, Protus, Kimbrel, et al., 2017). Nurses act as collaborating members of the interdisciplinary health care team, providing emotional support to patients and families, facilitating clarification of values and preferences, and providing

comfort-focused physical care. Once sedation has been induced, the nurse should continue to comfort the patient, monitor the physiologic effects of the sedation, support the family during the final hours or days of their loved one's life, and ensure communication within the health care team and between the team and the family.

Requests for Assistance in Dying

A major debate in palliative and end-of-life care is the acceptability of a patient's requests to hasten death. Health care recognizes the right to choose for or against medical treatments when a patient is of sound mind and can relay a rationale for or against treatments. Further, patients may choose to withdraw or withhold life-sustaining treatments and allow natural death if such therapies are not aligned with their wishes. When faced with a progressive life-limiting illness, some cannot fathom suffering at the end-of-life and explore options to hasten death. Language around assistance in dying has evolved over the years and it is important to recognize the following terms:

Chart 13-9 ETHICAL DILEMMA



Does Intent Matter When Palliative Care Leads to Death?

Case Scenario

You are a staff nurse working for an outpatient home hospice service. G.R. is a 68-year-old man admitted to home hospice service two months ago with advanced multiple myeloma. As a consequence of his disease he also has extensive destruction of bony matrix resulting in widespread pain, numerous vertebral fractures, and spinal cord compression. For the first six weeks that G.R. was managed in hospice, you and the hospice staff have successfully kept his pain and other symptoms under reasonable control. However, over the past two weeks his pain has been more difficult to control, despite escalating dosages of opioids. He has not been able to sleep, and is restless, dyspneic, and anxious. The hospice physician was consulted, and G.R. and his husband agreed to intravenous opioids, sedatives, and anxiolytics to relieve G.R.'s suffering, knowing that he would also lose consciousness as a consequence of this treatment. However, they stated that though they were not pleased that G.R. would never regain consciousness, that result was offset by their hope was the G.R. might die peacefully. You administer the prescribed intravenous medications, and slowly administer the minimum dosages of these medications to relieve symptoms. G.R. loses consciousness and dies peacefully 48 hours later with his husband holding his hand at his bedside.

Discussion

There is a great deal of apprehension among both clinicians and laypersons around what constitutes comfort care (i.e., palliative care) at end-of-life and what may constitute either assisted suicide or euthanasia. The premise is that palliative care should ameliorate pain and suffering in the patient who is terminally ill, but it should not significantly shorten the patient's life.

The principle of double-effect highlights that some actions have two effects - one good, the other evil. The good and evil consequences are not considered in terms of proportions; if they were, and the evil outcome is death, then the good outcome could never be considered sufficient to justify the action. Rather, the intention behind the action is given paramount consideration. If the intention is to cause death, then the act is not morally justifiable. However, if the intention is to relieve suffering, then the act might be sanctioned.

Analysis

- Describe how the ethical principles of autonomy, beneficence, and nonmaleficence may intersect or be at odds with each other in this case. Discuss how palliative sedation meets the moral justification of the four criteria for the principle of double effect (see [Chapter 1, Chart 1-7](#)).

- Would you describe your role in this case as complicit in assisted suicide, euthanasia, or providing palliation? What distinction, if any, might there be among these three acts?
- Note that the ANA (2015) *Code of Ethics* clearly states that nurses should not *intentionally* cause a patient's death. What were your "intentions" in this case? How might intentionality be instrumental in making this act of titrating medications that provided palliation morally defensible? Are there resources you could identify that might be of assistance to you if you were to feel moral distress about your actions?

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Resources

See [Chapter 1, Chart 1-10](#) for Steps of an Ethical Analysis and Ethics Resources.

- *Physician aid in dying (PAD), medical aid in dying (MAD), and physician-assisted dying:* A physician prescribes a lethal dose of oral medication that the patient self-administers, for the purpose of ending someone's life.
- *PAS:* A physician prescribes medications, that the patient self-administers, to end their life at the person's voluntary and competent request.
- *Euthanasia:* Greek for “good death”; has evolved to mean the intentional killing by act or omission of a dependent human being for their alleged benefit.

Although assisted suicide is expressly prohibited under statutory or common law in the majority of states, calls for legalized aid in dying have highlighted inadequacies in end-of-life care. In 1994, Oregon voters approved the Oregon Death with Dignity Act, the first and—until 2009 only such legislative initiative to pass. This law provides for access to PAS by terminally ill patients under very controlled circumstances. After numerous challenges, the law was enacted in 1997. Of 2127 Oregonians who have received written prescriptions under the terms of the law since it was passed in 1997, 1459 have self-administered physician-prescribed lethal medication and have died (Oregon Public Health Division, 2018).

In recent years, additional states have adopted MAD laws, and Montana's Supreme Court has ruled that PAD is not a crime. International MAD is legal in Canada, the Netherlands, Belgium, Luxembourg, Switzerland, Columbia, and Victoria in Australia. Proponents of PAS argue that people who are terminally ill should have a legally sanctioned right to make independent decisions about the value of their lives and the timing and circumstances of their deaths, and its opponents argue for greater access to symptom management and psychosocial support for people approaching the end-of-life.

In its 2013 position statement on *Euthanasia, Assisted Suicide, and Aid in Dying*, the ANA acknowledged the complexity of the assisted suicide debate but clearly stated that nursing participation in assisted suicide is a violation of the Code for Nurses. The ANA position statement further stressed the important role of the nurse in supporting effective symptom management, contributing to the creation of environments for care that honor the patient's and family's wishes, as well as identifying their concerns and fears (ANA, 2013). Per the ANA, "Nurses have an obligation to provide humane, comprehensive, and compassionate care that respects the rights of patients but upholds the standards of the profession in the presence of chronic, debilitating illness and at end-of-life" (ANA, 2013, p. 1).

Similarly, the HPNA favors the value of comprehensive end-of-life care, as opposed to physician-assisted death (HPNA, 2017). The HPNA acknowledges that nurses may care for patients exploring or choosing physician-assisted death and thus concludes that nurses must be able to provide unbiased education yet not take an active role in this end-of-life option. American Academy of Hospice and Palliative Medicine (AAHPM, 2016) has taken a position of "studied neutrality" on assisted death, recommending that clinicians carefully assess the fear and suffering that have led patients to request assisted suicide and to address these without hastening death.

The International Association for Hospice and Palliative Care (IAHPC) released a position statement on euthanasia and PAS, in which they recommend access to palliative care and medication, such as opioids, prior to the legalization of both euthanasia and/or PAS. Further, they recommend that these acts should not be carried out on palliative care units (De Lima, Woodruff, Pettus, et al., 2017).

Voluntary Stopping of Eating and Drinking

Another potential, however quite controversial, option of last resort is voluntary stopping of eating and drinking (VSED) when a person cannot imagine prolonged dying and suffering for a life-limiting illness. Patient inquiry about VSED should be met with thorough assessment of both decisional capacity and presence of mental illness (Quill, Ganzini, Troug, et al., 2018; Wax, An, Koiser, et al., 2018). Aggressive palliative treatments for all types of suffering—physical, psychological, social, spiritual, and existential

—should be explored and offered before pursuing VSED. Typically, a person will die 10 to 14 days after starting VSED during which the most distressing symptoms include thirst and delirium (Quill et al., 2018; Wax et al., 2018). Given this expected prognosis, patients may have the opportunity to complete legacy work prior to initiating VSED.

The ANA included VSED in a 2017 position statement on *Nutrition and Hydration at the End of Life* in which the organization upholds respect for autonomy, relief of suffering, and expert care at the end of life. Nurses involved in the care of a patient who self-initiates VSED should support the family while providing expert symptom assessment and management. One example of a patient who may choose to pursue VSED is a patient who has been diagnosed with a progressive disease, such as ALS, and who finds a slow decline intolerable.



COVID-19 Considerations

The coronavirus disease 2019 (COVID-19) pandemic has altered the delivery of palliative and end-of-life care. Patients with severe COVID-19 pneumonia are managed with endotracheal intubation and mechanical ventilation and have a high case fatality rate (see [Chapter 19](#)) (Bajwah, Wilcock, Towers, et al., 2020; Richardson, Hirsch, Narasimhan, et al., 2020). Common end-of-life symptoms for those with severe COVID-19 include dyspnea, cough, anxiety, and delirium (Bajwah et al., 2020). During the pandemic, symptom management at the end-of-life has required revisions due to logistical challenges such as medication shortages and preservation of personal protective equipment (Chidiac, Feuer, Naismith, et al., 2020). Due to high risk of infectious transmission, family members may not be permitted to see loved ones at the end-of-life (Richardson et al., 2020; Wakam, Montgomery, Biesterveld, et al., 2020). Furthermore, family meetings are primarily virtual, where proxies must make difficult decisions such as resuscitation and intubation, and even removal of life support (Richardson et al., 2020). Communication guides for proactive goals of care discussions related to possible COVID-19 infection (Back, Tulsky, & Arnold, 2020), recommendations for “webside” manner (Chua, Jackson, & Kamdar, 2020), innovative use of virtual reality to stimulate previously unachieved dreams or allow families to virtually share one last moment together (Wang, Teo, Teo, et al., 2020). Meanwhile, health care providers are struggling from high physical workload demands while simultaneously experiencing moral and psychological distress (Adams & Walls, 2020). Clinicians working with patients with COVID-19 voice concerns about the risk of infecting self and family and stress due to constant change as health care systems evolve to meet patient needs during a pandemic situation (Foxwell, 2020). Health systems have developed innovative processes to support staff at the front lines of

working with patients with COVID-19 and at end-of-life, such as a 24-hour hotlines and pet therapy.

Clinicians' Attitudes toward Death

Clinicians' attitudes toward the terminally ill and dying remain the greatest barrier to improving care at the end-of-life. Kübler-Ross illuminated the concerns of the seriously ill and dying in her seminal work, *On Death and Dying*, first published in 1969. At that time, it was common for patients to be kept uninformed about life-threatening diagnoses, particularly cancer, and for primary providers and nurses to avoid open discussion of death and dying with their patients. Kübler-Ross's work revealed that, given open discussion, adequate time, and some help in working through the process, patients could reach a stage of acceptance in which they were neither angry nor depressed about their fate.

The growth of palliative and hospice care programs has led to greater numbers of health care providers becoming comfortable with and skilled in assessing patients' and families' information needs and disclosing honest information about the seriousness of illness (Brighton & Bristowe, 2016). However, in many settings, clinicians still avoid the topic of death in the hope that patients will ask or find out on their own. Despite progress on many health care fronts, many who work with patients who are seriously ill and dying recognize a persistent conspiracy of silence about dying.

How to communicate truthfully with patients and encourage patient **autonomy** (the right of the person to make choices) in a way that acknowledges where they are on the continuum of acceptance is a challenge. Despite continued reluctance of health care providers to engage in open discussion about end-of-life issues, patients want information about their illness and end-of-life choices and are not harmed by open discussion about death (Hamel, Wu, & Brodie, 2017; The Conversation Project National Survey, 2018). Timing of sensitive discussion takes experience, but speaking the truth can be a relief to patients and families, enhancing their autonomy by making way for truly informed consent as the basis for decision making.

Patient and Family Concerns

Terminal illness is experienced uniquely by each person and their loved ones. Family members are key stakeholders in honoring patients' wishes, caring for patients, and engaging in care planning. Family members are impacted by a loved one's illness and require support throughout the course of caregiving. Recognizing maladaptive coping in family members and providing resources for the family whether through social work staff or palliative care specialists is key to preventing worsening distress at the time of death. Palliative care

interventions have been reported to improve communication, improve person-centeredness and decrease length of ICU stay without significantly impacting caregiver psychological distress (White, Angus, Shields, et al., 2018).

Patient and family awareness of **prognosis** is a key factor in acceptance of and planning for death. For patients who have been informed about terminal illness, their understanding of treatment goals and prognosis is dynamic and may sometimes require reinforcement. Even when patients have been told that the intent of a therapy is palliative, they may think *I'll be the one to beat the odds*. As many as 37% of patients with metastatic cancer receiving palliative therapies misperceive their cancer as potentially curable (Yennurajalingam, Lu, Prado, et al., 2018). Just as patients and families may have differing perspectives on prognosis, they may also have different perceptions of severity of symptoms. Family caregivers tend to report distress and quality of life as worse than patient reports at the end-of-life (Hack, McClement, Chochinov, et al., 2018). This may be due to the family member's distress or anticipatory grief.

Historically, the approach in the United States to serious illness has been described as *death denying*—that is, the health care system has been built on management of acute illness and the use of technology to cure (when possible) and to extend life. As a result, life-threatening illness, life-sustaining treatment decisions, dying, and death occur in a social environment in which illness is largely considered an enemy. Many common expressions reflect this dominant sociocultural view. For example, people talk about the *war* against cancer or *fighting* illness, and when patients choose not to pursue the most aggressive course of medical treatment available, many health care providers and indeed patients and families perceive this as *giving up*. A care/cure dichotomy has persisted in which health care providers may view cure as the ultimate good and care as second best, a good only when cure is no longer possible. In such a model, alleviating suffering is not as valued as curing disease. Patients who cannot be cured feel distanced from the health care team, and when curative treatments have failed, they may feel that they too have failed. Patients and families may fear that any shift from curative goals to comfort-focused care will result in no care or lower-quality care, and that the clinicians on whom they have come to rely will abandon them if they withdraw from a focus on cure.

The statement in late-stage illness that exemplifies this care versus cure dichotomy is *nothing more can be done*. This all-too-frequently used statement communicates the belief of many clinicians that there is nothing of value to offer patients beyond cure; however, in a care-focused perspective, there is always more that can be done. This expanded notion of healing implies that healing can take place throughout life. There are many opportunities for physical, spiritual, emotional, and social healing, even as body systems begin to fail at the end-of-life.

TABLE 13-8 Select Resiliency Skills

Individual Skills	System Level Opportunities
<ul style="list-style-type: none">• Assessing personal strengths• Recognizing clinical emotional triggers• Applying mindfulness skills at work• Establishing health boundaries and reasonable expectations• Finding meaning in daily work• Continuing to adapt and cultivate resiliency skills	<ul style="list-style-type: none">• Supporting autonomy• Structuring rewards• Promoting collaboration among peers and other disciplines• Establishing fairness in the workplace• Acknowledging that patient-centered care takes time to explore patient's individual values• Allowing flexibility

Adapted from Back, A. L., Stienhauser, K. E., Kamal, A. H., et al. (2017). Why burnout is so hard to fix. *Journal of Oncology Practice*, 13(6), 348–351.

Professional Caregiver Issues

Issues of importance to professional caregivers include burnout, promoting resiliency, and nurses supporting themselves.

Burnout and Promoting Resiliency

Burnout is defined as the triad of emotional exhaustion, cynicism, and ineffectiveness at work. By the time one notices burnout it is usually too late. In order to prevent burnout, there is a body of work to promote resilience, thereby providing clinicians with the skills needed to have work–life balance and the opportunity to remain productive and satisfied with their career (Back, Steinhauser, Kamal, et al., 2017). See Table 13-8 for select resiliency skills and workplace factors to prevent burnout.

While the prevalence of burnout among nurses working in palliative care and hospice is unknown, researchers have attempted to assess the prevalence of burnout among all clinicians working in palliative care. One study found that physicians were more likely to exhibit signs of burnout; however, factors for both physicians and clinicians from other disciplines (advanced practice provider, nurse, chaplain, social worker) that contributed to burnout are younger age, working weekends or overtime, and feeling isolated (Kamal, Bull, Wolf, et al., 2020). Overall, hospice and palliative clinicians were found to have a burnout rate of approximately 39% (Kamal et al., 2020).

The ANA (2017a) published *A Call to Action: Exploring Moral Resilience toward a Culture of Ethical Practice*, which highlights potential for burnout among nurses and makes recommendations for both individuals and institutions to foster resilience, as well as recommendations for research. This

document also contains a toolkit for resources for the nurse and proposed actions.

Supporting Ourselves

In addition to healthy work environments that promote resiliency, nurses are often encouraged to develop self-care practices. Self-care can be any number of activities or practices that a person maintains while not at work; examples include practicing yoga or meditation, engaging in hobbies, or simply not checking email when away from work. A recent qualitative study found palliative care clinicians (primary providers and nurses) seek practices that promote well-being and allow restoration of self so as to better care for patients (Mills, Wand, & Fraser, 2018). The palliative care clinicians sought meaning through work-life balance and building relationships; they sought self-care during work (such as establishing boundaries, negotiating workload, and reflecting on practice) and outside of work (such as meditation, rest, separating self from work, and positive social relationships) (Mills et al., 2018).

Whether practicing in a trauma center, ICU, or other acute care setting, home care, hospice, long-term care, or the many locations where patients and their families receive ambulatory services, nurses are closely involved with complex and emotionally laden issues surrounding loss of life. To be most effective and satisfied with the care they provide, nurses should attend to their own emotional responses to the losses witnessed every day. In hospice settings, where death, grief, and loss are expected outcomes of patient care, interdisciplinary colleagues rely on one another for support by learning coping skills from one another and speaking about how they were affected by the lives of those patients who have died. In many settings, staff members organize periodic memorial services to support bereaved families and other caregivers, who find comfort in joining one another to remember and celebrate the lives of patients.

CRITICAL THINKING EXERCISES

1 ipc Your patient is a 60-year-old woman with lung cancer who is hospitalized with increasing shortness of breath. She is currently receiving second-line chemotherapy treatment and recently completed palliative radiation to rib metastases. She lives with her husband and has two adult children who live out of state. As you are administering her morning medications, she asks, “Why did God do this to me? What did I do to deserve this?” How will you facilitate an interprofessional discussion to address the patient’s spiritual concerns? What members of the interdisciplinary team are essential to include?

2 ebp Your patient is a 52-year-old man with idiopathic pulmonary fibrosis who is being evaluated for a lung transplant. He arrives at the lung transplant clinic with his friend from work. He is a security guard, but has been calling out frequently as he can’t make hourly rounds. He lives alone in a two-story home with one bathroom on the second floor. Due to dyspnea on exertion, he has not been able to go upstairs and now sleeps on a recliner and hasn’t showered in weeks. He has an estranged daughter whom he has not spoken to in over 10 years. Dyspnea also interferes with his appetite and he has been drinking sports drinks and eats fast food when his friends bring it over. How would you complete an evidence-based symptom assessment? What interventions would you consider to treat his dyspnea based on the evidence?

3 pq You are a new hospice nurse in your third month making home hospice visits. Your patient is a 73-year-old woman who has had multiple strokes. She previously expressed to her family that she would not want to live on prolonged life support. She has four devoted children who have set up a schedule so that someone will be with her 24 hours a day. When you enter the room, the patient is somnolent with periods of apnea. Her daughter at the bedside tells you that her mother was screaming out last night, calling for her parents and trying to get out of bed. The daughter feels that her mother is more comfortable now. She is intermittently tearful as she talks with you. At one point she asks, “Do you think she’s angry with us? That must be why she was agitated last night. . . . I knew I shouldn’t have listened to my siblings—My mom wants to live!” What stage of dying is this patient experiencing? How would you educate the daughter about stages of dying? How would you assess for bereavement needs?

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*Asterisk indicates nursing research.

**Double asterisk indicates classic reference.

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Resources

- American Academy of Hospice and Palliative Medicine (AAHPM),
www.aahpm.org
- American Hospice Foundation, www.americanhospice.org
- Americans for Better Care of the Dying (ABCD), www.abcd-caring.org
- Association for Death Education and Counseling (ADEC),
www.adec.org/default.aspx
- Caring Connections: A program of the National Hospice and Palliative Care Organization, www.caregiver.org/caring-connections-0
- Center to Advance Palliative Care (CAPC), www.capc.org
- Children's Hospice International (CHI), www.chionline.org
- Compassion & Choices, www.compassionandchoices.org
- End-of-Life Nursing Education Consortium (ELNEC),
www.aacnnursing.org/ELNEC
- Family Caregiver Alliance (FCA), www.caregiver.org
- Get Palliative Care (Blog and resources for palliative care),
www.getpalliativecare.org
- Harvard Medical School Center for Palliative Care, Dana Farber Cancer Institute,
[/pallcare.hms.harvard.edu](http://pallcare.hms.harvard.edu)
- Hospice and Palliative Credentialing Center (HPCC),
www.advancingexpertcare.org/HPNA/HPNA/About_Us/About.aspx
- Hospice and Palliative Nurses Association (HPNA), www.advancingexpertcare.org
- Hospice Association of America, <http://hospice.nahc.org/>
- Hospice Compare (Medicare ratings), www.medicare.gov/hospicecompare
- Hospice Education Institute, www.guidestar.org/profile/22-2701794
- Hospice Foundation of America (HFA), www.hospicefoundation.org
- International Association for Hospice & Palliative Care (IAHPC),
www.hospicecare.com/home
- National Association for Home Care & Hospice, www.nahc.org
- National Consensus Project for Quality Palliative Care,
www.nationalcoalitionhpc.org/ncp
- National Hospice and Palliative Care Organization (NHPCO), www.nhpco.org
- National Prison Hospice Association, www.npha.org

Office of End-of-Life and Palliative Care Research (OEPCR),

www.ninr.nih.gov/researchandfunding/desp/oepcr

Palliative Care NOW Fast Facts and Concepts, www.mypcn.org/fast-facts

POLST (Physician Orders for Life-Sustaining Treatment Paradigm),

www.polst.org

Promoting Excellence in End-of-Life Care, www.promotingexcellence.org

Supportive Care Coalition, www.supportivecac coalition.org

TIME: Toolkit of Instruments to Measure End of Life Care (Brown University),

www.chcr.brown.edu/pcoc/resourceguide/resourceguide.pdf

We Honor Veterans (Partnership between Veterans Administration and NHPCO),

www.wehonorveterans.org

World Health Organization (WHO) Palliative Care, www.who.int/news-room/fact-sheets/detail/palliative-care